Family Doctor

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Focus:

Case Reports in Family Medicine

FEATURE ARTICLES:

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From the Executive Vice President

By Vito Grasso, MPA, CAE

Editor's Note:

As many of you may know, Vito Grasso, EVP of the NYSAFP, tragically lost his daughter Rebecca in August. Rebecca had been a fixture at Academy events and activities since she was young, and was editor of the NYSAFP's electronic newsletter for several years.

The board and staff of the NYSAFP wish to express our sincere condolences to Vito, Sue and family and will miss Becky's indomitable spirit within our ranks.

Rebecca Grasso, DPT

Rebecca Grasso, the daughter of NYSAFP EVP & CEO Vito Grasso and Susan Grasso, died suddenly and unexpectedly on August 23rd. Rebecca was also the editor of NYSAFP News for several years. She was diagnosed with neurofibromatosis-2 in 2010 when she was 19 years old. She was 34 at the time of her death.

Rebecca was a pioneer in NF2 research and advocacy. She was the first patient admitted to Dr. Scott Plotkin's Avastin trial shortly after her diagnosis. Today, Avastin is commonly used to treat NF2. She was among the first patients admitted to Dr. Plotkin's Brigatinib trial. Brigatinib is also used now to treat NF2. She was also among the first patients to try the combination of Avastin and Brigatinib.

Rebecca was a source of inspiration for many other NF patients and their families. She counseled many who reached out to her for advice in dealing with the disease. She was an articulate advocate for funding for the NF Research Program in the Congressionally Directed Medical Research Program. She was honored with the Paul Bodner Memorial Award in 2019 for her NF advocacy and the inspiring manner in which she lived her life. She was also honored by the NYS Assembly and Senate in 2018 for her advocacy for NF research.

Rebecca's family has designated NF2BioSolutions as their charity of choice to receive donations in Rebecca's honor and memory: https://nf2biosolutions.org/donate/

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Albany Report

By Reid, McNally & Savage

September 26, 2025

The New York State (NYS) Legislature adjourned the 2025 session in mid-June with over 800 individual bills passed by both houses out of approximately 17,000 bills introduced since session started in January. At this point, most bills still await action by Governor Hochul before the end of the year, including the Medical Aid in Dying (MAID) Act, the Wrongful Death Act, the FAIR Business Practices Act which would permit the Attorney General to bring actions or proceedings for unfair business practices, and the Responsible AI Safety and Education Act which would require large AI developers to build in safety measures against potential risks, and others.

The focus right now however, is largely on the future impacts of deep federal budget cuts enacted by President Trump this summer as well as recent federal policy changes. While state legislators likely won't have to return to Albany for a special session this fall, Governor Hochul and the Division of Budget have found ways to close the $750 million gap this fiscal year. With the development of Governor's Executive Budget for next year underway, she and State Health Commissioner James McDonald have warned people to prepare for a tough 2026 budget. Difficult decisions have already been made including the termination of the State Innovation Waiver and return to a Basic Health Program. This will allow approximately 1.3 million New Yorkers to remain enrolled in the program, however, 450,000 New Yorkers will lose access to zero-premium comprehensive health insurance.

Another focus is the 2025 New York City mayoral election. With Election Day on November 4th, Zohran Mamdani maintains a big lead in the race and recently secured critical endorsements from Governor Kathy Hochul, NYS Senate Majority Leader Andrea Stewart-Cousins, and Assembly Speaker Carl Heastie. After current Mayor Eric Adams' announcement this month that he won't drop out of the race, chances are increasing that Mamdani may beat former Governor Andrew Cuomo.

A summary of ongoing advocacy efforts and priorities that we will continue to address with NYSAFP in the coming session follows below:

Advocacy Update

MAID Act: Following the bill's passage by both houses this year, we have been working closely with the New York Alliance for MAID coalition to maintain momentum around the legislation, address disinformation and developments in the media, and show widespread and varied support for MAID. NYSAFP is currently working on an opinion piece in collaboration with MSSNY and has submitted letters-to-the-editor over the summer to news outlets urging the Governor to enact the bill. NYSAFP has shared a letter with the Governor's office urging her to sign the legislation quickly and Academy leadership met with her staff in August to discuss any concerns they might have and how implementation has gone in states that have legalized MAID. Our media advocacy and grassroots efforts will ramp up this fall in coordination with the coalition with opportunities for NYSAFP members to attend rallies, send letters to the Governor, and more.

Vaccine Advocacy: NYSAFP and RMS continue to lead a vaccine coalition in New York (Let's Get Immunized NY) to help support education and advocacy around immunizations for children and adults and have been

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working diligently to ensure vaccine access and coverage in NYS given recent uncertainty with federal vaccine changes. NYSAFP secured a meeting with the NYS Department of Health (DOH) Commissioner's Office in late August to discuss what is being done at the state level and how the Academy can support DOH. Ultimately, discussions were positive and this opened up opportunities for the Academy to be a resource and collaborate with DOH in the future as they continue to navigate the large number of unknowns surrounding vaccine recommendations. We also discussed the Academy's priority for the state to pursue the public purchase of vaccines with the Health Commissioner, who previously implemented a similar program in Rhode Island.

In early September, Governor Hochul took steps to ensure vaccine accessibility in NYS including allowing pharmacists to prescribe and administer Covid-19 vaccines and releasing Covid-19 guidance for New Yorkers in partnership with the Northeast Public Health Collaborative. The NYS Department of Financial Services issued guidance to insurers expecting them to cover 100% of the costs of vaccines. The guidance is consistent with the fall 2025 Covid-19 vaccination guidance from the American Academy of Family Physicians, the American Academy of Pediatrics, and the American College of Obstetrics, and Gynecology. Legislation (S8496-A/ A9060-A) has also been introduced by Senator Hinchey and Assembly member Paulin to address Advisory Committee on Immunization Practices (ACIP) references in New York law by creating an alternative for DOH authority.

Additionally, as a result of this meeting, Vaccine Subcommittee member, Dr. Jamie Loehr began the process of applying to be nominated to an open position on the NYS Immunization Advisory Council. If appointed, he would serve as a valuable representative of the Academy and family medicine as a whole.

As Let's Get Immunized New York moves into year 5 in 2026, we'll continue to work with partners on advancing the bill that would require adult vaccine reporting to the State Immunization Registry and opposing efforts to weaken New York's vaccine laws. We expect to see a legislative package or proposals in the Governor's Executive Budget in January to address the ongoing issues with ACIP and are planning to make recommendations for what should be included.

Wrongful Death Bill: Despite strong opposition from NYSAFP, partners in medicine as well as hospitals, insurers and others, an amended version of the "wrongful death" bill, vetoed three times now by Governor Hochul, was reintroduced and passed by the Legislature in the final days of the session. We have already sent the Governor a letter on behalf of the Academy asking her to again veto the bill, and are working in coalition with MSSNY, other specialty societies, and others to register strong opposition. Further, in September over 25 Academy members sent grassroots letters to the Governor asking her to again veto the bill. We will continue these grassroots efforts this fall with a particular emphasis on the dramatic effects expanding damages awardable in wrongful death actions would have, particularly with the impending federal funding cuts. Governor Hochul likely won't request this contentious bill to come before her until the end of the year.

Primary Care Recruitment and Training: The Academy has long supported legislation (S7701/A2230) to establish a personal income tax credit for clinicians who provide preceptor instruction to students. The bill was passed by the Senate in 2024 and we will work to re-up this effort with bill sponsors and other supportive organizations ahead of the 2026 budget process. We have also worked to support increased funding for primary care recruitment and retention efforts and are happy to share that we successfully broadened the criteria for Doctors Across New York (DANY) for 2025 which allows limited liability partnerships (LLPs) and physicians working for LLPs to be eligible for a DANY award.

Insurance & Payment Reforms: We are continuing to advocate for a single payer system (S3425/A1466) and pursue greater investments in primary care by supporting legislation to require a minimum investment of the health care spend in the State for primary care (S1634/A1915-A). We also continue to advocate for insurance simplification and reforms to remove insurance barriers to access care and the time-consuming processes imposed on physician practices. We will continue these efforts in 2026.

Reproductive & Gender-Affirming Care: NYSAFP has signed on to a letter to Governor Hochul issued by the New York Civil Liberties Union urging her to sign the following bills as soon as possible given unprecedented attacks on abortion access and LGBTQ communities:

1. **Health Information Protection (S929, Krueger/ A2141, Rosenthal) –** This bill amends the general business law to create a legal framework for New Yorkers to reclaim and retain control of their healthcare information by requiring electronic apps or websites that provide a diagnosis or retain health information to receive affirmative consent by the user to retain such information. Electronic apps or websites would also be required to provide users with the ability to rescind such consent.
2. **Hospital Rule-Based Exclusions (S3486, Hinchey/ A3862, Rozic) –** This legislation amends the public health and insurance laws to require DOH to collect a list of hospital rule-based exclusions from each hospital and publish the list on the DOH website of general hospitals that have these exclusions and state specifically what they are. This provide patients and the public with information prior to admission to a hospital.
3. **Reproductive and Gender-Affirming Care Protections (S4914-B, Hoylman-Sigal/ A5480-C) –** This bill amends several areas of law to prevent the state from engaging with hostile actors attempting to restrict access to reproductive health care and gender-affirming care. It would also build on professional discipline and medical malpractice protections in New York's shield laws by extending these to more providers that may be engaged in the delivery of gender-affirming or reproductive health care.

We would like to thank the NYSAFP Board, Advocacy Commission, Home Office and the full membership for your strong advocacy this year. We look forward to continuing to work with you to pursue priorities of import to family physicians and your patients into 2026.

Biopsy-Proven Hansen's Disease in an Elderly Woman with Myelofibrosis and Chronic Hepatitis B: A Rare Case in an Immigrant from the Dominican Republic

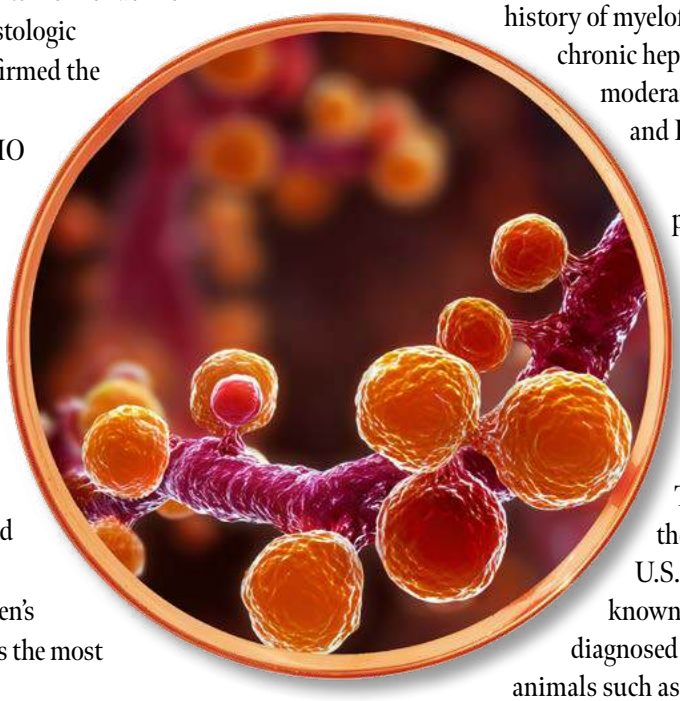
By Nellya Ablayeva, MD and Ravilya Caine, MD

Abstract

We present a case of an 83-year-old woman with multiple comorbidities, including myelofibrosis and chronic hepatitis B, who developed non-painful facial plaques later diagnosed as lepromatous Hansen's disease. Despite living in the United States for over a decade and having no known exposure to typical reservoirs such as armadillos, she was diagnosed via skin biopsy. This case highlights the importance of maintaining a high index of suspicion for Hansen's disease in patients from endemic regions, even years after immigration. Histologic staining, including FITE and CD68, confirmed the diagnosis. The patient is undergoing evaluation for multidrug therapy per WHO guidelines.

Introduction

Hansen's disease, caused by *Mycobacterium leprae*, is a chronic granulomatous infection with a declining global incidence but persistent cases in endemic areas such as South Asia, Brazil, and the Caribbean.[1,2] In 2020, more than 200,000 new cases were reported globally, with the highest burden in India, Brazil, and Indonesia.[1] Lepromatous Hansen's disease, the multibacillary form, represents the most infectious and systemic variant.[2,3]



In non-endemic countries such as the United States, diagnosis is often delayed because of low clinical suspicion and overlapping features with other granulomatous skin disorders.[2,4] Moreover, zoonotic transmission from armadillos has been described in southern U.S. states,[3] although this exposure was not relevant in our case.

This case is important because it highlights an unusual presentation of lepromatous Hansen's disease in an elderly, immuno-compromised woman with multiple comorbidities, living in the U.S. for over a decade without known exposure. The absence of classic neurological findings emphasizes how immunosuppression may alter disease manifestations and delay recognition. This report contributes to the limited literature on Hansen's disease in elderly immigrants and underscores the role of family medicine in recognizing atypical presentations of neglected tropical diseases in non-endemic settings.

Case Presentation

Patient Information: An 83-year-old woman with a past medical history of myelofibrosis, myeloid metaplasia, chronic hepatitis B (on entecavir), moderate pulmonary hypertension, and HFpEF.

Chief Complaint: Facial plaques present for approximately 10 months, described as "bumps" that were non-painful, non-pruritic, and without associated erythema.

History and Social Context: The patient immigrated from the Dominican Republic to the U.S. 12 years prior. She denied known contact with individuals diagnosed with Hansen's disease or with animals such as armadillos.

Physical Examination: Multiple non-erythematous plaques were present over the right cheek, left infraorbital region, nasal bridge, and eyebrows. No sensory loss was noted over lesions. Peripheral nerve thickening was absent. Neurological exam was intact. No systemic signs of infection were observed.

Initial Differential Diagnosis: Chronic cutaneous sarcoidosis, cutaneous lymphoma, lupus pernio, granulomatous rosacea, and atypical infections (deep fungal or mycobacterial).[5]

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Investigations:

- Skin Biopsy (Right Cheek): FITE stain strongly positive for acid-fast bacilli; CD68 showed diffuse histiocytic infiltrate; AFB and GMS were negative.
- Laboratory Findings: Hepatitis B panel: surface antigen non-reactive, surface antibody reactive, core total antibody reactive. HCV antibody: non-reactive. QuantiFERON-TB Gold Plus: indeterminate. G6PD: 19.9 U/g Hgb (normal 7.0–20.5).

Diagnosis: Histopathology confirmed lepromatous Hansen's disease.

Discussion

This case illustrates that Hansen's disease should remain on the differential in immigrant populations from endemic regions, regardless of the interval since immigration.[2,5] Classic features of lepromatous Hansen's disease include numerous cutaneous lesions and diffuse peripheral nerve involvement.[2,4] However, our patient lacked sensory loss or nerve thickening, which are considered hallmark signs of advanced disease.[2]

Her underlying comorbidities, including myelofibrosis and chronic hepatitis B, may have altered her immune response and contributed to this atypical presentation. Immunosuppression is known to modify the granulomatous reaction and blunt neural involvement in Hansen's disease.[2,6] As a result, the absence of classic neurologic findings delayed suspicion and could have led to misdiagnosis.

The differential diagnosis for chronic facial plaques in elderly or immunocompromised patients includes sarcoidosis, cutaneous T-cell lymphoma, lupus pernio, granulomatous rosacea, deep fungal infections, and non-tuberculous mycobacterial infections.[5] Only histopathologic analysis, with a strongly positive FITE stain, allowed definitive identification of *M. leprae*.

Importantly, this case underscores the central role of family medicine in the early evaluation of patients with complex comorbidities. Family physicians are often the first point of contact for elderly immigrant populations. Their broad training in dermatology, infectious diseases, and chronic disease management allowed for the recognition of atypical skin lesions and appropriate referral for biopsy.

Treatment of multibacillary Hansen's disease requires WHO-recommended multidrug therapy (rifampin, dapsone, clofazimine).[1,7] Clofazimine access in the U.S. is limited and usually requires special arrangements.[7] Our patient is undergoing evaluation for therapy initiation under infectious disease guidance.

This case demonstrates the collaborative nature of care, where family medicine serves as the gateway for early recognition and coordinated multidisciplinary management of rare infectious diseases in non-endemic settings.[2,6] From a literature perspective, this case is significant because reports of lepromatous Hansen's disease in elderly immigrants with concurrent myelofibrosis and chronic hepatitis B are exceedingly rare. It expands the understanding of how immunocompromised states can mask classic features such as peripheral neuropathy, leading to atypical presentations. By documenting this case, we add to the global knowledge base on delayed and modified presentations of Hansen's disease, which is essential for clinicians practicing in both endemic and non-endemic regions.

Conclusion

Hansen's disease, though rare in the U.S., should be considered in patients with chronic cutaneous lesions and a history of residence in endemic regions. Immunocompromised states may mask classic findings, leading to diagnostic delay. Prompt biopsy and specialized staining remain essential for diagnosis.[1,2,6]

Endnotes

1. World Health Organization. Guidelines for the diagnosis, treatment and prevention of leprosy. New Delhi: WHO Regional Office for South-East Asia; 2018.
2. Scollard DM, Adams LB, Gillis TP, Krahenbuhl JL, Truman RW, Williams DL. The continuing challenges of leprosy. Clin Microbiol Rev. 2006;19(2):338–381.
3. Truman RW, Singh P, Sharma R, et al. Probable zoonotic leprosy in the southern United States. N Engl J Med. 2011;364(17):1626–1633.
4. Lockwood DNJ. Leprosy. BMJ. 2002;324(7353):968–970.
5. Maymone MBC, Laughter M, Venkatesh S, Dacso MM, Rao PN, Stryjewska BM, Hugh J, Dellavalle RP, Dunnick CA. Leprosy: Clinical aspects and diagnostic techniques. J Am Acad Dermatol. 2020 Jul;83(1):1-14. doi:10.1016/j.jaad.2019.12.080. PMID: 32229279.
6. White C, Franco-Paredes C. Leprosy in the 21st century. Clin Microbiol Rev. 2015;28(1):80–94.
7. Noto S, Goto M, Mori T. Clofazimine: current status and future prospects. J Antimicrob Chemother. 2020;75(5):1109–1117.

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The authors express their deep gratitude to Abdulwahhab Alabid, MD, PGY2 FM for assistance in obtaining patient consent for the case report.

A Rare Case of Normoprolactinemic Galactorrhea with Recurrent Mastitis and Headache in a Young Postpartum Woman

By Gyasi Kodua, MD, MHS; Nwe Nwe Yu, MD and Hoang Nhu (Natalie) Hua, MD

Abstract

Normoprolactinemic galactorrhea is a rare condition with unclear etiology, characterized by persistent lactation despite normal serum prolactin. We report a 19-year-old postpartum woman with bilateral milky discharge persisting two years after delivery, recurrent mastitis, and headaches with visual changes. Laboratory evaluation including hCG, thyroid, liver, and renal function, as well as brain MRI, were normal. Conservative management with breast hygiene counseling and antibiotics for mastitis was effective, suggesting increased peripheral sensitivity to prolactin as a possible mechanism. This case highlights the diagnostic challenges of atypical presentations and underscores the importance of multidisciplinary evaluation to prevent misdiagnosis and guide management.

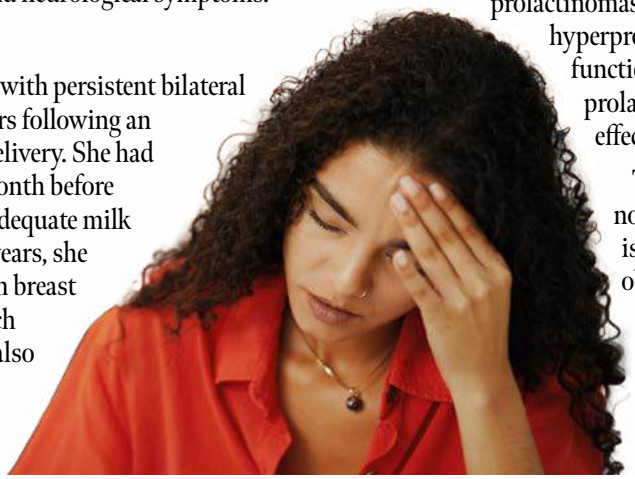
Introduction

Galactorrhea, defined as inappropriate milk secretion outside of pregnancy or breastfeeding, is usually associated with hyperprolactinemia from causes such as pituitary adenomas, hypothyroidism, renal failure, chest wall lesions, or medication effects. While hyperprolactinemia-induced galactorrhea is well studied, lactation despite normal prolactin levels termed normoprolactinemic galactorrhea remains poorly understood and underreported. Proposed mechanisms include heightened breast tissue sensitivity to prolactin or altered hypothalamic dopaminergic regulation.

The condition is uncommon, and prevalence is uncertain. Zervoudis et al. (2014) suggest it may be underestimated, as patients with mild symptoms often do not undergo evaluation. The clinical significance lies in excluding serious pathology such as pituitary tumors and recognizing complications including mastitis, which can cause chronic pain, abscess formation, and scarring. This case is notable for the coexistence of normoprolactinemic galactorrhea, recurrent mastitis, and neurological symptoms.

Case Presentation

A 19-year-old woman presented with persistent bilateral milky nipple discharge for two years following an uncomplicated full-term vaginal delivery. She had breastfed for approximately one month before discontinuing due to perceived inadequate milk quality. Over the subsequent two years, she experienced recurrent mastitis with breast pain, erythema, and swelling, which responded to oral antibiotics. She also reported intermittent frontal headaches rated 6–7/10 with transient blurred vision.



She denied use of dopamine antagonist medications, herbal galactagogues, or illicit drugs. Examination showed stable vitals, bilateral milky discharge from multiple ducts, mild breast tenderness, and no masses. The neurological exam was normal. Investigations revealed normal serum prolactin (13.10 ng/mL), thyroid and renal function. Brain MRI with contrast excluded pituitary adenoma or other sellar lesions.

Management included targeted antibiotics for mastitis, education on breast care and hygiene, and monitoring of neurological symptoms, with neurology referral offered if symptoms worsened. At six-month follow-up, galactorrhea persisted but mastitis episodes decreased with conservative measures.

Discussion

The pathophysiology of normoprolactinemic galactorrhea is unclear. In typical cases, prolactin stimulates alveolar epithelial cells to produce milk. In women with normal serum prolactin, potential explanations include increased peripheral sensitivity of breast tissue, intermittent unmeasured prolactin bursts due to altered hypothalamic regulation, or persistent glandular activity after pregnancy. Rarely, estrogen excess may enhance prolactin action despite normal circulating levels.[2,3]

Differential diagnoses include physiologic postpartum lactation (which may last beyond one year), chest wall stimulation or trauma, medication-induced lactation (notably with SSRIs), hypothyroidism with episodic prolactin surges, and chronic mastitis with ductal changes.[1] In this patient, thyroid function, medication review, and neuroimaging were unremarkable, supporting an idiopathic cause.

The patient's headaches with visual disturbances appropriately raised concern for pituitary or parasellar masses. While prolactinomas typically present with hyperprolactinemia, the "hook effect" or non-functioning adenomas may yield normal serum prolactin.[2] MRI was therefore indicated and effectively ruled out such lesions.

The patient's recurrent mastitis is noteworthy. Mastitis in non-lactating women is uncommon and usually linked to ductal obstruction, bacterial colonization, or duct ectasia.[5] Persistent milk production increases the risk of milk stasis, which predisposes to infection, most often by *Staphylococcus aureus*. Recurrent inflammation may also lead to chronic

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Chronic Vitamin A Toxicity in a 76-Year-Old Male

By Sheila Ramanathan, DO

Abstract:

Dietary history is an important component of the social history of a patient. A 76-year-old male was noted to have chronic hypercalcemia of unknown origin. The patient underwent a thorough workup when levels failed to normalize despite regular observation. The diagnosis was found to be vitamin A toxicity secondary to consuming braunschweiger sausage, which provided nearly 350% of the recommended daily dietary intake of vitamin A. This had occurred for at least three years per the patient's recollection, which was corroborated by his intermittent laboratory testing. Despite dietary changes the patient was found to have significant osteoporosis.

Introduction:

Vitamin A toxicity is seen as an acute ingestion, although it is exceedingly rare. There were fewer than ten cases per year from 1976 to 1987.[1] Vitamin A has two forms provitamin A, also known as carotenoids, and preformed vitamin A. Preformed vitamin A is sourced from animal products such as meat, cereals, dairy products, and eggs. Carotenoids must be metabolized to form vitamin A and thus are far less likely to build up to toxic levels. The current dietary recommended allowance for women is 2700 international units.[1] Higher doses are associated with a dramatic increase in teratogenicity resulting in secondary congenital disabilities. Medication-induced vitamin A toxicity through oral retinoids is commonly a concern when treating skin conditions, especially in women of childbearing age. However, chronic toxicity is implicated in multiple organ systems including bone health as it stimulates bone resorption with resulting hypercalcemia. This contributes to osteoporosis and hip fractures. Elevated triglycerides and elevated liver enzymes can be a presenting sign of vitamin A toxicity as well. On physical exam eruptive xanthomas as well as abdominal pain from pancreatitis can be seen with hair loss and fissuring of the oral mucosa.[1] A rare but potential complication is pseudotumor cerebri as severe headaches can be a presenting feature. Encouragingly, discontinuation of the vitamin can reverse adverse effects.

Case Report:

The 76-year-old professor established care in a rural outpatient office. He presented with a medical history of hypertension, hyperlipidemia, and gastrointestinal reflux disease, all of which were under good control at the time of his visit. He denied any history of alcohol or tobacco use. He was up to date regarding his colonoscopy which had been completed one year prior and was not due for an additional four years. His surgical history consisted of an appendectomy, knee replacement, and tonsillectomy. For his initial physical exam the patient's vitals were within normal limits. He took daily atorvastatin 20 mg, famotidine 20 mg, hydrochlorothiazide 25 mg, atenolol 25 mg, vitamin C 500 mg, a multivitamin, and lisinopril 40 mg. The patient demonstrated no new physical exam abnormalities and lab work was obtained. He was instructed to follow up in six months for reassessment of his blood pressure and chronic conditions.

The lab work obtained from his initial visit indicated an elevated white count of 11.8 x109/L, hemoglobin of 14.2 g/dL, mean corpuscular volume at 99.1 fL, and his absolute neutrophil and monocyte count were 7.3 and 1.3 cells/uL. His calcium was 11.5 mg/dL, and his non-fasting glucose was 104 mg/dL. His BUN was 21 mg/dL and his creatinine was normal at 0.92 mg/dL. His liver enzymes were normal. His B12, folate, and cholesterol panel were within normal limits. His albumin was 4.9 g/dL. The patient was advised that he should repeat his complete blood count and undergo a comprehensive metabolic profile to reassess his white blood count and his calcium level abnormalities, as well as discontinue any over the counter agents containing calcium to treat his acid reflux. This was completed six months later prior to his follow up visit. His follow up visit revealed no abnormalities on his physical exam or vitals and it was determined that he had a longstanding history of mildly intermittent hypercalcemia ranging from normal at 10.2 mg/dL to 11.5 mg/dL for the past three years when he had been under the care of a different primary care clinician. His follow up testing indicated a normalization of his complete blood count however his calcium levels remained elevated at 11.2 mg/dL so further lab testing was ordered. His serum protein electrophoresis, vitamin D 1,25, PTH related protein, and thyroid testing was within normal limits. His vitamin D 25 was mildly reduced at 26 ng/mL.

His lab work indicated primary hyperparathyroidism and he was instructed to discontinue the hydrochlorothiazide and initiate on amlodipine. He was instructed to follow up in the nurse's clinic for repeat blood pressure and be screened for fevers, chills, and night sweats which were negative per history. He was also advised to complete a twenty-four hour urinary calcium level. He was advised to avoid activities that can exacerbate hypercalcemia including volume depletion, inadequate hydration, prolonged bedrest, and a high calcium diet. He was warned concerning risk of renal nephrolithiasis that comes with hypercalcemia.

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His twenty four hour urinary calcium level was found to be elevated at 384 mg/day with a GFR of 87 mL/min. With the diagnosis of primary hyperparathyroidism confirmed, the patient was referred to see endocrinology. While awaiting the appointment with endocrinology the patient's comprehensive metabolic panel was checked to assess the effect of lifestyle changes. His vitamin A level was checked for completeness as per the primary hypercalcemia algorithm on UpToDate prior to being assessed by endocrinology. His calcium remained elevated at 10.7 mg/dL. His vitamin A levels were found to be toxic at 90.1 mcg/dL. The normal range for his lab was less than 69.5 mcg/dL. A careful discussion regarding possible sources of vitamin A ensued where the patient claimed to be "basically vegetarian." After a lengthy discussion the patient admitted to eating braunschweiger liver sausage daily. When evaluated for daily vitamin A intake it was calculated that he was receiving 350% of the recommended daily dietary intake of vitamin A from this source. A bone density scan was performed and he was found to have a bone mineral density at the level of the femoral neck 3.52 standard deviations below normal. His spine bone mineral density was found to be 0.82 standard deviations above normal. He subsequently discontinued consumption of the liver sausage.

The patient met with endocrinology who confirmed likely vitamin A toxicity with resulting severe osteoporosis, hypercalcemia, and hyperuricalcemia. The patient had noted reduction in vitamin A levels to a normal range. His hypercalcemia persisted however and he was found to have a parathyroid adenoma on imaging.

Discussion:

Vitamin A can be toxic in individuals consuming preformed vitamin A present in organ meats or other animal products. This is almost impossible with vegetarian diets as vitamin A needs to be metabolized from beta carotene. Toxicity from beta carotene is quite rare given that excretion is more likely to be the outcome even with visible carotonemia. Chronic vitamin A toxicity occurs when more than the 50,000 international units of the recommended dietary allowance is consumed on a daily basis as opposed to acute toxicity or teratogenic poisoning. Signs of chronic toxicity may include ataxia, alopecia, hyperlipidemia, hepatotoxicity, bone and muscle pain, visual impairments, hypercalcemia, and many other nonspecific signs and symptoms. Circulating vitamin A levels may not reflect true toxicity as vitamin A is stored in the liver and thus may release varying levels of the vitamin even after ingestion has ceased. Underlying liver or kidney disease can worsen vitamin A effect as can tetracycline and alcohol use. Additionally high retinol levels are associated with an increased risk of fracture in men compromising bone health as seen in this patient's case.

Endnotes

1. Olson JM, Ameer MA, Goyal A. Vitamin A Toxicity. [Updated 2021 Dec 29]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK532916/

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ductal changes, pain, or abscess formation. Preventive hygiene strategies were effective in reducing her episodes.[5]

Management of normoprolactinemic galactorrhea depends on severity. Asymptomatic women require only reassurance, as many improve spontaneously.[1,3] In symptomatic cases causing social or psychological distress, dopamine agonists such as cabergoline or bromocriptine may be effective even when prolactin is normal.[4] Supportive strategies include breast care education, empiric antibiotics for mastitis, neurological monitoring for persistent headaches or vision changes, and psychological support when symptoms affect quality of life.[1]

The literature suggests that spontaneous resolution within one to three years is common, but pharmacologic therapy may be required in persistent cases.[3] Rojansky et al. demonstrated that bromocriptine reduced galactorrhea in normoprolactinemic women, supporting the role of dopamine agonists.[4] StatPearls (2023) emphasizes excluding secondary causes before initiating therapy.[1] Recurrent mastitis, although rarely discussed, is clinically important; a case by Al-Eyd et al. (2019) described similar findings and highlighted the link between galactorrhea and infection risk.[5]

Prognosis and Conclusion

The overall prognosis is favorable, especially with effective mastitis prevention and reassurance regarding the benign nature of the condition. Nonetheless, persistent symptoms may affect quality of life, body image, and sexual health, necessitating holistic care. Long-term follow-up is advisable for patients with neurological complaints even if initial imaging is negative.

This case underscores the importance of thorough evaluation to exclude secondary causes of galactorrhea, recognition of mastitis as a potential complication of milk stasis, and the role of supportive care in management. Clinicians should be aware that normal prolactin levels do not exclude clinically significant galactorrhea, and patient education is essential to preventing complications

Endnotes

1. StatPearls. Galactorrhea. NCBI Bookshelf. 2023.
2. Molitch ME. Disorders of prolactin secretion. In: Melmed S, Polonsky KS, Larsen PR, Kronenberg HM, eds. Williams Textbook of Endocrinology. 13th ed. Elsevier; 2017:246-277.
3. Zervoudis S, Iatrakis G, Economides P, Navrozoglou I, Galazios G. Idiopathic normoprolactinemic galactorrhea: An underestimated condition? Obstet Gynecol Int. 2014;2014:548785. doi:10.1155/2014/548785.
4. Rojansky N, et al. Bromocriptine therapy in normoprolactinemic galactorrhea. Fertil Steril. 1989;51(4):604-606.
5. Al-Eyd G, et al. Recurrent mastitis secondary to persistent galactorrhea: a case report. Breast J. 2019;25(6):1170-1172.

Reference

American Academy of Family Physicians. Evaluation and management of galactorrhea. Am Fam Physician. 2022

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An Atypical Case of Median Arcuate Ligament Syndrome

By Ashima Dogra, MD, MPH; Japleen Kaur, MBBS and AnnMarie Zimmermann, MD

Abstract

Median arcuate ligament syndrome, or MALS, is a relatively rare and benign condition that primarily affects young females with a thin body habitus. Symptoms are usually postprandial and manifest as vomiting, nausea, weight loss, and even abdominal pain. Here we present a rare case of MALS in a 76-year-old overweight male with symptoms that were not only postprandial but also post-exercise and, on occasion, at rest. Symptoms were not just limited to nausea/vomiting but also consisted of pre-syncopal episodes with bowel/bladder incontinence. MALS was detected after an extensive workup through the CTA abdomen when looking for chronic mesenteric ischemia.

Introduction

Median arcuate ligament syndrome (MALS) results from extrinsic compression of the celiac artery by fibrous bands of the diaphragmatic crura, specifically the median arcuate ligament.[1] The ligament lies anterior to the aortic hiatus at the T12-L1 level.[2] MALS is a rare condition with a wide range of nonspecific symptoms, making diagnosis particularly challenging.[3] It is considered a diagnosis of exclusion and typically presents with gastrointestinal symptoms such as postprandial abdominal discomfort, nausea, vomiting, and weight loss.[4] Symptoms may also be triggered by physical exertion. Symptoms can often be debilitating and most commonly affect females between the ages of 20 and 60 with a slender body habitus.[4] Diagnostic imaging modalities include duplex ultrasonography, CT angiography (CTA), and magnetic resonance angiography (MRA).[5] Definitive treatment is usually surgical MAL release plus coeliac ganglionectomy.[5,6]

Case Report

A 76-year-old Caucasian male with a history of obesity, essential hypertension, and chronic bilateral lower extremity lymphedema presented to the primary care clinic with recurrent episodes of nausea, vomiting, and presyncope, occurring postprandially, post-exercise, and occasionally at rest. Notably, these episodes were intermittent and sometimes accompanied by bowel and bladder incontinence. Each episode lasted between 5 to 30 minutes. The patient also reported heart rate fluctuations, ranging from the low 30s to high 90s, which appeared unrelated to his symptomatic episodes. He denied associated symptoms such as chest pain, orthopnea, abdominal or back pain, shortness of breath, or focal neurological deficits.

The patient had recently developed an unprovoked deep vein thrombosis (DVT) in the left great saphenous vein, for which he was anticoagulated. He also recalled a similar unprovoked DVT episode 10 years prior while living in Asia, and was on anti-coagulants for a few months at that time.

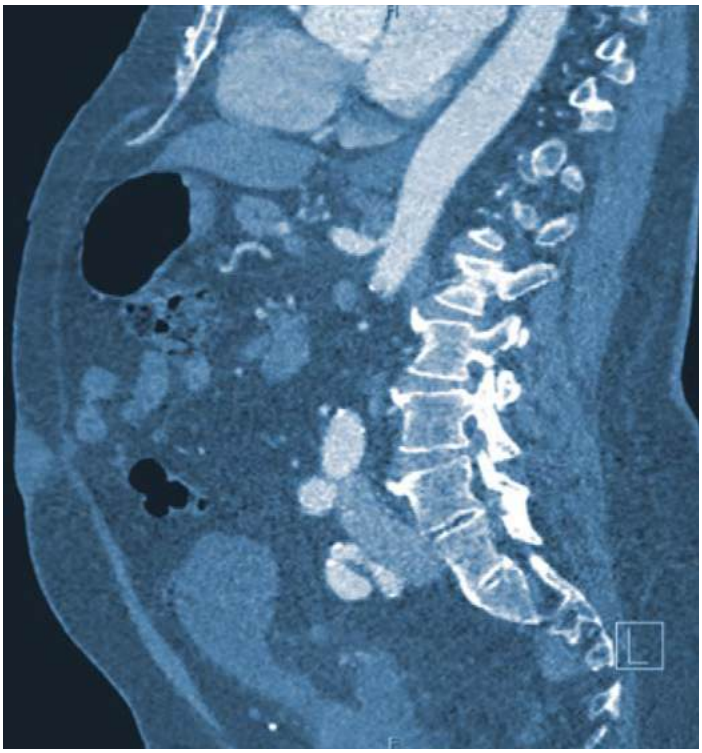

Figure 1: CTA abdomen revealing significant extrinsic compression adjacent to the origin of the celiac axis with distal compensatory dilation.

The only lifestyle change reported was recent involvement in swimming 3–4 times per week, and he reported that some episodes occurred after these activities. Physical and neurological examinations were unremarkable. A trial of proton pump inhibitor therapy provided partial relief of nausea but did not alleviate other symptoms. H. pylori testing was negative.

Extensive laboratory evaluation, including CBC, CMP, serum gastrin, insulin, somatostatin, and 24-hour cortisol levels, was within normal limits. A gastric emptying study showed no abnormalities. Cardiac workup included a Holter monitor, which revealed frequent premature atrial contractions and a rare premature ventricular contraction, without evidence of supraventricular or ventricular tachyarrhythmias. There were no episodes of atrial fibrillation or significant pauses. The patient had a mean heart rate of 53 bpm with periods of sinus bradycardia, though these did not correlate with his symptoms. Echocardiogram revealed a preserved ejection fraction (50–55%) and Grade 1 diastolic dysfunction. Due to the presyncopal episodes, carotid duplex ultrasonography was performed, revealing only mild stenosis and no evidence of atherosclerotic plaque with antegrade flow within both vertebral arteries. Cardiology was consulted for

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evaluation of possible chronic mesenteric ischemia, and they ordered a CT angiogram of the abdomen.

CTA of the abdomen revealed significant extrinsic compression adjacent to the origin of the celiac axis with distal compensatory dilation, findings consistent with MALS [Figure 1]. Additionally, the imaging revealed pronounced tortuosity of the common iliac, external iliac, and internal iliac arteries with severe extrinsic compression at the external iliac vein. A variant May-Thurner physiology with severe intrinsic compression of the external iliac vein was revealed as well, which was likely causing recurrent DVTs.

The patient was counseled on surgical options, and a referral to vascular surgery was initiated. Further management is ongoing.

Discussion

This case highlights an atypical presentation of MALS in an elderly, overweight male, demographically and clinically distinct from the classical presentation in younger, thin females. While common symptoms such as postprandial nausea, vomiting, and presyncope were observed, the presence of bowel and bladder incontinence was unusual. It may suggest a broader systemic response or multifactorial pathology.[3] This case reinforces the complexity and diagnostic difficulty of MALS due to its broad symptom spectrum.[7] It underscores the importance of maintaining a high index of suspicion for MALS in patients with unexplained gastrointestinal and syncopal symptoms, especially after more common and life-threatening conditions have been ruled out. The co-existence of May-Thurner physiology further complicates the clinical picture and may indicate a shared vascular susceptibility in this patient.

Overall, this case contributes to the limited body of literature on MALS in elderly males, emphasizing the need for greater awareness and further research into its variable presentations and management strategies.

Endnotes

1. Maddox K, Farrell TM, Pascarella L. Median Arcuate Ligament Syndrome: Where Are We Today? *The American Surgeon™*. 2024;91(2):284-291. doi:10.1177/00031348241292728
2. Kim EN, Lamb K, Relles D, Moudgill N, DiMuzio PJ, Eisenberg JA. Median Arcuate Ligament Syndrome-Review of This Rare Disease. *JAMA Surg*. 2016;151(5):471-477. doi:10.1001/jamasurg.2016.0002 https://doi.org/10.1001/jamasurg.2016.0002
3. Rodriguez JH. Median arcuate ligament syndrome: A clinical dilemma. *Cleve Clin J Med*. 2021;88(3):143-144. Published 2021 Mar 1. doi:10.3949/ccjm.88a.21001 https://doi.org/10.3949/ccjm.88a.21001
4. Upshaw W, Richey J, Ravi G, et al. Overview of Median Arcuate Ligament Syndrome: A Narrative Review. *Cureus*. 2023;15(10):e46675. Published 2023 Oct 8. doi:10.7759/cureus.46675 https://doi.org/10.7759/cureus.46675
5. Goodall R, Langridge B, Onida S, Ellis M, Lane T, Davies AH. Median arcuate ligament syndrome. *J Vasc Surg*. 2020;71(6):2170-2176. doi:10.1016/j.jvs.2019.11.012 https://doi.org/10.1016/j.jvs.2019.11.012
6. Metz FM, Blauw JTM, Brusse-Keizer M, et al. Systematic Review of the Efficacy of Treatment for Median Arcuate Ligament Syndrome. *Eur J Vasc Endovasc Surg*. 2022;64(6):720-732. doi:10.1016/j.ejvs.2022.08.033 https://doi.org/10.1016/j.ejvs.2022.08.033
7. Iobst TP, Lamb KM, Spitzer SL, Patel RN, Alrefai SS. Median Arcuate Ligament Syndrome. *Cureus*. 2022;14(2):e22106. Published 2022 Feb 10. doi:10.7759/cureus.22106 https://doi.org/10.7759/cureus.22106

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Upcoming Events

2025

Virtual Commission Meetings
October - TBD

Virtual Board Meeting
November 2

2026

Winter Weekend
Hilton Saratoga Springs
January 16-18, 2026

Winter Board Meeting
Renaissance Albany
February 22, 2026

Advocacy Day
Albany
February 23, 2026

A Nineteenth-Century Ovarian Cyst

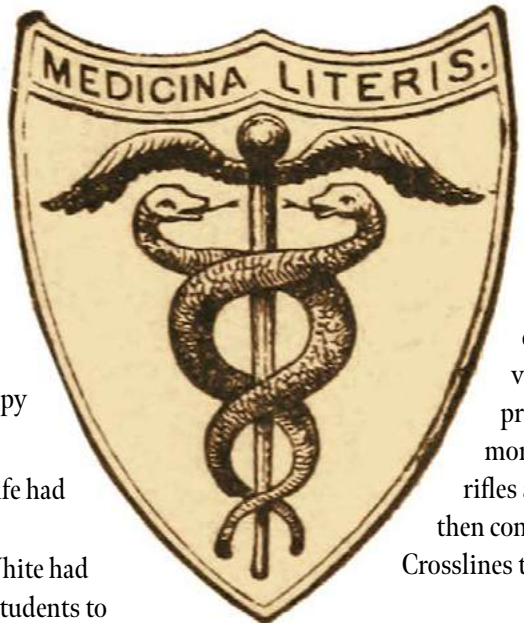
By Thomas C. Rosenthal, MD

One winter day in 1850, a lad, no more than 10 years old, burst into Dr. Allen's office, demanding he come immediately. "Momma's having a baby and the baby-lady wants you to come right away!" he shouted. Doc Allen threw a saddle on his horse and followed the boy to a cabin off the main road a little past the church in Griffins Mills. Before proper introductions, the agonized midwife explained Sally Crossline's prolonged labor and failure to progress. She knew something was terribly wrong.

Sally's husband also knew something was wrong and did nothing to reduce the anger and fear that hung in the cabin air. Doc Allen's history revealed that Sally had scant monthly bleeding all through the last few months, although no bleeding had occurred in recent weeks. He applied his short wooden stethoscope to the woman's bulging abdomen, but failed to detect fetal heart sounds or contractions. Asking for a pail of water, he described his every movement, hoping to calm the cabin's atmosphere of anxiety with a litany of words. After washing his hands; he applied fresh lard to his right hand, and rolled Sally onto her left side. Allowing no opportunity for comment or protest, he commenced an exam.

Sally's abdomen certainly gave the appearance of a term gravid condition, but beyond a closed cervix, the mass he now manipulated between his pelvic and abdominal hands was not a fetus. Motioning for Sally's husband to approach, Dr. Allen explained that there was no baby. Rather, Sally had a watermelon size tumor arising, he suspected, from her left ovary. The Crosslines needed to confront a new reality.

The condition was rare, but Dr. Allen explained recent advances just might alleviate Sally's condition. It would require another consultation, that of Buffalo Medical College professor Dr. James Platt White. At a medical society meeting some months back, Dr. White described an operation, under ether, that could remove tumors like the one afflicting Sally. Dr. White, he believed, could return Sally to a productive and happy life.



Devastated and embarrassed, the midwife had been silent. She now offered an untimely comment, reminding Dr. Allen that Dr. White had been accused of allowing several medical students to exam a woman during her labor. Newspapers published several accusations of ethical and moral debauchery about Dr. White's teaching methods. In response, White brought a claim of libel against his accuser. The subsequent trial gained nothing for Dr. White and public opinion waffled, but newspapers around the world reprinted the provocative details.

At this, Allen took the hands of both husband and wife while admitting the midwife's allegation was true. However, he assured the couple, Dr. White was an exceptional teacher, and a skilled surgeon who has as much experience remedying Sally's problem as any doctor in America. Allen emphasized that, "It may take months or a year but Sally is unlikely to survive this growth. Your choices are difficult but they are not complicated."

That evening, Presbyterian Pastor Coure from Griffins Mills paid a visit to Dr. Allen's home. He opposed the intervention for three reasons. First, he knew such operations to be dangerous. Second, heroic interventions challenged God's will. Third, Dr. White's morals seemed suspect.

Allen conceded that Dr. White suffers the self-confidence that comes with the summative skill he has gained in using the knife to cure patients. None-the-less, it must also be conceded that newspapers sensationalize events to boost their sales. The woman who agreed to the student demonstration had received the best care possible. Dr. White charged her no fee in an arrangement that was mutually agreeable, and his skills were above reproach. The discussion, over tea, lasted an hour, ending with Pastor Coure offering no clue if his opinion had changed.

The next morning, the east bound stagecoach driver presented Dr. Allen with a note from the pastor. It contained three words: "Proceed with arrangements." Allen sent an urgent telegram to Dr. White whose response was immediate. He was eager to add Sally to his case series.

Arriving by stagecoach the next day, the usually brisk and formal Dr. White proved charming and personable when he and Allen visited the Crossline cabin. Sally was preparing a rabbit her husband had shot that morning, giving White a moment to discuss rifles and shotguns with Mr. Crossline. White then confirmed Dr. Allen's exam and told the Crosslines that surgery was the only remedy. Trending

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close to a brag, he described his low post-operative rate of inflammation and promised to use the most modern antiseptic protocols developed by Lister at a hospital in Edinburgh. Ether would make the operation painless and Sally would be given a new form of opium called morphine to relieve pain after surgery. At this point Pastor Coure, summoned by the same lad who originally burst into Allen's office, arrived, prompting Dr. White to repeat all he had just told the Crosslines.

The next day Sally and her neighbor, Jane Arnold, left Dr. Allen's drugstore on the afternoon stagecoach. Jane was to provide support and assure propriety during the ordeal while Mr. Crossline tended to the farm and the children. The two women were lodged at a rooming house near the college and through the next week they were visited by students and Dr. Frank Hastings Hamilton, professor of surgery at Buffalo Medical College. Hamilton was to assist Dr. White. Both Sally and Jane were given a tour of the surgical suite, which was unlike any room they had ever seen. It had a deep well where Drs. White and Hamilton would perform the surgery and a large south-facing window. The well was surrounded on three sides by benches mounted on a steep incline on which several dozen medical students were perched. With careful attention to modest draping, Dr. White repeated Sally's exam while lecturing the students about pelvic anatomy, ovarian cysts, and surgical techniques. The two women noted that Dr. White was gentle and kind when addressing Sally and instantly a formal task master when addressing the students.

Dr. White's management of the situation never made Sally feel ill-at-ease as White explained these cysts resulted from a corruption of normal catamenia. Professor Meigs of Philadelphia had found similar tumors to contain a gallon of fluid and debris and that a Dr. Ephraim McDowell had performed the first operation to remove a cyst nearly forty years ago in Danville, Kentucky. Since first being used in surgery in 1846, ether now allowed for improved surgical technique, though post-surgical fever and inflammation were serious risks.

On Tuesday afternoon, both Dr. White and Dr. Hamilton came by the boarding house with final instructions. They told Sally she was to eat nothing more until after surgery and that a hot bath would be arranged prior to her departure for surgery. They left Sally with woolen underwear, drawers, stockings, and a nightgown she was to wear after the bath.

In the operating room the next morning, Sally was pleased to see Dr. Allen, who Dr. White had invited to administer the ether. Following Dr. Hamilton's protocol, Allen packed cotton wadding into a glass container, then soaked it with either. After a few twitches, Sally was asleep and two mirrors were positioned, one to focus the light and another to allow the students to see the procedure.

Their sleeves rolled up, White and Hamilton washed their hands in dilute carbolic acid, which had also been used to wet the linen cloth on which their surgical instruments were spread. After washing Sally's abdomen with carbolic acid, Dr. White handed the knife to Dr. Hamilton, who made an incision down the midline from the umbilicus to the pubis. Then Dr. White separated the linea alba so perfectly that very little bleeding occurred. With hands moving like a well-rehearsed dance, the two men exposed the tumor, identified the uterus, lifted the cyst, tied off blood vessels with carbolic soaked catgut and in twenty minutes the incision was closed.

As ether's fog lifted, Dr. Allen assured Sally that the procedure was a success. At that point, Sally was removed from the theater. The intact cyst was placed on the operating table as Dr. White complimented himself for removing the cyst intact. He now cut open the cyst, immediately filling the room with a foul odor typical of multilobulated cysts that contained fragments of hair, teeth, and skin mixed in a sebaceous, sticky fluid. Dr. White informed the students that more than half of the cysts reported by Dr. Meigs were of similar character.

Accompanied by students, Dr. White visited Sally in the rooming house each day for the next week, at which time she returned home to her family. Dr. Allen checked her weekly for signs of fever or inflammation for six weeks. In early May, while making calls in Griffins Mills, Dr. Allen paid a visit to the Crossline family and found Sally out in the field planting corn. As he returned home, Dr. Allen felt quite pleased with himself. He had recognized a problem and orchestrated a solution. Good fortune had given him the opportunity to take advantage of modern nineteenth century medicine and the new opportunities anesthesia and antisepsis had created. His role as a village doctor had expanded, allowing him to remedy even unusual cases by securing and assisting in the services provided by specialists he trusted.

References

Meigs, C. D. (1867). Obstetrics; The Science and the Art. Philadelphia, Henry C. Lea.

Ridenbaugh, M. Y. (1890). The Biography of Ephraim McDowell, M.D. The Father of Ovariotomy. New York, Charles L. Webster and Company.

Rosenthal, T. (2025). Cyrenius Chapin, Buffalo's First Doctor and War of 1812 Hero. Albany, SUNY Press.

Rosenthal, T. C. (2020). Bloodletting and Germs: A Doctor in Nineteenth Century Rural New York. Philadelphia, BookBaby.

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From Musculoskeletal Complaint to Oncologic Diagnosis: Prostate Cancer Masquerading as Hip Pain

By Sarin Itty, DO; Olutosin Quadri, DO; Pamela Otti, MD and Wesley Ho, MD

Abstract

Hip pain, a common complaint in primary clinics, can mask serious pathology such as metastatic cancer. This case report describes a 69-year-old male whose hip pain was ultimately diagnosed as metastatic prostate cancer. It highlights the importance of maintaining a broad differential, recognizing oncologic red flags, and knowing when to go beyond conservative treatment. It emphasizes the critical role of family physicians in early cancer detection, explores the diagnostic pitfalls of prostate cancer and reviews the utility of PSA testing.

Introduction

Hip pain, which is a common complaint in the family practice clinic, can mask underlying serious pathology, including metastatic cancer. It is important to keep a broad differential, especially in someone presenting with a new onset of bone pain. Family physicians play a critical role in recognizing an atypical presentation of serious underlying conditions. Early suspicion and investigation can impact outcomes, even when symptoms appear musculoskeletal or benign. This case emphasizes how subtle and nonspecific symptoms (e.g., isolated hip pain) can be the first sign of widespread metastatic disease. The case report will discuss the oncologic red flags of back pain and incorporate it into musculoskeletal assessments and reinforce the role of a family physician to initiate the appropriate cancer workup without delay. It will discuss when to go beyond conservative treatment for "typical" appearing joint pain and will discuss diagnostic pitfalls of prostate cancer and the utility of PSA testing in atypical presentations.

Case Report

A 69-year-old male presented in April of 2024 with right hip pain. Initially, the patient had attributed his symptoms to overexertion; however, the patient is active at baseline and noted that this pain felt distinct from his typical musculoskeletal injuries. He initially attempted conservative management, including NSAIDs, massage, hip flexor exercises, acupuncture, and osteopathic manipulative therapy, but experienced no improvement. The patient also changed his footwear, considering the possibility that the symptoms were activity-related or due to inadequate support during sports. About three months later, the pain had not improved and had started to involve the left hip, despite use of Tylenol and ibuprofen. He reported worsening of back pain, bilateral hip pain and also headaches. The pain was described as originating from the bone, and felt unlike any previous musculoskeletal injuries he had experienced.

Given that the symptoms were unresolved with conservative management, the patient's primary care provider had explored the potential of Lyme disease. Labs were ordered including ESR/Lyme screen (normal results) and CMP, which was notable for an elevated ALP at 1372. CBC demonstrated elevated metamyelocytes, myelocytes, and thrombocytopenia at 124. Previous CMPs (2022 and prior) had demonstrated normal ALP levels, prompting secondary evaluation for Paget's disease of the bone and also further evaluation for other types of cancers (mother had hx of pancreatic cancer). Labs were expanded to evaluate hepatic/pancreatic vs extrahepatic causes. Skull, lumbosacral, femur, and tibia/fibula x-rays were ordered. TSH, free PSA, GGT, CA-19-9 were normal.

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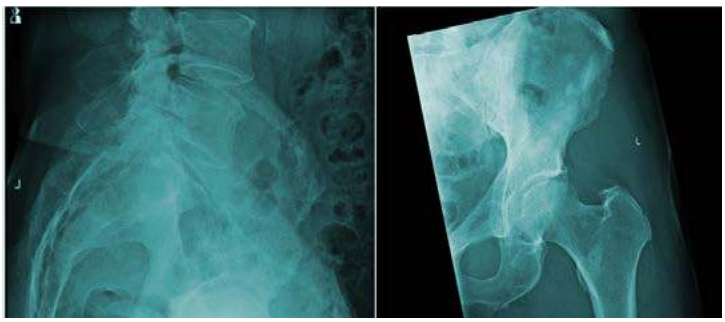

Figure 1: X rays of the lumbar spine and pelvis which demonstrate metastasis as sclerotic lesions.

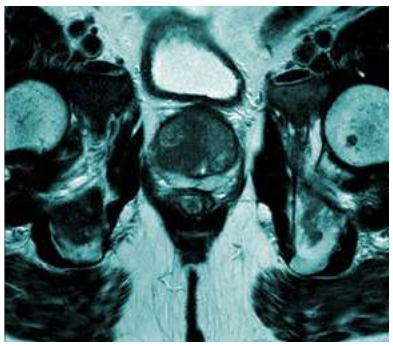

Figure 2: Coronal T2 view MRI images

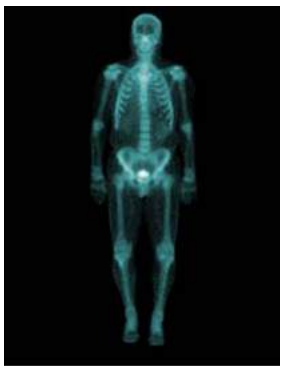

Figure 3: Bone Scan from 1/20/25

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Total PSA was elevated at 1269. PTH was elevated at 130. During all this time, the patient had no urinary symptoms such as nocturia or frequency. There was no family history of prostate cancer; he was non-obese and never smoked. He denied weight loss; however, X-ray of the lumbar spine demonstrated patchy sclerotic changes overlying the pedicles of the lumbar spine, as well as in the sacrum and pelvis, again suspicious for metastatic disease versus Paget's disease. Refer to *Figure 1* to view the x-ray.

Due to the significant elevation of PSA at 1269, it was presumed that the patient had metastatic prostate cancer rather than Paget's disease. Urology was promptly consulted with urgent evaluation arranged within the week. Digital rectal exam (DRE) per urology demonstrated a "smooth prostate with asymmetry, right side higher, firm. Increased firmness on the left side, 35-40 grams. No tenderness to palpation."

Treatment with degarelix 240mg was initiated and the patient was started on an androgen receptor pathway inhibitor daily (abiraterone 1000mg), along with prednisone 5mg BID. MRI prostate in August 2024 revealed a "PI-RADS 5 lesion in the right transverse mid gland extending apex to base capsular bulge without gross extraprostatic extension with positive enhancement, PI-RADS 4 lesion within the left posterior lateral mid gland peripheral zone with capsular abutment without gross extraprostatic extension. Extensive osseous metastatic disease in the visualized bony pelvis and proximal femurs. Prostate volume: 55 mL." MRI images are shown in *Figure 2*. After MRI was obtained and insurance approval was obtained, Eligard (leuprolide acetate), a GnRH agonist, was also initiated. Transrectal ultrasound demonstrated a prostate volume of 42.9 cc, with hypoechogenic areas existing which could represent areas of malignancy in the left lateral base. Six biopsies were obtained. The pathology report had demonstrated a Gleason score 3+4=7 in 2 of 6 cores. PET Illucix scan in October 2024 had demonstrated findings of diffusely increased uptake in multiple areas of the prostate, which is consistent with diffuse prostate cancer.

By October, PSA had improved to 7.98 and ALP had become 989. Initial testosterone prior to starting treatment measured at 407, which dropped to <2.5 in October. Denosumab 120mg injections were ordered to preserve bone health. By June 2025, the patient's PSA had dropped to 0.2, indicating an appropriate response to treatment. A referral to oncology for further discussion of chemotherapy and XRT options was placed. DEXA scan was obtained considering the patient's antihormonal therapies and it was negative for osteoporosis. The patient obtained a bone scan in January 2025, which is shown in *Figure 3*. The patient was able to return back to his baseline functional level during and after treatment; he continues to enjoy daily sports and engages in regular physical activity.

Discussion

Prior to 2008 , the USPSTF recommendation for prostate cancer screening was annually with PSA; however, this changed by 2012 to no screening at all, as the harms of screening outweighed the benefits.[1] PSA testing does have some limitations and pitfalls in that there is low specificity (can be elevated in other conditions such as BPH or prostatitis) and that there are variations amongst age and race; also, a normal PSA does not mean that cancer is ruled out. Unfortunately, this decrease in screening led to a rise in the number of metastatic cancers detected.[2] Current guidelines state that men 55-69 should make individual decisions to be screened based on shared decision making (SDM) discussions with their healthcare provider.

It is essential that patients engage in regular discussions with their healthcare provider to improve recognition and timely management of symptoms. However – for effective discussions – providers should have clear knowledge about the less common clinical features of prostate cancer. This patient presented without the typical urinary prostate cancer symptoms. In addition to this, he had no family history of prostate cancer, was not African American, and led a healthy lifestyle. He was stratified as low risk for prostate cancer; based on the USPSTF recommendations and SDM discussions between patient and provider, both parties likely reached the conclusion that he did not require regular prostate cancer screening with PSA.

As per a study published in *Clinical Genitourinary Cancer*, focusing on those with advanced prostate cancer, "commonly reported symptoms were fatigue (73%), urinary symptoms (63%), sexual function symptoms (62%), and bone pain (52%)."[3] However, of those that had bone metastasis, most of them had presented with pain (73%) before being diagnosed with metastatic prostate cancer.[3] Hip pain is often commonly attributed to arthritis. The average patient will self-medicate with pain reliever attributing these symptoms to "getting old."

Socioeconomic status and level of educational background heavily influences health literacy and a patient's ability to navigate their concerns and in turn, impact their chances of survival and positive outcomes. In a retrospective cohort study published in *Cancer* (2025), 9603 patients with various cancers (including breast, renal, prostate, lung, renal, colorectal, etc.) concluded low literacy was associated with all-cause mortality.[4] Our patient's high health literacy level most likely resulted in a better health outcome on recognition of the uniqueness of his symptoms and the quality of SDM.[3] Community outreach programs and education can help mitigate the impacts of poor health literacy on cancer detection and survival.

For atypical presentations, PSA can be a critical diagnostic clue, even if the symptoms are atypical and non-urinary in nature. For hip pain, it is important to maintain a broad differential but also know when to go beyond conventional treatments. Significant red flag symptoms for low back pain include: age <18 or >50, pain not resolved by analgesia, history of trauma or recent spinal interventions (surgery, injections), history of coagulopathy or

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abdominal aortic aneurysm, symptoms or history of malignancy (night sweats, weight loss, etc.), history of immunodeficiency (diabetes mellitus [DM], IVDU), recent infection, or fever and cord compression/cauda equina symptoms (bowel/bladder/erectile dysfunction, saddle anesthesia, progressive bilateral leg weakness).[5] There have also been several case reports regarding malignancy masquerading as hip arthritis.[6] It is important for family physicians to be cognizant of malignancy which can contribute to hip pain.

Endnotes

1. United States Preventive Services Task Force. Prostate Cancer: Screening. May 8, 2018. U.S. Preventive Services Task Force. Accessed 2025. https://www.uspreventiveservicestaskforce.org/uspstf/recommendation/prostate-cancer-screening.
2. Desai, Mihir M., Gennaro E. Cacciamani, Kiran Gill, et al. 2022. "Trends in Incidence of Metastatic Prostate Cancer in the US." *JAMA Network Open* 5 (3): e222246. https://doi.org/10.1001/jamanetworkopen.2022.2246.
3. Drudge-Coates, Lawrence, William K. Oh, Bertrand Tombal, et al. "Recognizing Symptom Burden in Advanced Prostate Cancer: A Global Patient and Caregiver Survey." *Clinical Genitourinary Cancer* 16, no. 2 (2018): e411–19. https://doi.org/10.1016/j.clgc.2017.09.015
4. Al Hussein Al Awamlh, B., K. A. Moses, J. Whitman, T. Stewart, S. Kripalani, and K. Idrees. 2025."Health Literacy and All-Cause Mortality among Cancer Patients." *Cancer* 131, no. 6 (March 15, 2025): e35794. https://doi.org/10.1002/cncr.35794.
5. Sikina, Matthew, MD, and MAJ John Kiel, DO, MPH. 2022. "Re-evaluating Red Flags for Back Pain." *American College of Emergency Physicians*, August 17. https://www.acep.org/sportsmedicine/newsroom/newsroom-articles/august2022/re-evaluating-red-flags-for-back-pain.
6. Meals, Roy A., David S. Hungerford, and Mary Betty Stevens. 1978. "Malignant Disease Mimicking Arthritis of the Hip." *JAMA* 238 (3): 244–246.https://jamanetwork.com/journals/jama/fullarticle/358721.

References

Chamberlain, Rachel. 2021. "Hip Pain in Adults: Evaluation and Differential Diagnosis." *American Family Physician*, January 15. https://www.aafp.org/pubs/afp/issues/2021/0115/p81.html

Lowrance, William, Robert Dreicer, David F. Jarrard, et al. *Updates to Advanced Prostate Cancer: AUA/SUO Guideline (2023). The Journal of Urology* 209, no. 6 (June 2023): 1082–1090. https://doi.org/10.1097/JU.0000000000003452.

Pinsky, Paul F., and Howard Parnes. 2023. "Screening for Prostate Cancer." *New England Journal of Medicine* 388, no. 15 (April 12): 1405–14. https://doi.org/10.1056/NEJMcp2209151.

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Anorexia in Patient with Feeding Tube

By Haoming Liu, DO; Alina Intisar, MD; Crystal Pang, DO; Zubia Sagarwala, DO and Anubhav Agarwal, MD

Abstract

Anorexia nervosa is an eating disorder characterized by persistent restriction of energy intake relative to requirements leading to a significantly low body weight, an intense fear of gaining weight or becoming fat, and disturbance in the way in which one's body weight or shape is experienced.[1] We present a case of a 44-year-old female with a history of caustic injury requiring GJ-tube placement for feeding and extremely low body weight (BMI <10) who has exhibited persistent behaviors that interfere with energy intake and physiological and psychiatric manifestations clinically consistent with anorexia nervosa. This case presented diagnostic and management challenges, warranting multidisciplinary effort to optimize the patient's long-term recovery.

Introduction

Anorexia nervosa is a serious and potentially fatal eating disorder, a group of psychiatric disorders defined as disturbance in eating or eating-related behaviors that impair physical or psychosocial functioning.[2] The diagnostic criteria of anorexia nervosa in DSM-5 by the American Psychiatric Association is defined as a) restriction of energy intake relative to requirements, resulting in significantly low body weight; b) intense fear of gaining weight or persistent behavior that interferes with weight gain; and c) disturbance in self-perceived weight or shape, or denial of the seriousness of current low body weight. Severity is based on body mass index (BMI) derived from World Health Organization categories for thinness in adults: Mild: BMI greater than or equal to 17 kg/m^2, Moderate: BMI 16–16.99 kg/m^2, Severe: BMI 15–15.99 kg/m^2, Extreme: BMI less than 15 kg/m.[2,3] Anorexia nervosa is associated with a high incidence of coexisting psychiatric conditions including depression, anxiety, and suicide attempts and medical complications notably electrolyte abnormalities, bradycardia, endocrine disturbances, and decreased bone density.[4] Based on current literature review, although anorexia has been studied in the general population, there is a lack of research specifically addressing anorexia in patients receiving tube feedings and the complexity of care in this population.

We present a patient who has persistent behavior and physiological complications consistent with a severe case of anorexia nervosa, which is complicated by a history of caustic injury requiring bolus feeding at the time of presentation. Timely diagnosis is key to initiating treatments including nutrition support, psychotherapy, and pharmacotherapy.

Case

A 45-year-old female with past medical history significant for caustic injury and esophageal burn in 2004, following tracheostomy and GJ Peg tube placement, severe malnutrition, and multiple admissions for tube site infection, presented with abdominal pain, dizziness and fall without loss of consciousness or head strike. At baseline, the patient is independent with ADLs including ambulation, and tube feeds with care. Initial vitals were notable for BP 90/59, HR 65, weight 54lbs, height 5'1", and BMI 10.20kg/m^2. Physical exam was significant for an ill-appearing, thin, frail woman with a tracheostomy (not requiring supplemental oxygen and non-ventilator dependent) in place with erythema, crusting, and drainage noted at the GJ tube site. CTAP showed incidental finding of middle and lower lobe pneumonia, otherwise consistent with prior noted cholelithiasis without cholecystitis and no acute pathology. The patient was admitted for community acquired pneumonia and failure to thrive with severe malnutrition. She was empirically started on piperacillin and tazobactam and vancomycin. Shortly after being admitted, the patient was noted to be hypotensive with MAP < 60 with systolic BP in the 70s and intermittently bradycardic to the 40s, although responsive to activities. The patient was upgraded to the medical intensive care unit, where she was started on hydrocortisone for suspected adrenal insufficiency. Infectious work up showed positive for human rhinovirus/enterovirus, while sputum culture and blood cultures were negative. Antibiotics were discontinued after completing a 5-day course. Transthoracic echocardiogram showed LVEF of 60%. The patient was subsequently taken off from Levophed, however vitals remained labile with blood pressure ranging from 80-90s/50-60s and resting heart rate in 50s. Cosyntropin stimulation test was negative. The patient was started on hydrocortisone and fludrocortisone given concerns of adrenal insufficiency secondary to severe malnutrition. In addition, the patient had a persistently elevated lactate level, which was attributed to increased metabolic demand. This trend was discontinued because the patient remained stable.

Throughout the hospital course, the patient had frequently exhibited behaviors that would go against medical care and measures to ensure adequate nutritional intake, including disconnecting tube feeds without alerting nursing staff, taking out IV lines, and requesting frequently to go home. The patient stated

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Table 1. DMS-5 Diagnostic Criteria for Anorexia Nervosa, Types, and Remission.[5]

DSM-5 Diagnostic Criteria for Anorexia Nervosa and Remission		
Disorder Class: Feeding and Eating Disorders		
A. Restriction of energy intake relative to requirements, leading to a significant low body weight in the context of the age, sex, developmental trajectory, and physical health (less than minimally normal/expected).	B. Intense fear of gaining weight or becoming fat or persistent behavior that interferes with weight gain.	C. Disturbed by one's body weight or shape, self-worth influenced by body weight or shape, or persistent lack of recognition of seriousness of low bodyweight.
Restricting type During the last 3 months, has not regularly engaged in binge-eating or purging.		**Binge-eating/purging type** During the last 3 months, has regularly engaged in binge-eating or purging.
Partial remission After full criteria met, low bodyweight has not been met for sustained period, BUT at least one of the following two criteria still met: Intense fear of gaining weight/becoming obese or behavior that interferes with weight gain OR Disturbed by weight and shape.		**Full remission** After full criteria met, none of the criteria met for a sustained period of time.

that she was unable to tolerate feeds in house, however adamantly reported that she was doing bolus feeds at home. As a result there was a steady decline in body weight in the first two weeks of her stay. A detailed psychosocial interview revealed that the patient was not able to display understanding of her medical illness and severity of her need for continued medical nutrition support. A diagnosis of anorexia nervosa was made with concern for major depressive disorder (see Table 1).

Recognizing the importance of nutritional support since adherence remained to be a hurdle to recovery, the medical team worked closely with the patient, family, and nutritionists to establish a structured feeding routine that would meet the patient's daily calorie demand. A regimen of continuous feeding was proposed with 60ml/hr x 12 hr/day. The patient was unable to tolerate this due to abdominal discomfort however was agreeable to reducing the rate and prolonging the timeline of feeds, eventually adjusted to 40ml/hr x 18hr/day. The patient was also started on fluoxetine 10mg po daily as recommended by the psychiatry team. The patient gradually improved in adherence with management and had weight gain of 4-5lb. She was eventually discharged to a skilled nursing facility for nursing care and ongoing nutrition monitoring and medical supervision.

Severity is based on body mass index (BMI) derived from World Health Organization categories for thinness in adults; corresponding percentiles should be used for children and adolescents: Mild: BMI greater than or equal to 17 kg/m2, Moderate: BMI 16–16.99 kg/m2, Severe: BMI 15–15.99 kg/m2, Extreme: BMI less than 15 kg/m2.[5] Purging is self-induced vomiting or misuse of laxatives, diuretics, or enemas.[5]

Table 2. Patient's Hospital Admissions: Reasons for Admission and Associated Weight Changes Over Multiple Years.

Date of admission	Reason for admission	Weight	BMI
03/2023	Abdominal wall cellulitis	100 lb (45.4 kg)	18.89 kg/m2
06/2022	Sepsis due to cellulitis	91 lb 3.2 oz (41.4 kg)	13.60 kg/m2
10/2021	Sepsis due to cellulitis	72 lb (32.7 kg)	14.06 kg/m2
05/2020	AKI and pseudomonas wound infection	89 lb (40.4 kg)	16.82 kg/m2
08/2014	Abdominal wall cellulitis	64 lb (29.0 kg)	12.10 kg/m2

Discussion

The definition of anorexia nervosa was revised for DSM-V to no longer require verbalized fear of weight gain if behaviors that interfere with weight gain can be observed (see Table 1).[6] Our patient met the criteria for the restriction type of anorexia nervosa given restriction of energy intake relative to requirements despite being on enteral nutrition through tube feeding, persistent behavior that interferes with weight gain (such as self-removing feeding tube due to discomfort or irritability), and a denial of the serious implications of her low body weight. The diagnosis of anorexia nervosa was not made until the most recent admission. Previous times, the focus at hand was treating her acute illness such as sepsis, and it was not recognized until now that the underlying anorexia was likely putting her at risk of such infections and hospitalizations in the past.

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A summary of the patient's weight and BMI on previous admissions in the past 10 years can be seen in Table 2. Notably, the patient had a body weight as low as 29.0 kg in 2014 and highest of 45.4 kg more recently in 2023, signifying her long-lasting state of malnutrition and waxing-waning nature of her condition.

Anorexia nervosa is associated with significant medical complications, particularly affecting the cardiovascular system with bradycardia and hypotension as common manifestations.[7] In this case, the patient demonstrated marked hemodynamic instability, with a new baseline mean arterial pressure of less than 60 mmHg and a heart rate of 50 beats per minute. Moreover, the patient also presented with other common manifestations of anorexia nervosa including dry skin, hair loss, and feeling cold due to low body fat and poor nutrition. Given the multiple comorbidities, the patient's medical stability, age, and duration of illness must be considered in deciding the treatment of anorexia nervosa.[8] It became necessary for us to closely monitor her weight and frequently reassess her feeding regimen to ensure adequate caloric and nutrient intake while minimizing the risk of refeeding syndrome.

Conclusion

The estimated lifetime prevalence for anorexia nervosa in adult women is 1.42% according to large US cohort studies.[9] Diagnosis and management could be complicated given a patient's inability to recognize the seriousness of low body weight, treatment resistance, frequent medical complications, and in this case, the presence of a GJ tube. By taking the time to zoom out of the patient's acute presentation, we were able to see clearly that she met the diagnostic criteria of anorexia nervosa in DSM-5. Recognizing anorexia was essential to directing the course of treatment for associated medical manifestations and psychiatric conditions, focusing on support and counseling considering the patient's unique history. Although the road to recovery and healthy weight may take some time, recognizing the different complexities makes this case unique and valuable to learn from.

Endnotes

1. Feltner C, Peat C, Reddy S, et al. Screening for Eating Disorders in Adolescents and Adults: An Evidence Review for the U.S. Preventive Services Task Force [Internet]. Rockville (MD): Agency for Healthcare Research and Quality (US); 2022 Mar. (Evidence Synthesis, No. 212.) Appendix A Table 1, Summary of DSM-5 Diagnostic Criteria for Eating Disorders. Available from: https://www.ncbi.nlm.nih.gov/books/NBK578994/table/appa.tab1/
2. Linda Mustelin, Yasmina Silén, Anu Raevuori, Hans W. Hoek, Jaakko Kaprio, Anna Keski-Rahkonen, The DSM-5 diagnostic criteria for anorexia nervosa may change its population prevalence and prognostic value, Journal of Psychiatric Research,Volume 77,2016,Pages 85-91,ISSN 0022-3956,https://doi.org/10.1016/j.jpsychires.2016.03.003.
3. Attia E, Becker AE, Bryant-Waugh R, Hoek HW, Kreipe RE, Marcus MD, Mitchell JE, Striegel RH, Walsh BT, Wilson GT, Wolfe BE, Wonderlich S. Feeding and eating disorders in DSM-5. Am J Psychiatry. 2013 Nov;170(11):1237-9. doi: 10.1176/appi.ajp.2013.13030326. PMID: 24185238.
4. Phillipou A, Schmidt U, Neill E, Miles S, McGorry P, Eddy KT. Anorexia Nervosa—Facts, Frustrations, and the Future. JAMA Psychiatry. Published online June 04, 2025. doi:10.1001/jamapsychiatry.2025.0812
5. Substance Abuse and Mental Health Services Administration. DSM-5 Changes: Implications for Child Serious Emotional Disturbance [Internet]. Rockville (MD): Substance Abuse and Mental Health Services Administration (US); 2016 Jun. Table 19, DSM-IV to DSM-5 Anorexia Nervosa Comparison. Available from: https://www.ncbi.nlm.nih.gov/books/NBK519712/table/ch3.t15/
6. US Preventive Services Task Force. Screening for Eating Disorders in Adolescents and Adults: US Preventive Services Task Force Recommendation Statement. JAMA. 2022;327(11):1061–1067. doi:10.1001/jama.2022.1806
7. Yahalom M, Spitz M, Sandler L, Heno N, Roguin N, Turgeman Y. The significance of bradycardia in anorexia nervosa. Int J Angiol. 2013 Jun;22(2):83-94. doi: 10.1055/s-0033-1334138. PMID: 24436590; PMCID: PMC3709923.
8. Cost J, Krantz MJ, Mehler PS. Medical complications of anorexia nervosa. Cleve Clin J Med . 2020;87:361–366. doi: 10.3949/ccjm.87a.19084.
9. US Preventive Services Task Force. Screening for Eating Disorders in Adolescents and Adults: US Preventive Services Task Force Recommendation Statement. JAMA. 2022;327(11):1061–1067. doi:10.1001/jama.2022.1806

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Special thanks to our nurses, case managers, social workers and nutritionist team in caring for this patient.

Adenocarcinoma of Esophagus in an Adolescent: Unexpected Age for Esophageal Cancer

By Ani A. Bodoutchian, MD, MBA, FAAFP

Abstract

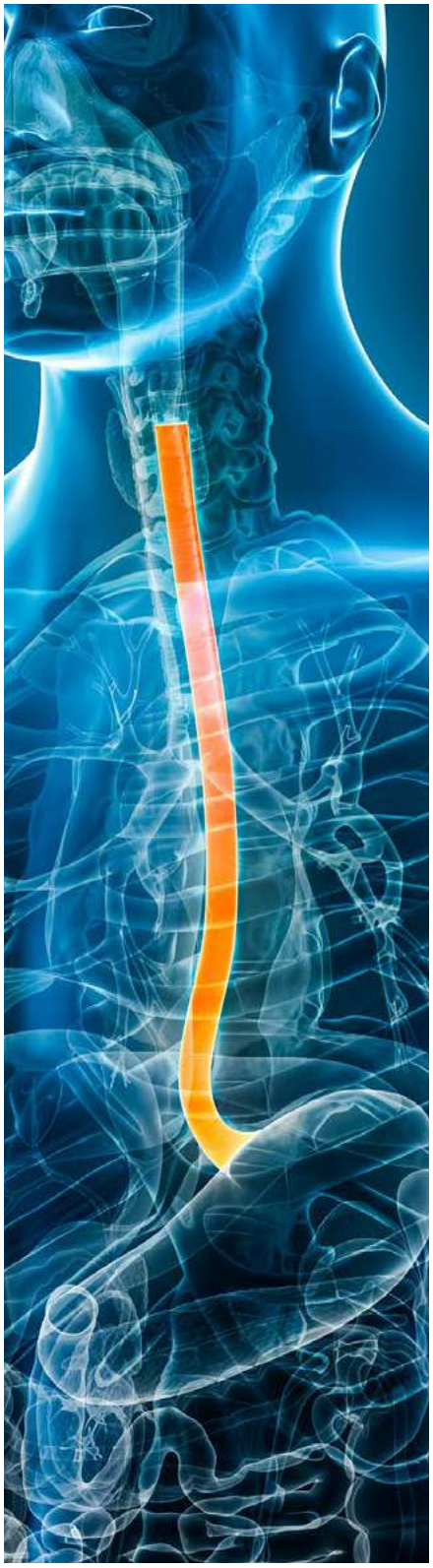
Esophageal malignancies are commonly seen in the sixth to eighth decades of life and are extremely rare at a young age and in children. Few cases have been reported worldwide. An 18-year-old male presented with a complaint of abdominal pain, nausea, fatigue, difficulty swallowing solids and metallic taste in the mouth. Laboratory studies showed hemoglobin of 7.4 grams per deciliter (g/dl). Endoscopy revealed a large necrotic mass in the gastro-esophageal junction. Pathology diagnosis of poorly differentiated adenocarcinoma with extensive tumor necrosis.

Limited data and review regarding esophageal cancer in pediatric and young populations must not make us complacent. Age should not preclude us from considering and ruling out this deadly cancer. The prognosis of esophageal cancer is poor as it metastasizes in the early stages of disease. The goal is to consider and understand this rare disease in the young. It is important for anyone who treats children and adolescents with a history of dysphagia, fatigue, and anemia to consider a malignant esophageal process. Early diagnosis is vital and life-saving given the hallmark accelerated mortality rate of esophageal cancer as a result of metastasis in its early stages.

Introduction

Esophageal malignancies are commonly seen in the geriatric population.[1] It is extremely rare in young patients and not many cases have been reported worldwide to date.[1,2,3]

Esophageal cancer (EC) has distinguished itself as being extremely aggressive, with high mortality and poor prognosis due to its late presentation of symptoms.[4,5] The incidence of this cancer ranks eighth around the world. It is the sixth most common for mortality.[2,4,6] Although there have been many advances in modern medicine, the 5-year prognosis for survival is at best 20%.[5,7] The two most common cell types are esophageal adenocarcinoma (EAC) and esophageal squamous cell carcinoma (ESCC).[2,4,5] Other esophageal cancers include: sarcomas, small cell carcinomas as well as, carcinomas, melanomas, leiomyosarcomas, carcinoids, and lymphomas.[4]



Histologically, ESCC is the predominant esophageal cancer around the world.[4,5] In developing countries, almost 80% of esophageal cancer is ESCC. In contrast, in the Western industrial countries, EAC is more common.[1,2,4,5]

Between the genders, EAC predominates amongst male patients (M: F =7:1) while ESCC is evenly distributed between the genders.[4,5,8] Racially, the incidence of EAC is higher in Caucasians than African Americans,[4,5] whereas ESCC is higher in African Americans than Caucasians by 3:1 ratio.[4]

Case Report

An 18-year old Caucasian male with a BMI of 23.57 complained of reflux for one year intermittently. He was seen by his family physician and reassured that his reflux was due to stress at school. He admitted to occasional drinking, three – four alcoholic drinks per month, and smoking marijuana three times a month, on occasion. He denied cigarette smoking and vaping. The family history was positive for gastro esophageal reflux disease (GERD).

His presentation to the emergency department was due to complaints of severe abdominal/chest pain. The patient's pain was sharp, spasmodic, intermittent, and associated with nausea but no vomiting or regurgitation.

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He reported difficulty swallowing solids but not to liquids. He also complained of a metallic taste in his mouth. His history included worsening fatigue, dizziness, and shortness of breath for the previous one month. He described a need to rest after a shower due to his severe fatigue. He denied change in stool color or visible blood. The patient admitted to losing some weight but again attributed it to not being hungry due to stress.

On physical exam, the patient was a pale age-appropriate well-developed young man. Pertinent positive findings were: epigastric pain, pallor, mild tachycardia, hemoglobin of 7.4 grams per deciliter (g/dl) and positive stool guaiac.

The patient was hospitalized for symptomatic anemia and started on proton pump inhibitors and transfused. Endoscopic gastroduodenoscopy revealed a large, friable, cavitating necrotic mass penetrating beyond the muscularis propria located in the region of the gastro-esophageal junction. The mass was circumferential in the gastric cardia extending 3cm into the stomach. The final histopathology report findings were adenocarcinoma poorly differentiated with extensive tumor necrosis involving esophagogastric junction and gastric mucosa. After positron emission tomography (PET)/computed tomography (CT), his cancer was staged at T3, N1, M0. His care was transferred to the surgical oncology unit specializing in gastroenterological oncology. The patient began neoadjuvant chemotherapy plus radiotherapy to be followed by surgery. This patient died four months after his initial diagnosis due to progressive disease and poor response to chemotherapy and radiation.

Discussion

The pathogenesis of esophageal cancer is associated with irritation of the esophageal epithelium leading to inflammation. In turn, this chronic irritation and inflammation transforms into dysplasia and then finally to in situ malignancy.

In general, EC is associated with older age, obesity, heavy alcohol use, smoking, consumption of extremely hot beverages, diet high in red meat consumption, low intake of fresh fruits and vegetables, low socioeconomic status, spicy food, lack of physical activity, tylosis, achalasia, lye stricture lesions, GERD, Barrett's esophagus, esophageal diverticula, Plummer-Vinson syndrome, and human papilloma virus (HPV).[2,3,4,5,9,10] Additionally, exposure to heavy metals, opium, yerba maté, carbonated drinks, pickled foods, myotoxins, paints, lacquers, toluene, synthetic adhesives, sulphuric acid, exposure to N-nitrosamines and polycyclic aromatic hydrocarbons are also linked to EC.[4,5,9,10]

The clinical management of esophageal cancer is based on its staging in accordance with the Tumor - Node - Metastasis (T-N-M) system developed by the American Joint Committee on Cancer (AJCC).[11] Various modalities are part of the armamentarium to stage this cancer, including computer tomography, endoscopy and positron emission tomography.[12] The approach to treatment would be determined by the histopathology as well as on the magnitude of the disease as it relates to metastasis.

In the past decade, there are novel, more direct and focused methodologies in the treatment of EC,[13] including targeting signaling pathways with monoclonal antibodies against epidermal growth factor receptor (EGFR), vascular endothelial growth factor (VEGF), human epidermal growth factor receptor 2 (HER-2), mammalian target of rapamycin (mTOR), and fibroblast growth factor receptor-2 (FGFR2) just to name a few.[12] Moreover, there are other targeted therapies under investigation that include, but not limited to, epigenetics with DNA methyltransferase inhibitors and microbial ecosystem directed treatment.[12]

Currently, combined therapy that includes radiation therapy, chemotherapy, immunotherapy and surgery can improve the prognosis of some patients. Optimally, a multifaceted approach would be best depending on the stage of the cancer and the wishes of the patient.[13] However, despite genomic therapies, systemic adjuvant and neoadjuvant therapies to surgery, the overall prognosis to EC remains dismal.[12,13,14]

During literature review, there were no management guidelines nor recommendations for the pediatric/adolescent age group on esophageal cancer treatment due to its extreme rare occurrence.[1,2,3,14] Of note, the pediatric/adolescent group reacts poorly to chemotherapy, as was the case here, and die due to progressive disease.[14] Pediatric and adolescent patients have unique physiology, and there is a noticeable need for adult and pediatric oncologists to combine efforts in the management of this disease.[14]

Although esophageal malignancies are exceptionally scarce at a young age and in children / adolescents, the literature findings revealed that an 8-year-old girl from India was the youngest patient with reported esophageal tumor[1,3] with dysphagia as the principle symptom followed by clinical worsening with weight loss, anemia and dehydration.[10,11,13,14]

Epidemiological studies have shown that childhood esophageal tumorigenesis is mainly influenced by environmental factors, some of which are referenced above, over an extended period of time of at least 15-20 years.[3,4,9,10,14] These factors, which cause chronic esophagitis, along with nutritional deficiency may be responsible for conditions more favorable for the development of esophageal cancer.[1,4,9,10,14] Furthermore, deficits in vitamin C, vitamin E, vitamin B2, retinol and zinc, may also be associated

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with the disease.[4,5] However, the triggers for the onset of teenage esophageal cancer are yet unknown.[1,6] While EC is a rare occurrence in childhood, it still must be considered.[1,6]

There may also be a "genetic" factor when clustering EAC within several family members.[5] Genetic propensity combined with risk factors, may suggest a vulnerability variance to esophageal cancer in various individuals[12,15] There is an inference that epigenetics may also play a role.[12,15]

Although adolescent EAC and ESCC may have some parallel features to those presenting in adults, in younger age group there are instances whereby distinctive etiological influences varied.

Our case, and those identified in the literature in the Journal of Pediatric Hematology Oncology by Issaivanan et al., clearly illustrate the inability to determine precise factors influencing EAC or ESCC.[14] Our case report supports this statement as our patient had no abusive caustic characteristics to predispose him to EAC.

Similar to our patient, a case reported by Theilen et al. identified a 21-year-old with EAC but no associated Barrett's esophagus.[8,14,17] Additionally, Isaivanan et al. concluded that they were "unable to identify" any distinctive etiological influences for EAC including reflux or Barrett's esophagus.[14]

Conclusion

While there is limited data and review regarding esophageal cancer in pediatric and the young population, age should not make physicians complacent or preclude us from considering and ruling out this deadly cancer. Although there are many advances regarding treatment, early diagnosis is vital and lifesaving. This is crucial given the hallmark of accelerated mortality rate of esophageal cancer as a result of metastasis in its early stages.

The goal is to consider and understand this rare disease in the young with its nuances. Although there may be similar features to adult esophageal cancer, not all characteristics are similar. It is important for anyone who treats children and adolescents, with a history of prolonged reflux and seemingly innocuous risk factors, to consider a malignant esophageal process.

Endnotes

1. Allam AR, Fiaz FM, Khawaja FI, Sultan A. Esophageal Carcinoma in a 15-Year-Old Girl: A Case Report and Review of the Literature. Annals of Saudi Medicine 2000: 20(3-4): 261-264.
2. Dawsey SP, Tonui S, Parker RK, et al. Esophageal Cancer in Young People: A Case Series of 109 Cases and Review of the Literature. PloS One 2010: 5(11): e14080.
3. Singh O, Gupta S, Baghel P, Shukla S, Paramhans D, Mathur RK. Esophageal Carcinoma in a 16-Year-Old Girl 8 Years after Gastrotomy. Journal of Clinical Oncology 2010: 28(1): e1-e3.
4. Zhang Y. Epidemiology of Esophageal Cancer. World Journal of Gastroenterology 2013: 19(34): 5598–5606.
5. Then EO, Lopez M, Saleem S, et al. Esophageal Cancer: An Updatd Surveillance Epidemiology and End Results Database Analysis. World Journal Oncology 2020: 11(2): 55–64.
6. Asombang AW, Kayamba V, Lisulo MM et al. Esophageal squamous cell cancer in a highly endemic region. World Journal of Gastroenterology 2016: 22(9): 2811–2817.
7. Cancer of the esophagus [cancer stat facts. SEER.] (n.d.). *Surveillance, Epidemiology, and End Results (SEER)* Available from URL: https://seer.cancer.gov/statfacts/html/esoph.html
8. Theilen TM, Chou AJ, Klimstra S, LaQuaglia M. Esophageal Adenocarcinoma and Squamous Cell Carcinoma in Children and Adolescents: Report of 3 Cases and Comprehensive Literature Review. Journal of Pediatric Surgery Case Reports 2016: 5(1):23-29.
9. Kamangar F, Chow WH, Abnet C, Dawsey S. Environmental Causes of Esophageal Cancer. Gastroenterology Clinics of North America 2009: 39(1)27-vii
10. Hedawoo JB, Nagdeve NG, Sarve GN. Squamous cell carcinoma of esophagus in a 15-year-old boy. Journal of Indian Association of Pediatric Surgeons 2010: 15(2): 59-61.
11. Napier KJ, Scheerer M, Misra S. Esophageal cancer: A Review of epidemiology, pathogenesis, staging workup and treatment modalities. World Journal Gastrointestinal Oncology 2014: 6(5):112-120.
12. Yang YM, Hong, P, Xu WW. et al. Advances in targeted therapy for esophageal cancer. Signal Transduction and Target Therapy 2020: (5): 229.
13. Layke JC, Lopez PP. Esophageal Cancer: A Review and Update. American Family Physician 2006: 73(12):2187-2194.
14. Issaivanan M, Redner A, Weinstein T, et al. Esophageal carcinoma in children and adolescents. Journal Pediatrics Hematology Oncology 2012: 34(1):63-67.
15. Cui QBS, Wu C, Lin D. Genomic alterations and precise medicine of esophageal squamous cell carcinoma, Journal of Bio-X Research 2018: 1(1): 7-11.
16. Rodziewicz TL, Houseman B, Hipskind JE. Medical Error Reduction and Prevention. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing 2022: (1):1.
17. Medina AL, Troendle DM, Park JY, Thaker A, Dunbar KB, Cheng E. Eosinophilic esophagitis, Barrett's esophagus and esophageal neoplasms in the pediatric patient: a narrative review. Transl Gastroenterol Hepatology 2021: (6):32.

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Colchicine-Induced Neuromyopathy Following Treatment for Pericarditis

Dante Scarnati and Sangharsha Thapa, MD

Abstract

Myopathy and neuropathy are both rare adverse effects that may be associated with colchicine use. The existing literature on this condition is sparse outside of case reports, contributing to unfamiliarity and delays in making the correct diagnosis. We report the case of a 77-year-old woman who developed both weakness and paresthesias after starting colchicine treatment for pericarditis. The diagnosis was supported by EMG findings of myotonic discharges after the patient was hospitalized. Earlier visits to the emergency room and clinic were missed diagnostic opportunities, which might have prevented hospitalization.

Introduction

Colchicine is an anti-inflammatory drug commonly prescribed to treat certain inflammatory diseases such as gout, pericarditis, Familial Mediterranean fever, and Beçhet syndrome.[5,6] Colchicine's anti-inflammatory effects come from inhibition of microtubule polymerization, disruption of neutrophil migration, and impairment of inflammasome formation in neutrophils and monocytes.[5,6] The most common side effects reported with colchicine include nausea, vomiting, and diarrhea.[5,6] While not as common as the gastrointestinal side effects, colchicine is also known to cause neurologic and musculoskeletal side effects, such as myalgia, myopathy, myotonia, neuropathy, paresthesias, and peripheral neuritis.[6] Most of the literature available on neuromyopathy comes from case reports, highlighting this as a relatively rare phenomenon.[3] The lack of familiarity with this presentation can result in a delay in diagnosis, frequent trips to clinics/hospitals as symptoms progress, and extensive workups. We report on a case of colchicine-induced neuromyopathy in a patient with multiple trips to the emergency room and primary care clinic after receiving treatment for pericarditis.

Case Report

A 77-year-old Caucasian woman presented to the hospital with a chief complaint of 1 month of progressive weakness of her upper and lower extremities along with tingling sensations in her upper extremities. The patient reports that a little over a month ago she developed shortness of breath and chest pain that prompted her to seek hospitalization. She was admitted to the hospital and diagnosed with acute pericarditis. She was treated with a short course of steroids and discharged from the hospital on colchicine and ibuprofen. A few days after returning home, the patient first noticed a constant tingling sensation in her fingertips that migrated towards both of her forearms. The patient noticed that she felt weaker after first noticing the tingling in her fingers and forearms. She reported that she was having difficulty standing up from sitting and walking because her legs felt heavy and difficult to move. The weakness seemed to worsen and she soon required a walker to ambulate. She later noticed that she felt weakness in her arms. She first reported difficulty lifting her arms over head which failed to resolve, making it difficult to comb or wash her hair. Subsequently, she developed weakness in her hands and fingers, making it difficult to pick up utensils and eat. The patient initially went to the emergency room for her symptoms of weakness and tingling. After a normal EKG, CT of head, and blood work ruled out life-threatening emergencies, she was discharged with instructions to follow up with her primary care provider. The patient was seen by her family medicine physician, and her symptoms were thought to be a complication of pericarditis. A workup was started to rule out Lyme disease, hypothyroidism, and myositis as a cause of her symptoms. The patient's weakness progressed to the point where she was unable to ambulate without assistance, and she was admitted for further workup of her symptoms.

The patient has a past medical history of hypertension, hyperlipidemia, hypothyroidism, and chronic kidney disease after a left nephrectomy for renal cancer. The patient's chronic medications included amlodipine 5 mg daily, atorvastatin 40 mg daily, levothyroxine 112 mcg daily, propranolol 80 mg daily, and loratadine 10 mg PRN. Since discharge from the hospital one month prior for pericarditis, she had also been taking colchicine 0.6 mg twice a day and ibuprofen 600 mg every 8 hours as needed for pain. The patient had no other surgical history aside from her left nephrectomy over 7 years ago. She also denied having any allergies, alcohol consumption, smoking history, and recreational drug use.

On admission, she was afebrile with a heart rate of 69, respiratory rate of 18, blood pressure of 153/81, and oxygen saturation of 91% on room air. On exam, she was resting comfortably in no apparent distress; she was fully alert and oriented to person, place, time and situation with no impairments to her attention, language, and speech. Examination of cranial nerves II-XII revealed no abnormalities. Sensation to light touch, pinprick, and vibration was intact in bilateral upper and lower extremities. Muscular strength in upper extremities was noted to be 4/5 for bilateral abduction of the arms at the shoulders, 4/5 for bilateral elbow flexion, 3/5 for bilateral elbow extension, 5/5 for bilateral wrist flexion and extension, and 4/5 for bilateral hand grip. Muscular strength in lower extremities was noted to be 2/5 for bilateral hip flexion, 3/5 for bilateral knee extension, 4/5 for bilateral knee flexion, 4/5 for bilateral dorsiflexion, and 4/5 for bilateral plantarflexion. Reflexes were normal in the upper extremities, but the patellar and Achilles reflexes were diminished in the bilateral lower extremities. The patient had no abnormalities in the examination of her heart, lungs, and abdomen.

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On admission, the patient's labs were notable for elevated inflammatory markers with a sedimentation rate of 43 and C-reactive protein of 0.74. Her creatine kinase levels were elevated at 425. The patient also had an elevated BUN at 41 with a normal creatinine of 1.05. She had mildly elevated liver enzymes with an AST of 68, ALT of 69, and alkaline phosphatase of 154. The rest of her labs, including her complete blood count, coagulation studies, and basic metabolic panel were within normal limits. The patient had an MRI of the C-spine which showed multi-level degenerative changes with no abnormal contrast enhancements of the spine. Electromyography was significant for myotonic discharges.

The patient was diagnosed with colchicine-induced neuromyopathy based on her symptoms of paresthesia and progressive bilateral weakness of extremities, the timing of her symptoms shortly after starting the colchicine therapy, and the findings on her EMG/NCS that were consistent with drug-induced myopathy from colchicine. For treatment, colchicine was immediately discontinued, and the patient was discharged to an acute rehab facility to improve her bilateral upper and lower extremity strength. With the cessation of the offending agent and working with PT/OT for a few weeks, the patient was able to improve the strength in her extremities and make a recovery.

Discussion

The rarity of colchicine-induced neuromyopathy plays a significant role in the lack of familiarity among physicians. In the case of this patient, she had gone to both the emergency room and her primary care provider to address her increasing muscular weakness and paresthesia. The tests and labs that were ordered reflected the most common and life-threatening potential causes, including stroke, volume depletion, Lyme disease, hypothyroidism, and inflammatory myositis. While these serious conditions were ruled out, the patient's symptoms progressed since the root cause was not addressed. It was not until the patient was hospitalized, the correct diagnosis was made, and colchicine was stopped that the patient made a recovery. The diagnosis was only made in the hospital after the patient's EMG showed myotonic discharges, which were a finding noted in myopathy associated with colchicine use in case reports.[2,3] The EMG may have been done as an outpatient if timely suspicion of colchicine-induced neuromyopathy had occurred, which would have saved the costs and risks associated with hospitalization. Alternatively, a trial of stopping the colchicine may have resolved symptoms without a need for a diagnostic EMG, saving the patient from undergoing the costly and unpleasant procedure along with preventing the hospitalization.

Most of the knowledge on colchicine-induced neuropathy and myopathy come from case reports. A recent systematic review of these case reports was published in 2023.[3] The systematic review from McEwan et al. found that the risk of colchicine-induced neuromyopathy was increased in patients with co-morbidities, such as kidney or liver disease, and patients with concomitant medication use of drugs known to cause myopathy.[3] The heightened risk is thought to be due to colchicine being metabolized and excreted by the liver and kidneys, which when impaired increase its serum concentration.

Certain drugs, when taken along with colchicine, may contribute to the development of myopathy symptoms. For example, drugs that already are known to cause myopathy, like corticosteroids, statins, and cyclosporine, were the most common co-medications seen in patients with myopathy from colchicine.[3] Other drugs that inhibit the CYP3A4 enzyme may also contribute to myopathy by increasing the plasma concentration of colchicine, leading to toxicity at smaller doses.[1,3,4,5] In the case of the patient, she had multiple risk factors as she had CKD from a left nephrectomy and chronic use of a statin.

The key takeaway from this case is the importance of taking a proper history and doing a medication reconciliation with patients. It is not feasible for physicians to memorize all adverse effects of every single medication, let alone the very rare but still serious adverse effects. A careful history from this patient reveals that her new symptoms developed after she started a new medication after being discharged from the hospital. Even if physicians would not immediately recognize colchicine as a cause of both neuropathy and myopathy, the timing of her symptoms shortly after starting a new drug should prompt a review of the medication and whether it is known to be associated with such symptoms.

Endnotes

1. Choi, S SL, K F Chan, H K Ng, and W P Mak. 1999. "Colchicine-Induced Myopathy and Neuropathy." *Hong Kong Medical Journal = Xianggang Yi Xue Za Zhi* 5 (2): 204–7. https://pubmed.ncbi.nlm.nih.gov/11821595/.
2. Jung, Dar-Eun, Seung-Hee Na, Yun-Jeong Hong, Seong-Hoon Kim, Tae-Won Kim, and Young-Do Kim. 2024. "Colchicine-Induced Neuromyopathy with Myotonic Discharges in a Patient Using Concomitant Diuretics." *Journal of Electrodiagnosis and Neuromuscular Diseases* 26 (1): 9–13. https://doi.org/10.18214/jend.2023.00129.
3. McEwan, Tim, Jaspreet Bhambra, David F. Liew, and Philip C Robinson. 2023. "Systematic Review of Colchicine Neuromyopathy: Risk Factors, Duration and Resolution." *Seminars in Arthritis and Rheumatism* 58 (February): 152150. https://doi.org/10.1016/j.semarthrit.2022.152150.
4. Olmos-Martínez, José M., Helena Molina, Cristina Salas, José M. Olmos, and José L. Hernández. 2019. "Acute Colchicine-Induced Neuromyopathy in a Patient Treated with Atorvastatin and Clarithromycin." *European Journal of Case Reports in Internal Medicine* 6 (3): 1. https://doi.org/10.12890/2019_001066.
5. "Colchicine." n.d. www.dynamed.com. https://www.dynamed.com/drug-monograph/colchicine.
6. "Colchicine: Drug information." 2025. Uptodate.com. 2025. https://www.uptodate.com/contents/colchicine-drug-information.

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The "Fracture of Necessity": A Case of Galeazzi Injury

By Justin Ali, DO and Erik Augspurger, MD

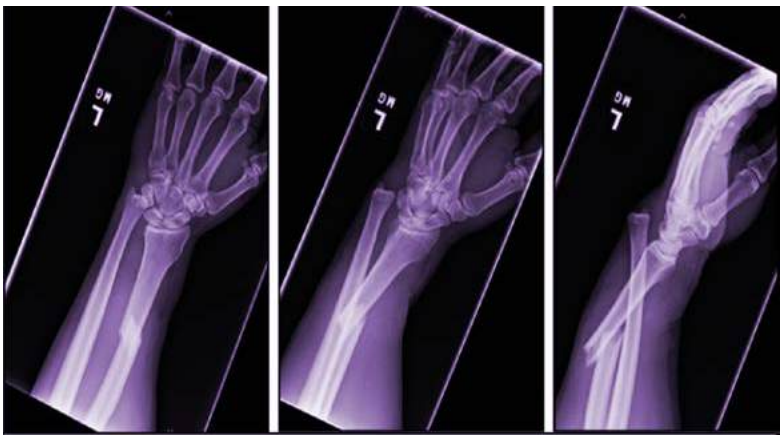

Figure 1A. Initial pre-reduction radiograph of the left forearm and wrist obtained in the ED, demonstrating the radial shaft fracture with associated distal radioulnar joint disruption.

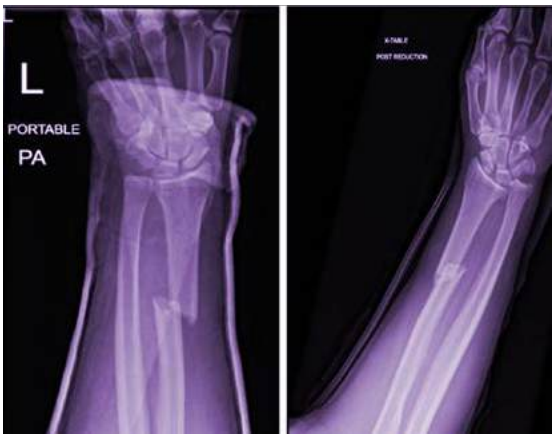

Figure 1B. Post-reduction radiograph of the left forearm and wrist obtained in the ED, demonstrating improved radial alignment with persistent concern for distal radioulnar joint instability.

Figure 1C. Example post-operative radiograph demonstrating a Type II Galeazzi fracture following open reduction and internal fixation, with restoration of radial alignment and stabilization of the distal radioulnar joint[2].

Abstract:

Galeazzi fracture-dislocation involves a distal radial shaft fracture and distal radioulnar joint (DRUJ) disruption. Early identification and surgical intervention are crucial due to high complication rates with non-operative management. A 45-year-old male sustained a type II Galeazzi fracture after a fall. The patient underwent closed reduction and splinting in the ER with outpatient open reduction and internal fixation of the radius and DRUJ stabilization by orthopedics. Galeazzi fractures require careful clinical examination and appropriate stabilization, with timely imaging and referral, to prevent long-term complications and ensure proper management in primary care and sports medicine settings.

Introduction:

Galeazzi fractures are uncommon but clinically significant injuries characterized by a fracture of the distal third of the radial shaft accompanied by a disruption of the distal radioulnar joint (DRUJ).[1] Often presenting with an obvious deformity, this injury is commonly referred to as the 'fracture of necessity', emphasizing the need for surgical intervention to restore forearm stability and function.[2] Mechanistically, Galeazzi fractures typically result from a fall onto an outstretched hand (FOOSH) injury with the forearm in pronation, a very common scenario in contact sports and accidental falls.[3]

Two primary Galeazzi fracture patterns have been described. Type I fractures, caused by axial loading in supination, lead to posterior displacement of the radius and anterior dislocation of the distal ulna. In contrast, type II fractures, from axial loading in pronation, produce anterior displacement of the radius and dorsal dislocation of the distal ulna.[4]

Despite their relatively low incidence, it is essential for primary care providers to recognize Galeazzi fractures promptly as they are orthopedic emergencies due to the complexity of DRUJ disruption and the need for precise anatomical realignment. Additionally, while they may initially appear as isolated radial shaft fractures on imaging, failure to identify the associated DRUJ injury can result in missed or delayed surgical referral leading to chronic instability, functional impairment, and poor

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long-term outcomes.[5] Early identification and timely referral to orthopedic surgery are critical to ensure appropriate operative intervention and optimize recovery.[6]

This case report describes a classic mechanism and presentation of a type II Galeazzi fracture in an active adult following a fall, emphasizing not only the mechanism and radiographic findings but also the importance of early recognition by frontline providers in facilitating definitive care.

Case Report:

A 45-year-old male with a history of hypertension presented to the inpatient family medicine service after falling from a motorized bicycle traveling approximately 30 miles per hour. He reported landing on his outstretched left hand and immediately experienced significant forearm and wrist pain with a visible deformity. He also endorsed mild discomfort in his left hip and knee but denied other injuries or loss of consciousness. On examination, he was neurovascularly intact with no sensory deficits in the median, ulnar, or radial distributions. The left upper extremity exhibited an obvious deformity over the distal forearm and wrist with dorsal prominence suggestive of ulnar dislocation. Moderate swelling was noted without open wounds or skin compromise. The skin was warm and well-perfused, compartments were soft, and motor function was preserved. There was crepitus at the mid-forearm and tenderness to palpation over the distal radius and wrist.

Initial radiographs of the left forearm and wrist revealed an acute fracture of the distal radial diaphysis with 1.3 cm of dorsal displacement, 2.7 cm of foreshortening, and dorsal apex angulation (Figure 1A). There was clear disruption of the distal radioulnar joint (DRUJ), with dorsal and distal dislocation of the ulna. These findings were consistent with a type II Galeazzi fracture-dislocation, a rare but urgent orthopedic injury typically resulting from axial loading of the forearm in pronation.

The patient received IV fluids and analgesia in the ED. Orthopedic surgery was consulted and performed a closed reduction and placed a splint in the ED to restore gross alignment, relieve pain, and protect surrounding soft tissues. Post-reduction imaging showed improved alignment of the radius and DRUJ, with residual 1.7 cm of radial foreshortening and 5 mm of residual radial displacement (Figure 1B). The DRUJ appeared reduced with persistent positive ulnar variance. The patient remained neurovascularly intact following the reduction. Exact anatomic reduction is not required at this stage, as definitive management with open reduction and internal fixation would be performed in the operating room, where precise restoration of radial length, alignment, and distal radioulnar joint stability could be achieved. Due to the high-energy mechanism of injury and elevated creatine kinase (CK) level of 583 U/L on presentation, he was admitted for observation to monitor for compartment syndrome and rhabdomyolysis.

On his morning labs, his CK trended upward to 1009 U/L, likely related to the acute fracture and upper/lower extremity muscle injury from the trauma, while his CBC and CMP remained within normal limits. He remained clinically stable with no signs of compartment syndrome. After overnight observation, the patient was discharged in a forearm splint and sling with instructions to remain non-weight-bearing and to follow up with orthopedic surgery within one to two days for discussion of definitive operative management. Definitive operative management of the fracture was deferred during this hospitalization due to limited available staff. He was counseled on warning signs of compartment syndrome and instructed to return to the emergency department if concerning symptoms develop.

Thereafter, he presented for follow up with orthopedic surgery four days later as directed and underwent open reduction and internal fixation of the distal diaphysis of the radius and the DRUJ (Figure 1C).[2] He was placed into a sugar-tong splint for protection of the DRUJ and underwent an outpatient occupational therapy rehabilitation plan.

This patient demonstrates a classic mechanism and presentation of a type II Galeazzi fracture, highlighting the importance of early recognition and timely referral by primary care and sports medicine providers. Although the radial fracture is often apparent on imaging, the associated DRUJ instability can be easily overlooked, emphasizing the need for a high index of suspicion in any forearm fracture with wrist pain or deformity. Clinical features that may raise concern include ulnar-sided wrist pain, prominence or abnormal mobility of the distal ulna at the wrist, and pain or crepitus at the distal radioulnar joint elicited during stress testing, all of which were present in this patient.

Discussion:

Galeazzi fractures, though relatively rare, represent a critical subset of forearm injuries that demand prompt recognition by primary care providers and surgical management by orthopedic surgery. These injuries are defined by a fracture of the distal third of the radial shaft in conjunction with a dislocation or subluxation of the distal radioulnar joint (DRUJ). This fracture-dislocation injury has earned the phrase 'the fracture of necessity' due to the necessity for operative fixation in nearly all cases to restore normal forearm biomechanics and prevent long-term functional deficits. The mechanism typically results from an axial force through the forearm, often with the wrist in pronation. The consequence is an anterior displacement of the fractured radius accompanied by a posterior dislocation of the ulna, hallmarks of a type II injury, as demonstrated in this case.

From a family medicine and primary care sports medicine perspective, Galeazzi fractures pose a diagnostic challenge, particularly in the acute setting where swelling and pain may obscure deformity, and attention may be focused on the more

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obvious radial shaft fracture. However, failure to identify the DRUJ instability can lead to significant long-term morbidity including chronic pain, instability, limited range of motion and impaired grip strength. Providers in frontline settings must maintain a high index of suspicion when evaluating any distal radial fracture, especially those resulting from high-energy mechanisms such as a fall from a motorized bicycle, as seen in this case. Additionally, a careful clinical exam that includes assessment of DRUJ stability and neurovascular status, followed by appropriate forearm imaging, is essential. A demonstration of the clinical examination used to assess DRUJ stability is presented in this video:[7] https://www.youtube.com/watch?v=nz53aeq-30k.

This case highlights the vital role of primary care physicians, particularly those working in inpatient, urgent care, or primary care sports medicine settings, in initiating early and appropriate management. While the definitive treatment of Galeazzi fractures falls within the domain of orthopedic surgery, initial stabilization, diagnosis, and timely referral fall squarely within the scope of primary care. Understanding the importance of forearm alignment, maintaining a low threshold for imaging when the mechanism is concerning, and recognizing subtle signs of joint disruption are key components of comprehensive musculoskeletal care.

More broadly, this case report reinforces a foundational principle in fracture management, recognizing that not all fractures are isolated bone injuries. Many involve adjacent joints or soft tissue structures that can complicate recovery. Just as a distal radius fracture may signal underlying DRUJ injury, other fracture patterns throughout the body require attention to joint alignment, ligamentous stability, and neurovascular integrity. For primary care physicians, integrating mechanism of injury, physical exam findings, and radiographic interpretation is crucial to delivering effective, timely care and ensuring optimal outcomes for patients.

Endnotes

1. JR Fowler and AM Ilyas, "Distal Radius Fractures: A Clinical Overview," *American Family Physician* 103, no. 6 (2021): 345–53, https://www.aafp.org/pubs/afp/issues/2021/0315/p345.html.
2. Orthobullets, "Galeazzi Fractures," accessed June 2025, https://www.orthobullets.com/trauma/1029/galeazzi-fractures.
3. American Medical Society for Sports Medicine, *Pediatric Primary Care Sports Medicine Handbook* (May 2022), https://sportsmedref.amssm.org/wp-content/uploads/2022/05/Pediatric-Primary-Care-Sports-Medicine-Handbook.pdf.
4. Core EM, "Forearm Fractures," July 28, 2015, https://coreem.net/core/forearm-fractures/.
5. AC Rettig and JR Raskind, "Galeazzi Fracture-Dislocation: A New Clinical-Significance Classification," *American Journal of Orthopedics* 30, no. 5 (2001): 401–6, PMID: 11326352.
6. HPJ Walsh, DM Eastwood, and CM Booth, "Galeazzi Fractures in Children," *Journal of Bone and Joint Surgery*. British Volume 78, no. 5 (1996): 791–93, PMID: 13416321.
7. "Distal Radioulnar Joint Test / DRUJ Test." 2019. *YouTube video*, June 5. https://www.youtube.com/watch?v=nz53aeq-30k.

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Chief Concern of Weight Gain Uncovers Fast Growing ACTH-Secreting Pituitary Macroadenoma

By Myranda Steingraeber, MD and Nora Callinan, MD

Abstract:

Weight gain is a common concern among patients in a primary care setting. Here we discuss a 26-year-old female who presented with the chief concern of abnormal weight gain. After weight monitoring and a detailed review of diet and exercise, she had an abnormal random cortisol test and an abdominal CT which was negative for adrenalomas. Subsequent tests revealed abnormal fasting AM cortisol and abnormal dexamethasone suppression. In rapid succession over the next several weeks, she developed galactorrhea, amenorrhea, and visual changes. An MRI was ordered, confirming a pituitary adenoma abutting the optic chiasm. Ultimately, the mass (1.7cm) was resected.

Introduction:

The overall prevalence of pituitary adenomas is estimated to be 16% in the general population.[1,6] While prolactinomas and nonfunctional pituitary adenomas are the most common types, ACTH-secreting pituitary adenomas comprise only 1-2%, and microadenomas are far more common than macroadenomas.[4] Pituitary macroadenoma are defined as pituitary adenomas with a diameter of ≥ 10mm. They make up roughly 48% of clinically evident pituitary adenomas, and occur in 1 in 1100 individuals in the general population based on database studies.[2,7] The majority of incidentally discovered pituitary adenomas are microadenomas. Macroadenomas are more likely to be symptomatic due to their size, leading to mass effect including visual field defects, headaches, and hypopituitarism. They are more commonly detected in men and at older ages when compared to microadenomas. Although the clinical features and management of pituitary adenomas are well documented, cases involving predominantly ACTH-mediated effects are less common, and presenting symptoms are more vague.

We describe a case of pituitary macroadenoma uncovered from extensive workup in a young woman presenting to her family physician, whose primary concern was weight gain. She had numerous prior visits for headaches. The authors believe this case to be significant in highlighting the importance of expanding differentials in the setting of persistent bothersome symptoms that are not responding as expected. **In this case the patient's persistence, self-advocacy, and diligent adherence to tracking lifestyle interventions lead to an investigation of less common etiologies. Time to diagnosis was impacted by interruptions in continuity of care, logistical challenges obtaining certain labs, and availability bias. The progression from nonspecific symptoms to a diagnosis of functional pituitary disease highlights the importance of continuity of care, detailed symptom tracking, and a high index of suspicion in patients with evolving endocrine features.**

Case Report:

A 26-year-old woman without significant past medical history presented to her PCP in November 2022 to discuss headaches,

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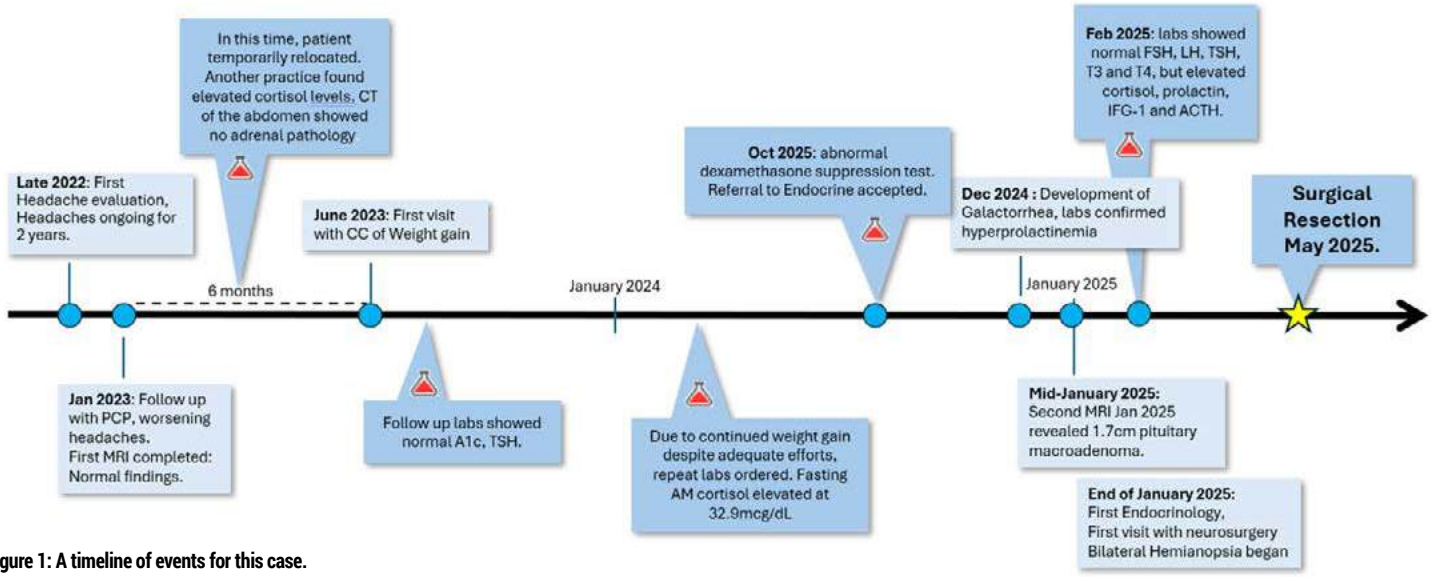

Figure 1: A timeline of events for this case.

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which had been going on for 2 years at that time. There were no red flag headache symptoms, and characteristics aligned with a clinical diagnosis of migraines. She was started on daily magnesium and riboflavin for migraine prophylaxis.[3,5,8] She followed up in January 2023 reporting worsening headache severity and frequency despite adherence to treatment. Given worsening symptoms, a brain MRI without contrast was ordered and completed within 2 weeks revealing no abnormalities. She was started on amitriptyline for migraine prophylaxis. She then moved out of state. While out of state, she was seen at another primary care office for weight gain and was found to have an abnormally elevated fasting morning cortisol level. A subsequent CT of the abdomen ruled out adrenal masses. She also saw ophthalmology for blurry vision and was diagnosed with primary open angle glaucoma.

In June 2023, six months after her MRI, she returned to our office with chief concern of weight gain. She was seen for a few follow up visits over the next several weeks and a detailed dietary and exercise history showed that she was maintaining caloric deficit and was still gaining weight. At this point she had lab work done showing normal TSH, low free T4 and normal hemoglobin A1c. Her vitals and exam were unremarkable. A repeat fasting morning cortisol level was ordered and was elevated at 32.9mcg/dL. An endocrinology referral was placed at that time. Due to limited resources in the area, they requested dexamethasone suppression testing be done before accepting the referral. Fortunately, detailed lab testing and interpretation was assisted by e-consult insights from these endocrinologists. This revealed a normal (not suppressed) fasting AM cortisol level of 19 mcg/DL and sufficient levels of serum dexamethasone (327 ng/dL). The endocrinology referral was accepted.

In December 2024, the patient followed up with her PCP, with a new symptom of galactorrhea. Exam remained unchanged, without abnormal fat distribution and without striae, and no breast abnormalities. Labs in December showed normal TSH and free T4 0.83 uIU/mL / 0.80 ng/dL, negative Beta-HCG, normal testosterone serum levels 66 ng/DL, normal FSH and LH 7.5 / 6.8 mIU/ml, and elevated serum prolactin level 99.7 ng/DL.

The abnormal serum prolactin level prompted a repeat MRI which was completed in January 2025 and revealed a 1.7 cm pituitary adenoma which was notably absent from the MRI done just 2 years earlier *(see Figure 2)*. This news was urgently relayed to the specialists, and she was seen by endocrinology and neurosurgery by the end of that month. Endocrinology tested salivary cortisol levels x 3 which were normal and 24 hour urinary free cortisol which was elevated at 382 mcg/24h, repeated serum prolactin which remained elevated at 109 ng/mL, serum FSH, LH,

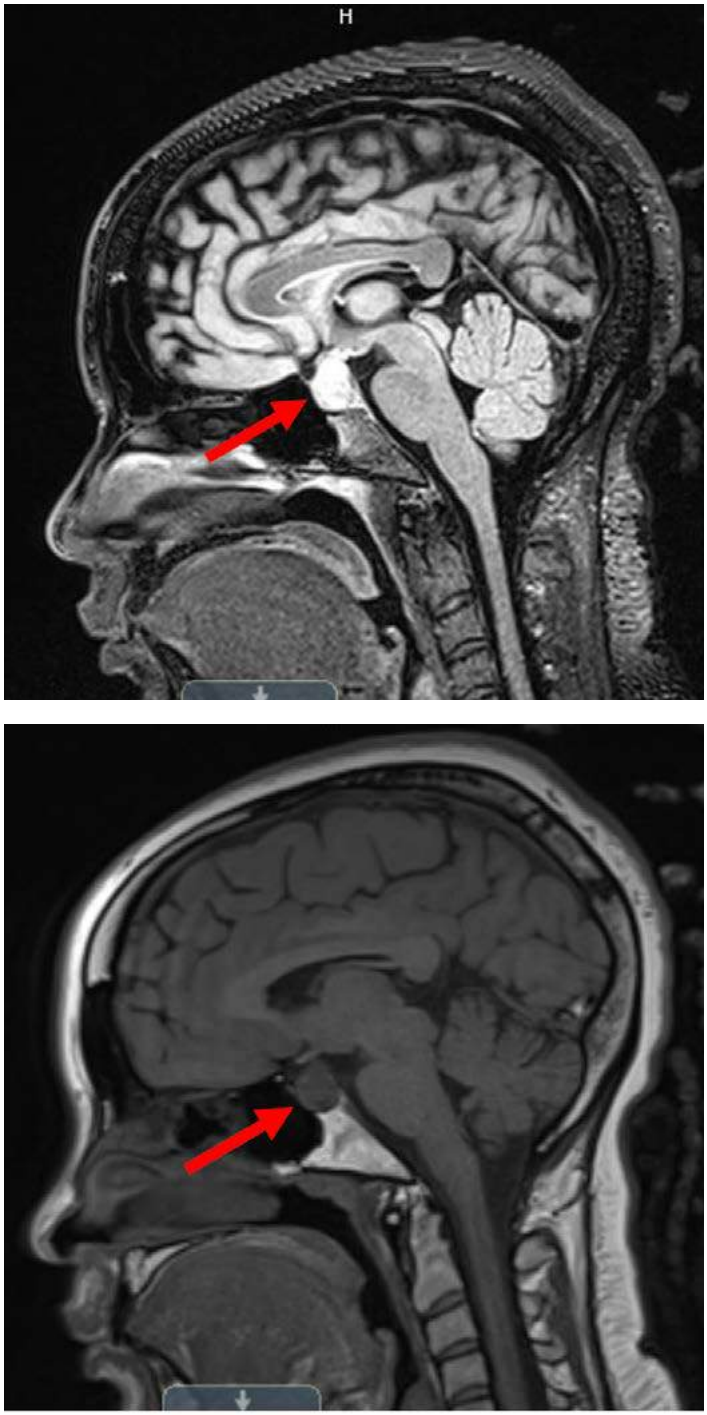

Figure 2: A sagittal view of this patient's second MRI with (upper image) and without (lower image) contrast, where in the 1.7cm pituitary adenoma can be seen (red arrow).

and TSH as well as free T3 and T4 which were all normal. They found that both IGF-1 (340 ng/ml) and ACTH (93 pg/ml) were elevated. In February at a PCP follow up visit, she reported new amenorrhea, and by April, when she saw neurosurgery, she had developed bilateral hemianopsia, new and different from the blurriness of her glaucoma.

Initially medical management was recommended by the multidisciplinary team, however the rapid development of multiple new symptoms and the presence of visual changes prompted plans

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for surgical resection. Neurosurgery referred her to otolaryngology as they planned for a transsphenoidal approach. **Despite the complexity inherent in multidisciplinary management, this patient provided frequent updates via portal messages and the authors' ability to access multiple EMRs allowed for easier coordination of care.** Her pituitary adenoma was resected successfully in May 2025, and pathology was consistent with pituitary adenoma. Her postsurgical course included intense headaches which resolved with time, and gradual return of vision and menstruation. She continues to follow with endocrinology because her cortisol levels remained elevated postoperatively, suggesting Cushing's disease. *(See Figure 1 for timeline)*

Discussion:

Weight gain is a common chief concern, with a growing number of patients seeking evaluation and treatment. It is frequently and often appropriately attributed to imbalances between caloric intake and energy expenditure, which remains the most common etiology, however physicians need to remain diligent in their investigation in case more rare pathologies are present. While history taking, particularly in diet and physical activity is emphasized in medical education—often most proficiently executed by medical students—these components are sometimes deprioritized in primary care settings due to time constraints. As a result, detailed assessments are often deferred to specialists. One practical alternative is to engage patients in self-monitoring using digital tools or structured handouts. Various mobile applications allow users to track dietary intake and physical activity, while publicly available resources, such as the CDC's food and exercise diaries, provide a simple and effective paper-based method. However, the utility of these tools varies significantly and is highly dependent on patient engagement and adherence.

This case is also notable for its predominantly ACTH-driven symptomatology significant growth of the adenoma over a relatively short interval. While pituitary adenomas, Cushing's disease, and their associated pathophysiology are well covered in medical education, clinical emphasis is often placed on hallmark features such as amenorrhea, galactorrhea, and visual disturbances due to optic nerve compression—symptoms which appeared later in this case. Even hallmark signs of Cushing syndrome were not present on physical exam due to this patient's extreme caloric restriction and high activity level (2-3 hours at the gym 5-6 days per week, often working with a personal trainer). This somewhat unusual presentation prompted repeat laboratory testing and close follow up. This case underscores the importance of thorough history taking and trusting the patient's account. The point is to not test everyone for this, but to identify when to test. **In this case, the lack of improvement over time despite adherence to interventions prompted further testing.**

Other challenges in this particular case were logistical issues obtaining certain labs in the patient's daily life. For example, it was difficult to properly time fasting morning labs with her morning routine and lab hours. The availability and accessibility to specialists also posed challenges and concerns about delays in care. Larger systemic factors include the inconsistent availability of health insurance and access to healthcare including specialists for our patients: any delay caused by a lapse of insurance or by disagreements between insurances and the providing networks in the area could have caused this patient irreversible damage to her optic nerve or allowed a rapidly growing tumor to increase in size exponentially. The patient in this case fortunately maintained insurance and should be applauded for her persistence in seeking care when the lifestyle changes were insufficient, and in her self-advocacy and record keeping while on this journey.

Endnotes:

1. Ezzat S, Asa SL, Couldwell WT, et al. The prevalence of pituitary adenomas. *Cancer*. 2004;101(3):613-619. doi:https://doi.org/10.1002/cncr.20412
2. Gruppetta M, Vassallo J. Epidemiology and radiological geometric assessment of pituitary macroadenomas: population-based study. *Clinical Endocrinology*. 2016;85(2):223-231. doi:https://doi.org/10.1111/cen.13064
3. Ha, H, and Gonzalez, A. Migraine Headache Prophylaxis. *American Family Physician*. 2019; 99(1): 17-24. https://www.aafp.org/pubs/afp/issues/2019/0101/p17.html#afp20190101p017-b29
4. Lake MG, Krook LS, Cruz SV. Pituitary Adenomas: An Overview. *American Family Physician*. 2013;88(5):319-327. https://www.aafp.org/pubs/afp/issues/2013/0901/p319.html
5. Rajapakse T, Pringsheim T. Nutraceuticals in migraine: a summary of existing guidelines for use. *Headache*. 2016;56(4):808-816.
6. Russ S, Shafiq I. Pituitary Adenoma. PubMed. Published March 27, 2023. https://www.ncbi.nlm.nih.gov/books/NBK554451/
7. Tritos NA, Miller KK. Diagnosis and Management of Pituitary Adenomas. *JAMA*. 2023;329(16):1386. doi:https://doi.org/10.1001/jama.2023.5444
8. von Luckner A, Riederer F. Magnesium in migraine prophylaxis—is there an evidence-based rationale? A systematic review. *Headache*. 2018;58(2):199-209.

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A Rare Presentation of Type 2 Acromioclavicular Joint Cyst in a 70-Year-Old Female

By Lorena Abou Asaff, MD; Moiz Ahmed Zahid, MD and Haidy G. Rivero, MD

Abstract

Acromioclavicular (AC) joint cysts are rare in elderly patients and often result from persistent shoulder pathology. They are classified as type 1, which is confined to the AC joint with an intact rotator cuff, and type 2, which arises from a full-thickness rotator cuff tear. A 70-year-old female presented with 3 months of right shoulder pain and a mass-like sensation on the anterior aspect of the shoulder. On physical examination, a 4x5 cm cystic mass was noted over the right AC joint, with negative shoulder tests. MRI revealed a high-riding humeral head, decreased subacromial space, and a 5.5 cm septated ganglion cyst arising from the AC joint, along with rotator cuff tears and the "geyser sign." MRI can aid in determining AC joint cyst types.

Introduction

Rotator cuff tears and acromioclavicular (AC) joint degeneration are common occurrences in clinical practice. AC joint cysts, however, are a rare presentation in elderly patients (average age of 68.5), with an increased incidence in males, resulting from persistent shoulder pathophysiology. AC joint cysts are uncommon, with fewer than 50 cases reported in the literature. They were first described in 1986 and later categorized into two groups by Hiller et al.[1,2] Type 1 cysts occur in the setting of an intact rotator cuff and are limited to the AC joint, with increased synovial fluid production leading to cyst formation, usually caused by degenerative changes within the AC joint, such as osteoarthritis. Type 2 cysts, on the other hand, occur from full-thickness chronic rotator cuff tears and cuff tear arthropathy, resulting in upward migration of the humeral head. Specifically, the loss of supraspinatus muscle function causes upward migration of the humeral head due to the unopposed action of the deltoid muscle,[2,4,5] causing, over time, irritation and degeneration of the inferior capsule of the AC joint. This leads to the formation of a communication between the glenohumeral (GH) joint and the AC joint, and the migration of synovial fluid into the AC joint, resulting in cyst formation. The degenerative changes in the AC joint then act as a one-way valve, holding the synovial fluid in the cyst.[2,4,6] Based on the literature, type 2 cysts are more common than type 1 cysts.

Case Report

A 70-year-old female presented to the clinic with a 3 month progression of right shoulder pain and a mass-like lesion over the anterosuperior aspect of the shoulder. She reported associated pain and a burning sensation in the area. The pain was described as constant, moderate in severity, non-radiating, worsened with repetitive joint movement, and alleviated with rest. The patient

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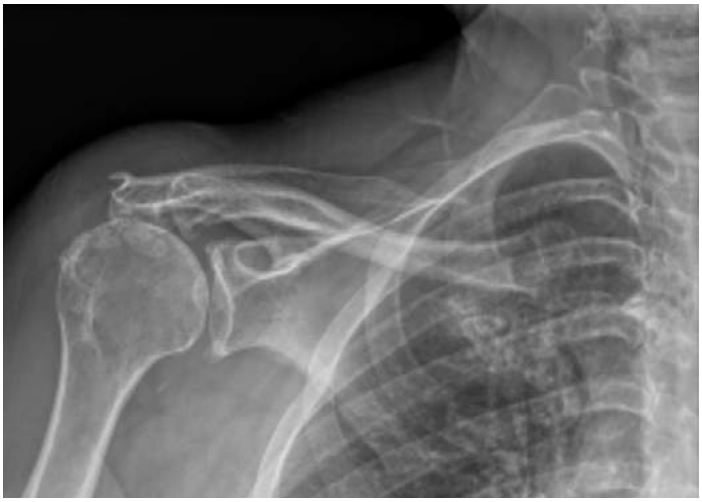

Figure 1: X-ray of the right shoulder showing superior migration of the humeral head and AC joint arthropathy.

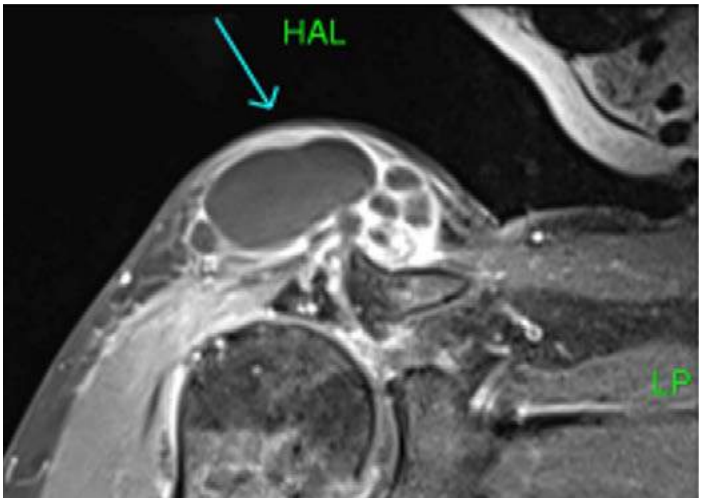

Figure 2: MRI with contrast showing a 5.5 × 5.5 cm septated ganglion cyst extending from AC joint with geyser sign.

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denied using pain medications (topical or oral agents). The patient reported that symptoms gradually became bothersome with time, interfering with her right shoulder function during everyday activities. The patient denied weakness, numbness, or tingling in the right arm, noted no difference in strength compared to the left arm, and denied any constitutional symptoms or nocturnal pain. She also denied trauma or recent surgeries to the affected area. Physical examination was notable for a 4x5 cm cystic mass over the right AC joint, slightly tender to palpation, with minimal mobility. The mass transilluminated and was non-pulsatile. Right shoulder range of motion and strength were normal, with no signs of rotator cuff weakness or tear, and no shoulder pain with provocative tests, including the painful arc, drop-arm, empty can, and Hawkins tests. Imaging of the right shoulder was performed. X-ray revealed moderate glenohumeral arthropathy, a high-riding humeral head, and decreased subacromial space findings compatible with a rotator cuff tear (Figure 1). MRI with and without contrast revealed a 5.5 cm x 5.5 cm septated ganglion cyst in the subcutaneous tissues of the anterosuperior aspect of the shoulder, arising from the AC joint. Osteoarthritis was present at the AC joint, along with chronic full-thickness supraspinatus and infraspinatus tendon tears and cephalad migration of the humeral head. These findings reflect the "geyser sign," resulting from fluid extending from the glenohumeral joint into the subacromial-subdeltoid bursa through the rotator cuff tear, and subsequently through a defect in the acromioclavicular joint capsule into the superficial subcutaneous tissues (Figure 2). At a follow-up visit to review MRI results, the patient reported that her shoulder pain had resolved; however, the size of the cystic mass remained unchanged. She was referred to orthopedic surgery for consideration of cyst excision along with rotator cuff repair.

Discussion

The differential diagnosis of AC joint cysts should include hematoma, synovial cyst, ganglion, lipoma, or tumor, as reported in the literature.[7] In this patient, the differential diagnosis included soft tissue and bone tumors, considering the patient's age and the rapidly growing mass. MRI confirmed superior migration of the humeral head due to chronic full-thickness tears of the supraspinatus and infraspinatus. Chronic AC joint osteoarthritis was also present, allowing communication between the glenohumeral and AC joints, as evidenced by the geyser sign on MRI, guiding the diagnosis toward a type 2 cyst in the AC joint. Management of AC joint cysts varies. Type 1 cysts can be managed with cyst excision and/or distal clavicle resection.[4,7] This patient presented with a type 2 AC cyst, which is managed by addressing the underlying pathology. Aspirating or excising a type 2 cyst carries a high risk of recurrence due to the persistent communication between the GH joint and the AC joint.[1,2,3,4,6] In our patient, given the unchanged cyst and underlying chronic rotator cuff tear, the decision was made to refer the patient to orthopedic surgery to avoid recurrence of the cyst. However, on a follow-up visit, the patient reported minimal symptomatology and good functional status, declining surgical approach.

Conclusion

Evaluation of rotator cuff integrity is essential in patients presenting with an AC joint cyst. MRI plays a key role in identifying the geyser sign, which helps differentiate between cyst types (type 1 and 2) and therefore, their respective management. Type 2 cysts, which are associated with rotator cuff tears, require surgical repair of the underlying defect to prevent recurrence.

Endnotes

1. H. J. Cooper et al., "The MRI Geyser Sign: Acromioclavicular Joint Cysts in the Setting of a Chronic Rotator Cuff Tear," American Journal of Orthopedics 40, no. 6 (2011): E118–E121.
2. A. D. Hiller et al., "Acromioclavicular Joint Cyst Formation," Clinical Anatomy 23, no. 2 (2010): 145–152.
3. H. Mullett et al., "Arthroscopic Treatment of a Massive AC Joint Cyst," Arthroscopy 23, no. 4 (2007): 446.e1–446.e4.
4. E. V. Craig, "The Geyser Sign and Torn Rotator Cuff," Clinical Orthopaedics and Related Research, no. 191 (1984): 213–215.
5. A. A. Sayed et al., "Geyser Sign on MRI in a 65-Year-Old Female," Cureus 14, no. 4 (2022): e23751.
6. O. Cvitanic et al., "AC Joint Cyst: GH Communication Revealed by MR Arthrography," Journal of Computer Assisted Tomography 23, no. 1 (1999): 141–143.
7. B. Ockert et al., "AC Joint Cyst: Case Report and Literature Summary," Der Orthopäde 38, no. 10 (2009): 974–979.

References

N. Maziak et al., "AC Joint Cyst in a Patient with Rotator Cuff-Tear Arthropathy: A Rare Cause of Discomfort," BMJ Case Reports (2018): bcr2018226188.

F. Postacchini et al., "AC Joint Cyst Associated with Rotator Cuff Tear," Clinical Orthopaedics and Related Research, no. 294 (1993): 111–113.

R. A. Singh et al., "Management of a Massive AC Joint Cyst: Geyser Sign Revisited," Shoulder & Elbow 5, no. 1 (2013): 62–64

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A Rare Case of Spontaneous Heterotopic Pregnancy Presenting with Hemoperitoneum

By Robin Peterson, DO; Hoang Nhu (Natalie) Hua, MD, FAAFP and Jonathan Eli-Phillips, MD, FACOG

Abstract

This case describes a heterotopic pregnancy in a 33-year-old G3P2002 presenting at 10 weeks gestation with abdominal pain, dizziness, and shortness of breath. Imaging identified a viable intrauterine pregnancy and a right adnexal mass concerning for ectopic gestation versus hemorrhagic cyst. Laparoscopy revealed a ruptured tubal ectopic pregnancy with hemoperitoneum, requiring salpingectomy and ovarian cystectomy. Postoperative recovery was uncomplicated with preservation of the intrauterine pregnancy. Heterotopic pregnancy occurs in approximately 1 in 30,000 spontaneous pregnancies.[1] This case highlights the importance of maintaining suspicion in low-risk populations to reduce morbidity.

Introduction

Heterotopic pregnancy, defined as the coexistence of intrauterine and extrauterine gestations, is rare and potentially lifethreatening condition. The spontaneous incidence is estimated at approximately *1 in 30,000 pregnancies*,[1] increasing to *1 in 100* in the context of assisted reproductive technology (ART).[2] Risk factors mirror those for ectopic pregnancy and include pelvic inflammatory disease (PID), prior ectopic pregnancy, tubal or abdominopelvic surgery, and use of fertility treatments.[3] However, up to 33% of cases occur in women without identifiable risk factors.[3] This case represents an uncommon presentation of heterotopic pregnancy following spontaneous conception, occurring in the setting of a single, lower-risk factor and without a history of ART or PID.

Case Report

A 33-year-old gravida 3 para 2 woman at 10 weeks and 3 days of gestation by first-trimester ultrasound presented to the emergency department with a one-day history of dizziness, diarrhea, shortness of breath, and progressive abdominal pain. She described the pain as spasm-like, initially localized to the right upper quadrant (RUQ) and radiating to her neck, later becoming more diffuse. She also reported bladder discomfort with urination but denied fever, chills, vomiting, vaginal bleeding, or prior complications in her previous pregnancies. Her past surgical history was significant for two cesarean deliveries and prior ovarian cyst removal. She was not receiving fertility treatments.

On physical examination, she appeared uncomfortable but was hemodynamically stable. Her abdomen was diffusely tender with greatest intensity in the right upper and lower quadrants. There was mild voluntary guarding but no rebound tenderness. Bowel sounds were present.

Initial laboratory testing demonstrated hemoglobin 11.9 g/dL, hematocrit 33.2%, platelet count 148 × 10^3/µL, and white blood cell count 10.16 × 10^3/µL. Electrolytes were within normal limits.

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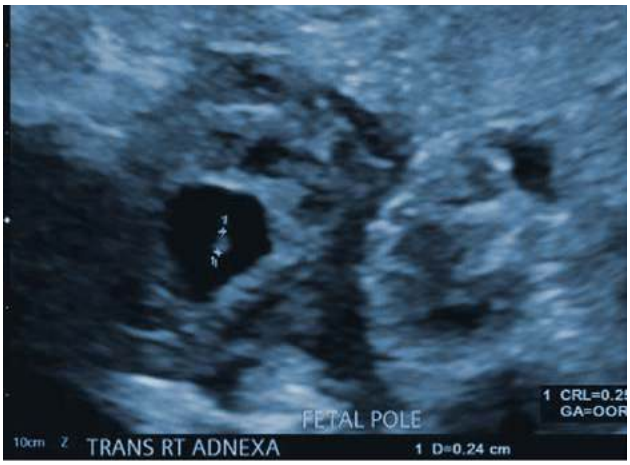

Figure 1: Transabdominal ultrasound of right adnexa showing fetal pole within the fallopian tube.

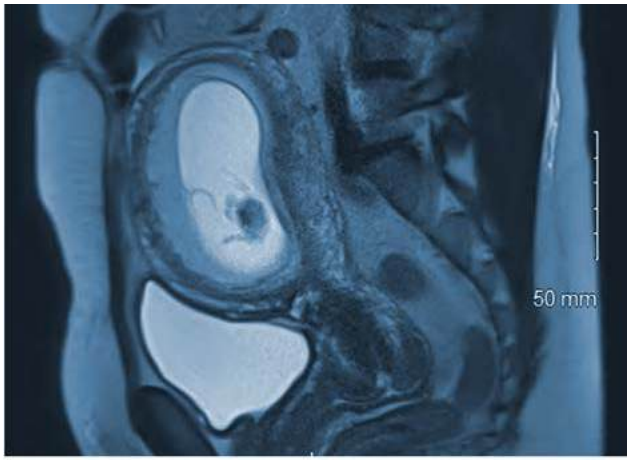

Figure 2: Sagittal view of T2 weighted MRI without contrast showing IUP at approximately 10 weeks gestation.

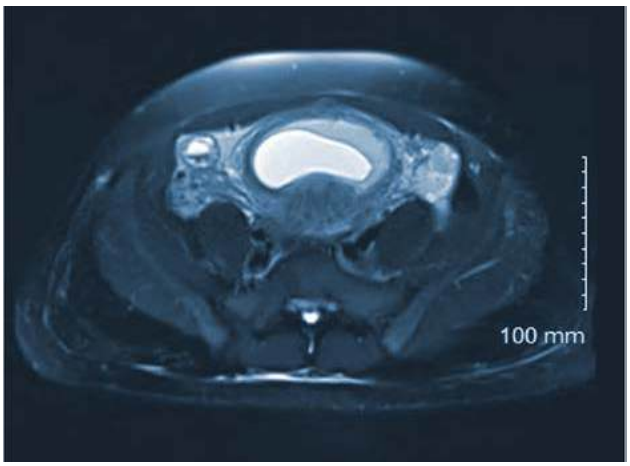

Figure 3: Axial view of T2 weighted MRI without contrast showing an IUP and right sided 17 mm complex paraovarian mass suspicious for ectopic gestation

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Imaging studies began with a transabdominal obstetric ultrasound, which revealed a viable intrauterine pregnancy (IUP) at 10 weeks and 3 days with fetal heart rate of 171 bpm. Adjacent to the superior margin of the right ovary, a second gestational sac was identified with a yolk sac and small fetal pole (figure 1), without detectable cardiac activity. Moderate free fluid was visualized in the pelvis. A right upper quadrant gallbladder ultrasound demonstrated free fluid in the upper abdomen without cholelithiasis, biliary dilation, or renal pathology.

Given these findings, the differential diagnosis included heterotopic pregnancy versus ruptured hemorrhagic ovarian cyst. The patient was initially scheduled for diagnostic laparoscopy. However, once in the operating room, she was unable to tolerate lying flat due to significant right-sided abdominal pain radiating to the shoulder and neck. General surgery was consulted for possible appendicitis, and the case was temporarily deferred pending further evaluation.

MRI of the abdomen and pelvis was obtained to avoid radiation exposure. Imaging confirmed a normal appendix, early viable intrauterine pregnancy (figure 2), moderate hemoperitoneum extending to the subhepatic space, and a 17 mm complex right paraovarian cyst with high T1 signal consistent with hemorrhagic content (figure 3). The leading considerations remained ruptured ectopic pregnancy versus hemorrhagic cyst.

Repeat laboratory testing showed a drop in hemoglobin to 10.2 g/dL and platelets to $123 \times 10^3/\mu L$. Several hours later, hemoglobin decreased further to 9.8 g/dL and platelets to $96 \times 10^3/\mu L$, consistent with ongoing intra-abdominal bleeding. The patient remained hemodynamically stable but continued to experience significant abdominal pain.

The patient was taken to the operating room for diagnostic laparoscopy. Intraoperatively, 700 mL of hemoperitoneum consisting of fresh blood and clot was evacuated. An 8 cm complex right paraovarian mass involving the right fallopian tube was visualized with active bleeding from the fallopian tube. The uterus was enlarged, consistent with early pregnancy, and the left fallopian tube and both ovaries (except for the right-sided mass) appeared grossly normal. The appendix was visualized and normal in appearance.

A laparoscopic right salpingectomy and right ovarian cystectomy were performed. The fallopian tube containing the mass was grasped with atraumatic forceps, and the necrotic, friable tissue was dissected using a LigaSure device. The cyst wall was carefully separated from ovarian tissue, with cauterization of the base to achieve hemostasis. The specimen was removed in an endoscopic retrieval bag through an enlarged right lower quadrant port site. The peritoneal cavity was thoroughly irrigated with warm saline until clear effluent was observed, and meticulous hemostasis was confirmed before desufflation. Port sites were closed in layers, and the patient was extubated and transferred to recovery in stable condition.

Pathologic examination confirmed a ruptured ectopic tubal gestation with cystic gestational sac and organizing blood clot/fibrinous material. No intact ovarian tissue was present in the specimen.

Postoperatively, the patient remained stable. Fetal heart tones were obtained on postoperative days 0 and 1, both in the 150s. She tolerated oral intake, ambulated without difficulty, and was discharged home in stable condition on postoperative day 1 with instructions for outpatient obstetric follow-up. The intrauterine pregnancy continued without complication at the time of discharge.

Discussion

This case illustrates several key diagnostic and management considerations for heterotopic pregnancy in a spontaneous conception with a lower-risk, non-tubal risk factor. Although 71% of patients with heterotopic pregnancy have at least one ectopic pregnancy risk factor,[3] one-third of patients present without any known risk factors.[3] This underscores the importance of maintaining heterotopic pregnancy in the differential diagnosis of early pregnancy abdominal pain, regardless of risk profile.

Diagnostic Considerations

The detection of an IUP should not exclude evaluation of the adnexa. In this case, pelvic ultrasound demonstrated both an IUP and an adnexal mass, prompting further investigation. TVUS is the preferred modality for early diagnosis, offering superior resolution over transabdominal imaging. In postIVF populations, TVUS demonstrates sensitivity of 92.4% and specificity of 100%,[4] though realworld sensitivity may be lower in spontaneous conceptions due to less frequent highresolution early scans. Historically, more than 70% of diagnoses were made intraoperatively, but improvements in ultrasound technology have increased preoperative detection rates.[2,6] Nevertheless, false reassurance from the IUP can lead to missed or delayed diagnosis, as occurred in up to 33% of cases in one review.[3]

Surgical Management

Ruptured tubal heterotopic pregnancies require prompt surgical intervention to prevent hemodynamic compromise and preserve the IUP when possible. Laparoscopy is the preferred approach in hemodynamically stable patients, offering shorter recovery times and reduced postoperative pain compared with laparotomy.[2] Laparotomy remains indicated for unstable patients or in cases of massive hemoperitoneum. In large case series, salpingectomy was the most common intervention, performed in 47.5% of surgical cases,[5] with salpingostomy reserved for unruptured cases or when fertility preservation is paramount.

Concerns regarding the safety of laparoscopy during pregnancy have been addressed by guidelines recommending maintenance of intraabdominal CO_2 pressure at or below 10–15 mmHg,[6] use of open (Hasson) entry when possible, and careful patient positioning to

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avoid aortocaval compression.[7] Available evidence suggests that necessary nonobstetric surgery under general anesthesia is safe in pregnancy when these precautions are observed.[7]

Implications for Clinical Practice

Family medicine and emergency clinicians are the first point of contact for many pregnant patients. This case reinforces the need for complete adnexal evaluation even when an IUP is visualized, particularly in symptomatic patients. Early recognition and timely surgical management can decrease maternal morbidity and mortality.

Endnotes

1. Reece, E.Albert, Roy H. Petrie, Meredith F. Sirmans, Mieczyslaw Finster, and W.Duane Todd. "Combined Intrauterine and Extrauterine Gestations: A Review." *American Journal of Obstetrics and Gynecology* 146, no. 3 (June 1983): 323–30. https://doi.org/10.1016/0002-9378(83)90755-x.
2. Maleki, Aryan, Noorulain Khalid, Chandni Rajesh Patel, and Essam El-Mahdi. "The Rising Incidence of Heterotopic Pregnancy: Current Perspectives and Associations with in-Vitro Fertilization." *European Journal of Obstetrics & Gynecology and Reproductive Biology* 266 (November 2021): 138–44. https://doi.org/10.1016/j.ejogrb.2021.09.031.
3. Talbot, K., R. Simpson, N. Price, and S. R. Jackson. 2011. "Heterotopic Pregnancy." *Journal of Obstetrics and Gynaecology* 31 (1): 7–12. doi:10.3109/01443615.2010.522749.
4. Li, X. H., Y. Ouyang, and G. X. Lu. "Value of Transvaginal Sonography in Diagnosing Heterotopic Pregnancy after *In-vitro* Fertilization with Embryo Transfer." *Ultrasound in Obstetrics & Gynecology* 41, no. 5 (April 22, 2013): 563–69. https://doi.org/10.1002/uog.12341.
5. Barrenetxea, Gorka, Lorea Barinaga-Rementeria, Arantza Lopez de Larruzea, Jon Ander Agirregoikoa, Miren Mandiola, and Koldo Carbonero. "Heterotopic Pregnancy: Two Cases and a Comparative Review." *Fertility and Sterility* 87, no. 2 (February 2007). https://doi.org/10.1016/j.fertnstert.2006.05.085.
6. Guidelines for the use of laparoscopy during pregnancy - a sages publication. Accessed August 5, 2025. https://www.sages.org/publications/guidelines/guidelines-for-diagnosis-treatment-and-use-of-laparoscopy-for-surgical-problems-during-pregnancy/.
7. Reitman, E., and P. Flood. "Anaesthetic Considerations for Non-Obstetric Surgery during Pregnancy." *Survey of Anesthesiology* 56, no. 3 (June 2012): 129–30. https://doi.org/10.1097/01.sa.0000413412.89458.f1.

References

Hassani, KarimIbn Majdoub, Abderrahim El Bouazzaoui, Mohammed Khatouf, and Khalid Mazaz. "Heterotopic Pregnancy: A Diagnosis We Should Suspect More Often." *Journal of Emergencies, Trauma, and Shock* 3, no. 3 (2010): 304. https://doi.org/10.4103/0974-2700.66563.

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Collaborative Care Model for Management of Fetal Trisomy 18: Lethal Fetal Anomalies in Early Pregnancy with Advanced Maternal Age

By Sushama Thandla, MD, MPH, FAAF; Heather Link, MD, MPH and Katherine Wilkie, BMBS

Introduction

Family Physicians are trained to provide routine obstetric care and at times are the first physicians to diagnose an early pregnancy during a primary care visit. Routine prenatal care guidelines from the American Association of Family Physicians categorize pregnancy at advanced maternal age, as high risk for fetal aneuploidy, birth defects, maternal hypertension, gestational diabetes, miscarriage, IUGR, and still birth.[1] Advanced maternal age has been historically defined as women who are 35 years or older at the estimated date of delivery. Current guidelines for management of pregnancy at age 35 years or older were published in August 2022 by the Obstetric Care Consensus, as a joint report by the American College of Obstetrics and Gynecology (ACOG) and the Society for Maternal Fetal Medicine.[2]

Family physicians are likely to diagnose a new early pregnancy at their office in women 35 years or older and need to promptly recognize that pregnancy at advanced maternal age carries an increased risk of adverse perinatal outcomes. Accordingly, family physicians are uniquely positioned to provide early risk identification, timely referral, and evidence-based interventions for these patients with pregnancy at advanced age.[1] We report an obstetric case diagnosed by the patient's family physician at advanced maternal age in the first trimester, and the subsequent management of anencephaly, with fetal trisomy 18 and omphalocele, contributing to the literature on optimizing outcomes through a collaborative care model between family medicine and maternal fetal medicine specialties.

Case Report

A 44-year-old gravida 6 para 4014 with chronic hypertension presented for evaluation at her family physician's office for amenorrhea of about 2½ months. She reported irregular menses after the removal of Nexplanon a year and a half ago and was sexually active with her husband. Her blood pressure was normal and urine HCG testing confirmed pregnancy at

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the office visit. A point of care ultrasound (US) at this visit by the family physician revealed a viable intrauterine pregnancy with fetal heart activity, an overall active fetus and she decided to return for prenatal care. Dating ultrasound (about 4 days later) on the day of her initial OB visit, established a gestational age (GA) of 12 weeks gestation (by crown rump length), which also detected a large omphalocele prompting further work-up. Prenatal genetic screening was briefly addressed at the visit and the patient planned to return to lab with her husband to discuss the consent for cell-free DNA testing.[3]

Urgent consultation with maternal fetal medicine (MFM) was directly scheduled by the family physician for the omphalocele in the following week (GA 13 weeks), meanwhile prenatal genetic screening with a cell-free DNA (cfDNA) test (with a sensitivity of 99% for combined trisomy 21/trisomy 18/ trisomy 13) was drawn at 12 weeks 3 days with informed consent from the patient. At the initial appointment with maternal-fetal medicine specialists, the patient had another obstetric ultrasound at 13 weeks, which in addition to confirming the large omphalocele, also revealed anencephaly, an endocardial cushion defect and absent nasal bones. The MFM consultant reviewed ultrasound findings with the patient, of anencephaly as a lethal anomaly and the additional risks to pregnancy due to advanced maternal age, fetal heart defects and omphalocele. Follow up MFM appointment at 14 weeks 2 days was made to discuss abnormal prenatal cell-free DNA test results that were conclusive for high risk for trisomy 18. At this visit, options for cytogenetic testing and genetic counseling were offered, which the patient declined. The lethality of the ultrasonographic findings were again reviewed.

Over the next two weeks, the patient remained in contact with her family physician by phone multiple times, spoke in-person twice in the office including once with her spouse, as she contemplated her decision for continuation of the pregnancy. Some days, the patient was unable to leave work for appointments. She met with her pastor and called her family who resides out of the country. At every discussion, she was presented with the risks to her fetus and her health with continuation of pregnancy, especially the future non-viability of anencephaly, risk of her developing pre-eclampsia and other late complications. She deferred genetic counseling because she felt it would not help her and that her doctors already gave her a lot of information. At 16½ weeks, she met with Maternal Fetal Medicine specialist for the third time, when an ultrasound was viewed by the patient, noted the persistence of the previously diagnosed findings and MFM specialist again addressed her questions in detail. This discussion focused on both the lethality of the US findings and the permanence of these conditions to the fetus. Patient reported to the family planning department the following day for surgical termination of pregnancy at 16 weeks 5 days. She also opted for placement of an IUD after the procedure.

In summary, the patient received comprehensive obstetric care (beginning in the last week of the first trimester), multidisciplinary care co-ordination for counseling regarding the prognosis, management options, and potential outcomes associated with her pregnancy at age 44 complicated by Trisomy 18, anencephaly, and omphalocele.[4,5] After multiple discussions with maternal fetal specialists, the primary care family physician established rapport with this patient for co-ordination of care, facilitating support from her family as well as her spiritual faith. Her cultural background, job situation and literacy were major challenges during her tough decision-making process. The patient deliberated for almost 2 weeks, deeply disturbed by the prognosis and had difficulty understanding the diagnosis, taking her time to decide about the options. She ultimately decided to go ahead with termination of pregnancy early in the second trimester.

Discussion

This case illustrates the central role of family physicians in the early, guideline-driven management of pregnancies at advanced maternal age and early collaboration with maternal fetal medicine specialists. Obstetric Care Consensus suggests that pregnancy with anticipated delivery at age 35 years and older be recognized as a risk factor for adverse maternal, fetal and neonatal outcomes, which is a grade 2C recommendation.[2] It highlights the need for family physicians to be vigilant and obtain prenatal genetic screening expeditiously. Our case was screened appropriately and identified a lethal neural tube defect associated with trisomy 18.

Despite an early referral to MFM after the first trimester ultrasound findings of a lethal congenital anomaly, this patient's case required time to address barriers of health literacy, employment, and religious beliefs within her community. The social complexities of her situation required additional support from her family physician. Her physician worked with her to identify facilitating aspects like care co-ordination, spousal, family and spiritual support in her personal life. The barriers were also addressed through a thorough explanation of the clinical findings by MFM specialists, who provided the essential additional time and effort. The collaboration between her family physician and MFM specialist provided the patient with many

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opportunities to clarify her questions. The patient needed advocacy to schedule appointments, complete paperwork for time-off from her job, and to use her disability leave. Addressing health equity is a Grade 1B recommendation by the Obstetric Care Consensus for pregnancy at age 35 years or older.[2] In our case, addressing her individual situation, the collaborative care addressed health equity as she eventually overcame literacy barriers, understood the outcomes of continued pregnancy and felt empowered to take her own decision to make an informed choice about her pregnancy. Our patient had challenges in understanding the risks and outcomes of pregnancy continuation which needed specific discussions with her physicians, who counseled her individually and eventually assisted her.

For other practices facing similar challenges, a nurse coordinator at the primary care physician's office or at the specialist's office could help with coordinating office-appointments. A social worker could help with assisting the patient in completing paperwork for time-off from her job, and in knowing that she was protected from losing her job through the FMLA. And physicians could use this additional help for providing patients with the non-clinical information mentioned above.

Additionally, completion of appropriate genetic screening and prompt referral for fetal anomaly with early counseling impacted timing for pregnancy termination at an earlier gestation, resulting in shorter procedure time with lesser risk of procedural complications. In a study of 268 cases, shorter time to genetic diagnosis in early pregnancy by a screening approach (using nuchal translucency, cell free DNA and or first or second trimester serum test) was found to lead to pregnancy termination approximately 2 weeks earlier that those diagnosed without the screening approach.[6]

This case report illustrates a collaborative care model between family medicine and maternal fetal medicine specialties for the first trimester management of a pregnancy at advanced maternal age complicated by trisomy 18 with a lethal neural tube defect which was directly established by the family physician. If available, this case and other complex obstetric cases would benefit from an expanded care team including social workers and nurse care coordinators to assist in managing the non-clinical barriers that arise. This collaborative, multidisciplinary approach provided this patient with a higher level of care which was in accordance with the Obstetric Care Consensus recommendations providing multidisciplinary counseling, nondirective support, and individualized management planning, including the discussion of pregnancy continuation or termination.

Endnotes

1. Ramírez, Sarah Inés. "Prenatal care: an evidence-based approach." American family physician 108, no. 2 (2023): 139-150.
2. Gantt, Angela, Torri D. Metz, Jeffrey A. Kuller, Judette M. Louis, Alison G. Cahill, Mark A. Turrentine, American College of Obstetricians and Gynecologists, and Society for Maternal-Fetal Medicine. "Obstetric Care Consensus# 11, Pregnancy at age 35 years or older." American Journal of Obstetrics and Gynecology 228, no. 3 (2023): B25-B40.
3. Blackwell, Sean, Vincenzo Berghella, Joseph Biggio, Aaron Caughey, Sabrina Craigo, Jodi Dashe, Cynthia Gyamfi-Bannerman et al. "# 36: Prenatal aneuploidy screening using cell-free DNA." American Journal of Obstetrics and Gynecology 212, no. 6 (2015): 711-716.
4. Rieder, Wawrzyniec, Sabine Vasseur Maurer, Eric Giannoni, and David Baud. "Fetal omphalocele: review of predictive factors important for antenatal counseling?." Obstetrical & gynecological survey 77, no. 11 (2022): 683-695.
5. Cook, Rebecca J., Joanna N. Erdman, Martin Hevia, and Bernard M. Dickens. "Prenatal management of anencephaly." International Journal of Gynecology & Obstetrics 102, no. 3 (2008): 304-308.
6. Grossman, Tracy B., and Stephen T. Chasen. "Abortion for fetal genetic abnormalities: type of abnormality and gestational age at diagnosis." American Journal of Perinatology Reports 10, no. 01 (2020): e87-e92.

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Beneath the Rash: Majocchi's Granuloma

By Ala Almansoob, MD; Thomas Rzatkiewicz, DO and Katherine Reeve, MD

Abstract

Topical corticosteroids are often the first treatment for skin conditions, but they are not always effective. We present a 15-year-old wrestler with a persistent, itchy, scaly rash lasting over a year. It was initially diagnosed as tinea corporis or eczema but persisted despite topical antifungals and steroids. A dermatology consult raised suspicion of Majocchi's granuloma, a deep fungal folliculitis usually caused by *Trichophyton rubrum* and aggravated by corticosteroids.[2,5] Chronicity, treatment failure, and steroid use supported the diagnosis. The rash resolved with oral terbinafine. This case underscores the need to reassess diagnoses when standard therapies fail. The discussion highlights similar pediatric cases, disease pathophysiology, superficial versus nodular variants, common misdiagnoses, and the role of biopsy and fungal culture in establishing a definitive diagnosis.[2,3,5]

Introduction

Various forms of tinea are universally common in all genders and ages, especially in athletes who participate in contact sports.[8] Majocchi's granuloma is one of the infrequent types of tinea. It is a follicular dermatophyte infection that invades the deep dermis.[2,3] *Trichophyton rubrum* is the most common causative pathogen of this infection.[2,3,5] It is often exacerbated by inappropriate, unnecessary use of topical corticosteroids.[2,5] It presents as a persistent, scaly, erythematous lesion that is unresponsive to standard topical antifungal therapy.[2,5]

Case Report

This case report presents a 15-year-old male high school wrestler who presented to the resident clinic with approximately a year-long history of recurrent pruritic, scaly rashes involving the posterior surface of right ear, right forearm, and left knee. Initial clinical diagnoses included tinea corporis and eczema. The patient initially presented in May 2024 with a pruritic erythematous patch posterior to the right auricle with a faint ring of erythema surrounding (refer to Figure 1). There was no fluctuance, warmth, or discharge. He was treated with 1-week course of nystatin-triamcinolone with minimal improvement. Three weeks later, he presented with a similar lesion on his right knee for which he was given turbinafine 1% cream twice daily for one week. Again, improvement was limited. In July 2024 the ear and knee lesions persisted and there was also the onset of a new erythematous nodule on the right forearm, which was thought to be an insect bite or other unrelated concern and was observed (refer to Figure 3). In August 2024, eczema was considered as an alternative etiology and he was started on clobetasol cream 0.05% for 2 weeks and later triamcinolone 0.5% cream for 2 weeks. Again, improvement was limited. The rash did not worsen over the course of the year, but did not fully resolve. In December 2024, the right ear rash remained erythematous with areas of clearing. The right forearm rash had progressed from the initial small nodule to a large erythematous, scaly patch on the distal right forearm with areas of clearing (refer to Figure 2). He was treated with miconazole 2% cream and worsened over four days between office visits. Overall his rashes were treated intermittently with topical antifungals and moderate to high potency topical corticosteroids, with minimal or no improvement. The rashes became more extensive and refractory over time. Therefore, the patient was scheduled for an in-office biopsy of the lesion.

Prior to performing the biopsy, dermatology was informally consulted for further recommendations. After their review of patient's history and photos of the rashes, Majocchi's granuloma was suspected in light of the exam findings, chronicity, steroid exposure, and persistent rash. The diagnoses of Majocchi's granuloma is primarily clinical, supported by a detailed history and precise skin examination.[9] Given high clinical suspicion of Majocchi's granuloma, biopsy was deferred, particularly in this pediatric patient, where avoidance of invasive procedures is preferable. The diagnosis was made clinically and a 3-week course of oral terbinafine 250mg daily was initiated after obtaining baseline liver function tests. At follow-up, the patient reported complete resolution of symptoms.

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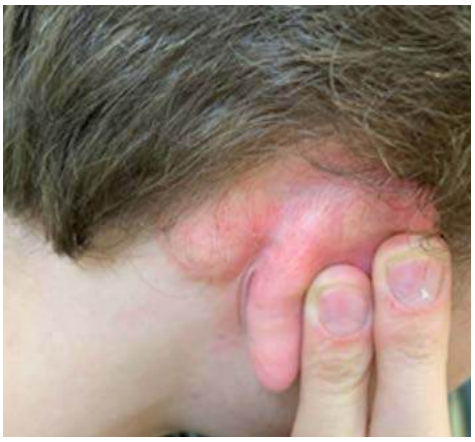

Figure 1 - Right Ear Rash

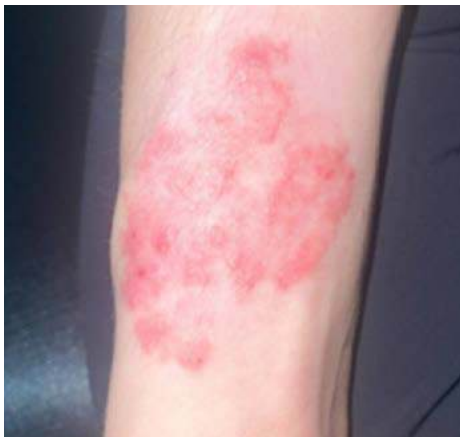

Figure 2 - Right Arm Rash December 2024

Figure 3 - Right Arm Rash July 2024

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Discussion

This case unfolds the diagnostic challenges of Majocchi's granuloma by family physicians, particularly in individuals with concurrent dermatologic conditions.[2,5] Inappropriate use of topical steroids can mask typical features and further exacerbate dermatologic fungal infections.[2,5] Timely recognition and initiation of systemic antifungal therapy are crucial steps for conclusive management and prevention of chronic skin complications.[2,3,5]

Majocchi's granuloma can be a diagnostic challenge due to its myriad presentations and rarity. It is seen more commonly in adults but has been described in children. One case involved a 6-year-old child who presented with a facial erythematous and scaly rash following long-term high potency topical corticosteroid and metronidazole use for presumed tinea. The child was then diagnosed with Majocchi's granuloma and successfully treated with oral terbinafine.[1] Another report presented a 12-year-old girl with facial inflammatory papules misdiagnosed as acne vulgaris. Symptoms improved with topical steroids but recurred upon cessation prompting further work up with a biopsy that confirmed Majocchi's granuloma.[4] A third pediatric example involved a child with facial injury and laceration followed by nodular Majocchi's granuloma. Histopathological examination with GMS stains confirmed the diagnosis and antifungal therapy was effective.[7]

Majocchi's granuloma is most commonly caused by *Trichophyton rubrum*, a dermatophyte that penetrates the epidermis, stratum corneum and into hair follicles and dermis.[2,3,5] Mechanical trauma such as friction, prolonged topical corticosteroid use, or pre-existing dermatophyte infection enables entry of the pathogen.[2,5] Once dermatophytes enter the follicle, they exploit keratin-rich environments for replication triggering a granulomatous inflammatory response.[2,3,5] Majocchi's granuloma manifests in two primary forms: follicular (superficial) and nodular (subcutaneous).[2,3,5] The follicular form is common in immunocompetent hosts, presenting as perifollicular papules, pustules, or scaly erythematous plaques. The nodular type, which is more often seen in immunocompromised individuals, presents with deeper dermal or subcutaneous nodules, sometimes forming abscesses.[2,3,5]

Majocchi's granuloma is a great mimic, leading patients to receive inappropriate treatments which prolong or, in the case of topical steroids, exacerbate symptoms.[2,5] Frequent misdiagnoses include tinea corporis, eczema, acneiform eruptions, bacterial folliculitis, granulomatous disorders, lupus tumidus, or cutaneous lymphoma.[2,5] A wrestling environment can further complicate diagnosis due to similarities with tinea gladiatorum.[10]

Although not required, diagnosis is usually confirmed with a skin biopsy.[2,5] Histologic features include granulomatous inflammation in the dermis and fungal components within hair follicles.[2,3] Specific stains such as periodic acid–Schiff (PAS) and Gomori methenamine silver (GMS) are used.[2,5] Fungal cultures from biopsy tissue or lesion scrapings identify the dermatophyte species, guiding treatment.[2,3]

Majocchi's granuloma is an infrequent but important consideration in chronic, treatment-resistant skin conditions.[2,3,5] Pediatric cases, though rare, share common features of misdiagnosis, steroid exposure, and delayed systemic treatment.[1,4,6,7] Awareness of the different variants, common differential diagnoses, pathophysiologic mechanisms, and the critical role of biopsy and culture can shorten the duration to arrive at diagnoses and initiate treatment.[2,3,5] The prolonged course of symptoms in our case, contrasted with complete resolution achieved using oral terbinafine, highlights the importance of early initiation of systemic therapy as the cornerstone of management.[2,3,5]

Endnotes

1. André NF, Canato M, Zanatta DA, Gomes IF, Abage KT, Carvalho VO. Majocchi granuloma on a child's face. Dermatol Online J. 2018;24(12). doi:10.5070/D32412042448
2. Boral H, Durdu M, Ilkit M. Majocchi's granuloma: current perspectives. Infect Drug Resist. 2018;11:751-760. doi:10.2147/IDR.S164220
3. Zheng YY, Li Y, Chen MY, et al. Majocchi's granuloma on the forearm caused by Trichophyton tonsurans in an immunocompetent patient. Ann Clin Microbiol Antimicrob. 2020;19:39. doi:10.1186/s12941-020-00382-y
4. Anees MA. Case of inflammatory acne or something else? Contemp Pediatr. October 12, 2022. Accessed August 17, 2025. https://www.contemporarypediatrics.com/view/case-of-inflammatory-acne-or-something-else-
5. Gnanasegaram M, Gomez J. Majocchi granuloma. DermNet. Published December 23, 2018. Accessed August 17, 2025. https://dermnetnz.org/topics/majocchi-granuloma
6. Pereira MBTC, Campos GA, Kanaan ICS. Majocchi granuloma in the face of an immunocompetent child: A case report. Sci Arch Int Open Access J. Published July 11, 2024. Accessed August 17, 2025. https://www.scientificarchives.com/article/majocchi-granuloma-in-the-face-of-an-immunocompetent-child-a-case-report
7. Song X, Zhao L, Geng S. A five-year-old girl with erythematous papules on cheek. In: Case Reports in Dermatology. Springer; 2022:19-22. doi:10.1007/978-3-030-89089-6_3
8. Nowicka D, Bagłaj-Oleszczuk M, Maj J. Infectious diseases of the skin in contact sports. Adv Clin Exp Med. 2020;29(12):1491-1495. doi:10.17219/acem/129022
9. Ssuleman. (2025, January 6). *Majocchi granuloma*. OASIS DERMATOLOGY GROUP PLLC. https://oasisderm.com/majocchi-granuloma-2/
10. Zalewski A, Goldust M, Szepietowski JC. Tinea Gladiatorum: Epidemiology, Clinical Aspects, and Management. Zalewski, Adam, et al. "Tinea Gladiatorum: Epidemiology, Clinical Aspects, and Management." *Journal of Clinical Medicine*, vol. 11, no. 14, 14 July 2022, p. 4066, https://doi.org/10.3390/jcm11144066. Accessed 13 Dec. 2022.

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May–Thurner Syndrome in a Patient with Systemic Lupus Erythematosus: A Case of Recurrent Iliofemoral Deep Vein Thrombosis and Phlegmasia

By Nellya Ablayeva, MD; Victoria Lovallo; Ravilya Caine, MD and Anghel Valentin, MD

Abstract

May-Thurner syndrome (MTS) is an underdiagnosed anatomical cause of left-sided iliofemoral deep vein thrombosis (DVT), often requiring endovascular intervention. Autoimmune disorders such as systemic lupus erythematosus (SLE) increase thrombotic risk and complicate management.

We report a 62-year-old female with SLE, rheumatoid arthritis, and pulmonary fibrosis who presented with three weeks of progressive left lower extremity swelling and pain. Initial duplex ultrasound was significant for lower extremity DVT. On physical exam, there were early signs of phlegmasia, prompting CT venogram that revealed extensive DVT from the left iliac to popliteal veins, with compression suggestive of MTS. Despite anticoagulation with heparin and urgent interventional radiology-guided thrombectomy, re-thrombosis occurred, necessitating transfer for vascular stenting. The patient's anemia and refusal of blood transfusion added complexity to anticoagulation management.

This case underscores the importance of early detection of MTS in patients with autoimmune diseases presenting with extensive DVT. Comprehensive, multidisciplinary management is critical to prevent limb loss and optimize patient outcomes.

Introduction

May-Thurner syndrome is a clinical condition caused by thickening and occlusion of the anatomical morphology of the iliac vein brought on by compression of the iliac vein between the underlying bone and overlying arterial system. Many cases are asymptomatic; however, the possibility of severe morbidity exists and has been observed in cases where compression allows for significant focal intimal proliferation of the vein leading to venous stasis and the development of deep venous thrombosis and post-thrombotic sequelae.[1] The most common morphology is compression of the left common iliac vein (LCIV) by the right common iliac artery (RCIA) against the lumbar vertebra. May-Thurner Syndrome should be evaluated in any person presenting with left-sided DVT because radiological studies of patients with a left lower extremity DVT showed May-Thurner Syndrome in up to 76% of cases.[2]

May-Thurner syndrome commonly affects women between the ages of 30 and 50.[3] It initially presents with leg heaviness and swelling in response to activity and is relieved by leg elevation. Chronic venous insufficiency and varicose veins may eventually develop. Alternatively, DVT can occur as the first presentation, beginning with acute pain and swelling in the left lower extremity from the start. A history of hypercoagulable state is typically elicited, including recent pregnancy, oral contraceptive use, or autoimmune disease.

Diagnosis involves using various imaging modalities including Doppler ultrasound, magnetic resonance venography, and CT venography. Treatment of severe symptomatic MTS depends highly on the presentation and may include multimodal treatment strategies including anticoagulation, thrombolysis, and consideration of endovascular surgical intervention.[3]

In patients with co-existing hypercoagulable states, May-Thurner Syndrome may contribute to the development of thrombosis, as outlined in pregnant women as an example of a hypercoagulable state.[4,6] Autoimmune diseases such as SLE provide another pathway to establishing a hypercoagulable state;[5] however, May-Thurner Syndrome in the context of SLE has yet to be deeply explored.

We present a case of a patient with previously diagnosed SLE who presented with extensive DVT with MTS which evolved into the severe DVT complication, phlegmasia. This case serves as a warning against the potentially severe outcomes of MTS with thrombosis and highlights the complexities that arise during management. We encourage primary care providers to recognize those at risk for this condition and its sequelae, with the goal of early detection and swift treatment to enhance patient outcomes.

Case Report

A 62-year-old Hispanic female with a history of SLE, rheumatoid arthritis, pulmonary fibrosis, fibromyalgia, and Raynaud's phenomenon presented on March 13, 2025 with three weeks of progressive swelling and pain in her left leg. She attributed symptom onset to prolonged sitting while caring for her hospitalized daughter. She denied trauma, recent travel, chest pain, dyspnea, or fever.

Clinical Evaluation

- *History:* Prolonged immobility while caregiving; no recent trauma, travel, chest pain, dyspnea, or fever.
- *Physical exam:* Left leg swelling, tenderness, early mottling; distal pulses intact; hemodynamically stable.
- *Laboratory findings:* Hemoglobin 9.7 g/dL (nadir 6.7 g/dL), platelets 567 K/µL, pH 7.25, lactate 3.3 mmol/L.
- *Imaging:*
  - Duplex ultrasound — occlusive thrombus from left common femoral → popliteal vein and left great saphenous vein.
  - CT venogram — extensive thrombus from left iliac → popliteal veins with left common iliac vein compression by the right common iliac artery, consistent with MTS.

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Results

Day	Event Summary and Interventions
1-2	IV heparin started; urgent thrombectomy/thrombolysis/ angioplasty for phlegmasia risk. ICU transfer for postprocedure hypotension & pain.
3	CTA showed persistent thrombosis & severe iliac vein stenosis → stent recommended.
3-7	Rethrombosis on anticoagulation; anticoagulant changed to enoxaparin; hemoglobin dropped to 6.7 g/dL; Patient declined transfusion and transfer was delayed.
8	Patient stable but with persistent lower extremity swelling; Patient transferred for vascular stenting. Tolerated procedure well and was discharged on Eliquis with vascular and hematology follow up

Final diagnosis: Extensive left iliofemoral and popliteal DVT secondary to MTS with SLEassociated hypercoagulability, complicated by phlegmasia cerulea dolens and recurrent thrombosis despite initial intervention.

Discussion

May-Thurner syndrome (MTS) should be considered in all patients presenting with left-sided iliofemoral deep vein thrombosis (DVT), especially in those with known hypercoagulable states. In our patient, the combination of systemic lupus erythematosus (SLE)-related prothrombotic tendency and mechanical venous compression resulted in extensive thrombosis and recurrence despite prompt intervention.

Previous reports have described MTS complicated by extensive thrombosis in postpartum women,[4] young otherwise healthy patients,[1,3] and individuals without systemic risk factors.[2] However, reports of MTS in the context of SLE are rare. A systematic review by Kaltenmeier et al.[5] emphasizes that optimal management requires both thrombus removal and correction of the underlying venous compression. When compression is not promptly relieved, re-thrombosis can occur — as in our patient, where transfer delays prevented timely stenting, mirroring outcomes reported in similar cases.[1,2,4]

The current standard of care in symptomatic MTS involves:

1. Clot removal — typically via catheter-directed thrombolysis or mechanical thrombectomy.
2. Relief of compression — using balloon angioplasty and stenting.

Failure to address the underlying anatomical cause substantially increases the risk of recurrence and long-term morbidity.

Primary care physicians are often the first to evaluate unilateral leg swelling. Awareness of MTS risk factors — particularly autoimmune disease — can prompt earlier imaging and vascular referral, potentially preventing limb-threatening complications such as phlegmasia cerulea dolens and reducing post-thrombotic syndrome risk.

Clinical lessons from this case and literature review:

- Maintain high suspicion for MTS in patients with unilateral iliofemoral DVT, particularly those with autoimmune comorbidities or other hypercoagulable states.
- Employ advanced imaging early (CT venography, MR venography, or intravascular ultrasound) to identify anatomical causes.
- Definitive management requires both thrombus removal and correction of venous compression.
- Expedite stent placement to reduce the risk of re-thrombosis.
- Primary care providers play a critical role in early recognition and referral.

Endnotes

1. Khan M, Khan S, Al-Shaikh TM, et al. May-Thurner Syndrome: A Case of an Underdiagnosed Cause of Deep Vein Thrombosis. Cureus. 2021;13(10):e18994. doi:10.7759/cureus.18994. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC8569277/
2. Harbin MM, Lutsey PL. May-Thurner syndrome: History of understanding and need for defining population prevalence. J Thromb Haemost. 2020 Mar;18(3):534-542. doi:10.1111/jth.14709. Available from: https://pubmed.ncbi.nlm.nih.gov/31821707/
3. O'Sullivan GJ, Semba CP, Bittner CA, Kee ST, Razavi MK, Sze DY, Dake MD. Endovascular Management of Iliac Vein Compression (May-Thurner) Syndrome. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan. Available from: https://www.ncbi.nlm.nih.gov/books/NBK554377/
4. Zander KD, Staat B, Galan HL. May-Thurner Syndrome resulting in acute iliofemoral deep vein thrombosis in the postpartum period. Obstet Gynecol. 2008;111(2 Pt 2):565-569. doi:10.1097/AOG.0b013e318160d6d5.
5. Cervera R, Espinosa G. Cardioembolic stroke in systemic lupus erythematosus: lessons from the Euro-Lupus Cohort. Lupus. 2011;20(5):529-532. doi:10.1177/0961203310388570. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC3413961/
6. McLendon K, Goyal A, Bansal P, Attia M. May-Thurner Syndrome. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC305329/

References

Al-Nouri O, Milner R. May-Thurner syndrome: case report and review of the literature. Vasc Med. 2020;25(3):276-281. doi:10.1177/1358863X19898221. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC7155831/#R03

Moudgill N, Hager E, Gonsalves C, Larson R, Lombardi J, DiMuzio P. May-Thurner syndrome: case report and review of the literature involving modern endovascular therapy. Vasc Endovascular Surg. 2023;57(2):141-147. doi:10.1177/15385744231123451. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC10400067/

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Multifactorial Lower Extremity Swelling in a Young Athlete: A Case of Suspected Morel-Lavallée Lesion with Probable Recurrent Lyme Disease

By Kittu Rao, MD, MPH and Scott Darling, MD

Abstract

A 20-year-old female collegiate soccer player with a childhood history of Lyme disease presented with right ankle pain and calf swelling after a 5-hour car ride. Doppler ultrasound ruled out deep vein thrombosis but revealed a possible Morel-Lavallée lesion, and later confirmed by MRI as a 10x20 cm serosanguineous fluid collection. Aspiration drained 70 mL, but fluid reaccumulated. Knee MRI showed a dissecting popliteal cyst, synovitis, and bursal inflammation, with elevated inflammatory markers suggesting recurrent Lyme disease. Empiric doxycycline was initiated, pending serology. This case highlighted the need for a comprehensive evaluation and follow-up for swelling in athletes.

Introduction

Lower extremity swelling in athletes is commonly linked to trauma, such as sprains or contusions, but infectious causes like Lyme disease are often overlooked, especially in patients with prior exposure. Lyme disease, caused by *Borrelia burgdorferi*, can lead to recurrent arthritis years after initial infection, presenting with joint swelling, pain, and inflammation that mimic other conditions.[2] Morel-Lavallée lesions, resulting from shear forces that separate skin from underlying fascia, create subcutaneous fluid collections that are rare and difficult to diagnose due to their nonspecific symptoms.[1,11] Similarly, popliteal cysts, which form from intra-articular fluid extravasation, are often associated with meniscal tears or joint inflammation and can complicate the clinical picture.[4] These conditions, when combined with a history of Lyme disease, pose significant diagnostic challenges, as symptoms may overlap with autoimmune or traumatic etiologies.

This case involved a 20-year-old female collegiate soccer player with a childhood history of Lyme disease who presented with right ankle pain and calf swelling after a prolonged car ride. The combination of a Morel-Lavallée lesion, a dissecting popliteal cyst, and suspected recurrent Lyme disease is rare and underscores the complexity of managing multifactorial swelling in athletes. The case required advanced imaging, including Doppler ultrasound to rule out vascular issues and MRI to delineate soft-tissue and joint pathologies.[8,10] Laboratory testing for Lyme serology and inflammatory markers was critical to assess for infectious causes. At the same time, the initial false-positive anti-dsDNA result highlighted the risk of misdiagnosing autoimmune diseases like systemic lupus erythematosus (SLE).[5]

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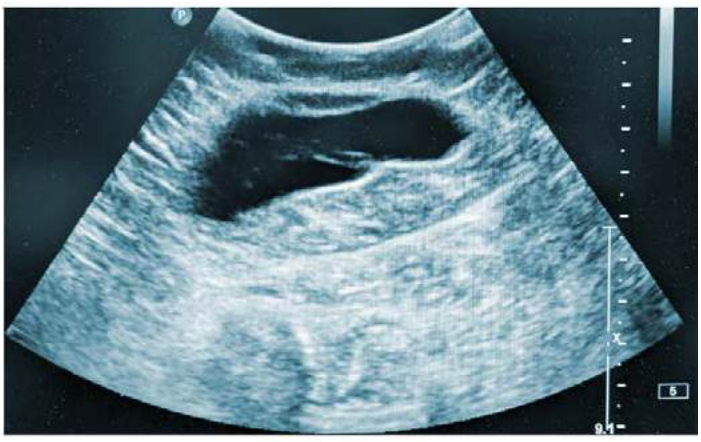

Figure 1: Lower Extremity Doppler Ultrasound Showing Extensive Edema Suggestive of Morel-Lavallée Lesion

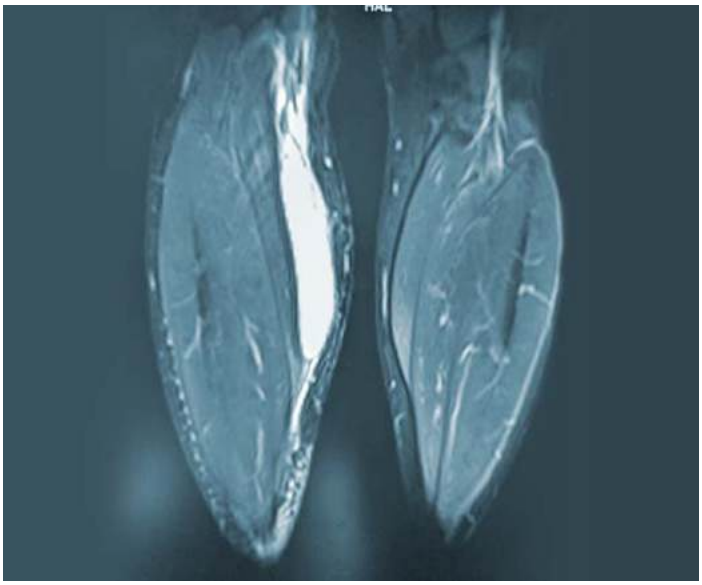

Figure 2: Tibiofibular MRI Confirming Morel-Lavallée Lesion with Ultrasound-Guided Aspiration of Serosanguineous Fluid Collection

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This case contributes to the fields of family medicine and sports medicine by illustrating the importance of a thorough diagnostic approach. It emphasizes the integration of point-of-care ultrasound and MRI for rapid and accurate identification of complex pathologies, alongside targeted laboratory testing to confirm or rule out infectious and autoimmune conditions.[13] The involvement of primary care, sports medicine, and rheumatology specialists was essential to navigate diagnostic uncertainty and tailor treatment to the patient's athletic needs. By addressing both traumatic and infectious factors, this case highlights the need for multidisciplinary collaboration to prevent misdiagnosis, optimize functional outcomes, and support the athlete's return to play.[9]

Case Report

A 20-year-old female collegiate soccer player from Germany, with a childhood history of Lyme disease treated without complications, presented with right calf swelling and ankle pain following a 5-hour car ride. She denied numbness, cold sensation, or recent trauma, was a non-smoker, and used oral contraceptives. Physical examination revealed a 2+ right knee effusion, non-tender, with preserved range of motion. Lower extremity Doppler ultrasound excluded deep vein thrombosis but identified extensive edema from the knee to the calf, suggestive of a Morel-Lavallée lesion. Within one week, swelling decreased, but pain worsened upon resuming soccer activity (Figure 1).

Tibiofibular MRI confirmed a 10x20 cm serosanguineous fluid collection in the medial gastrocnemius, diagnostic of a Morel-Lavallée lesion (Figure 2). Ultrasound-guided aspiration drained 70 mL of serosanguineous fluid. One week post-aspiration, fluid reaccumulated, with mild-to-moderate pain managed with a compression sleeve. Knee MRI revealed a dissecting popliteal cyst with leakage, synovitis, medial plica irritation, and inflammatory signals in the TCL-semimembranosus, semitendinosus, and pes anserine bursae, suggestive of inflammatory arthropathy, likely recurrent Lyme disease.

Laboratory findings included an elevated erythrocyte sedimentation rate of 82 mm/h and elevated C-reactive protein; anti-double-stranded DNA was initially reported positive but later deemed a false positive due to cross-reactivity in Lyme testing. Given her childhood history of Lyme disease, recurrent Lyme arthritis was strongly suspected, and empiric doxycycline (100 mg twice daily for 14 days) was initiated, pending full Lyme serology (IgG, IgM antibodies). Rheumatology evaluation noted no autoimmune family history. The differential diagnosis included recurrent Lyme disease (childhood history, bursal inflammation, synovitis), rheumatoid arthritis (synovitis, elevated ESR/CRP), seronegative spondyloarthropathy, systemic lupus erythematosus (SLE) (initially anti-dsDNA positive, now questioned), and traumatic bursitis from soccer-related trauma.

The final diagnosis was a Morel-Lavallée lesion, popliteal cyst with leakage, and probable recurrent Lyme disease, pending serology. The patient resumed soccer with a compression sleeve but experienced persistent pain and fluid reaccumulation. Activity modification was advised, and follow-up with primary care and rheumatology was scheduled; however, she returned home to Germany. The next step in testing would have included further Lyme and autoimmune workup (IgG, IgM antibodies, complement C3 and C4, sedimentation rate, CRP).

Discussion

This case illustrated the diagnostic complexity of lower extremity swelling in a young athlete with a childhood history of Lyme disease, presenting with a traumatic Morel-Lavallée lesion, a dissecting popliteal cyst, and probable recurrent Lyme disease. The Morel-Lavallée lesion likely resulted from soccer-related shear forces, creating a serosanguineous fluid collection between subcutaneous tissue and fascia. Its persistence post-aspiration highlighted management challenges in active individuals, as activity exacerbates reaccumulation.[1] The popliteal cyst and synovitis indicated intra-articular pathology, while elevated inflammatory markers (ESR 82 mm/h, CRP) and bursal inflammation suggest recurrent Lyme disease, supported by the patient's childhood Lyme history.[2]

Morel-Lavallée lesions are frequently misdiagnosed as hematomas or edema, which delays appropriate treatment. Recent studies have highlighted the importance of MRI in confirming these lesions and differentiating them from vascular or lymphatic causes.[1] The 30% recurrence rate in athletes underscores the necessity for prolonged activity modification or minimally invasive sclerotherapy for cases that do not respond to standard treatments.

The popliteal cyst, identified through knee MRI, likely resulted from increased intra-articular pressure caused by synovitis, which is a characteristic of Lyme arthritis.[4] A systemic process often involves inflammatory signals in multiple bursae, while bursitis that is local only affects one site.[2]

During the initial workup, a test for anti-dsDNA antibodies came back positive, suggesting a possible autoimmune condition. However, it was later found to be a false positive due to cross-reactivity with Lyme disease tests. Other conditions, such as rheumatoid arthritis, were considered less likely because the typical signs were absent.[6] Using point-of-care ultrasound was an ideal first-line modality, as it quickly identified the Morel-Lavallée

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lesion and is an excellent tool for detecting these fluid pockets.[8,10] An MRI was done to get a clearer picture of the knee's soft tissues and joint structures, which confirmed the popliteal cyst, which is often linked to joint swelling or meniscus issues.[4] Doppler ultrasound ruled out blood vessel problems, such as DVT. The false-positive anti-dsDNA test showed why it is so important to double-check results to avoid mistakenly diagnosing an autoimmune disease.[5]

The treatment plan focused on non-surgical options. Compression stockings were used to manage the Morel-Lavallée lesion, and doxycycline was prescribed for suspected Lyme disease recurrence. Recent studies suggested that doxycycline injections can help alleviate and treat challenging cases of Morel-Lavallée lesions by reducing fluid buildup.[3,12] If tests confirmed Lyme arthritis, a longer course of antibiotics, such as doxycycline or ceftriaxone for 28 days, might have been needed.[2] Follow-up care in Germany was recommended, including new Lyme tests, complement levels, and more autoimmune workups, to guide next steps.

This case offers valuable lessons for family doctors and sports medicine specialists. Persistent knee swelling in someone with a Lyme disease history needs a wide-ranging approach, considering injuries, infections, and inflammatory conditions. Point-of-care ultrasound is a quick and effective tool for identifying potential Morel-Lavallée lesions, especially in urgent settings like the ER or sports clinics.[8,10] MRI is still the best for detailed views of joint and tissue issues, such as popliteal cysts.[4] The misleading positive anti-dsDNA test shows how confirmatory testing is critical to avoid misdiagnosing conditions like SLE.[5] Of note, the primary care doctor and sports medicine experts focused on the patient's athletic needs and initial workup, while rheumatology helped sort out the infection and inflammation issues. This collaboration ensured the patient received optimum care, addressing both immediate symptoms and long-term goals.[13] Educating the patient about the possibility of Lyme disease returning and the role of serology testing/hormones in inflammation was also important.[7]

Endnotes

1. Koc BB, et al. Morel-Lavallée lesions: diagnosis and management in 2024. J Orthop Res. 2024;42(7):1567-1575.
2. Steere AC, et al. Lyme disease in 2025: diagnostic challenges and treatment updates. N Engl J Med. 2025;392(3):245-254.
3. Smith R, Jones T. Advances in the management of Morel-Lavallée lesions: a focus on minimally invasive techniques. Clin Orthop Relat Res. 2024;482(11):2045-2053.
4. Lee JH, Kim SY. Imaging of popliteal cysts: current approaches and clinical implications. Radiol Clin North Am. 2025;63(1):89-97.
5. Wormser GP, Shapiro ED. Diagnostic pitfalls in Lyme disease: false positives and cross-reactivity. Clin Infect Dis. 2024;79(5):1234–1240.
6. Aletaha D, Smolen JS. Diagnosis and management of rheumatoid arthritis: 2024 updates. Arthritis Rheumatol. 2024;76(8):1123-1132.
7. Petri M, Smith J. Hormonal influences on systemic lupus erythematosus: 2024 perspectives. Lupus. 2024;33(10):825-831.
8. Bronstein E, Kounelis-Wuillaume S, MaGill C, Leggit JC. Diagnosis and Treatment of Sports-Related Morel Lavallée Lesion With Point-of-Care Ultrasound: A Case Study. Mil Med. 2025 Jul 23:usaf378. Doi: 10.1093/milmed/usaf378. Epub ahead of print. PMID: 40700685.
9. Davies DM, Mills M. Repair of a gluteal Morel-Lavallee lesion with complex perineal laceration in a 44-year-old female: a case report. J Surg Case Rep. 2025 Jul 8;2025(7):rjaf483. PMID: 40631070; PMCID: PMC12234443.
10. Agrait Gonzalez MF, Rivera Ortiz N, Santiago J, Malaret Hernandez G. An Unusual Cause of Knee Pain Identified by Point-of-Care Ultrasound in the Emergency Department. Cureus. 2025 Jun 28;17(6):e86934. PMID: 40583917; PMCID: PMC12206076.
11. Ning T, Mei S, Ma M, Ruan X, Lan S, Fu Y. Diagnostic ultrasound characteristics of Morel-Lavallée lesions: Case and dataset analysis. Medicine (Baltimore). 2025 Jun 20;104(25):e42906. PMID: 40550049; PMCID: PMC12187309.
12. Bouchard MD, Pow C, Gilbert J, Slawaska-Eng D, Vivekanantha P, Fageeh R, Yan J. Characteristics, Patterns and Optimal Treatment Strategies of Morel-Lavallee Lesions: A Systematic Review. J Orthop Trauma. 2025 May 6. Epub ahead of print. PMID: 40333732.
13. Sadjo SA, Muller F, Lemelle JL. Morel-Lavallee Lesion of the Hip in Children: About a Case and Literature Review. J Indian Assoc Pediatr Surg. 2024 Nov-Dec;29(6):651-653. Epub 2024 Nov 5. PMID: 39691936; PMCID: PMC11649047.

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The Role of Family Medicine Physicians in Detecting Congenital Heart Disease in Rural Newborns

By Daniela Falcone and Michelle Lombardo, MD

Abstract:

Limited access to pediatric care in rural areas can delay diagnosis of serious conditions. In a family medicine clinic, a 1-month-old infant with no follow-up since birth was found to have a grade 2/6 systolic murmur along the left sternal border. She was born at term via unassisted delivery in a car en route to a birthing center. Inadequate weight gain was suspected at the visit, and the murmur prompted referral, echocardiographic confirmation of critical pulmonary stenosis, and timely cardiologic intervention. This case underscores the importance of thorough newborn assessments in primary care, particularly for infants born outside traditional settings.

Introduction:

Almost 2% of births in the United States (US) occur outside of hospitals, whether planned or unplanned.[1] Rates are substantially higher in rural areas, approaching 3%, compared to only 0.15% in urban regions.[2] Contributing factors include limited access to medical centers, rural poverty, multiparity, and long travel times to delivery units.[3] Rurality has also been identified as a risk factor for delayed access to care and decreased attendance at well-child visits.[4] As such, family medicine physicians often serve as the first line of pediatric assessment, playing a critical role in detecting heart murmurs and distinguishing benign findings from those suggestive of congenital heart disease.[5]

Congenital heart disease (CHD) represents nearly one-third of all congenital anomalies in the US.[6] Pulmonary stenosis, a rare CHD, occurs in approximately 1 in 2,000 live births and accounts for about 8% of all CHD cases.[7] It may occur in association with other congenital defects such as tetralogy of Fallot, or as part of genetic syndromes like Noonan syndrome.[6] However, pulmonary stenosis presents in isolation in 7% to 12% of cases.[7]

Here, we present a case of critical pulmonary stenosis detected during a routine new-patient family medicine visit for a one-month-old infant. The patient's care was complicated by an out-of-hospital birth, delayed initiation of pediatric care, and barriers unique to rural health settings.

Case Report:

A female infant was born at full term to a 28-year-old G3P3003 mother. The mother had received prenatal care at a birthing center and planned to deliver there; however, while commuting from a rural area, she delivered in the car en route. According to birthing center records, the infant's Apgar scores were 9 at both 1 and 5 minutes. Birth weight was 8 lb 3 oz (3713 g). The following day, the patient returned to the birthing center for routine newborn testing. Parental report confirmed passage of meconium and successful breastfeeding. Critical congenital heart disease screening and a newborn hearing screen were normal. No murmurs were appreciated on exam. She was afebrile with a heart rate of 150 bpm. Her weight was 7 lb 11 oz (3543 g) and she was cleared for discharge home. The infant also screened negative on the New York State Newborn Screening Panel.

Due to maternal hospitalization, insurance issues, and long travel distance, the infant experienced a delay in establishing primary care. She presented at one month of age to a suburban family medicine office with her mother and grandmother for a new patient visit. She had not been evaluated by a medical provider since the first 24 hours of life. During this time, she was fed a combination of donor breast milk and formula via bottle. Her caregivers reported one episode of a bloody-streaked stool.

On examination, a grade 2/6 systolic murmur was auscultated along the left sternal border. Peripheral pulses were strong, the skin was warm and well-perfused, and no cyanosis was observed. The mother denied any family history of congenital heart disease. At the time of the visit, the patient was 34 days old and weighed 9 lb 2 oz (4140 g), which raised concern for poor weight gain, as this reflected an average gain of less than 1 ounce per day. A sacral dimple was also noted on physical exam.

The patient was urgently referred to pediatric cardiology and was evaluated seven days later. At that visit, she appeared comfortable and in no acute distress. Her physical exam was largely unchanged from the family medicine assessment. However, pulse oximetry revealed an oxygen saturation of 87% on room air. Echocardiography demonstrated critical pulmonary valve stenosis with severe right ventricular hypertension and flattening of the interventricular septum. A patent foramen ovale with bidirectional shunting was also noted. Left ventricular systolic function was normal. The mildly reduced oxygen saturation was consistent with right-to-left atrial shunting. There was no evidence of a patent ductus arteriosus. The patient was admitted to the hospital directly from this visit.

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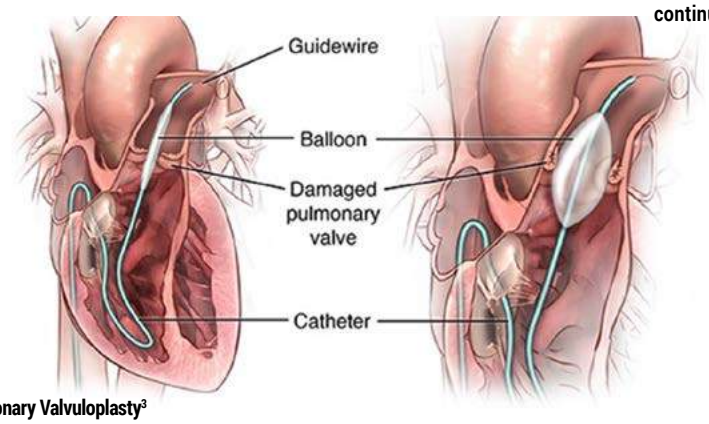

Figure 1: Pulmonary Valvuloplasty[3]

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Three days later, the patient underwent cardiac catheterization with balloon valvuloplasty of the pulmonary valve (Figure 1).[8] She tolerated the procedure well and was successfully weaned off nasal cannula. Post-procedure, her oxygen saturations normalized, she resumed feeding without difficulty, and demonstrated appropriate weight gain. The sacral dimple noted earlier was deemed benign via ultrasonography during hospitalization. After 48 hours of monitoring, she was discharged home in stable condition.

At her most recent cardiology follow-up, echocardiography revealed mild residual pulmonary valve stenosis, which is expected to remain stable without need for further intervention. She will continue routine cardiology follow-up at one year of age. The patient remains under the care of her family medicine clinic, where the life-threatening murmur was first identified. She has achieved all developmental milestones to date and continues to grow appropriately.

Discussion:

In summary, this case highlights an infant who experienced an out-of-hospital birth and delayed follow-up to primary care, resulting in the diagnosis of critical pulmonary stenosis. Timely intervention led to a successful outcome. This case underscores the essential role of family medicine physicians in recognizing pathologic murmurs, distinguishing them from innocent murmurs, and facilitating early and potentially lifesaving interventions.

The American Academy of Pediatrics recommends that healthy newborns be seen by a primary care physician within 48 to 72 hours after hospital discharge.[9] Missed or delayed visits can result in missed vaccinations, inadequate weight monitoring, and delayed recognition of congenital heart disease.[4] In this case, several factors contributed to delayed follow-up, including long travel distance, prolonged maternal hospitalization, insurance challenges, and a recent family relocation. Rural children, in particular, face disproportionately worse health outcomes.[10] They are more likely to live in low-income, medically underserved areas and have higher rates of hospital readmission compared to their urban counterparts.[10] A shortage of healthcare providers in rural areas has been well documented.[11] Family medicine physicians are the specialty most likely to practice in rural counties, yet while nearly 20% of the U.S. population resides in rural areas, only 11% of physicians do.[11] This creates a significant gap in access to care.[11]

Approximately 9% of infants are noted to have a heart murmur.[5] However, evidence suggests that physicians' auscultation skills are often suboptimal.[12] Neonatal murmurs can be particularly challenging to interpret, and studies show that 37% of infants with murmurs but no other syndromic features are ultimately diagnosed with congenital heart disease, with about 3% requiring procedural intervention during infancy.[5] Although these cases are relatively uncommon, careful cardiac auscultation and pulse oximetry screening remain simple, accessible, and potentially lifesaving tools.[5] In this case, a thorough newborn exam performed in a family medicine office likely saved the infant's life. The key takeaway for family medicine physicians is the importance of a comprehensive newborn assessment, including adequate heart auscultation and routine use of pulse oximetry screening.

Congenital heart disease (CHD) is one of the most common types of congenital anomalies, with pulmonary stenosis accounting for approximately 8% of cases.[6,7] Typical signs of CHD include a cardiac murmur, poor feeding, failure to thrive, and cyanosis.[7] However, these findings may be absent or subtle in the immediate newborn period, leading to missed diagnoses during initial screening.[13] In this case, a soft murmur and borderline inadequate weight gain at one month were the only early warning signs. Importantly, neither pulse oximetry nor newborn screening detects all CHDs, particularly right-sided lesions.[13] Non–ductus-dependent CHDs, such as the pulmonary stenosis seen in this infant, are especially likely to be missed by critical congenital heart disease screening, which relies on pulse oximetry.[13] This infant's screening was negative at the birth center following her unintended out-of-hospital delivery. Notably, pulmonary valve stenosis is among the CHDs most frequently associated with delayed diagnosis.[14]

In summary, rural health barriers such as poverty, transportation challenges, and limited access to physicians can contribute to delayed neonatal evaluation. Out-of-hospital births further increase this risk by limiting immediate access to standardized newborn assessments. Timely coordination between birth centers, hospitals, and primary care providers is essential to prevent delays in care. Family physicians should perform thorough newborn examinations and promptly refer any suspicious murmurs for cardiology evaluation. This case illustrates how attentive primary care can facilitate timely intervention and ultimately provide lifesaving care.

Endnotes

1. Lang, G., E.A.t. Farnell, and J.D. Quinlan, *Out-of-Hospital Birth*. Am Fam Physician, 2021. 103(11): p. 672-679.
2. Gutvirtz, G., et al., *Unplanned Out-of-Hospital Birth-Short and Long-Term Consequences for the Offspring*. J Clin Med, 2020. 9(2).
3. Grünebaum, A. and F.A. Chervenak, *Enduring safety concerns for out-of-hospital births in the United States*. American Journal of Obstetrics & Gynecology, 2024. 231(2): p. e70-e71.
4. DeGuzman, P.B., et al., *Rural Disparities in Early Childhood Well Child Visit Attendance*. Journal of Pediatric Nursing, 2021. 58: p. 76-81.
5. Ford, B., S. Lara, and J. Park, *Heart Murmurs in Children: Evaluation and Management*. Am Fam Physician, 2022. 105(3): p. 250-261.
6. van der Linde, D., et al., *Birth prevalence of congenital heart disease worldwide: a systematic review and meta-analysis*. J Am Coll Cardiol, 2011. 58(21): p. 2241-7.
7. Marchini, F., et al., *Pulmonary Valve Stenosis: From Diagnosis to Current Management Techniques and Future Prospects*. Vasc Health Risk Manag, 2023. 19: p. 379-390.
8. *Valvuloplasty*. 2025; Available from: https://www.hopkinsmedicine.org/health/treatment-tests-and-therapies/valvuloplasty.
9. Taylor, A. and J. Parekh, *Follow-up Care of the Healthy Newborn, in Neonatology for Primary Care*. 2020, American Academy of Pediatrics. p. 0.
10. Peltz, A., et al., *Characteristics of Rural Children Admitted to Pediatric Hospitals*. Pediatrics, 2016. 137(5): p. e20153156.
11. Barreto, T., et al., *Distribution of Physician Specialties by Rurality*. J Rural Health, 2021. 37(4): p. 714-722.
12. Mangione, S. and L.Z. Nieman, *Cardiac auscultatory skills of internal medicine and family practice trainees. A comparison of diagnostic proficiency*. Jama, 1997. 278(9): p. 717-22.
13. Arvind, B., A. Saxena, and S. Ramakrishnan, *Utility of pulse-oximetry screening in newborns with nonductus-dependent cyanotic congenital heart defects: A reason to alarm?* Ann Pediatr Cardiol, 2022. 15(1): p. 41-43.
14. Liberman, R.F., et al., *Delayed Diagnosis of Critical Congenital Heart Defects: Trends and Associated Factors*. Pediatrics, 2014. 134(2): p. e373-e381.

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When a Bed Becomes the Medicine: The Impact of Low-Barrier Shelter on Chronic Wound Healing in a Patient Experiencing Homelessness

By Anjali Prakash, MBBS and Sandhya Kumar, MD, MPH

Abstract

Chronic dependent edema, often associated with conditions such as heart failure, kidney disease, and cirrhosis, can be worsened by unstable sleeping arrangements among people experiencing homelessness. Prolonged upright sleeping exacerbates edema and can lead to ulceration. We present a 51-year-old woman with diabetes, hypertension, and schizophrenia who developed recurrent venous ulcers while residing in a drop-in center with only chairs for sleep. Despite treatment, ulcers recurred until she transitioned to a low-barrier shelter with a bed, where her edema resolved and wounds healed. This case highlights sleeping conditions as an underrecognized driver of chronic wounds.

Introduction

Homelessness is a significant challenge faced by a growing number of people in the US, with over 770,000 individuals experiencing homelessness on a single night in January 2024.[1] In New York State the number of people experiencing homelessness more than doubled between January 2022 and January 2024 to 158,019 people.[1] People experiencing homelessness face a number of health complications including cardiovascular disease, diabetes mellitus, pulmonary conditions, mental health disorders, infectious diseases, weather-related conditions, and injuries.[2-3] Homelessness complicates the management of chronic illnesses and increases vulnerability to new health problems, leading to substantial morbidity and mortality.[2]

Dependent edema is a condition that commonly affects people experiencing homelessness, especially if unable to consistently sleep in a bed. In dependent positions intravascular hydrostatic pressure increases, promoting fluid movement from the intravascular to interstitial space and leading to the accumulation of interstitial fluid in gravity-dependent areas, most commonly the lower extremities.[4-6] Sleeping conditions of people experiencing unsheltered homelessness, such as in cars, on benches, or on subway seats, contribute to the increased likelihood of dependent edema. The growing use of purposely designed 'hostile architecture' in many communities amplifies this problem (e.g. benches with central armrests intended to deter sleeping).[7] Those in indoor spaces like drop-in centers—where only chairs are available—may also develop dependent edema due to the inability to regularly lie flat.[8-9]

A number of comorbid conditions can complicate the management of dependent edema including chronic venous insufficiency, heart failure, obstructive sleep apnea, nephrotic syndrome, hepatic cirrhosis, and medication-induced edema.[4-5] Substance use, particularly injection drug use, can also contribute to the development of chronic venous ulcers.[10] These comorbid conditions are more common in people experiencing homelessness, further compounding the problem and increasing the likelihood of complications. Chronic peripheral edema can lead to chronic venous ulcers, cellulitis and erysipelas, as well as skin changes, for example venous stasis dermatitis and lipodermatosclerosis.[5] Chronic peripheral edema and its complications can also cause pain, gait impairment, and mobility limitations.[11]

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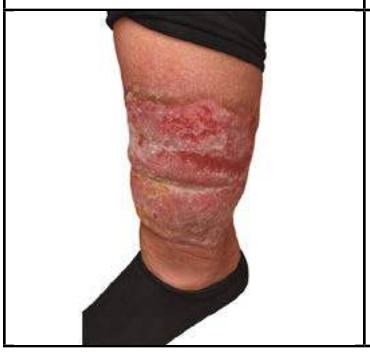
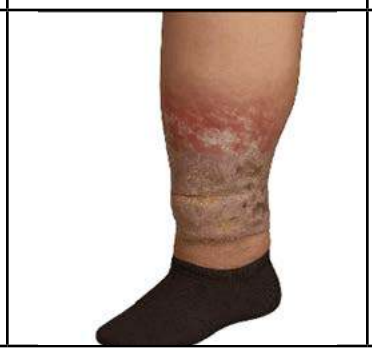
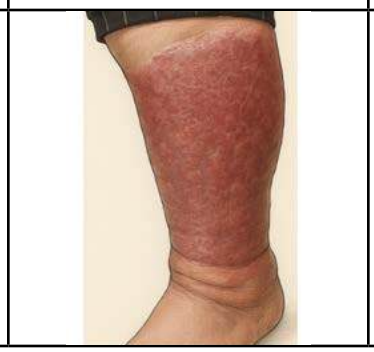
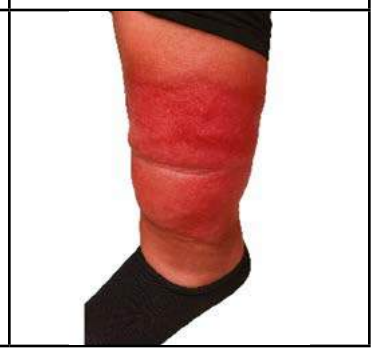
Figure 1.

At Time of Initial Presentation	1 Week After Initial Presentation	1 Month After Initial Presentation	2 Months After Initial Presentation
			

Images recreated with AI in order to promote anonymity.

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This case report aims to illustrate how a structural intervention - providing a bed for a patient experiencing unsheltered homelessness - helped resolve chronic venous ulceration.

Case Report: Ms. Alexander

The name and certain details of the patient described here are modified to maintain anonymity.

Ms. Alexander was escorted by a case manager for evaluation of lower extremity wounds due to concern for a strong odor.

Patient Background

Ms. Alexander is a 51-year old female with a history of diabetes, hypertension, and schizophrenia. Ms. Alexander became unhoused for the first time at age 46 after her parents both passed away, and she spent a period of time living on the streets. While New York City (NYC) does have a "right to shelter," like many individuals who live unsheltered, Ms. Alexander was not interested in staying in a shelter. This may have included concerns about fears of violence or theft within the shelter, fear or discomfort related to sharing spaces in congregate shelters, lack of privacy or autonomy, mistrust of institutions, or preference for familiar environments. For Ms. Alexander, this may have also included her unmanaged symptoms of her mental health condition that may have made it difficult to navigate shelter systems or trust staff. Ms. Alexander entered a drop-in-center in NYC at age 48, where she had access to hot meals, access to showers and laundry, case management, and onsite medical and mental health services. This drop-in-center, like other similar DICs in NYC operated 24 hours a day and seven days a week, and was designed to provide immediate respite and services to individuals experiencing homelessness. However, regulations require that DICs not offer any sleeping accommodations, meaning there are no beds or cots available in DICs, and as a result, Ms. Alexander slept in a chair.

Clinical Evaluation and Management

Ms. Alexander has had multiple hospitalizations across the local hospitals, primarily related to mental illness and related to wounds of her lower extremities. She was intermittently engaged in medical and mental healthcare at the local city hospital and at the DIC. Her medications included metformin, lisinopril, and risperidone. She declined any other primary care services outside of wound care.

On presentation, Ms. Alexander described pain in both lower legs and itching of the skin. On exam, Ms. Alexander was afebrile and hypertensive. Bilateral lower extremities demonstrated chronic venous stasis changes with ulceration (Figure 1). The skin appeared reddish with hemosiderin deposition, starting just above the ankle and extending up the lower leg. The skin appeared thickened with granulation tissue, and there was significant edema with oozing. Ulcerations were shallow with irregular borders. There was no purulent drainage. The odor was limited to the socks and shoes, and the cleaned wound did not have any odor. The wound was cleaned with soapy water to remove crusted exudate and dead tissue adhered to the skin. A&D ointment was applied to the periwound, gauze was applied to the wound, and ACE bandages were applied for gentle graduated compression, with increased pressure at the ankle and less pressure extending up the lower leg. Ms. Alexander was provided with supplies and provided with instruction to continue the same routine daily with one-week follow-up.

Outcome and Follow-up

In one-week follow-up, Ms. Alexander's wounds improved dramatically, with significant reduction in pain as well as edema, erythema, and drainage. Ulcerations had largely healed. However, as she continued to sleep in a chair, her lower extremity wounds recurred. This cycle repeated multiple times, compounded by the challenges of maintaining a wound care routine in the context of unmanaged mental health symptoms.

For the three years that Ms. Alexander was residing in the DIC, a case management team was working closely with her to establish trust and identify an alternative setting that would be acceptable to Ms. Alexander. As a result, within a year of presentation, Ms. Alexander transitioned to a "Safe Haven," which is a low-barrier shelter designed for individuals who may not engage with the traditional shelter system. Ms. Alexander's safe haven offered a small, private room with modest furnishings, on-site case management, and ongoing connection with medical care.

The ability to sleep in a bed broke the years-long cycle of wounds that had plagued this patient. As the wounds healed, her lower extremity pain resolved and she had marked improvement in her mobility, social engagement, and participation in healthcare and case management. She now resides in permanent supportive housing, which provides long-term, affordable housing with on-site support services that promote stability and independence in the community.

Discussion

Homelessness among New Yorkers arises from a constellation of structural and individual factors, with common experiences including unaffordable housing,[12] structural racism,[13] unmanaged mental illness,[14] history of criminal legal involvement,[15] domestic violence,[16] addiction,[14] and forced displacement.[17] This case illustrates the structural and social factors that contribute to disease burden in people experiencing homelessness, and highlights the critical role environmental determinants play in the resolution of severe dependent edema and chronic venous ulcers.

Recommendations for the treatment of venous ulcers include graduated compression therapy, leg elevation, and wound care, which may include tissue debridement, dressings, and infection prevention and treatment.[6,18-20] People experiencing homelessness face significant barriers to optimal management; however, tailored homeless healthcare through drop-in clinics, shelter-based clinics, and mobile outreach programs have been shown to improve accessibility to treatment.[21-23] Tailored homeless healthcare can also enhance the management of modifiable risk factors, including smoking cessation and optimal control of hypertension, diabetes mellitus, and heart failure.[3] Effective management of comorbidities is essential for both ulcer healing and prevention.[6,20] Expanding training programs to promote skills in homeless healthcare and improving primary care training in wound care, harm reduction, and trauma-informed care can also enhance management and lead to improved outcomes.[3,23-24]

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Access to a bed is vital for prevention and treatment of dependent edema and subsequent chronic venous ulcers in people experiencing homelessness. Low-barrier shelters—with modifications designed to reduce the restrictions that often keep people from accessing services—and Housing First approaches, which provide immediate access to housing without preconditions, have been shown to decrease hospitalizations and improve health outcomes for people experiencing homelessness.[3,24-25] Along with advocating against hostile architecture, the promotion of these solutions at a policy-level should be an aim for family physicians.

Endnotes

1. "New Yorkers in Need: Homelessness in New York State," New York State Comptroller, January 2025, https://www.osc.ny.gov/files/reports/pdf/new-yorkers-in-need-homelessness-nys.pdf.
2. Michael Liu and Stephen W. Hwang, "Health Care for Homeless People," *Nature Reviews Disease Primers* 7, no. 5 (January 14, 2021), https://doi.org/10.1038/s41572-020-00241-2.
3. Jason S. Lanham, Paige White, and Brody Gaffney, "Care of People Experiencing Homelessness," *American Family Physician* 106, no. 6 (December 2022): 684–93, https://www.aafp.org/pubs/afp/issues/2022/1200/homelessness.html.
4. Hiten Patel, Christopher (C.J.) Skok, and Anthony DeMarco, "Peripheral Edema: Evaluation and Management in Primary Care," *American Family Physician* 106, no. 5 (2022): 557–64, https://www.aafp.org/pubs/afp/issues/2022/1100/peripheral-edema.html.
5. Kathryn P. Trayes et al., "Edema: Diagnosis and Management," *American Family Physician* 88, no. 2 (2013): 102–10, https://www.aafp.org/pubs/afp/issues/2013/0715/p102.html.
6. Eri Fukaya and Raghu Kolluri, "Nonsurgical Management of Chronic Venous Insufficiency," *New England Journal of Medicine* 391, no. 24 (December 19, 2024): 2350–59, https://doi.org/10.1056/nejmcp2310224.
7. Winnie Hu, "'Hostile Architecture': How Public Spaces Keep the Public Out," *The New York Times*, November 8, 2019, https://www.nytimes.com/2019/11/08/nyregion/hostile-architecture-nyc.html.
8. Remy C. Martin-Du Pan, Raymond Benoit, and Lucia Girardier, "The Role of Body Position and Gravity in the Symptoms and Treatment of Various Medical Diseases," *Swiss Medical Weekly* 134, no. 1 (2004): 543–51. https://doi.org/10.4414/smw.2004.09765,
9. James W. Baish, Timothy P. Padera, and Lance L. Munn, "The Effects of Gravity and Compression on Interstitial Fluid Transport in the Lower Limb," *Scientific Reports* 12, no. 1 (2022): 4890, https://doi.org/10.1038/s41598-022-09028-9.
10. Barbara Pieper et al., "Impact of Injection Drug Use on Distribution and Severity of Chronic Venous Disorders," *Wound Repair and Regeneration* 17, no. 4 (July 2009): 485–91, https://doi.org/10.1111/j.1524-475x.2009.00513.x.
11. Soroush Besharat et al., "Peripheral Edema: A Common and Persistent Health Problem for Older Americans," *PLOS ONE* 16, no. 12 (2021): e0260742, https://doi.org/10.1371/journal.pone.0260742.
12. "National Low Income Housing Coalition," The Gap. n.d, accessed August 18, 2025, https://nlihc.org/gap.
13. Erika J. Edwards, "Who Are the Homeless? Centering Anti-Black Racism and the Consequences of Colorblind Homeless Policies," *Social Sciences* 10, no. 9 (2021): 340, https://doi.org/10.3390/socsci10090340.
14. Lauren M. Kaplan et al., "Unmet Mental Health and Substance Use Treatment Needs among Older Homeless Adults: Results from the HOPE HOME Study," *Journal of Community Psychology* 47, no. 8 (2019): 1893–1908, https://doi.org/10.1002/jcop.22233.
15. L. Couloute, "Nowhere to go: Homelessness among formerly incarcerated people," Prison Policy Initiative, August 2018, https://www.prisonpolicy.org/reports/housing.html.
16. Danielle Chiaramonte et al., "Examining Contextual Influences on the Service Needs of Homeless and Unstably Housed Domestic Violence Survivors," *Journal of Community Psychology* 50, no. 4 (2021): 1831–53, https://doi.org/10.1002/jcop.22637.
17. Delara Samari and Sebastian Groot, "Potentially Exploring Homelessness among Refugees: A Systematic Review and Meta-Analysis," *Journal of Social Distress and Homelessness* 32, no. 1 (2021): 135–50, https://doi.org/10.1080/10530789.2021.1995935.
18. Susan Bonkemeyer Millan, Run Gan, and Petra E. Townsend, "Venous Ulcers: Diagnosis and Treatment," *American Family Physician* 100, no. 5 (2019): 298–305, https://www.aafp.org/pubs/afp/issues/2019/0901/p298.html.
19. Steven Bowers and Eginia Franco, "Chronic Wounds: Evaluation and Management," *American Family Physician* 101, no. 3 (2020): 159–66, https://www.aafp.org/pubs/afp/issues/2020/0201/p159.html.
20. Thomas F. O'Donnell et al., "Management of Venous Leg Ulcers: Clinical Practice Guidelines of the Society for Vascular Surgery® and the American Venous Forum," *Journal of Vascular Surgery* 60, no. 2 (2014): 3S-59S, https://doi.org/10.1016/j.jvs.2014.04.049.
21. Wisoo Shin et al., "Drop-in Wound Care: Calgary's Wound Care Model Centred around People Experiencing Homelessness," *International Wound Journal* 22, no. 4 (2025): e70179, https://doi.org/10.1111/iwj.70179.
22. Rebekah A. Kaufman et al., "The Role of Street Medicine and Mobile Clinics for Persons Experiencing Homelessness: A Scoping Review," *International Journal of Environmental Research and Public Health* 21, no. 6 (2024): 760, https://doi.org/10.3390/ijerph21060760.
23. Valeriya Kopanitsa et al., "A Systematic Scoping Review of Primary Health Care Service Outreach for Homeless Populations," *Family Practice* 40, no. 1 (2022): 138–51, https://doi.org/10.1093/fampra/cmac075.
24. Carolyn Ingram et al., "Priority Healthcare Needs amongst People Experiencing Homelessness in Dublin, Ireland: A Qualitative Evaluation of Community Expert Experiences and Opinions," *PLOS ONE* 18, no. 12 (2023): e0290599, https://doi.org/10.1371/journal.pone.0290599.
25. Stephen W. Hwang and Tom Burns, "Health Interventions for People Who Are Homeless," *The Lancet* 384, no. 9953 (October 2014): 1541–47, https://doi.org/10.1016/s0140-6736(14)61133-8.

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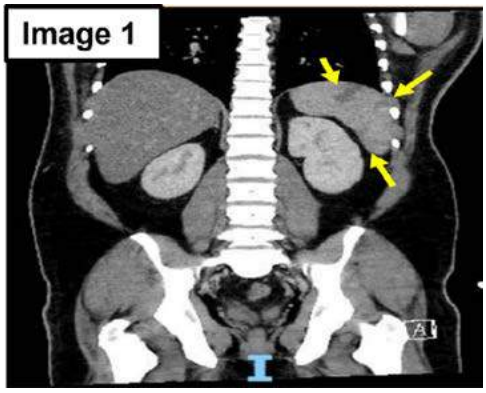
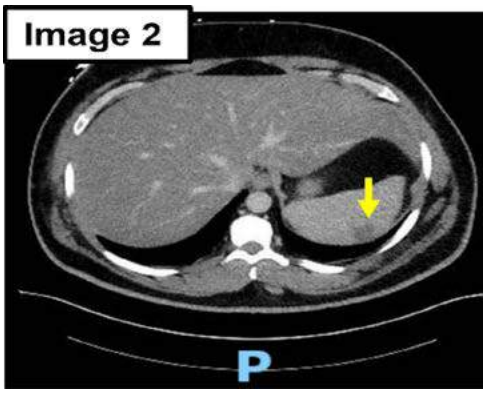
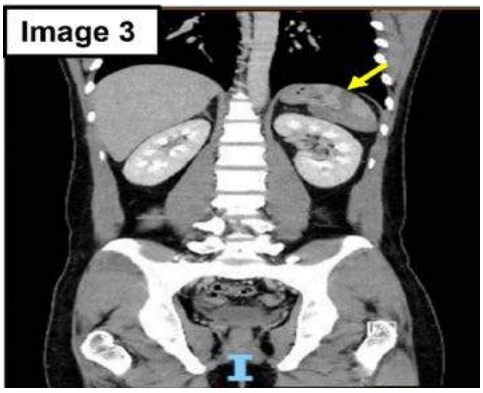
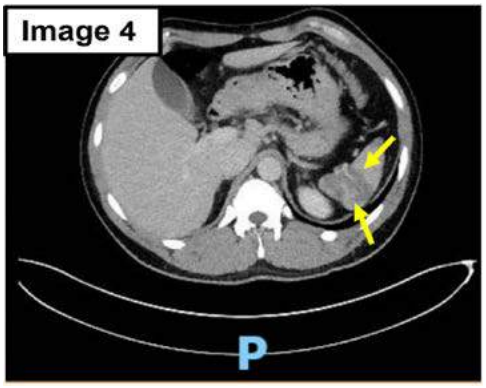
Recognizing the Forgotten Organ:
Case Studies of Splenic Infarction

By Mayur Rali, MD, FAAFP; Cristina Marti-Amarista, MD; Abigail Hamilton, MD, MBA and Margaret Donat, MD, FAAFP









Abstract

Splenic infarction (SI) is a rare but significant cause of left upper quadrant abdominal pain, often associated with thromboembolic diseases, hematological disorders, and inflammatory conditions. We present two cases of spontaneous SI with distinct etiologies: one in a 42-year-old male with type 2 diabetes and reactivated Epstein-Barr virus (EBV) infection, and the other in a 26-year-old male following energy drink consumption, with calcified lung granulomas. Both patients had additional risk factors for thrombosis. This case series highlights the complexity of SI and the importance of identifying underlying causes, which can guide targeted therapies and improve patient outcomes.

Introduction

The spleen is an often-neglected organ with immunomodulatory, metabolic, and endocrine function.[1] Splenic infarction is a rare cause of abdominal pain, although its exact prevalence remains unclear due to the absence of large-scale population studies. It is most associated with thromboembolic diseases, hematological disorders, and inflammatory conditions.[2,3] We present two cases of spontaneous splenic infarcts, one of idiopathic etiology and one secondary to reactivation of infectious mononucleosis.

Case #1

A 42-year-old male with a history of type 2 diabetes mellitus presented to the emergency department with left upper quadrant (LUQ) abdominal pain lasting for two days, accompanied by fever, malaise, and nausea. The review of symptoms, as well as the family and social history, was unremarkable. On examination, the patient was febrile (100.6°F) and tachycardic (110 bpm), with a clear pharynx, no lymphadenopathy, and tenderness upon palpation of the LUQ.

Initial workup revealed monocytosis, an elevated D-dimer (6490 ng/ml, reference <250), an elevated ESR (38 mm/hr, reference 0-20), and transaminitis (alkaline phosphatase 226 U/L, AST 59 U/L, ALT 110 U/L). Serologies for HIV, HCV, HBV, and COVID-19 were negative. Abdominal computed tomography (CT scan) demonstrated hepatosplenomegaly with multiple wedge-shaped splenic opacities, suggestive of splenic infarctions (Images 1 and 2). The patient was started on ceftriaxone, and further evaluations, including transthoracic echocardiogram, lower extremity venous Doppler, blood and urine cultures, and a hypercoagulability workup, were all negative.

The patient was discharged after four days of hospitalization, with the Epstein-Barr virus (EBV) panel still pending. One week later, during a follow-up visit, the patient reported resolution of abdominal pain. The EBV panel was reviewed at that time and revealed positive virus capsid IgG and IgM antibodies, positive nuclear antigen IgG, and positive early antigen IgG, consistent with a diagnosis of reactivated mononucleosis.

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Case #2

A 26-year-old male with no significant past medical history presented to the emergency department with LUQ abdominal pain, accompanied by subjective fever, chills, nausea, and vomiting. The symptoms began a few hours after consuming an energy drink, which he typically drinks several times a week. The patient denied any trauma, respiratory symptoms, diarrhea, blood in the urine or stool, or weight loss. He reported occasional alcohol use and worked as a butcher.

On physical examination, there was tenderness to palpation in the LUQ. Initial workup revealed leukocytosis with neutrophilia (WBC 16.84 K/uL; NEU 92.9%), along with elevated inflammatory markers (procalcitonin 0.09 ng/mL, CRP 0.9 mg/dL), but normal liver function tests. Serologies for HIV, HCV, HBV, and COVID-19 were negative. Abdominal CT showed multiple splenic infarcts (Images 3 and 4), while contrast-enhanced CT of the chest revealed multiple calcified granulomas in the right lung, along with calcified lymph nodes in the right hilum and mediastinum.

The patient was started on piperacillin/tazobactam (TZP) and full-dose enoxaparin. A hypercoagulopathy workup and blood cultures returned negative results, and sickle cell disease was ruled out. After one dose of TZP, the patient's leukocytosis was resolved. He was discharged on warfarin and referred to an outpatient hematology clinic for further evaluation of his spontaneous splenic infarcts.

Discussion

In this case series, we present two males who developed spontaneous splenic infarction under distinct clinical scenarios: one with reactivated Epstein-Barr virus (EBV) infection, and the other with granulomatous disease concerning for mycobacterium or sarcoidosis, both of whom had additional risk factors such as type 2 diabetes mellitus (T2DM) and energy drink consumption.

Regarding case #1, splenic infarction has been described in association with infectious mononucleosis, typically in immunocompromised patients or those with preexisting risk factors for thrombosis.[4] The patient's diabetes may have contributed to a hypercoagulable state, as individuals with diabetes have an increased tendency for thrombosis due to platelet hyperactivity and endothelial dysfunction.[5]

In case #2, the patient's history of energy drink consumption in the context of splenic infarction is noteworthy. Energy drinks are known to increase catecholamine release, leading to vasoconstriction and hypercoagulability, both of which may predispose individuals to thrombotic events.[6] The calcified lung granulomas present the possibility of previous tuberculosis or sarcoidosis, both associated with chronic inflammation and immune-mediated vascular changes that predispose to thromboembolic events.

Conclusion

This case series underscores the complexity and variety of factors that contribute to spontaneous splenic infarction highlighting the need for a thorough workup to determine the underlying etiology. By recognizing the diverse etiologies of SI, family physicians can significantly improve patient outcomes through early detection and targeted therapy.

Endnotes

1. Tarantino, G., Savastano, S., Capone, D., & Colao, A. (2011). Spleen: A new role for an old player?. World journal of gastroenterology, 17(33), 3776–3784. https://doi.org/10.3748/wjg.v17.i33.3776
2. Chapman J, Helm TA, Kahwaji CI. Splenic Infarcts. [Updated 2023 Jul 17]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK430902/
3. Chun-Han C, et al. Spontaneous Splenic Infarction as an Uncommon Cause of Fever in a Cirrhotic Patient. Int Journal of Gastroenterology, 2017, 11(2):121-124 https://doi.org/10.1016/j.ijge.2016.09.005
4. Mashav, N., Saar, N., Chundadze, T., Steinvil, A., & Justo, D. (2008). Epstein-Barr virus-associated venous thromboembolism: a case report and review of the literature. *Thrombosis research, 122*(4), 570–571. https://doi.org/10.1016/j.thromres.2008.03.005
5. Carr M. E. (2001). Diabetes mellitus: a hypercoagulable state. Journal of diabetes and its complications, 15(1), 44–54. https://doi.org/10.1016/s1056-8727(00)00132-x
6. Pommerening, M. J., Cardenas, J. C., Radwan, Z. A., Wade, C. E., Holcomb, J. B., & Cotton, B. A. (2015). Hypercoagulability after energy drink consumption. The Journal of surgical research, 199(2), 635–640. https://doi.org/10.1016/j.jss.2015.06.027

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When the *WORST* Happens

By Richard Mittereder, MD

Abstract

This case is poignant to family medicine providers in particular due to our overly-dependent computer assisted diagnostic environment. It involves a healthy 37-year-old man who suffered sudden collapse at his home office. He was discovered to have a worst case scenario grade 4 glioblastoma brain cancer. There were a few preceding clues to his diagnosis that may have been useful to pick up prior to a very traumatic seizure that led to emergency hospital admission. Although this case was well handled by the medical teams once involved, it makes one aware of the need to be ever mindful to use our human clinical intuition in addition to the many computer and AI assisted methodologies at the in-person patient evaluation.

Introduction

Tumors of the central nervous system are the 10th leading cause of death worldwide, with 90% being brain tumors. Glioblastoma represents about 15% of brain tumors, but is the most common brain cancer and is the most aggressive. Despite recent advances in treatments for other cancers, patients diagnosed with glioblastoma continue to have a poor prognosis for survival. Mortality, even with treatment, is greater than 90% at 5 years; median survival is 12.6 months.[8]

Symptoms and signs for glioblastoma are generally nonspecific, but often include headache or personality changes, as occurred in this case report. The diagnosis typically is made by a combination of CT scan, MRI, and tissue biopsy.

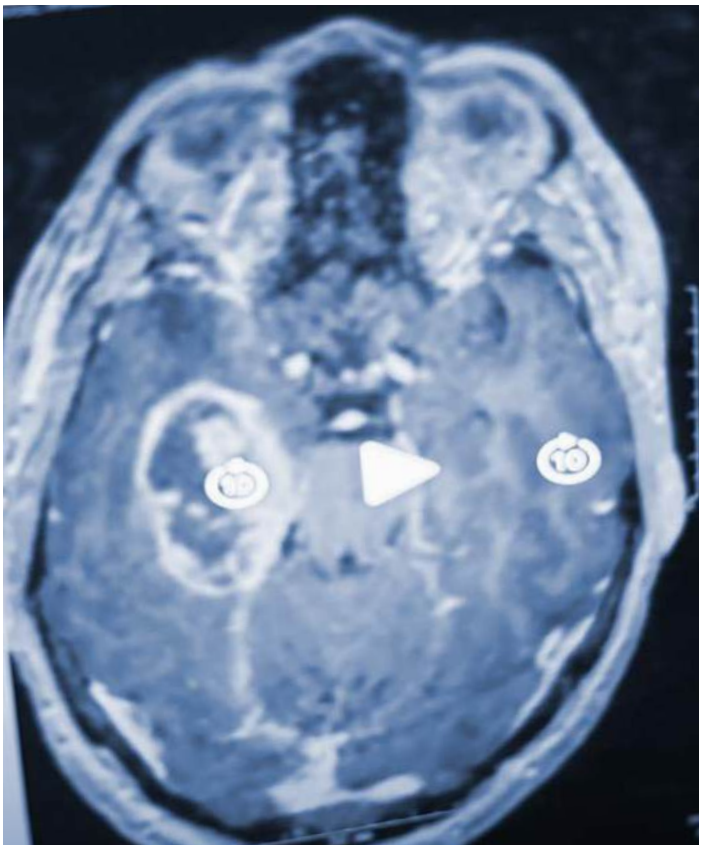
World Health Organization (WHO) Classification of Central Nervous System Tumors, fifth edition, 2021, redefined glioblastoma according to *molecular* testing with respect to isocitrate dehydrogenase (IDH) status. In addition, WHO defines glioblastoma as an adult grade 4 diffuse astrocytic glioma that is IDH-wildtype with one or more histologic or genetic features:[1,2]

- Microvascular proliferation
- Necrosis
- *TERT* promoter mutation
- *EGFR* gene amplification
- +7/-10 chromosome copy number change

Symptoms worsen rapidly, especially without treatment. Standard of care currently remains debulking surgery, if possible; then chemotherapy with temozolomide (TMZ) and concomitant adjuvant radiotherapy.

The cause of most cases of glioblastoma is unknown. Uncommon risk factors include the following:

- Genetic factors, e.g., neurofibromatosis[4]
- Exposure to ionizing radiation—e.g., therapeutic radiation[6]
- Exposure to non-ionizing radiation—e.g,.cell phone use (controversial)[3,5]
- Head injury, N-nitroso compounds, occupational hazards, electromagnetic field exposure (all inconclusive)[7]



Case Report

A previously well appearing 37-year-old gentleman was found down on the porch of his residence by a passing postal carrier. He had been witnessed moments before by his wife to be arguing on the phone with someone and then, suddenly, fell to the ground, developing a full generalized seizure with tonic-clonic movements of his limbs. EMS 911 was called and responded within minutes. He was quickly stabilized as the seizure abated. Assuming all possibilities, including drugs, the unconscious man was forcefully restrained to keep himself and EMS safe while treating him. He was stabilized and transported to the nearest emergency department. In subsequent evaluation, he was noted to have deformities and signs of trauma to both shoulders and his head. Early labs (metabolic panel, CBC) were within normal limits and his drug screen was negative. X-rays of the head and affected limbs revealed an intact skull, and his shoulders had trauma-induced bilateral fractures in the proximal humeri. A CT scan revealed 4 cm. amorphous, non-hemorrhagic mass in the right temporal area of the brain.

The man regained consciousness, albeit with mild disorientation and was admitted for further diagnostic testing and consultations.

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His past medical history:

– Essentially unremarkable except for hypercholesterolemia and anxiety.
– Medications included Lipitor 10 mg. qd, and Celexa 10 mg. qd

Family History:

– Remarkable for early CAD - PGf. died at age 39, MGf. died at age 47
– No significant cancer history

Social History:

– Attorney-at-law for 10 years
– Married for 7 years
– Physically fit, active jogger
– Daily cocktail
– Occasional use of recreational THC, no other drugs
– No high risk occupational exposures

Pertinent Review of Systems :

– Cardiovascular, pulmonary negative
– GI, GU negative
– Neurologic positive for daily headaches for 6 weeks
– Psychologic positive for being uncharacteristically argumentative for 1-2 weeks with wife/coworkers

Hospital Stay Synopsis:

His brain MRI showed a large right temporal lobe enhancing mass. See above photo. A neuro-surgical consult semi-urgently performed a biopsy resection of the tumor mass with gross cyto-pathology revealing a grade 4 glioblastoma. Neuro-oncology consult planned chemotherapy-radiation treatment course as an outpatient, pending biomarkers.

Out-patient Follow-up:

Pathology identification of biomarkers: IDH-1 mutation negative, confirming *wild-type* IDH; MGMT = unmethylated; PTEN,TERT, EGFR gene amplification identified, histologic microvascular proliferation/necrosis present.

All were consistent with STAGE WHO 4 high-grade glioma with a very poor prognosis. Oncologists predicted estimated survival at 7-12 months on treatment with a standard course of TMZ chemotherapy and radiation therapy. After completing this, the patient received a clinical trial using SURVAX-M plus adjuvant TMZ for glioblastoma (SURVIVE trial= NCT05163080). The trial terminated early due to recurrence of the tumor mass. He underwent a second debulking surgery, then further radiation and chemotherapy (lomustine). Despite this, recurrence and wider cancer spread was noted. The patient opted for hospice and died peacefully 15 months after his diagnosis was made.

Discussion

Based on the provided clinical information, the probability of brain cancer in a 37-year-old man with no family history or occupational risks is statistically quite low. However, brain cancer is a distinct possibility in the differential diagnosis of *any* individual presenting with complaints of daily headache for several weeks plus personality changes, regardless of other demographic data.

The other takeaway is that there are often mitigating factors. This individual was young and vital with a good Karnofsky score and excellent mental attitude all of which enabled him, with treatment, to gain 15 months of reasonably good quality of life. Thus, it is important to consider each case individually in view of the fact that family medicine practices are uniquely composed of wide cultural and age diverse populations.

Considerations for family medicine:

- Glioblastoma is rare: The likelihood of developing any type of cancerous brain tumor, including glioblastoma, is less than 1%. **HOWEVER, as in this case, our clinical antennae need to be up!**
- Glioblastoma incidence peaks at older ages: average age of diagnosis for glioblastoma is 64 years old. Incidence increases significantly with age. YET, it does happen in increasing #'s for younger populations raising concern about a potential increase in cases among younger adults. Specifically, **some reports suggest a rise in this diagnosis within the adolescent and young adult population (ages 15-39).**
- Additionally, though, there may simply be an increase in diagnosis due to greater access to neuro-imaging for clinicians, rather than a true increase in the incidence. Further epidemiological studies are needed.

Endnotes

1. WHO Classification of Tumours Editorial Board. Glioblastoma. *Central nervous system tumors*. 5th ed. International Agency for Research on Cancer; 2021. 6: 36-56.
2. Louis DN, Perry A, Wesseling P, Brat DJ, Cree IA, Figarella-Branger D, et al. The 2021 WHO Classification of Tumors of the Central Nervous System: a summary. *Neuro Oncol*. 2021 Aug 2. 23 (8):1231-1251. [QxMD MEDLINE Link].
3. Hardell L, Carlberg M. Mobile phone and cordless phone use and the risk for glioma - Analysis of pooled case-control studies in Sweden, 1997-2003 and 2007-2009. *Pathophysiology*. 2015 Mar. 22 (1):1-13. [QxMD MEDLINE Link].
4. Ostrom QT, Adel Fahmideh M, Cote DJ, Muskens IS, Schraw JM, Scheurer ME, et al. Risk factors for childhood and adult primary brain tumors. *Neuro Oncol*. 2019 Nov 4. 21 (11):1357-1375. [QxMD MEDLINE Link].
5. Kan P, Simonsen SE, Lyon JL, Kestle JR. Cellular phone use and brain tumor: a meta-analysis. *J Neurooncol*. 2008 Jan. 86(1):71-8. [QxMD MEDLINE Link].
6. Braganza MZ, Kitahara CM, Berrington de González A, Inskip PD, Johnson KJ, Rajaraman P. Ionizing radiation and the risk of brain and central nervous system tumors: a systematic review. *Neuro Oncol*. 2012 Nov. 14 (11):1316-24. [QxMD MEDLINE Link].
7. Fisher JL, Schwartzbaum JA, Wrensch M, Wiemels JL. Epidemiology of brain tumors. *Neurol Clin*. 2007 Nov. 25(4):867-90, vii. [QxMD MEDLINE Link].
8. Perkins A., Liu G. Primary Brain Tumors in Adults: Diagnosis and Treatment. *Am Fam Physician*. 2016;93(3):211-217B

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The Rash That Wasn't: A Primary Care Journey from Diagnostic Uncertainty to Active Recovery

By Minh Nguyen, MD and Soumya Sridhar, MBBS

Abstract

Muscle weakness with characteristic rash typically suggests dermatomyositis, but family physicians must recognize when initial presentations don't tell the complete story. A 71-year-old active male presented with progressive proximal weakness, V-shaped rash, and elevated CK (1002), initially suggesting dermatomyositis. Three critical turning points redirected diagnosis: peripheral smear showing bone marrow infiltration with ferritin spike to 5980, CT revealing bulky cervical lymphadenopathy with MRI pelvis showing 'passive congestion' secondary to marrow infiltration rather than inflammatory myopathy, and EMG confirming axonal motor polyneuropathy rather than myositis. Tissue biopsy confirmed classic Hodgkin's lymphoma with paraneoplastic motor neuropathy. The patient responded excellently to IVIG and outpatient chemotherapy, returning to pickleball at six months after treatment, demonstrating diagnostic flexibility and multidisciplinary coordination achieving excellent functional recovery.

Introduction

The classic triad of proximal muscle weakness, characteristic rash, and elevated muscle enzymes strongly suggests dermatomyositis, particularly in patients over 50 where paraneoplastic associations are common.[1,2] The 2017 EULAR/ACR classification criteria for dermatomyositis demonstrate high sensitivity (87%) and specificity (82%) for diagnosis.[11] While dermatomyositis affects 5–10 per million adults annually, paraneoplastic motor neuropathy in Hodgkin lymphoma represents fewer than 1% of cases, making this diagnostic challenge particularly relevant. This case demonstrates the critical importance of diagnostic flexibility when objective findings contradict initial clinical impressions and contributes to existing literature by illustrating systematic diagnostic reasoning that distinguishes true myositis from marrow-infiltrating malignancy.[3,4]

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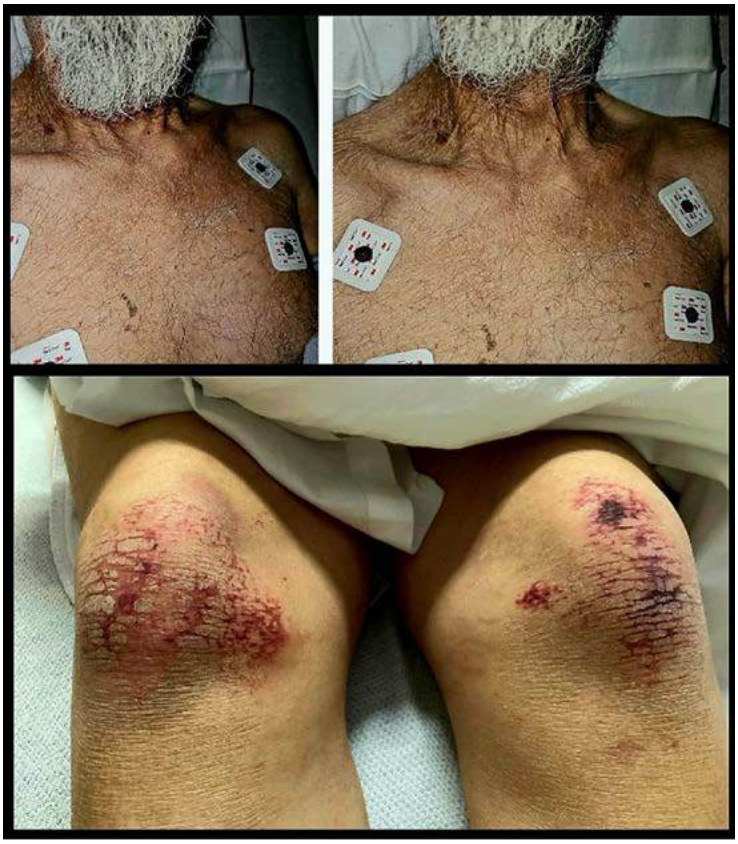

Figure 1: Clinical photographs showing a V-shaped erythematous rash over the anterior chest and shoulders ("shawl sign") and violaceous plaques over the knees consistent with Gottron-like papules/plaques, findings suggestive of dermatomyositis (published with patient consent).

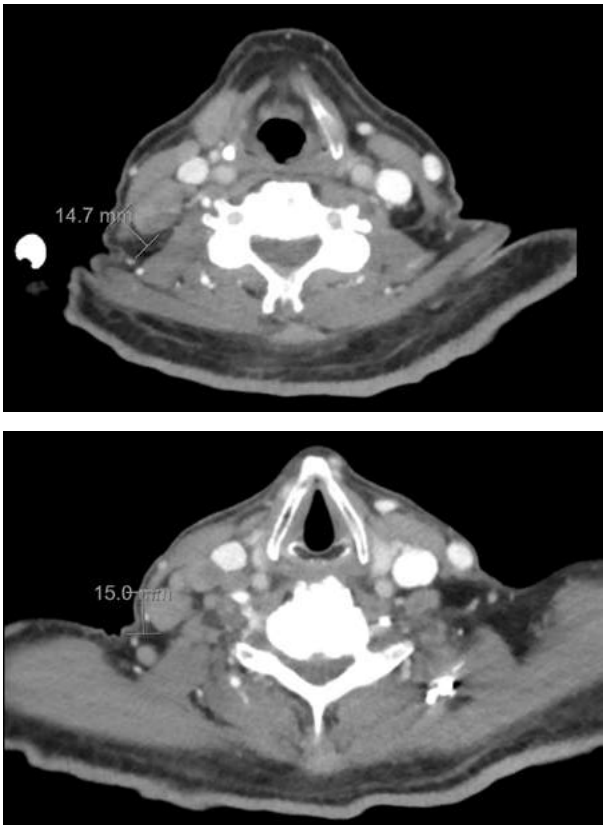

Figure 2: CT neck with soft tissue contrast demonstrating bulky cervical lymphadenopathy with multiple enlarged lymph nodes, with the largest measuring 1.5 cm, representing the lymphoproliferative process of classic Hodgkin lymphoma.

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Case Report

Initial Presentation

A 71-year-old previously active male (pickleball, cycling) presented following multiple falls over two days, including one down stairs. His family reported a three-month progressive decline with 25-pound weight loss, decreased appetite, and a new V-shaped 'tan' rash since November 2024. Past evaluation included constitutional symptoms, anemia, and liver abnormalities. In September 2024, prostate MRI showed 'multiple enhancing bone lesions highly suspicious for metastases,' but a January 2025 bone scan was negative.

On physical exam, the patient was ill-appearing and slightly jaundiced, with a V-shaped erythematous rash on the neck ('shawl sign'), violaceous scaly plaques on the elbows and knees (Gottron-like papules), and scalp scaling (Figure 1). Neurologic examination revealed truncal instability—he could not sit up from a supine position. Bilateral proximal weakness was noted (right 2/5 > left 3/5 in upper extremities, hip flexors 2/5 bilaterally), while distal strength in upper and lower extremities was preserved (5/5). Reflexes were brisk, and frontal release signs were present.

Initial laboratory studies showed CK 1002, LDH 658, hemoglobin 8.7, AST 217, ALT 65, alkaline phosphatase 290, total bilirubin 2.5, ferritin 925, ESR 91, and CRP 118.

The initial assessment strongly suggested dermatomyositis, likely paraneoplastic given his age and cancer history. The differential diagnosis included paraneoplastic dermatomyositis, idiopathic dermatomyositis, polymyositis, inclusion body myositis, drug-induced myopathy, motor neuron disease, and metabolic myopathy. Rheumatology and dermatology were consulted. Dermatology performed punch biopsy and emphasized comprehensive malignancy screening. Rheumatology noted atypical features, including unilateral weakness and skin lesions 'not classically Gottron's in appearance,' and recommended a myositis panel and autoimmune markers while holding immunosuppression pending further workup.

Hematologic Concerns

On hospital day 3, the patient developed SIRS with fever (38.4°C), hypotension (89/51 mmHg), and rising lactate (3.1 mg/dL). Hematology consultation revealed peripheral smear findings of nucleated red blood cells and immature white blood cells—a leukoerythroblastic picture pathognomonic for bone marrow infiltration.[5] This shifted the clinical focus from autoimmune disease to suspected hematologic malignancy. Hemoglobin dropped to 7.6, platelets to 61, and ferritin spiked from 925 to 5980. Ferritin >5000 is strongly associated with hematologic malignancy.[6] Notably, CK normalized (1002 to 30) despite persistent weakness—the CK normalization paradox pointing toward neurologic rather than myopathic etiology.[7] LDH remained elevated (658), and inflammatory markers persisted. A chronic DIC pattern emerged with elevated D-dimer (7.22 mg/L) and fibrinogen (510 mg/dL).

Imaging Revelations

CT of the neck showed bulky cervical lymphadenopathy with nodes up to 1.5 cm (Figure 2). Chest CTA revealed bilateral hilar lymph node enlargement. CT abdomen/pelvis revealed portal lymphadenopathy measuring 1.9 × 2.7 cm. MRI pelvis demonstrated osseous involvement with severe edema in proximal thigh musculature labeled 'passive congestion'—without inflammatory T2 enhancement, arguing against inflammatory myopathy.[8] These findings indicated multi-nodal lymphadenopathy with marrow involvement, and the passive congestion finding excluded inflammatory myositis.

Skin biopsy revealed orthohyperkeratosis without interface dermatitis or vacuolar changes—nondiagnostic for dermatomyositis—though follicular spicules suggested possible plasma cell disorder association.[1] Importantly, the absence of interface dermatitis definitively ruled out true dermatomyositis, indicating the rash was a paraneoplastic cutaneous manifestation mimicking dermatomyositis rather than an autoimmune inflammatory skin disease.

Specialist reassessments confirmed this evolving picture. Rheumatology signed off, citing CK normalization despite persistent weakness, atypical skin biopsy findings, and MRI evidence of passive congestion rather than inflammatory muscle edema, concluding this was not inflammatory myositis but likely a paraneoplastic process.

Neurologic Characterization

Neurologic exam revealed proximal weakness with upper motor neuron signs, including tongue fasciculations, brisk reflexes, and frontal release signs. EMG/NCS demonstrated a motor-predominant paraneoplastic pattern with severely reduced motor amplitudes (fibular 0.33 mV, tibial 1.6 mV, median 4.2 mV, ulnar 3.6 mV) and preserved sensory responses (radial 17 µV, sural 4 µV). Active denervation indicated ongoing nerve damage, with chronic reinnervation changes consistent with the three-month symptom timeline. Importantly, there was 'no irritability to suggest myositis,' definitively ruling out inflammatory myopathy. Secondary myopathic changes were noted only in the tensor fasciae latae muscles—the same anatomic location showing 'passive congestion' on MRI—confirming mechanical compression rather than primary muscle disease.

Clinical correlation showed EMG findings matched the motor-predominant weakness, MRI muscle edema pattern, CK normalization, and neurologic exam. All evidence converged on generalized axonal motor polyneuropathy consistent with paraneoplastic motor neuropathy.

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Diagnosis and Treatment

The team proceeded with IR-guided bone marrow and lymph node biopsies for definitive diagnosis. Autoimmune panels (ANA, dsDNA, SSA/SSB, RNP/Smith) were negative, and RF and C3/C4 were normal, ruling out systemic autoimmune disease. Free light chain analysis showed polyclonal elevation (Kappa 4.23, Lambda 4.51, Ratio 0.94), consistent with inflammatory response and ruling out multiple myeloma.

Lymph node biopsy revealed classic Hodgkin lymphoma with Reed-Sternberg cells positive for CD15, CD30, and Pax5. IVIG produced remarkable improvement within 48 hours, with the patient reporting he 'feels stronger than when he came in.' Asialo-GM1 antibody positivity confirmed an immune-mediated motor neuropathy mechanism.[10] The patient was discharged with physical therapy and oncology follow-up. PET-CT was scheduled for full staging, and hematology/oncology coordinated seamless outpatient chemotherapy. By six months, he had achieved complete neurologic recovery and returned to pickleball, with only mild residual tingling, demonstrating the excellent reversibility of paraneoplastic syndromes with prompt cancer treatment.

Discussion

This case illustrates several critical clinical lessons. Systematic evidence integration improves diagnostic accuracy through sequential refinement based on objective findings, emphasizing the importance of diagnostic flexibility when objective findings contradict initial impressions. The CK normalization paradox is a key clue: when CK normalizes despite weakness, neuropathy should be considered over myopathy.[7] Ferritin >5000 warrants urgent hematologic evaluation[6] and a leukoerythroblastic smear is pathognomonic for bone marrow infiltration.[5] MRI 'passive congestion' definitively excludes inflammatory myositis.[8] Expert diagnostic reasoning requires weighting pathognomonic findings (peripheral smear, tissue pathology) over supportive clinical evidence when they conflict. EMG findings must align with clinical, imaging, and laboratory trends. Paraneoplastic syndromes can create perfect clinical mimics, where skin and neurologic manifestations appear identical to autoimmune conditions but lack true inflammatory pathology. Therapeutic validation, such as the rapid IVIG response in this case, confirmed the immune-mediated mechanism.

Motor-predominant paraneoplastic syndromes in Hodgkin lymphoma are rare but reversible. This case exemplifies systematic diagnostic reasoning in complex diagnosis—sequential evidence integration based on objective findings led from initial dermatomyositis suspicion to confident final diagnosis. The case highlights how paraneoplastic syndromes can precede obvious malignancy signs and create near-perfect clinical mimics of autoimmune conditions. The systematic evaluation, diagnostic flexibility, and specialist integration led to early cancer diagnosis with excellent prognosis, demonstrating how coordinated multidisciplinary care can achieve optimal patient outcomes.[3,4,9] This case reinforces the critical role of primary care physicians in recognizing atypical presentations, advocating for timely multidisciplinary input, and guiding patients through complex diagnostic pathways.

Endnotes

1. Dalakas MC. Inflammatory muscle diseases. *N Engl J Med*. 2015;372(18):1734-1747.
2. Oldroyd AGS, et al. The idiopathic inflammatory myopathies. *Best Pract Res Clin Rheumatol*. 2019;33(1):101485.
3. Graus F, et al. Paraneoplastic neurological syndromes in Hodgkin and non-Hodgkin lymphomas. *Blood*. 2014;123(21):3230-3238.
4. Flanagan EP, et al. Paraneoplastic lower motor neuronopathy associated with Hodgkin lymphoma. *Muscle Nerve*. 2012; 46(5):823-827.
5. UpToDate. Approach to the adult with pancytopenia. Accessed August 2025.
6. Schram AM, Comstock P, Campo M, et al. Marked hyperferritinemia does not predict for HLH in the adult population. *Blood*. 2015;125(10):1548-1552.
7. Dimachkie MM, Barohn RJ. Idiopathic inflammatory myopathies. *Semin Neurol*. 2012;32(3):227-236.
8. Muscle MRI findings in inflammatory vs non-inflammatory myopathy. *AJR Am J Roentgenol*. 2008;190(6):1615-1621.
9. Ayyappan S, et al. Hodgkin lymphoma in older adults. *Hematol Oncol Clin North Am*. 2021;35(6):1077-1093.
10. Lucchinetti CF, et al. Autoimmune neuropathies and paraneoplastic syndromes. *Continuum (Minneap Minn)*. 2017;23(5):1343-1369.
11. Lundberg IE, Tjärnlund A, Bottai M, et al. 2017 European League Against Rheumatism/ American College of Rheumatology classification criteria for adult and juvenile idiopathic inflammatory myopathies and their major subgroups. *Arthritis Rheumatol*. 2017;69(12):2271-2282.

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Unmasking the Culprit: Occupational Exposure and Chronic Kidney Disease

By Deborah Hong, DO; Zuleen Chia Chang, MD and Lisa Shapiro, MS, DO

Abstract

A 39-year-old male from El Salvador with a history of agricultural work presented to our family medicine center with chronic kidney disease (CKD) stage 3b of unknown etiology (CKDu). The absence of typical risk factors like hypertension, diabetes, or relevant family history, coupled with evidence of tubular dysfunction, raised suspicion that an occupational etiology such as chemical/pesticide exposure or repetitive dehydration from sun exposure was responsible. This case emphasizes the importance of investigating atypical causes of CKD when common etiologies are absent.

Introduction

Chronic kidney disease (CKD) is frequently diagnosed by family medicine physicians, most often associated with diabetes and hypertension. However, CKD of unknown etiology (CKDu), characterized by progressive kidney damage without readily identifiable causes, poses a significant diagnostic and therapeutic challenge. CKDu has a high prevalence in male agricultural workers from El Salvador. A potential link to specific occupational and environmental exposures has been suggested.[1,3]

We will emphasize the importance of considering atypical etiologies (especially in individuals with occupational risk factors) during the diagnostic workup of CKD and highlight the need for early detection of CKDu, which when undetected and unchecked can have devastating consequences. It is estimated that more than 60,000 deaths related to kidney failure occurred in Central America between 1997 and 2013. El Salvador is disproportionately affected with a mortality rate that is 10 times higher than that in the United States.[2,3] The high mortality rate underscores the importance of addressing this public health problem.

Case Report

A 39-year-old asymptomatic male from El Salvador presented to our family medicine center to establish care. He did not report any concerns. Occupational history was significant for agricultural work in his native country and current employment as a landscaper. He denied other significant past medical history such as hypertension or diabetes and denied a family history of kidney disease. Medication history was positive only for rare use of non-steroidal anti-inflammatory drugs.

Initial examination revealed mildly elevated blood pressure (129/82 mmHg) with otherwise normal findings. Laboratory results showed elevated serum creatinine (2.02 mg/dL), blood urea nitrogen (25 mg/dL), and reduced estimated glomerular filtration rate of 42 mL/min/1.73m^2. Hypokalemia (3.2 mEq/L) was also noted. Hemoglobin A1c was in the prediabetic range (5.9%). Further investigation revealed proteinuria (protein/creatinine ratio 1.1) and albuminuria (albumin/creatinine ratio 267 mg/g) with elevated random urine potassium (23.5 mmol/L), suggestive of tubular dysfunction. Serum heavy metal testing was not performed. Despite potassium supplementation, renal function remained impaired over six months (serum creatinine 1.89-2.16 mg/dL, eGFR 39-45 mL/min/1.73m^2). A computed tomography scan of the abdomen and pelvis without contrast to evaluate for nephrolithiasis revealed hepatomegaly with diffuse steatosis but no hydronephrosis or renal calculi. Serum immunofixation was negative for monoclonal gammopathy.

Nephrology consultation suggested chronic interstitial nephritis secondary to chemical/pesticide exposure and/or repetitive dehydration from sun exposure as potential etiologies of his CKD stage 3b and CKDu, given the patient's agricultural history. It was not possible to distinguish the etiology more specifically. Management currently focuses on potassium replacement therapy and dietary potassium intake. Currently, the patient remains asymptomatic and continues to follow up with plans for further labs and imaging after 3 months of therapy.

Discussion

Chronic kidney disease of unknown etiology (CKDu) is a term used to describe chronic kidney disease in the absence of common risk factors like hypertension and diabetes and is characterized by tubular proteinuria, lack of hypertension and edema.[6] The condition predominantly affects young to middle-aged males, particularly those residing in rural areas and engaged in agricultural work. Several factors are implicated in the development of CKDu among agricultural workers:

- Dehydration: Repetitive episodes of dehydration, common with strenuous outdoor work, can lead to recurrent acute kidney injury and subsequent progression to CKD.[3,6]
- Agrochemical exposure: Exposure to fertilizers, pesticides, and herbicides can occur through inhalation, dermal absorption, or ingestion of contaminated food or water. The lack of proper safety regulations, inadequate use of personal protective equipment, and unsafe handling practices further increase the risk for agricultural workers.[3,6]
- Heavy metal exposure: In regions with volcanic soil like El Salvador, agricultural workers face an additional risk from heavy metal exposure. Metals such as lead, cadmium, and arsenic are naturally occurring in these soils and can contaminate water sources and crops. These heavy metals are known nephrotoxins and can accumulate in the body over time, leading to progressive kidney damage.[3,6]

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- Infectious diseases: Certain infections endemic to some agricultural regions, such as leptospirosis, hantavirus, and malaria, can cause renal damage if left untreated.[3]

CKDu is often diagnosed late due to its asymptomatic early stages and the limited access to healthcare in underserved, resource-limited areas.[5] Late-stage symptoms include fatigue, shortness of breath, gastrointestinal issues, weight loss, sleep disturbances, and ultimately, death.[6] While diagnosis can be made with elevated serum creatinine, proteinuria, hypokalemia, hyponatremia, hypomagnesemia, hyperuricemia, or reduced kidney size on ultrasound, not all these findings may be present.[3,5,6] A thorough history detailing occupational and environmental exposures is crucial. Renal biopsy is not routinely performed, but when it is, histopathological findings often reveal tubular atrophy, interstitial fibrosis, and global glomerulosclerosis.[5]

Management of CKDu focuses on slowing progression to end-stage renal disease (ESRD) requiring dialysis or transplantation by mitigating identifiable risk factors and limiting further exposures.[3,6] Preventive measures include ensuring access to safe drinking water, providing adequate shade and rest breaks during work, instituting workplace safety procedures with regard to heavy metals, fertilizers, pesticides, and herbicides and promoting proper hydration with electrolyte supplementation.[3] Access to dialysis remains a significant challenge for patients in resource-limited settings.[3,6]

Currently, no established guidelines exist for early CKDu screening. Screening appropriately defined high risk groups would potentially have benefit and research into this is needed. In addition, not enough is known regarding the link to fertilizers, pesticides, and herbicides. A study evaluating pendimethalin and atrazine exposure suggests a potential link, though further investigation is needed.[7] The poor enforcement of bans on pesticides of known risk, as well as limited literacy exacerbates the problem. In 2013, the Congress of El Salvador proposed a ban to prohibit nephrotoxic chemicals like paraquat and glyphosate but this was never enacted into law.[3] In Mejía et al.'s cross-sectional study, many agricultural workers in El Salvador had limited primary education due to high poverty rates and unknowingly mishandled hazardous pesticides without appropriate personal protective equipment. Many pesticides had restricted use but were still available in the market, allowing purchase of the pesticide without understanding the technical language of the labels and leading to their misuse.[4]

Greater efforts to increase awareness of and more research focused on CKDu is needed. Research is crucial to understanding the complex interplay of occupational and environmental factors in disease development. It would seem likely that early intervention would slow disease progression and improve outcomes and could be accomplished with the development and implementation of early detection and screening programs. Policies fostering the protection of agricultural workers including regulation and enforcement of pesticide use and improving occupational factors such as access to safe drinking water, are needed to address systemic issues.

This case highlights the need for a comprehensive, patient-centered approach to CKD diagnosis and management. Beyond addressing traditional risk factors, family physicians are well-positioned to assess and address the social determinants of health that contribute to CKDu, such as occupational exposures and limited access to resources. Timely diagnosis coupled with initiatives to address this public health problem can improve the health and well-being of individuals affected by this challenging condition.

Endnotes

1. Herrera, Raúl, Carlos M. Orantes, Miguel Almaguer, Pedro Alfonso, Héctor D. Bayarre, Irma M. Leiva, Magaly J. Smith et al. "Clinical characteristics of chronic kidney disease of nontraditional causes in Salvadoran farming communities." *MEDICC review* 16 (2014): 39-48.
2. Hoy, Wendy, and Pedro Ordunez. "Epidemic of Chronic Kidney Disease in Agricultural Communities in Central America. Case definitions, methodological basis and approaches for public health surveillance." (2017): 1-54.
3. Johnson, Richard J., Catharina Wesseling, and Lee S. Newman. "Chronic kidney disease of unknown cause in agricultural communities." *New England Journal of Medicine* 380, no. 19 (2019): 1843-1852.
4. Mejía, Roberto, Edgar Quinteros, Alejandro López, Alexandre Ribó, Humberto Cedillos, Carlos M. Orantes, Eliette Valladares, and Dina L. López. 2014. "Pesticide-Handling Practices in Agriculture in El Salvador: An Example from 42 Patient Farmers with Chronic Kidney Disease in the Bajo Lempa Region." *Occupational Diseases and Environmental Medicine* 02 (03): 56–70.
5. Paidi, Gokul, Anuruddhika I. Iroshani Jayarathna, Divya Bala Anthony Manisha R. Salibindla, Jashvini Amirthalingam, Katarzyna Karpinska-Leydier, Khadija Alshowaikh, and Huseyin Ekin Ergin. "Chronic kidney disease of unknown origin: a mysterious epidemic." *Cureus* 13, no. 8 (2021).
6. Priyadarshani, Watte Vidanelage Dinesha, Angela F. Danil de Namor, and S. Ravi P. Silva. "Rising of a global silent killer: critical analysis of chronic kidney disease of uncertain aetiology (CKDu) worldwide and mitigation steps." *Environmental geochemistry and health* 45, no. 6 (2023): 2647-2662.
7. Shearer, Joseph J., Dale P. Sandler, Gabriella Andreotti, Kazunori Murata, Srishti Shrestha, Christine G. Parks, Danping Liu et al. "Pesticide use and kidney function among farmers in the Biomarkers of Exposure and Effect in Agriculture study." *Environmental research* 199 (2021): 111276.

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Invasive Pulmonary Aspergillosis in a Relatively Immunocompetent Patient

By Melissa Di Santo, MD and Elizabeth Harding, MD

Abstract

Several countries have seen an increase in the incidence of invasive aspergillosis; however, many cases are overlooked and diagnosed only at the time of autopsy.[1] A 59-year-old Karen immigrant with a history of type 2 diabetes, hypertension, and hyperlipidemia was admitted multiple times for intractable left-sided headaches secondary to a left orbital apex mass. She eventually underwent a craniotomy for evacuation of the mass, with frozen pathology consistent with aspergillus species. Moreover, the pulmonary nodules and cavitary lung lesion on CT torso revealed a final sputum culture that grew *Aspergillus fumigatus*, which was likely the origin of the fungal disease. Considering her disseminated, angioinvasive *Aspergillus fumigatus*, she was treated with voriconazole. The low index of suspicion for invasive aspergillosis caused an unfortunate delay in diagnosis and treatment, with the potential for long-term sequelae.

Introduction

Since 2013, estimates have found 3,000,000 cases of chronic pulmonary aspergillosis and 250,000 cases of invasive pulmonary aspergillosis (IPA) globally.[2] In the US alone, the number of cases of aspergillosis is challenging to determine because aspergillosis is not a reportable disease.[3,4] The incidence of IPA in the US is nearly 1 to 2 cases per 100,000 individuals.[4,5]

This case emphasizes the importance of maintaining a high clinical index of suspicion for invasive aspergillosis despite the absence of classic risk factors. It is important to consider that IPA is no longer a disease isolated to immunocompromised patients. In our case, it should raise suspicion for aspergillosis if an immunocompetent individual presents with unresolving neurologic and/or pulmonary symptoms while on a relatively short course of systemic corticosteroid therapy.

Case Report

A 59-year-old Karen refugee to the United States, who had resided in the US since October 2006, presented to an outside hospital on 5/20/22 with left eye proptosis and left-sided headaches. The onset of headaches began in December 2021. At her initial neurology appointment on 5/9/22, she was given the clinical diagnosis of giant cell arteritis based on previously elevated ESR (43), unilateral headaches, and ocular symptoms.

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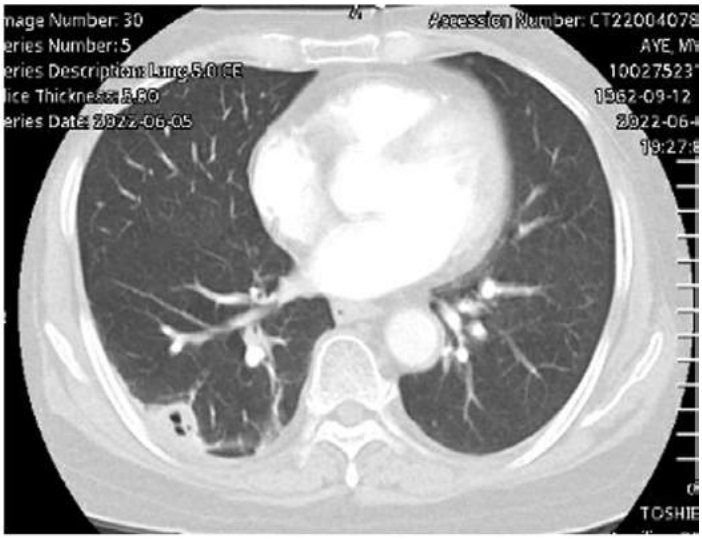
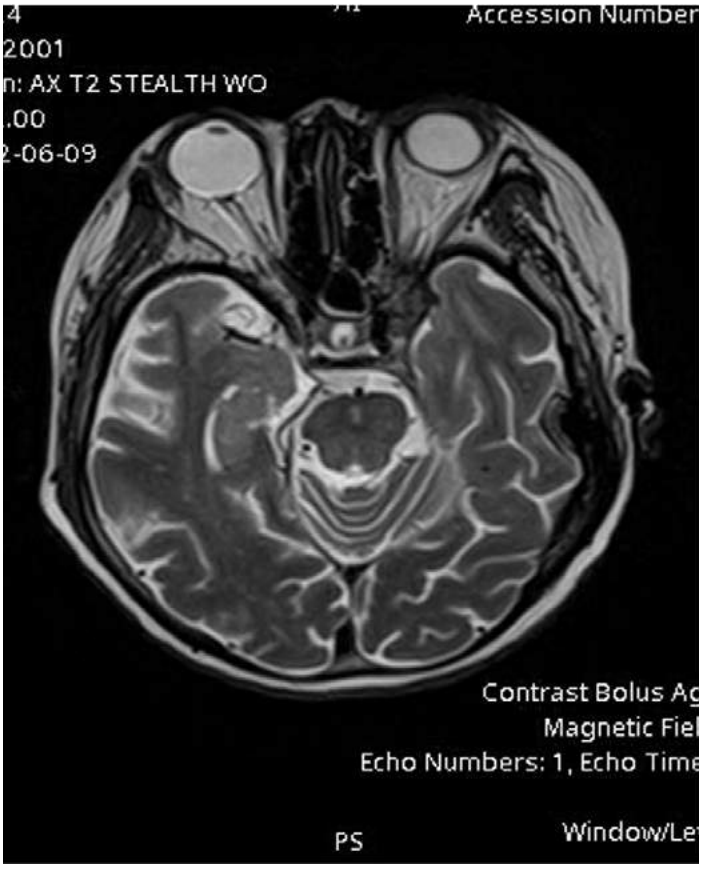
Figure 1 *(below)*. CTA chest with IV contrast from 6/5/22, read as: "Numerous bilateral pulmonary nodules, nodular/tree-in-bud opacities, and a cavitary right lower lobe lesion. These findings are nonspecific however suggestive of infectious/inflammatory process with other etiologies not excluded."

Figure 2 *(right)*. MRI brain from 6/9/2022, read as: "Enhancing lesion left anterior cavernous sinus/orbital apex again noted measuring approximately 2.4 x 1.4 cm maximal transverse dimension with dense, mildly heterogeneous contrast enhancement. The abnormality extends to the inferior orbital fissure. As noted on prior study (5/29/22) there is atrophy of the left optic nerve."





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She was started on high-dose prednisone until re-evaluation. When she presented to the outside hospital on 5/20/22, she was still taking high-dose prednisone. At that time, she had an MRI of the orbit demonstrating a 2.1 cm left orbital apex mass. She denied visual acuity changes, diplopia, or amaurosis fugax. Prednisone was increased from 60 mg to 125 mg daily, and she was admitted.

She also had a torso CT on 5/23/22, revealing no lymphadenopathy or obvious primary oncologic site. A 3 mm calcified granuloma was noted in the posterior left lower lobe of the lung with few scattered calcifications in the liver up to 7 mm in diameter. She was discharged on 5/27/22 with a Medrol Dosepak and advised to follow up with neurosurgery for management of her left orbital apex mass. However, 2 days later (5/29), she presented to our hospital with worsening left-sided headaches and new left-sided ptosis and dilated left pupil. Repeat MRI brain demonstrated *"left orbital apex and anterior cavernous sinus mass measuring up to 2.2 cm, with associated atrophy of the intraorbital optic nerve."* Based on these findings and her symptoms, she was treated with pain control and PO dexamethasone 2 mg BID, which seemed to help. She was discharged home again on 6/1 with Percocet and dexamethasone, as well as neurosurgery follow-up on 6/3 for preoperative planning.

She returned to the hospital 4 days later (6/5) with new-onset dyspnea, productive cough, and right-sided pleuritic chest pain. In the emergency department, she was hypoxic and had leukocytosis (13.8 x10^9/L). A respiratory viral panel, including COVID-19 PCR, was positive for influenza A. She also had an elevated D-dimer (2.6), which prompted the ordering of a CTA. CTA was negative for pulmonary embolism but showed a 1.8 cm right lower cavitary lesion, bilateral pulmonary nodules, and a small right pleural effusion (Figure 1). As previously mentioned, the CT torso from May 2022 reported no similar lesions, making these findings relatively new. Based on the above clinical findings, her new-onset hypoxia and cavitary lesion were thought to be secondary to influenza A complicated by a superimposed bacterial pneumonia at the time.

She was admitted and started on a 5-day course of Tamiflu alongside empiric coverage with vancomycin and Zosyn. Sputum culture, AFB stain, Quantiferon-TB, and Legionella antigen urine were obtained. The Legionella antigen urine was negative. After 48 hours, vancomycin and Zosyn were transitioned to PO Augmentin for an additional 5 days, given a negative MRSA swab and overall clinical improvement. She was stable for discharge on 6/8 with a plan for neurosurgery follow-up for tentative surgical intervention on 6/17. Quantiferon-TB and sputum culture were pending upon discharge on 6/8. The sputum culture showed early growth of a mold on 6/9.

She returned the next day (6/9) with persistent headaches despite prednisone and pain medication. This time she was admitted for 33 days (6/9-7/12). Repeat MRI brain revealed a stable retro-orbital lesion (Figure 2), and she was started on an increased dose of dexamethasone 4 mg every 6 hours. She continued PO Augmentin for her possible pneumonia until 6/14. She was eventually deemed medically optimized for neurosurgical resection of the orbital mass to relieve compression of the optic nerve and obtain an official pathologic diagnosis.

She underwent craniotomy on 6/17 with successful evacuation of the orbital mass. Frozen pathology was consistent with fungal abscess, specifically *Aspergillus species*. Infectious disease was consulted, and she was subsequently started on IV voriconazole and liposomal amphotericin B 7mg/kg/day. She was transitioned to PO voriconazole 200 mg BID following improvement of angioinvasive aspergillosis. Amphotericin B was discontinued on 6/20 once the final sputum and tissue culture grew *Aspergillus fumigatus*. Prior AFB stain, Quantiferon-TB, and Interferon-gamma TB resulted negative.

She was discharged home on 7/12/22 after spending nearly 12 days in the medical rehab unit. For her disseminated aspergillus infection, she continued at least 4 months of voriconazole per ID recommendations.

Fortunately, she fared well. Repeat MRI brain on 7/26/22 showed near complete removal of the previous left orbital apex mass, for which she did not require further intervention, but continued surveillance.

Discussion

The use of extended and high-dose corticosteroid therapy is a known risk factor that predisposes a patient to developing life-threatening invasive aspergillosis.[6,7] Steroids act by promoting the growth of *Aspergillus* and inhibiting the non-oxidative mechanisms by which macrophages work to kill *Aspergillus species*.[6,7]

Our case is remarkable because this patient was relatively immunocompetent despite her history of uncontrolled diabetes. She did not have classic risk factors for immunosuppression (i.e., recipient of stem cell or solid organ transplant, hematologic malignancy, chemotherapy, advanced AIDS, COPD/chronic lung disease, etc.) that would explain the development of this progressive disease.[6] She was empirically started on systemic steroids on 5/9/22

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following a presumed clinical diagnosis of giant cell arteritis. She had been on steroids up until the day of her craniotomy on 6/17/22. However, her use of systemic steroids without any known chronic lung condition did not seem to have a direct causal relationship to her development of invasive aspergillosis, as symptom onset occurred before the start of steroid therapy. Furthermore, on 5/20/22, just days after starting steroids, she had an MRI brain that already demonstrated a 2.1 cm left orbital apex mass.

Although there is no evidence that our patient's IPA was precipitated by corticosteroid treatment, there is a high likelihood that the use of a short course of systemic steroids did exacerbate her symptoms. After all, she was re-admitted for intractable headaches associated with new left-sided ptosis and dilated left pupil within one week of being discharged with a Medrol Dosepak. Additionally, her pulmonary findings did not appear to resolve on systemic steroids as evidenced by a cavitary lesion with bilateral nodules on CTA from 6/5/22 (Figure 1), which were relatively new compared to CT torso from 5/23/22.

Given that her aspergillus infection progressed rapidly after corticosteroid initiation, and considering the low likelihood that our patient had a preexisting chronic pulmonary disease, we searched the literature for documented cases in which a short duration of corticosteroid therapy contributed to the risk for IPA. For instance, an elderly woman succumbed to IPA after receiving a short course of steroid therapy for acute bronchitis and asthma exacerbation.[6] In addition, there was another elderly woman with no reported past medical history who was found to have invasive aspergillosis involving the lungs and brain after a short 2-month duration of intra-articular corticosteroid injection for joint pain.[8]

Conclusion

The patient was not severely immunocompromised and seemingly did not have classic risk factors for developing this life-threatening disease. The index of clinical suspicion for invasive aspergillosis was therefore low, resulting in an unfortunate delay in diagnosis and treatment initiation. She exemplified an unforeseen presentation of an uncommon infectious disease, which highlights the importance of maintaining these lesser-known diseases in our differential. It is important to consider that IPA is no longer a disease isolated to immunocompromised patients. This case serves as a lesson to consider aspergillosis if a relatively immunocompetent patient presents with refractory symptoms in the absence of classic risk factors or while on a short course of systemic corticosteroid therapy.

Endnotes

1. Danion, François, Claire Rouzaud, Amélie Duréault, Sylvain Poirée, Marie-Elisabeth Bougnoux, Alexandre Alanio, Fanny Lanternier, and Olivier Lortholary. "Why are so many cases of invasive aspergillosis missed?." Medical mycology 57, no. Supplement_2 (2019): S94-S103.
2. Bongomin, Felix, Sara Gago, Rita O. Oladele, and David W. Denning. "Global and multi-national prevalence of fungal diseases—estimate precision." *Journal of fungi* 3, no. 4 (2017): 57.
3. Centers for Disease Control and Prevention. Data and Statistics on Aspergillosis. Available: https://www.cdc.gov/aspergillosis/statistics/?CDC_AAref_Val=https://www.cdc.gov/fungal/diseases/aspergillosis/statistics.html [Accessed 20 June, 2024].
4. Denning, David W., Alex Pleuvry, and Donald C. Cole. "Global burden of allergic bronchopulmonary aspergillosis with asthma and its complication chronic pulmonary aspergillosis in adults." *Medical mycology* 51, no. 4 (2013): 361-370.
5. Rees, Judy R., Robert W. Pinner, Rana A. Hajjeh, Mary E. Brandt, and Arthur L. Reingold. "The epidemiological features of invasive mycotic infections in the San Francisco Bay area, 1992–1993: results of population-based laboratory active surveillance." *Clinical Infectious Diseases* 27, no. 5 (1998): 1138-1147.
6. Naaraayan, Ashutossh, Ronak Kavian, Jeffrey Lederman, Prasanta Basak, and Stephen Jesmajian. "Invasive pulmonary aspergillosis–case report and review of literature." *Journal of Community Hospital Internal Medicine Perspectives* 5, no. 1 (2015): 26322.
7. Lewis, Russell E., and Dimitrios P. Kontoyiannis. "Invasive aspergillosis in glucocorticoid-treated patients." *Medical mycology* 47, no. Supplement_1 (2009): S271-S281.
8. Choi, Young Rak, Jeong Tae Kim, Jeong Eun Kim, Heo Won Jung, Kang Hyeon Choe, Ki Man Lee, and Jin Young An. "Invasive aspergillosis involving the lungs and brain after short period of steroid injection: A case report." *Tuberculosis and respiratory diseases* 72, no. 5 (2012): 448-451.

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Could Dialysis Cause Koebner Phenomenon and Pyoderma Gangrenosum?

By Vivian Li, Alexander Reals, Chinonso Ndubuisi, MD and Joseph Canzoneri, DPM

Abstract

Pyoderma gangrenosum (PG) is a rare neutrophilic dermatosis, with incidence estimated to be 3 to 10 cases per million people per year.[9] Although some correlation between dialysis, renal failure, and PG has been suggested in literature internationally, the topic has not been fully explored.

This report highlights the diagnostic and therapeutic challenges posed by new onset PG in the context of renal failure and dialysis, emphasizing the importance of early recognition, and individually-tailored management strategies. Our findings aim to shed light on the likely link between PG and dialysis, emphasizing a need for further research to optimize care in such patients.

Introduction

Pyoderma gangrenosum (PG) is a rare neutrophilic dermatosis of noninfectious origin. PG can affect all areas of the skin, including mucous membranes.[12,13] The most commonly affected location is the lower extremities, particularly occurring in the pretibial area.[3] Clinical presentation is characterized by recurrent cutaneous ulcerations with mucopurulent or hemorrhagic exudates. These painful ulcers can present with undermined violaceous borders with surrounding erythema.[12,13]

In many cases, PG is associated with an underlying condition, most commonly inflammatory bowel disease, rheumatologic disease, hematologic disease, and malignancy.[12,13] PG has been demonstrated in patients with psoriatic arthritis,[10] systemic lupus erythematosus, anti-phospholipid syndrome,[11] and hypothyroidism.[8] Diagnosis is based on a history of underlying disease, clinical presentation, exclusion of similar diseases, and histopathology samples.[9] In one study of four patients who had undergone biopsies, pathology revealed dense neutrophilic infiltrates in three cases, and leukocytic vasculitis in one.[11]

Accurate epidemiological data are currently lacking. There is an estimated incidence of 3 to 10 cases per million people per year. Persons of any age can be affected; however, the peak incidence is between 20 and 50 years of age, with women being affected more often.[2]

Patients with PG are typically managed with a combination of medications that suppress the inflammatory process, supplemented by consistent wound care to optimize healing. Definitive treatment guidelines for PG are currently unavailable due to insufficient management data. Initial pharmacological therapy for limited PG is a topical corticosteroid.[7] If topical steroids are ineffective, topical tacrolimus, oral dapsone, or oral minocycline may be used. More extensive disease is treated with systemic glucocorticoids or oral cyclosporine. For refractory disease, ultimately, biologic agents may be used.[7] Complete healing with no recurrence is often reported, ranging from one to three months after diagnosis, as demonstrated in one study of multiple PG patients.[11]

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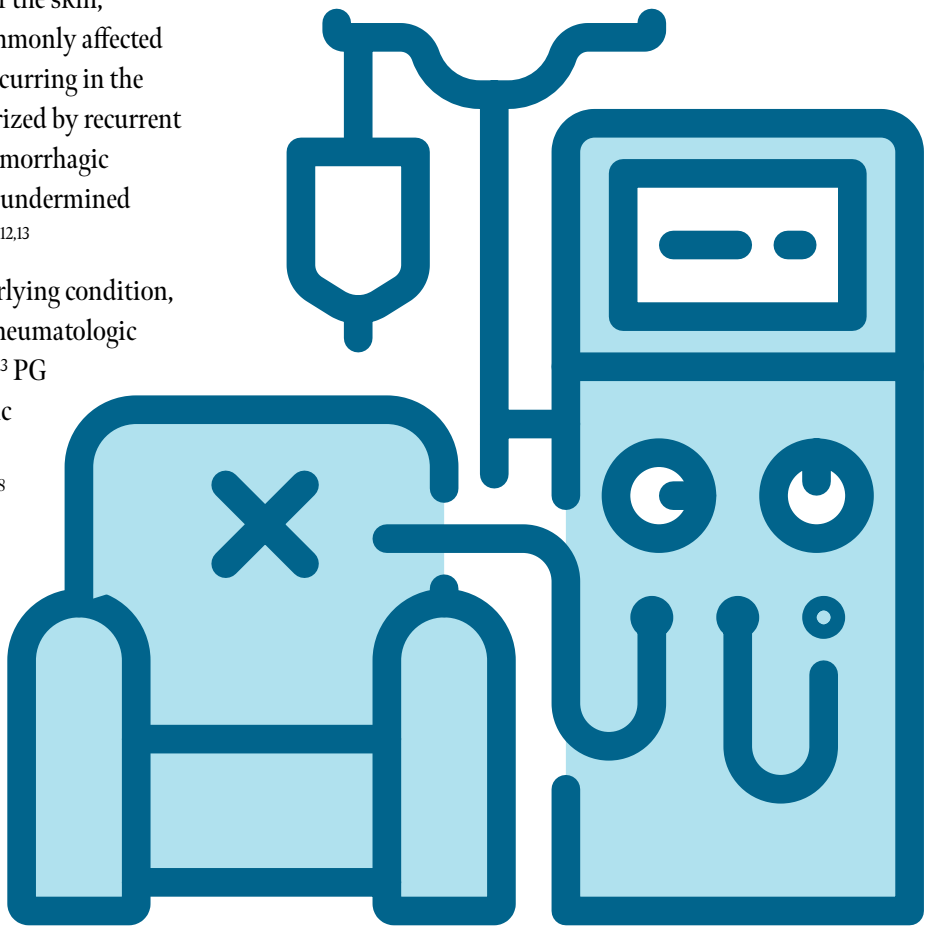

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Case Report

The patient is a 65-year-old male who presented to the wound care clinic in October 2024 with bilateral posterior calf ulcers. Past medical history is significant for end-stage renal failure with a glomerular filtration rate (GFR) low of 7 mL/min/1.73 m^2 in June 2024 (normal: >60 mL/min/1.73 m^2), at which point intermittent hemodialysis was initiated. A month later, he reported bilateral calf ulcers to his primary care physician, attributing their presence to pressure from sitting in the dialysis chair. Other significant medical history includes type 2 diabetes mellitus without long-term use of insulin, benign prostatic hyperplasia with incomplete bladder emptying, heart failure with reduced ejection fraction of 30%, chronic venous insufficiency, and peripheral artery disease.

Upon physical exam at his initial wound care visit, he had grade 2 Wagner ulcerations on his bilateral posterior calves (Table 1). The right posterior calf ulcer was measured to be 3.2 cm length x 4.4 cm width x 0.1 cm depth, and the left posterior calf ulcer was measured to be 2 cm x 2 cm w x 0.1 cm. Following the standard wound care treatment of debridement and daily Santel (collagenase ointment) for sloughing of necrotic tissue for four total once weekly visits, both ulcers began to worsen significantly. Koebner phenomenon, as demonstrated by the patient, is the process in which wound care treatment exacerbates a wound due to the autoimmune inflammatory nature of the underlying disease process.[1] At his fourth visit in December, the right ulcer measured 4.5 cm x 4 cm x 0.4 cm, and the left ulcer measured 6.5 cm x 5 cm x 0.1 cm. The patient was in remarkable pain, with both ulcer sites extremely tender to touch. Both ulcers demonstrated necrotic violaceous edges. A 5 mm punch biopsy was performed on the left posterior ulcer, and the results stated the presence of "fibroadipose tissue with gangrenous necrosis, abscesses and granulation tissue formation." Clinical presentation, along with the biopsy results, revealed the likely diagnosis of pyoderma gangrenosum.

The initial treatment plan involved steroid titration: oral prednisone 10 mg 1 pill daily for 3 days, then 1 pill three times a day for 3 days, titrating up to 2 pills three times a day for 3 days for a total of 60 mg a day. The patient was also started on prophylactic oral doxycycline 50 mg twice a day for 10 days. The expected outcome was marked improvement in bilateral ulcerations with completion of therapy.

At the two- week follow up visit in December 2024, physical exam revealed visible healing of both ulcers. He reported significantly less pain at both ulcer sites, and he was able to tolerate treatment well. The right ulcer measured 4.1 cm x 3.2 cm x 0.3 cm, and the left ulcer measured 7.5 cm x 5.3 cm x 0.3 cm. The left lesion appeared to be larger due to the sloughing of necrotic tissue, though the lesion was visibly more healed than previously observed. Patient continued oral prednisone (steroid), topical Santyl (collagenase ointment), topical lidocaine (anesthetic) for pain, and topical clobetasol (steroid) for peri-wound treatment.

He continued to improve at subsequent visits, reporting less necrotic tissue at both sites, and decreased peri-wound erythema and inflammation. He reported minimal to no pain. He continued to follow-up consistently with once a week wound care visits for debridement and management. Prednisone was titrated up to 40 mg daily.

In early January 2024, the patient presented to the wound care clinic with difficulty breathing. He demonstrated signs of fluid retention, evidenced by increased removal of excess fluid during dialysis, and increased bilateral lower extremity edema. Due to his past medical history of congestive heart failure with ejection fraction of 30%, and concern for worsening heart failure, he was tapered off prednisone. He was started on oral dapsone 50 mg daily, which he tolerated well. After a few weeks, dapsone was then increased to 100 mg daily. At the subsequent follow-up visit, he no longer demonstrated signs of fluid retention, and continued to experience improvement in his bilateral calf ulcers.

Discussion

Pyoderma gangrenosum (PG) can be difficult to diagnose given a typical presentation of a standard ulcer. **Demonstration of Koebner phenomenon is one manner in which an underlying autoimmune cause can be suspected. A biopsy is a non-diagnostic supplement to clinical diagnosis.** Few cases of pyoderma gangrenosum in relation to dialysis and renal failure have been represented in literature.

Previously, a case report published in 2002 in the British Journal of Dermatology suggested a possible correlation between dialysis and the exacerbation of PG. A woman presented with bilateral painful recurrent erythematous leg lesions after undergoing hemodialysis for 10 years, though both lesions were non-ulcerative with no drainage of pus, suggesting a vegetative form of PG.[5]

Another case report published in 2013 in a dermatology journal in Brazil presented a patient with splenic and renal nodules that presented simultaneously as purulent necrotic ulcers on both legs. Though the patient had no history of renal impairment, both splenic and renal nodules resolved with treatment of PG. The

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treatment regimen consisted of corticosteroids, cyclosporine, and sulfasalazine.[4]

A case report published in 2022 in the Canadian Journal of Kidney Health and Disease presented a patient who developed PG at their tunneled catheter site on their arm, with another similar lesion appearing on their abdomen, after initiating maintenance hemodialysis. The patient was treated with IV hydrocortisone, and was later switched to oral prednisone. Ultimately, they were on oral prednisone 50 mg every day, and hydroxychloroquine 200 mg twice a day. Complete healing of both ulcers occurred after 2 months.[1]

Due to the rarity of PG, no standardized treatment plan exists. General classes have been proven effective, such as the use of corticosteroids, though no specific route of administration, nor specific steroid has been shown to be more effective than others. Previous case reports have shown that an individualized approach has been the most beneficial to patients. The correlation between renal disease and PG has also been suggested, though no case reports to our knowledge has demonstrated a patient with history of chronic renal failure developing PG almost immediately after first-time initiation of hemodialysis.

Table 1: Meggitt-Wagner Scale

Score 1	Superficial ulcer
Score 2	Deep ulcer to tendon, bone, or joint
Score 3	Deep ulcer with abscess or osteomyelitis
Score 4	Forefoot gangrene
Score 5	Whole foot gangrene

The Meggitt-Wagner scale, first proposed in 1976, grades ulcers based on the following subjective features: wound depth, location, and the presence of gangrene. The Wagner scale is often utilized due to the simplicity of the scale.[6]

Endnotes

1. Alosaimi, Majed M., et al. "Pyoderma gangrenosum after insertion of a hemodialysis catheter: Koebner phenomenon, systemic inflammatory response syndrome, and a delay in diagnosis." *Canadian Journal of Kidney Health and Disease* 9 (2022): 20543581221120618..
2. Ashchyan, Hovik J., et al. "The association of age with clinical presentation and comorbidities of pyoderma gangrenosum." *JAMA dermatology* 154.4 (2018): 409-413.
3. Bhaskaran, Bindhu, et al. "Pyoderma gangrenosum: a clinician's nightmare." *Journal of Family Medicine and Primary Care* 5.3 (2016): 698-700.
4. Carvalho, Luciana Rabelo de, Virgínia Vinha Zanuncio, and Bernardo Gontijo. "Pyoderma gangrenosum with renal and splenic impairment-case report." *Anais Brasileiros de Dermatologia* 88 (2013): 150-153.
5. Goto, M., et al. "Vegetative pyoderma gangrenosum in chronic renal failure." *British Journal of Dermatology* 146.1 (2002): 141-143.
6. Karthikesalingam, A., et al. "A systematic review of scoring systems for diabetic foot ulcers." *Diabetic Medicine* 27.5 (2010): 544-549.
7. Maronese, Carlo Alberto, et al. "Pyoderma gangrenosum: an updated literature review on established and emerging pharmacological treatments." *American journal of clinical dermatology* 23.5 (2022): 615-634.
8. Mittal, Shashi, and Valerie Graham. "Skin lesions mimicking septic arthritis." *Journal of family practice* 55.10 (2006): 881-885.
9. Neil Crowson, A., Martin C. Mihm Jr, and Cynthia Magro. "Pyoderma gangrenosum: a review." *Journal of cutaneous pathology* 30.2 (2003): 97-107.
10. Spangler, John G. "Pyoderma gangrenosum in a patient with psoriatic arthritis." *The Journal of the American Board of Family Practice* 14.6 (2001): 466-469.
11. Tay, Daniel Zunsheng, Ki-Wei Tan, and Yong-Kwang Tay. "Pyoderma gangrenosum: a commonly overlooked ulcerative condition." *Journal of family medicine and primary care* 3.4 (2014): 374-378.
12. Wang, Feixia, et al. "Clinical Characteristics, Treatment, and Wound Management of Pyoderma Gangrenosum: A Case Series." *PloS One*, vol. 20, no. 6, June 2025, p. e0326203. *EBSCOhost*, https://doi-org.lecom.idm.oclc.org/10.1371/journal.pone.0326203.
13. Wollina, Uwe. "Pyoderma gangrenosum–a review." *Orphanet journal of rare diseases* 2 (2007): 1-8.

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