Family Doctor

A Journal of the New York State Academy of Family Physicians

Spring 2022

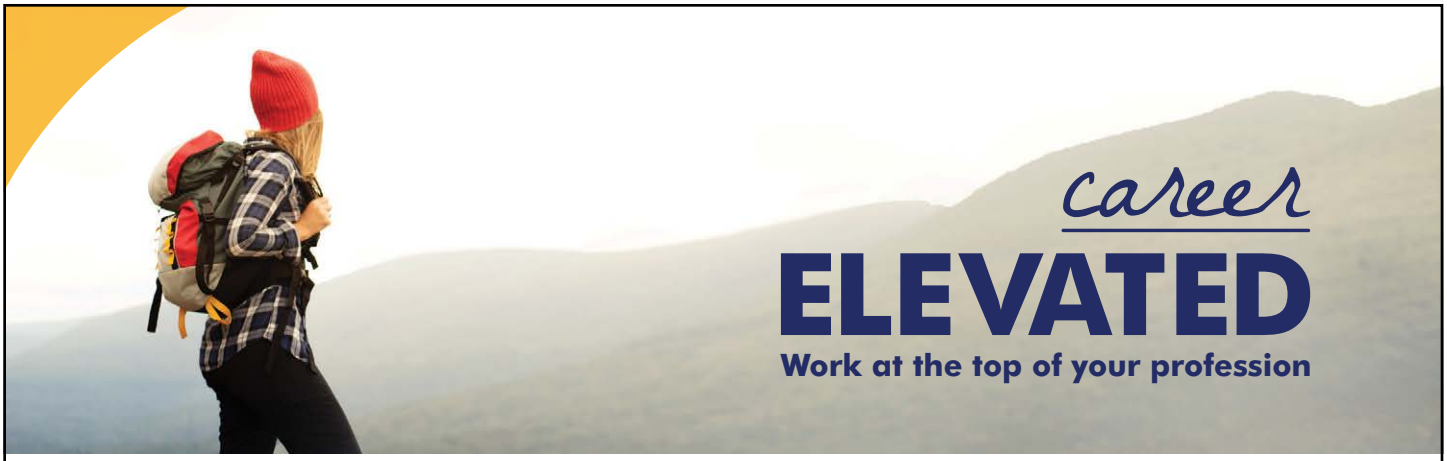
Volume ten, Number four

Focus:

Case Reports in Family Medicine

FEATURE ARTICLES:

New York State Academy of Family Physicians



GLENS FALLS HOSPITAL SEEKS PHYSICIANS TO JOIN OUR TEAM AT CAMBRIDGE MEDICAL CENTER

Practice Primary Care in Beautiful Cambridge, New York!

THE OPPORTUNITY

- Join an Albany Med Health System affiliate, ANCC Magnet®-recognized, 391-bed community hospital with a well-established practice in the Glens Falls Hospital Employed Provider Network.
- Full-time position, providing support to Primary and Urgent Care teams, flexible schedule options available.
- Attractive compensation package, benefits, sign-on bonus and **LOAN FORGIVENESS**.
- Strong nursing and administrative support.
- On-site medical imaging and point-of-care lab testing.
- Participate in telephone-only call schedule, approximately four weeks per year.

WANT TO KNOW MORE ABOUT THE REGION?

Cambridge, New York, situated between Saratoga Springs and Manchester, Vermont, is a safe, family-focused community with affordable housing and excellent schools. Cambridge is home to its own food co-op, an independent bookstore, a variety of restaurants and an abundance of arts and cultural experiences. Southern Vermont ski resorts, the Berkshires and the Adirondacks are all just a short drive away with hiking, camping, boating, skiing and golfing at your fingertips. Located within three hours from NYC, Boston and Montreal, and less than 1 hour from Albany International Airport, Cambridge is the perfect jumping-off point for you and your career.

FOR MORE INFORMATION CONTACT:

Antoinetta M. Backus, CPRP, Recruitment Manager
abackus@glensfallshosp.org | 518.926.6928
GlensFallsHospital.org/ProviderRecruitment

Glens Falls Hospital

ALBANY MED Health System

MAGNET RECOGNIZED
AMERICAN NURSES CREDENTIALING CENTER

AMERICA'S BEST-IN-STATE EMPLOYERS
Forbes 2021
POWERED BY STATISTA

From one essential worker to another,
we appreciate all that you're doing on the front line.

StrongerTogether

YOUR MILK COMES FROM A GOOD PLACE

AMERICAN DAIRY ASSOCIATION NORTH EAST

AmericanDairy.com

Family Doctor, A Journal of the New York State Academy of Family Physicians, is published quarterly. It is free to members of the New York State Academy and is distributed by mail and email. Non-member subscriptions are available for $40 per year; single issues for $20 each.

New York State Academy of Family Physicians
16 Sage Estate, Suite 202
Albany, New York 12204
www.nysafp.org
Phone: 518-489-8945
Fax: 518-888-7648

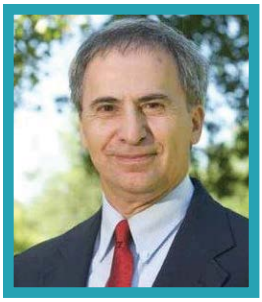
Letters to the Editor, comments or articles can be submitted by mail, fax or email to journaleditor@nysafp.org

Editor: Penny Ruhm, MS

Articles

Departments

Index of Advertisers



From the Executive Vice President

By Vito Grasso, MPA, CAE

The February 18-20 Ten-State Conference in Milwaukee was my first in-person Academy meeting since COVID, and pandemic related restrictions forced cancellation of live events. It was good to see people again and to renew friendships, although it took a while to get back into travel mode. Making my travel and lodging reservations used to be second nature but lapse of connection with those behaviors was evident as I stumbled through the online processes of booking flights, arranging ground transportation and making hotel reservations.

Much of the discussion at Ten-State was about the impact of COVID and the lessons learned. Additionally, we addressed DEI, scope of practice, telemedicine and the changing demographics of Academy membership. We also heard from participating chapters about current policy issues in their states. As is usually the case, many of those issues are common among Ten-State chapters and probably among Academy chapters generally.

Presentations on DEI developments within the Academy and among chapters were illuminating and stimulated constructive discussion. The AAFP has produced many educational programs, facilitated numerous discussions about racism and bias in health care and medical education, and accumulated a significant inventory of educational and training materials. Chapters, also, have been very active and reported progress in generating awareness and ongoing attention to the presence and impact of bias in the Academy. Much of the discussion focused on efforts to assure that the Academy and chapters accommodate advancement of minority members. Some chapters have been challenged in this area. In NY, where we have a diverse population, many medical schools, a plethora of residency programs and a high concentration of IMGs among residents and active members, there has been a larger pool of members from various minorities to engage with. Students and residents are generally interested in social justice and more willing to be active advocates for reform. Also, our Congress of Delegates (COD) affords a structure within which to pursue action on issues our members are passionate about. Our support of members who participate in our COD, through training in resolution writing and parliamentary procedure, elevates and expedites their preparation for leadership positions and cultivates their interest in the Academy as a vehicle for both their own leadership development and for advancement of a social justice agenda.

Discussion of legislation which chapters are dealing with surfaced some familiar topics. COVID pushed many familiar issues to the background as state legislatures and governors were consumed with emergency procedures and constituent anxiety. A number of chapters reported that they are once again dealing with legislation to expand the scope of practice of nurse practitioners, efforts to expand investment in primary care and relief of the administrative burden imposed on physicians by our multiple payer system.

In NY, the Nurse Practice Act, which provides for independent practice for nurse practitioners under certain circumstances, expires this year. Legislation has been introduced to make it permanent. It seems the arguments against such policy by the medical profession have not been persuasive. Indeed, proponents of independent practice and legislators who are undecided ask whether there is anything new to offer to rebut the idea that allowing independent practice would promote access to primary care, contain costs and satisfy consumer demand for more time and attention from clinical professionals. There does not appear to be evidence to suggest that independent practice is either a good idea or a bad one. Old arguments against it simply suggest that physicians have more training and are, therefore, better equipped to make an informed diagnosis and treatment plan. As more states, including NY, have allowed independent practice in varying degrees it may be time to come up with a different strategy. We have generally supported policy that is evidence-based. Perhaps we should apply that standard to scope of practice expansion. If NY should extend the Nurse Practice Act, we should insist that it include specific procedures to measure and evaluate the impact of independent practice on costs, access and consumer satisfaction. If the evidence materializes that independent practice contributes positively to achieving these public benefits, then the medical profession should concede the point and accept independent practice. If the evidence is not there, however, then the law should expire, and physician supervision should be restored.

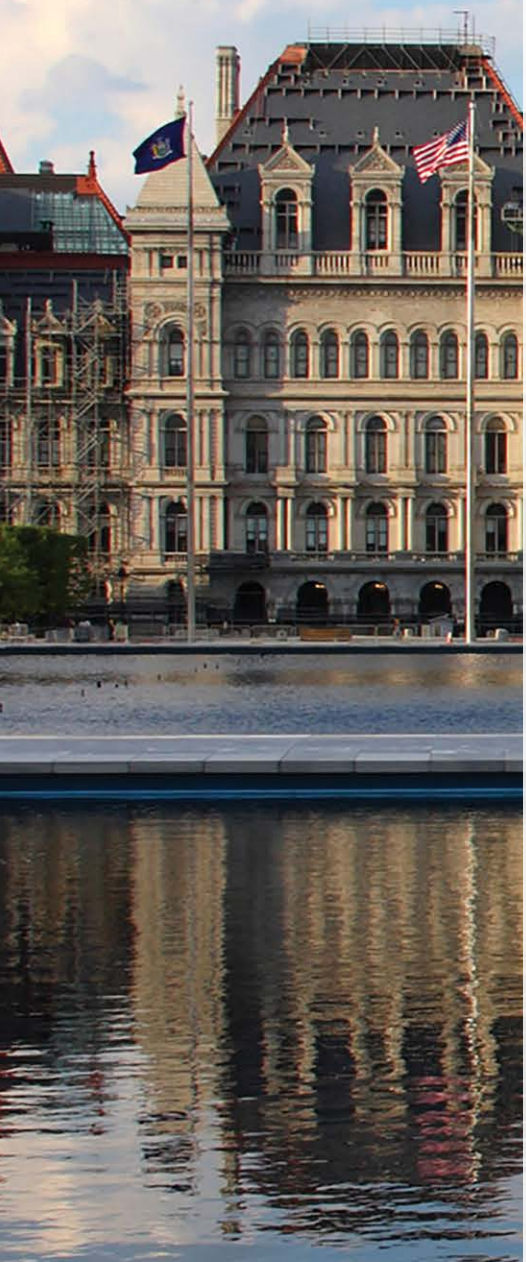
Investment in primary care is another hot topic which multiple states are addressing. The absence of a generally accepted definition of primary care has undermined the effectiveness of efforts to increase investment in primary care where such efforts have produced legislation or regulations to require specific levels of investment in primary care. It appears that most initiatives have focused on identifying a certain level of primary care spending as optimal and then requiring plans to reach that level of spending. Perhaps it would be more effective to require plans to offer separate coverage for primary care and for all other services. This would force plans to clearly identify specific primary care services and costs. It would also isolate spending on primary care in a manner that would accommodate better analysis of the impact of primary care spending on overall costs and clinical outcomes. Such an approach could also present a vehicle for applying regulation more effectively to support primary care.

Physician burn-out was an underlying concern with many issues discussed at Ten-State and there was much interest in efforts to reduce administrative burden. Several chapters, including NY, are supporting legislation to standardize many of the most common and most onerous administrative functions imposed upon physicians by plans.

Ten-State was, indeed, a refreshing opportunity to renew acquaintances and recover to the extent possible from the ravages of COVID. It was good to see old friends and have the social and professional engagement associated with live events. There were, of course, masking and social distancing requirements during travel and the conference but everyone seemed unburdened by the continued deference to safety as we make our way back. The substance of Ten-State, however, made it clear that as we restore some semblance of normalcy in our lives it is likely we will continue to encounter familiar unresolved issues and challenges in our advocacy for family medicine and the interests of patients.

Albany Report

By Reid, McNally & Savage



On January 5, 2022, the 2022 State Legislative Session began with Governor Hochul's first state of the state address and both the Senate and Assembly gaveling into the new session. Two weeks later on January 18th, Governor Kathy Hochul released her 2022-23 proposed executive budget totaling over $216 billion, which is $4 billion more than the final state budget last year. The plan includes enormous investments in health care, education, and NYS infrastructure.

Budget negotiations dominated discussions in Albany as the Senate and Assembly work to develop their "one house" budget proposals that were released in mid-March, which represent their response to the Governor's budget as well as their own budget priorities for the year. Following the release of the one-house budgets, the Governor, Senate, and Assembly commenced "three-way" negotiations on a final state budget to be passed by April 1, the deadline to begin the new fiscal year.

Leaders from NYSAFP including President Dr. Mumford, President-elect Dr. Symons, Advocacy Chair Dr. Menendez, EVP Vito Grasso and Reid, McNally & Savage had a busy fall of advocacy activities leading up to the 2022 session. This included a meeting with the new Governor's top health staff to introduce them to NYSAFP and discuss our top priorities including single payer, support for primary care funding investments and Doctors Across NY, vaccine promotion including the adult vaccine reporting bill, expanded access to reproductive healthcare and our recommendations for improved communications and pandemic planning with the State's physician community. In addition, we had meetings with single payer bill sponsor Assembly member Gottfried to discuss ways that NYSAFP could further assist with the bill's advancement and address some union opposition. Assembly member Gottfried recently announced his planned retirement at the end of 2022, currently the longest seated legislator in NY history.

Finally, we had meetings with the Office of Cannabis Management and other policy makers to discuss NYSAFP's white paper on the new adult use law, spearheaded by Dr. William Klepack, and we have been involved in various discussions on potential ways that New York physicians can support patients in other states with highly restrictive abortion policies, as facilitated by Dr. Linda Prine.

After a discussion on the very successful Advocacy Day held by NYSAFP on February 28th, we have included a detailed summary of the proposals currently in play and under consideration from the Governor's budget released in late January.

2022 NYSAFP Advocacy Day

The Academy held its Advocacy Day on Monday, February 28th and it was fully virtual again this year as the Legislative Office Building was still closed to the public. During the day, NYSAFP members advocated on state budget issues and key legislative priorities. Reid, McNally & Savage set up Zoom meetings with key legislators and staff with over fifty physicians, residents and students participating. There were 10 teams of meetings taking place simultaneously throughout the day.

Below are the leading NYSAFP priority issues that Advocacy Day participants discussed with their lawmakers.

Budget Priorities:

- Support for Medicaid primary care investment funding proposal.
- Support increased funding for Doctors Across NY program totaling $15.8 million in the executive budget.
- Request $2.2 million for Area Health Education Centers (AHEC) be added to the final budget.

- Support 1% Medicaid rate increase proposal in the executive budget, and restoration of prior year across the board Medicaid cuts.
- Support telehealth parity in the executive budget, which is critical for primary care.
- Oppose proposal to remove collaborative agreement requirements between nurse practitioners and physicians in primary care in the executive budget.
- Oppose restructuring of Physician Excess Medical Malpractice Insurance program in the executive budget.

Legislative Priorities:

- Support New York Health Act for universal coverage for all including ability of physicians to collectively bargain with the single payer (S5474, Rivera/ A6058, Gottfried).
- Support legislation to expand reporting of adult vaccines to the NYS/NYC immunization registries, similar to child vaccines (S75A, Hoylman/A279A, Gottfried).
- Support initiatives to provide administrative simplification/ streamlining of insurance plan policies and procedures and a requirement for public hearings around insurer premium increases. There is no legislation yet. Members educated legislators about NYSAFP's concept paper with these recommendations.

Executive Budget Proposals of Interest to NYSAFP

Physician Excess Medical Malpractice Program

The Governor's budget proposes to extend the program through June 30, 2023. It also proposes to restructure payments from one per year to two over two fiscal years if funds in the pool are sufficient and pursuant to eligibility requirements, beginning 7/1/22.

Primary Care Investment

The executive budget includes a proposal promoting access to primary care by increasing Medicaid fees for evaluation and management (E&M) codes for primary care to 70% of Medicare. The Governor's budget (per the Medicaid budget scorecard) provides $4.93 million for SFY23 and $6.6 million for SFY24 for this purpose.

Doctors Across New York

The Governor's budget increases funding for the program to $15,865,000, an increase from $7,252,000 in funding in the final state budget for SFY 2021-22.

Nurse Practitioner (NP) Independent Practice in Primary Care

The Governor's budget proposes to remove the requirement for NPs in primary care with over 3600- hours experience to maintain a "collaborative relationship" with physicians. Makes the program permanent by removing the expiration currently in statute.

Medication Administration and Non-Patient Specific Orders for COVID-19, Flu, and Upper Respiratory Testing

The Governor's budget proposes changes in laws to allow certified medication aides to administer routine medications under certain conditions and in certain institutional settings; allows physicians and NPs to issue non-patient specific standing orders for COVID-19, flu and upper respiratory illness testing, and allows registered professional nurses to collect specimens from patients for COVID-19 or flu testing with training and under the supervision of a registered nurse, NP or physician.

Interstate Medical Licensure Compact

The Governor's budget proposes that New York State will join the interstate medical licensure compact and interstate nursing licensure compact with other states in the compact for a streamlined licensure process with one application.

Medicaid Prescriber Prevails Changes

The Governor's budget proposes to eliminate prescriber prevails under FFS and MMC except for 9 classes of drugs currently allowed under MMC.

Restoration of Across-the-Board Medicaid Cuts & 1% Medicaid Rate Increase

The Governor's budget proposes to restore the 1.5% across the board Medicaid cut that was enacted in the 2021 state budget for all providers. In addition, the Governor proposes to provide an increase in Medicaid rates of an additional 1 percent for a two-year investment of $3.7 billion.

Telehealth Parity

The Governor's budget would establish reimbursement parity for telehealth services by requiring health plans, including those in Medicaid, to reimburse providers for services delivered through telehealth on the same basis, and at the same rate, as services delivered in person. The proposal states that telehealth shall not require reimbursement to a provider for certain costs including facility fees, costs reimbursed through APGs or other clinic reimbursement methodologies under Article 28 if such costs were not incurred in the provision of telehealth services due to neither the originating site of the patient/client, nor the distant site of the provider occurring within a facility or other clinic setting.

Health Care/Mental Health Workforce Bonuses

The Governor's budget includes funding for a one-time bonus of $3000 for workers who make up to $125,000 annually; employers with at least 20% of revenue derived from Medicaid, or subject to the certificate of need process are eligible to receive monies for bonus payments. It imposes obligations on employers and penalties for failure to identify, claim and pay a bonus for each employee. The OMIG is responsible for audits of payments and actions by employers. ARP and HCBS monies are referenced as sources. Income tax would not be applied.

Expanded Access to Naloxone/MAT

The Governor's budget would require pharmacies to stock a 30-day supply of naloxone and MAT/ buprenorphine, in accordance with DEA rules and wholesaler thresholds.

continued on page 8

continued from page 7

New York State Department of Health Oversight of Certain Professions

The Governor's budget proposes to move oversight of the professions, including physician licensure, from the New York State education law to the New York State public health law to provide for oversight and regulation of these professions by the New York State Department of Health instead of the State Education Department.

Third Trimester Syphilis Screenings

The Governor's budget proposes to amend public health law to require syphilis testing of pregnant patients by a physician or other authorized practitioner in the third trimester consistent with any guidance and regulations issued by the Department of Health.

Maternal Health Coverage

The Governor's budget proposes to require commercial coverage of maternal health care including postpartum coverage up to one year after birth under the Essential Plan or Medicaid.

Reproductive Health Protections

The Governor's budget would require every individual accident and health insurance policy that provides medical, major-medical, or similar comprehensive-type coverage in New York to provide coverage for abortions. Further, the bill would require that the coverage not be subject to copayments, coinsurance, or annual deductibles unless the policy is a high-deductible plan.

Essential Plan Expansion

The Governor's budget proposes to increase eligibility of the essential plan from 200% up to 250% of FPL subject to federal approval.

Child Health Plus Changes

The Governor's budget proposes to expand coverage to align with Medicaid coverage; eliminates the $9 monthly premium for eligible children whose family earns up to 223% of FPL; moves rate setting to DOH and allows DOH to modify rates in conjunction with the Department of Financial Service (DFS).

A reminder that these are proposals from the Governor's budget currently being negotiated with the Legislature. Some will be altered or not included in the final state budget due April 1, 2022. We will send further updated information out on the provisions of interest in the final budget.

For a comprehensive sector-by-sector summary of health/ mental health hygiene budget proposals, contact our journal editor at penny@nysafp.org.

58-Year-Old Female with Severe Hyponatremia and Prolonged Altered Mental Status

By Iziegbe Fenemigho, MD; Oluwatoyin Moyebi, MD, MPH; Amit Sharma, MD and Miller Laurence, MD

Abstract

Hyponatremia (serum sodium of <135mmol/L) is the most frequently encountered electrolyte abnormality in both outpatient and inpatient settings. Symptoms are very non-specific and can be as mild as prolonged fatigue, nausea, or headaches, or present with more severe symptoms such as altered mental status (AMS), seizures, psychosis and even death. We discuss a 58-year-old female with severe hyponatremia who presented with AMS. We review causes of hyponatremia including SIADH and provide algorithms for evaluation and treatment.

Evaluation and management of hyponatremia is often poorly understood and undertreated. Given its potentially dreadful manifestations, family physicians should be comfortable managing hyponatremic patients by thorough clinical evaluation, appropriate follow-up and medication adjustments if necessary.

Introduction

About 21% of patients have hyponatremia on initial presentation to ambulatory care[1,2] with an estimated 42.6% of hospitalized patients with hyponatremia at any given time,[3] and hyponatremia is directly proportional to mortality rate.[4] Its overall prevalence in the US ranges from 3.2 to 6.1 million persons with an estimated cost of management between $1.6 to $3.6 billion yearly.[5]

Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH) is the most common cause of hyponatremia[6], an often-dreaded manifestation of a wide variety of diseases and medication side effects.[7]

Case Report

A 58-year-old Hispanic woman with past medical history of hypertension, prediabetes, hypothyroidism, depression, and asthma presents to the emergency department with hiccups, vomiting, and diarrhea which started a few hours prior to presentation. Patient reported a prolonged feeling of unwellness before the sudden onset of symptoms. Vital signs were significant for mild hypertension of 154/93mmHg, otherwise normal. Examination findings were normal. Routine laboratory tests at that time showed a sodium of 126mEq/L (reference range 135-145 mEq/L), chloride of 87mEq/L (reference range 98-108 mEq/L), and glucose of 129mg/dl (reference range 70-120 mg/dl).

Pertinent history was significant for recent colonoscopy 2 days prior, and thiazide diuretics for hypertension. She was not on SSRIs and had no history of excessive water intake.

Patient was admitted due to persistent hyponatremia. Thiazide diuretic was discontinued and she was managed with normal saline infusion. Antibiotics were given following an assessment of probable gastroenteritis from recent colonoscopy and mildly elevated white cell count.

The patient's symptoms resolved and sodium improved to 132mEq/L. She was discharged home after 2 days of admission to discontinue thiazides and with a close follow-up appointment. However, two weeks later, the patient presented to the emergency room with marked altered mental status - not oriented to time, place or person. No history could be obtained except from medical records. Vital signs were significant only for elevated blood pressure of 182/81mmHg. On examination, there were no signs of dehydration, no signs of head trauma, pupils were equal and reactive to light. Other examination findings were normal.

Laboratory findings were significant for severe hyponatremia of 114mEq/L. Delirium work-up, TSH/T3/T4, blood sugar, urine analysis, and drug screen were normal. EKG was normal, as well as CT of the head. The patient was started on saline infusion and was admitted for further management. On the floors, serum osmolality was noted to be 259mOsm/L (reference range: 275-295mOsm/L) with urine sodium of 39mEq/L (reference range 25-150 mEq/L)

By the second day of admission, mentation improved and she was oriented to person and place. Urine osmolality noted to be 158mOsm/kg. Serum sodium improved to 122mEq/L. A working differential of adrenal insufficiency, SIADH, drug induced hyponatremia or hypothyroidism was made. Fluid restriction was started. T3, T4 and TSH results came back normal, serum cortisol and AM cortisol were normal at 18.7ug/dl and 18.4ug/dl respectively (reference range- T3: 60-181 ng/dl; T4: 0.80-2.00 ng/dl; TSH: 0.40-4.50 mIU/L; cortisol serum: 5.0-25.0 ug/dl).

By fourth day on admission, urine osmolality improved to 287mOsm/kg, serum sodium improved to 128mEq/L and urine sodium 70mEq/L. Our patient was now fully oriented and denied any use of psych medications since 2018. She was given 1 gram of sodium chloride tablet. A final diagnosis of SIADH was made.

By the next day, serum sodium improved to 134mEq/L and the patient was discharged home with close out patient PCP and endocrinology follow up appointments.

continued on page 10

continued from page 9

Discussion

Hyponatremia is divided into mild (130-134mEq/L), moderate (125-129mEq/L), and severe (<125mEq/L), however, the rate of reduction of serum sodium may be more predictive of severity of symptoms.[8]

Evaluation starts from a thorough history and physical exam. It is important to ask about medication history, medical conditions, social history, and to check for level of dehydration. Medications like SSRIs, diuretics and carbamazepine can cause hyponatremia, as well as the use of illicit drugs and alcoholism.[9,10,11]

Laboratory tests include complete metabolic panel, creatinine, serum osmolality, urine osmolality, urine sodium, thyroid panel, plasma cortisol and adrenocorticotropic hormone.[9,10]

Based on hydration status, hyponatremia can be classified into hypovolemic, euvolemic and hypervolemic. Hypovolemic hyponatremia results from acute volume loss such as diarrhea and vomiting. Management involves volume repletion with normal saline and salt tablets.[9,11] The most common cause of euvolemic hyponatremia is SIADH, however, it is also important to rule out less common causes such as glucocorticoid deficiency and hypothyroidism.[12] Hypervolemic hyponatremia is caused by volume overload states such as in liver cirrhosis, heart failure and kidney failure. Treatment involves fluid restriction and careful diuresis.[11,12]

Often times, discontinuing the offending medication or treating the underlying cause may improve sodium levels, however, most causes of hyponatremia result from inappropriately high levels of antidiuretic hormone (ADH).[13]

The relatively new medication class, vasopressin receptor antagonists (vaptans), can be used in management of SIADH, especially in cases that have failed to resolve after the first 24hrs of fluid restriction. It is available in both parenteral and oral medications for acute and chronic management of SIADH. Starting with a low dose it is titrated upwards based on the patient's sodium levels. This is particularly helpful for patients who have had persistent or recurrent hyponatremia.[14] When using vaptans, physicians need to carefully monitor serum sodium to avoid the risk of overcorrection. Vaptans should not be used in hypovolemic hyponatremia or hyponatremia due to excessive fluid intake or other known causes such as renal failure.[15]

Severe symptomatic hyponatremia requires gentle correction with 3% saline solution. Sodium correction must not exceed 10mEq/L over a 24-hour period to avoid central pontine myelinolysis. It is appropriate to have a correction goal of about 6mEq/L over 24 hrs.[16]

Regardless of etiology, the goals of treating hyponatremia are to prevent further reduction in sodium levels, improve symptoms, and to avoid iatrogenic brain injury from overcorrection.[16]

Conclusion

Family physicians should be comfortable with the evaluation and treatment of patients who present with hyponatremia in both inpatient and outpatient settings. The new medication class, vaptans, is a good option to manage patients at clinic. Appropriate short-term follow up is more cost effective than presenting to the emergency room with severe symptoms, thereby reducing the overall burden of hyponatremia.

Endnotes

1. Boscoe A, Paramore C, Verbalis JG. Cost of illness of hyponatremia in the United States. *Cost Eff Resour Alloc.* 2006;4:10. Published 2006 May 31. doi:10.1186/1478-7547-4-10
2. Hawkins RC. Age and gender as risk factors for hyponatremia and hypernatremia. Clin Chim Acta. 2003;337:169 –172
3. Upadhyay A, Jaber BL, Madias NE. Incidence and prevalence of hyponatremia. Am J Med. 2006;119(7 suppl 1):S30–S35.
4. Sumit Mohan MD, MPH, Sue Gu MD, MSPH, Amay Parikh MD, MBA, MS and Jai Radhakrishnan MD, MS American Journal of Medicine, The, 2013-12-01, Volume 126, Issue 12, Pages 1127-1137.e1
5. Cost of illness of hyponatremia in the United States. Boscoe A, Paramore C, Verbalis JG *Cost Eff Resour Alloc.* 2006 May 31; 4():10
6. Cuesta M, Thompson CJ. The syndrome of inappropriate antidiuresis (SIAD). Best Pract Res Clin Endocrinol Metab. 2016 Mar;30(2):175-87. doi: 10.1016/j.beem.2016.02.009. Epub 2016 Feb 27. PMID: 27156757.
7. Shchekochikhin DY, Kozlovskaya NL, Kopylov FY, Syrkin AL, Shilov EM. Giponatriemiia: klinicheski podkhod [Hyponatremia: A clinical approach]. Ter Arkh. 2017;89(8):134-140. Russian. doi: 10.17116/terarkh2017898134-140. PMID: 28914864.
8. Robert D. Zenenberg, Alessia L. Carluccio & Mark A. Merlin (2010) Hyponatremia: Evaluation and Management, Hospital Practice, 38:1, 89-96, DOI: 10.3810/ hp.2010.02.283
9. Pfennig CL, et al. Sodium disorders in the emergency department: a review of hyponatremia and hypernatremia. Emerg Med Pract. 2012; 14(10):1–26.
10. Assadi F. Hyponatremia: a problem-solving approach to clinical cases. J Nephrol. 2012;25(4):473–480.
11. Verbalis JG, et al. Diagnosis, evaluation, and treatment of hyponatremia: expert panel recommendations. Am J Med. 2013;126(10 suppl 1):S1–S42.
12. Spasovski G, et al.; Hyponatraemia Guideline Development Group. Clinical practice guideline on diagnosis and treatment of hyponatraemia. Eur J Endocrinol. 2014;170(3):G1–G47.
13. G.L. Robertson. Regulation of arginine vasopressin in the syndrome of inappropriate antidiuresis. Am J Med, 119 (2006), pp. S36-S42
14. Schrier RW, Gross P, Gheorghiade M, Berl T, Verbalis JG, Czerwiec FS, et al. Tolvaptan, a selective oral vasopressin V2-receptor antagonist, for hyponatremia. *N Engl J Med.* 2006 Nov 16. 355(20):2099-112.
15. Joseph G. Verbalis, Steven R. Goldsmith, Arthur Greenberg, Cynthia Korzelius, Robert W. Schrier, Richard H. Sterns, Christopher J. Thompso Diagnosis, Evaluation, and Treatment of Hyponatremia: Expert Panel Recommendations,The American Journal of Medicine, Volume 126, Issue 10, Supplement 1, 2013, Pages S1-S42, ISSN 0002-934
16. Sterns R.H., Silver S.M., Hix J.K. (2013) Treatment of Hyponatremia. In: Simon E. (eds) Hyponatremia. Springer, New York, NY. https://doi-org.eresources.mssm.edu/10.1007/978-1-4614-6645-1_12

Cameron Lesions: Something to Consider When Evaluating Patients with GI Bleed and Anemia

By Daniel E. Isaac, MD; Sherman M. Chamberlain, MD and Rodika Coloka-Kump, DO, FAAFP

Abstract

Cameron lesions are linear erosions or ulcerations of the mucosal folds lining the stomach at the point it is constricted by the thoracic diaphragm in patients with large hiatal hernias. Cameron lesions can cause chronic blood loss resulting in iron deficiency anemia. In more severe cases Cameron lesions can cause acute blood loss. Cameron lesions, although uncommon, may be the cause of anemia or GI bleed in patients with hiatal hernias. These lesions are an incidental finding in 5.2% of patients with hiatal hernias who undergo EGD examinations.

Introduction

Cameron lesions are an unusual cause of upper gastrointestinal bleeding and must be considered in the differential diagnosis of patients with anemia and hiatal hernias. Found in 5.2% of patients with hiatal hernias who undergo esophagogastroduodenoscopy (EGD) examinations, the prevalence of Cameron lesions seems to be dependent on the hernia sac, with an increased prevalence the larger the hernia sac.[1]

Often these lesions are incidentally found, however. If Cameron lesions were in the differential, the physician performing the EGD would be more vigilant during scoping resulting in more of these lesions being discovered on routine EGD. Perhaps, with increasing awareness of these potentially detrimental lesions, the frequency in which they are detected will increase.

Case Report

The patient, a 71- year- old female with a past medical history significant for asthma, hypertension, bilateral cataract surgery, anemia secondary to chronic blood loss and frequent use of naproxen sodium for headaches, presented to the emergency department for dizziness of 4 days' duration. The patient had a similar episode of dizziness and pre-syncope three months prior but did not seek medical attention. She denied melena, bright red blood per rectum, hematemesis, abdominal pain or a change in bowel habits. The patient denied chest pain, shortness of breath, nausea, vomiting, abdominal pain, fevers, chills or other symptoms at that time. She had recent outpatient labs obtained with her primary care physician that revealed severely low hemoglobin, prompting an emergency room evaluation. Emergency department assessment labs were significant for a hemoglobin of 4.2 G/DL (13.5-17.5 g/dl); MCV 68 fl (80.0-100.0 fl) with a high RDW% and elevated platelets KCUMM (acute phase reactant); and iron studies were obtained - ferritin 5.49 ng/ml (18-464 ng/ml), iron 19 ug/dl (49-181 ug/dl), and TIBC 490 ug/dl (261-462 ug/dl). Physical exam was unremarkable, including a normal abdominal examination.

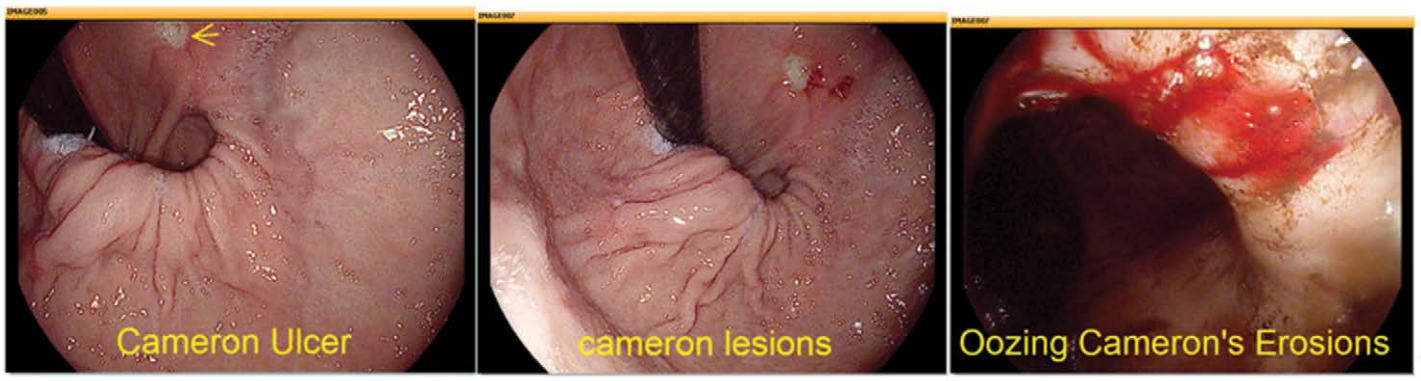
The patient was admitted for further management and received 3 units of packed red blood cells with an appropriate rise in hemoglobin to 7.5 g/dl. Esophagogastroduodenoscopy and colonoscopy revealed 5 cm hiatal hernia, two Cameron's gastric ulcers ranging 5-9 mm in the cardia, non-erosive gastritis in the gastric antrum, and moderate non-bleeding diverticulosis. No diverticulitis was noted.

The patient received Venofer prior to discharge from the hospital. Discharge medications included, pantoprazole 40mg once daily and ferrous sulfate 325mg (65mg Iron) twice daily. Upon discharge the patient reported resolution of dizziness and pre-syncopal symptoms.

Discussion

According to a retrospective cohort study preformed in 2015, researchers found a prevalence of 3.3% in patients with hiatal hernias. Within this cohort, 43 patients [mean age: 65.2(14.1) years, 49% females] had Cameron lesions identified at endoscopy, with a prevalence of 0.66% among all upper endoscopic procedures, and

continued on page 12



continued from page 11

3.3% among all patients with hiatal hernia.[7] The study went on to suggest that there was a correlation between the size of the hiatal hernia and the prevalence. Of the 857 small hiatal hernias, 10 (1.2%) had Cameron lesions. Among 300 moderate sized hiatal hernias, 14 (4.7%) had Cameron lesions. Large hiatal hernias had the highest prevalence: 19 of 149 (12.8%) had a Cameron lesion.[7]

The etiology of Cameron lesions remains unknown, although there are a few etiologies that have been researched. Our patient was Helicobacter pylori positive. Infection with H. pylori is one possible etiology that has been investigated. F. Panzuto found in a 2004 prospective study, that H. pylori infection in 61.9% (13 out of 20) of patients with large hiatal hernias and iron deficiency anemia, but anemia was still present in all patients despite successful eradication.[5] Another etiology of the development of Cameron lesions is mechanical trauma. Dr. Cameron proposed that this occurs because the gastric folds at the level of constriction of the diaphragm rubbed against each other. Based on observation of the stomach in 450 patients undergoing open surgery for hernia repair, researcher W. Colin suggested that pressure differences between the abdomen and thorax caused a sliding movement of the hernia during respiration and, thereby, distress of the mucosa leading to edema, petechiae and actual ulceration.[6]

Regardless of etiology, awareness of the condition when creating a differential diagnosis remains the goal of this case report. Patients with Cameron lesions usually present with chronic iron deficiency anemia, rarely as acute GI bleed. Multiple other risk factors such as non-steroidal anti-inflammatory drug use, alcohol consumption, and gastro-esophageal reflux disease (GERD) may be present concomitantly which makes initial differential diagnosis of Cameron lesions more difficult.[8] Cameron lesions are not often discovered with routine EGD, stressing the importance of consideration with patients with hiatal hernias. Due to the lack of circumferential involvement and their variable degrees of severity that may range from erosions to ulcerations, a complete evaluation of the neck of hernias, including anterograde, retrograde and perpendicular views, is important for the detection of Cameron lesions.[9]

In patients with obscure GI bleeding, Cameron lesions should be borne in mind as a possible diagnostic etiology. Occasionally it is not discovered at first endoscopy, however the "second look" or during pulsion enteroscopy the pathology is revealed.[2] In our patient, the gastroenterologist discovered the hiatal hernia and subsequently noted erosions at the level of the cardia. These erosions or lesions are not easily identified with routine EGD, and special considerations should be made to identify them, particularly with patients with known hiatal hernias. Endoscopy in a patient with hiatal hernia should involve meticulous visualization of the hernia neck and surrounding mucosa.[3] Of note, as many as 69% of patients underwent one or more previous upper endoscopy procedures before diagnosis of Cameron lesions was achieved.[4]

Cameron lesions should be considered when diagnosing a patient with anemia and GI bleed. These lesions are not routinely considered when a patient presents in such a fashion, so physicians should consider this cause of GI bleed and anemia, particularly when the patient is known to have a hiatal hernia.

Endnotes

1. Weston A P. Hiatal Hernia with Cameron ulcers and erosions. *Gastrointest Endosc Clinical N America*. 1996 Oct;6(4):671-9
2. Hernadez GH, Soto ICF, Garcia CAJ. Cameron Lesions: Keep in Mind in Chronic Anemia. *Journal of Hepatology and Gastrointestinal Disorder*.
3. Kapadia S, Jagroop S, Kumar A. Cameron ulcers: an atypical source for a massive upper gastrointestinal bleed. *World J Gastroenterol*, 2012 Sept 21; 18(35):4959-61
4. Zullo A, Manta R, De Francesco V, Fiorini G, Lahner E, Vaira D, Annibale B. Cameron lesion: A still overlooked diagnosis. Case report and systematic review of literature. *Clin Res Hepatol Gastroenterol*. 2018 Dec;42(6):604-609. Doi:10.1016/j.clinre.2018.05.002. Epub 2018 Jun 14
5. Panzuto F, Di Giulio E, Capurso G, et al. Large hiatal hernia in patients with iron deficiency anaemia: a prospective study on prevalence and treatment. *Aliment Pharmacol Ther* 2004;19:663-70
6. Cameron AJ, Higgens JA. Linear gastric erosion. A lesion associated with large diaphragmatic hernia and chronic blood loss anemia. *Gastroenterology* 1986;91:338-42
7. Gray D, Kushnir V, Kalra G. Cameron Lesions in Patients with Hiatal Hernias: Prevalence, Presentation, and Treatment Outcome. *Dis Esophagus*. 2015 Jul: 28(5): 448-452
8. Mehershahi S, Jog A, Ronderos D, Shaikh D, Ihimoyan A. Cameron Ulcers: Rare Case of Overt Upper Gastrointestinal Bleed in a Patient with Alcohol Use Disorder. *Cureus*. 2020 Apr; 12(4): e7644
9. Gupta P, Suryadevara M, Das A, Falterman J. Cameron Ulcer Causing Severe Anemia in a Patient with Diaphragmatic Hernia. *Am J Case Report*. 2015; 16: 733-736

References

Kimer N, Schmidt P, Krag A. Cameron lesions: an often overlooked cause of iron deficiency anaemia in patients with large hiatal hernias. *BMJ Case Rep*. 2010; 2010: bcr0620103129

Chun C, Conti C, Triadafilopoulos G. Cameron ulcers: you will find only what you seek. *Dig Dis Sci*. 2011;56:3450-52

Hamburg, NY Doctor, Wife Perish: Cholera

By Thomas Rosenthal, MD

Abstract

In 1832, when municipalities relied on free ranging pigs to clean the streets of garbage, horse manure, and morning pot dumps, Asiatic cholera entered North America at Quebec, spread to Albany then continued on along the Hudson River and Erie Canal. It took a horrible toll among the poor, ill fed, and intemperate, but soon struck the upper class. The epidemic burned out by 1834 only to return in 1849. Little had changed. Those living in villages and cities got their water from community wells, relieved themselves in community latrines and drank any water approved by a horse. The lingering 1849-1854 cholera epidemic killed ten percent of New Yorkers, including five Erie County physicians.

Introduction

In 2020, America's family doctors faced a rogue contagion for the first time in a century. Tackling the unknown and undefined is an essential role of primary care. Attentive caring backed by the best available science have always informed a good physician's tactics, but it has never been easy.

Hospitals began opening across New York in the 1840s, and grouping inpatients with similar symptoms revealed previously unrecognized patterns of disease. In the summer of 1849, the Buffalo Sisters of Charity Hospital admitted 233 cholera patients who received the recommended hourly doses of opium and calomel, a potent mercury and rhubarb cathartic meant to clear the bowels of poison. Half died. Dr. George Hawthorne, a London hospital doctor, ran out of calomel and noted better survival using only opium.[1] Philadelphia hospitals found homeopathic dilutions safer than calomel as evidence accumulated proving cholera spread by human evacuations.[2]

Case Report

Jabez Allen, MD practiced in East Aurora, NY from 1834 to 1885. His older brother, James, practiced ten miles away in Hamburg. In the lingering 1849 endemic they saw many patients with dysentery and others with profound rice water diarrhea and muscle spasms characteristic of cholera. People tried to neutralize cholera's miasmic taint by wearing camphor amulets, consuming Dr. Byrd's sulfur tablets, Old Jacob Townsend's sarsaparilla and alcohol containing elixirs. Homeopathic journals claimed one of Hahnemann's dilutions prevented cholera. Politicians burned pitch in the streets and blamed immigrants or moral laxity. President Taylor declared a day of national prayer as the AMA concluded cholera resulted from debauchery and intemperance; and was not contagious. Between 1849 and 1854 no twelve-month period passed without cholera striking somewhere. Tuberculosis killed more people, but cholera's sudden death terrorized.

In October of 1852 the eastbound stagecoach driver handed Jabez a note from James' wife, Rebecca. James was ill. Jabez saddled a horse, and made it to Hamburg within the hour. He found James listless, soiled by near constant watery evacuations, and sapped of energy by muscle spasms. Rebecca tried to keep him clean while James pleaded for relief, by death if necessary.

Rebecca filled in the details. Two days earlier James had been called to attend Mitchell Morgan, a drunkard who lived in the basement of Batlow's Tavern along with two women. The three ne'er-do-wells got by with some honest work, occasional pickpocketing and a regular trade in sexual favors. That morning Batlow decided to evict the trio, but the stench emanating from the basement repelled his effort. James was the only doctor Batlow could find who would attend the reprobates.

Once his eyes adjusted to the dimly lit basement, James observed three scantily clad bodies, one male and two females laying on a wet dirt floor. One woman was already dead and the other woman joined her before the hour was out. Morgan lasted until mid-afternoon. James could do little except provide a few doses of laudanum (opium in alcohol). Convinced by his own practical experience that cholera was spread by contagion, James spread carbonic acid powder over the bodies and around the basement. The village undertaker refused the bodies so James loaded them on a wagon that the constable drove to a nearby field where he burned wagon, bodies and all. There was no thank you, and no one to pay the doctor's fee.

Returning home, James bathed in dilute chlorinated lime water and dosed both he and Rebecca with hourly drachms of camphor sugar. Still, the next morning James woke with vomiting and fishy smelling, mucous flecked diarrhea typical of cholera. He dosed himself with 100 drops of laudanum, and terrified Rebecca with instructions to burn his clothes and bedding and scrub the room with carbolic acid after his death.

Jabez feigned invincibility, commanding Rebecca to leave the room. She refused. He applied the most up to date remedy, mixing bicarbonate soda with water and brandy, and did his best to get James to drink. Jabez's inward panic strained his outward confidence as his brother's life hung at the precipice of eternity. He gave his brother one dose of calomel; in case the new remedies were wrong.

The death of any patient leaves a wound. Doctors learn to carry on, suppress death's gloom and deny fallibility. It is the intense intimacy of being with a person at the moment of death that propels us to attend the next. Being a good shepherd at the boundaries of life is a personal honor

continued on page 14

continued from page 13

whether it be for a longtime patient, or one's brother. The good physician balances his effort to conquer with the need to comfort. The 48-year-old James faced death's passage nobly. He lapsed into a coma and died six hours later, mucous flecked stools draining his corpus until the end. Rebecca maintained a modicum of cleanliness and respectability.

By the next morning, October 7, 1852, forty-two-year-old Rebecca showed cholera's first symptoms, joining James into eternal life two days later. The constable ordered their home, and all their possessions, burned. Burial in the local cemetery was approved, but only if the bodies were soaked in chlorinated lime for 24 hours. Jabez bathed in chlorinated lime before returning home though experience suggested he was invulnerable to cholera. Perhaps one of those bouts of dysentery, so common in the nineteenth, had been a mild case of cholera.

Follow-up

In 1855, John Snow published, *On the Mode of Communication of Cholera*, depicting his epidemiological study around London's Broad Street well. Snow's work shifted blame from the victim to a contagion. Finally, in 1884 Robert Koch isolated the bacterium, *Vibrio cholerae*.

Years before it could be proven by science, community doctors observed that body fluids spread cholera, and encouraged communities to establish quarantine protocols. A partnership of science with compassionate medical practice is essential to optimizing both cure and caring.[3]

Disclaimer: Details of Dr. James Allen's death are sketchy. The events described here are most consistent with historical and medical facts. The Batlow and Morgan names have been changed. The family graves may be visited in the Hillcrest Cemetery and Mausoleum, Hamburg, NY.

Endnotes

1. Hawthorne, G. S. (1848). The True Pathological Nature of Cholera, and an Infallible Method of Treating It. Liverpool, John Churchill.
2. King, W. H. (1905). History of Homeopathy and Its Institutions in America. New York, Lewis Publishing Company.
3. Rosenthal, T. C. (2020). Bloodletting and Germs: A Doctor in Nineteenth Century Rural New York. Philadelphia, BookBaby.

Help! My Mouth is on Fire!

By Donna I. Meltzer, MD

Abstract

Burning Mouth Syndrome (BMS) is an idiopathic chronic pain disorder characterized by a burning sensation of the oral cavity but without detectable abnormalities of the oral mucosa. This disorder is more commonly seen in postmenopausal women and affected patients often have co-existing anxiety or depression. Primary BMS is a diagnosis of exclusion and remains very challenging to treat.

Introduction

Patients often present to their family physician for diagnosis and management of oral complaints. Oral lesions and discomfort can be minor and self-limiting or a sign of more serious systemic disease. It is important for primary care clinicians to be able to recognize and address oral health issues.

Case Presentation

A 60-year-old female presented to establish care and complained of a sore and burning mouth of several weeks duration. She was up to date on dental care and had tried different toothpastes without improvement in symptoms. The pain had begun insidiously and was localized to her tongue, oral mucosa and lower lip and was constant in nature. The patient denied food triggers and was adhering to a bland diet in an attempt to alleviate the burning sensation.

Her past medical history was notable for gastroesophageal reflux disease (GERD), osteoporosis, atrophic vaginitis, and open angle glaucoma. She had tried various H2 blockers and proton pump inhibitors and a trial of alendronate without improvement in oral or GERD symptoms. Her only other medications were topical estrogen and bimatoprost ophthalmic drops. The patient denied supplements other than calcium with vitamin D.

The patient is a nonsmoker and endorsed occasional social alcohol use. She is happily married with grown children and volunteers at her local church. Routine depression screening was unrevealing. She reported an overall healthy lifestyle with regular exercise and balanced diet.

She appeared healthy and well-nourished and had normal vital signs with a body mass index of 21 kg/m^2. She had good oral hygiene and there were no obvious oral lesions or signs of dry mouth. The patient was sent for investigative labs to further evaluate her symptoms as well as routine screening for diabetes and dyslipidemia. Referrals were also given for overdue breast and colon cancer screenings.

One month later the patient returned for lab review and reported no improvement in the burning mouth symptoms. Her weight remained stable. She had seen an oral surgeon who prescribed clonazepam mouthwash, but the burning persisted. There was no sign of anemia or iron deficiency and serum vitamin B12 was normal. Renal, liver, and thyroid functions were normal. Sjogren's antibody testing was within a normal range, but there was a mildly positive ANA (1:80, speckled pattern) titer. A stool antigen for *Helicobacter pylori (H. pylori)* was positive, but negative after triple antibiotic treatment. The patient had a normal hemoglobin A1c and mildly elevated total cholesterol.

Based on the history and normal examination of the oral cavity, a diagnosis of burning mouth syndrome (BMS) was entertained. Physical exam was unremarkable except for a 4x 5cm left breast mass which prompted further evaluation and a diagnosis of intraductal breast cancer. The patient was empirically offered gabapentin and a trial of antidepressant medication which did not improve her burning mouth.

In the ensuing weeks, endoscopic evaluation led to a diagnosis of celiac disease and the patient adopted a gluten free diet which improved her GERD symptoms, but not her burning mouth. In the subsequent months, the patient had a mastectomy with reconstructive surgery and chemotherapy. She was placed on tamoxifen and osteoporosis was treated with denosumab.

Several months later, the patient returned for follow up and reported that the burning mouth symptoms had subsided while undergoing chemotherapy but had since returned. A trial of alpha lipoic acid was recommended and has been helpful in reducing the burning to a more tolerable level.

Literature Review

BMS is a chronic pain disorder that is characterized by an uncomfortable burning sensation but with a clinically normal examination of the oral cavity.[1] The prevalence of BMS ranges from 0.7% to 15% depending on the population studied and diagnostic criteria.[2] The incidence of BMS also increases with age and has a strong predilection for postmenopausal women.

BMS tends to have a spontaneous onset and the cause and pathophysiology are not well understood.[1] Some theories point to a neuropathic origin (peripheral or central nervous system) and many studies mention psychiatric comorbidity (anxiety and depression). The tongue is most commonly affected and while the discomfort tends to be bilateral, it may be localized to any area within the oral cavity.[3] Burning can be constant or intermittent and varies in severity throughout the day.

The first major challenge for the clinician is to rule out secondary causes of burning mouth. A thorough history and physical exam and dental evaluation can help to exclude local or systemic disease that might present with a burning mouth. Table 1 outlines many disorders that cause burning mouth and should be considered in the differential diagnosis of BMS. This list may be used to direct a workup of BMS as it remains a diagnosis of exclusion of secondary causes. Anxiety, depression, and somatization disorder are common findings in patients with BMS. It is not known if these psychological factors are a consequence of having BMS or causal factors[2].

continued on page 16

continued from page 15

TABLE 1: Differential Diagnosis of Burning Mouth Syndrome[1,3,4]

Infection • Herpes simplex • Herpes zoster • Candidiasis • HIV • Coxsackie
Autoimmune disease • Systemic lupus erythematosus • Sjogren syndrome • Lichen planus • Celiac disease • Behcet syndrome
Nutritional deficiency • Vitamins B1, B2, B6, B12 • Folate • Zinc • Iron
Endocrine disorders • Diabetes • Thyroid disease
Gastrointestinal • GERD
Oral or Dental • Aphthous ulcer • Xerostomia • Dental prostheses • Poorly fitting dentures • Allergy (dental restoration materials) • Toothpaste • Mouthwash
Medication side effect • Antihistamines • Angiotensin-converting enzyme inhibitors • Angiotensin receptor blockers • Benzodiazepines
Psychological factors • Depression • Anxiety

A cure for BMS has been elusive given the limited understanding about the exact etiology of the disorder. Up to 30% of individuals report moderate improvement with or without treatment.[5] For the remaining, management involves pharmacological therapy, behavioral interventions and eliminating treatable secondary causes of burning mouth.

While various studies have demonstrated symptom improvement, there is a surprising lack of high-quality evidence to guide clinicians treating patients with BMS. One systematic review of 23 randomized controlled studies analyzed antidepressants, antipsychotics, anticonvulsants, benzodiazepines, cholinergics, dietary supplements, electromagnetic radiation, physical barriers, psychological therapies, and topical treatments and found insufficient evidence to support or refute any of these interventions.[6] Consequently, managing patients with BMS is often trial and error and directed towards symptom relief.[7] Various medications have been reported to alleviate burning mouth symptoms, but they may be of limited benefit (Table 2). Topical agents with clonazepam, lidocaine or capsaicin have been tried as a mouthwash with mixed results. Since BMS might be a neuropathic pain problem, tricyclic antidepressants have also been explored with inconclusive results. Alpha lipoic acid (ALA) is a fat-soluble vitamin-like antioxidant and in some studies showed promising results compared to placebo[8]. Cognitive behavioral therapy alone or in combination with other therapies might bring relief to some individuals.[9]

TABLE 2: Treatment Options for Burning Mouth Syndrome (BMS)[2]

Topical • Anxiolytic(clonazepam) • Anesthetic (lidocaine) • Atypical analgesic (capsaicin)
Systemic • Tricyclic antidepressants-amitriptyline • Serotonin antagonist and reuptake inhibitors- trazodone, sertraline, Serotonin norepinephrine reuptake inhibitor-duloxetine, etc. • Anxiolytic -clonazepam, diazepam, chlordiazepoxide • Anticonvulsants-gabapentin, pregabalin, topiramate • Antioxidants-alpha-lipoic acid (ALA) • Atypical anagesics/ antipsychotics - capsaicin, olanzapine • Vitamin supplementation

Discussion

During the initial visit with this patient, baseline labs were ordered to eliminate possible secondary causes of burning mouth. A diagnosis of BMS was suspected after laboratory evaluation did not demonstrate anemia, vitamin deficiencies or metabolic disturbances. Although ANA can be associated with mixed connective tissue disease, the low ANA titer was not felt to be clinically significant considering all the other negative rheumatologic markers. Over the course of a year, the patient had incidental findings of celiac disease and H. pylori and was also diagnosed and treated for breast cancer. Celiac disease and H. pylori infections have been associated with burning mouth symptoms but should improve with treatment. Her reflux symptoms did abate after eradication of H. pylori, but strict adherence to a gluten free diet had no effect on the burning mouth.

Psychological comorbidities often seem to be the rule in primary care and not the exception. This patient had good social support and did not have significant mental health problems which simplified management. BMS is a condition that often leads to frustration on part of the patient, but also the family physician who might not have a remedy that alleviates the problem. Good communication and empathy can be very helpful when dealing with any chronic disease and BMS is no exception.

It is curious that there is no evidence to support recommending hormone replacement therapy to women with BMS given the association with menopause. It is also unclear why the patient had improvement in burning mouth during chemotherapy with return of symptoms when cancer treatment was completed. Ultimately, the supplement alpha lipoic acid was empirically tried in this patient with notable improvement.

continued on page 18

Increasing Awareness of Colorectal Cancer in Younger Populations

By Kriti Badola and Paul Cohen, DO, MBA, DABFM, FAAFP

Abstract

Historically young onset colorectal cancer was believed to be rare, however recent studies have shown increasing rates of diagnosis in those younger than 50 since 1994. Early onset CRC has also been found to present with a poorer prognosis despite therapeutic advances. Here, we report a case of a 29-year-old female presenting with symptoms of bloody stool for two weeks. Our purpose is to emphasize the importance of screening for symptoms of colorectal cancer in younger populations as the face of colon cancer patients has changed drastically in the last 30 years.

Introduction

Colorectal cancer (CRC) is the third most common cancer and cause of cancer death worldwide in both genders. Despite the prevalence of screening tests such as fecal occult blood test (FOBT) and colonoscopies allowing for early detection, deaths among people younger than age 55 have increased 2% per year from 2007-2016.[1] While the overall frequency of CRC has been declining, early onset CRC continues to rise representing a population of patients with unmet clinical needs. By the year 2030, the incidence of colon cancer is expected to double and rectal cancer incidence is expected to quadruple from 11% and 18% respectively.[2] Although some cases may have a hereditary basis, the majority appear to arise sporadically. Needless to say this presents a serious threat to our healthcare system; this prompted the American Cancer Society(ACS) to change its recommendation from age 50 to age 45 for people at average risk in 2018. Currently routine colonoscopy is not recommended for individuals younger than age 45, resulting in advanced stages of colorectal cancer especially when diagnosed before age 20.[3] With this brief case review we would like to emphasize the alarming rise in the incidence of early onset colorectal cancer, and the importance of raising awareness and creating newer guidelines in response to the changing demographic of patients impacted by CRC.

Case Report

A 29 year-old Caucasian female went to her primary care physician because of sudden onset of blood in her stool for two weeks. She denied any weight loss, fever, abdominal pain, nausea, vomiting, and changes in weight. Before the onset of symptoms, no signs of abnormality were noted during her physical one month prior. She also had no history of chronic disease, and no known family history of cancer. She took no medications, but did take Zoloft (50 mg daily) temporarily in college, birth control (Alaycen 1 mg/0.035 mg) for 10 years, and anti-nausea (Diclegis 10 mg daily) during weeks 9-15 of pregnancy. She had a normal pregnancy and delivered a healthy full term baby one year prior to presenting with disease symptoms.

For the prior 7 years she did not consume any alcohol and followed a well- balanced diet. She did admit to excessive exposure to tanning beds 2-3 times a week in high school, as well as college. She also admitted to binge drinking in high school and in college, consuming at times over 14 drinks a week. Her family history includes a 65-year-old father with pre diabetes, and a 56-year-old pre diabetic mother. Her sister, 27 was diagnosed with interstitial cystitis.

Her primary care physician performed a rectal exam and fecal occult blood test, which was positive. She was immediately referred to a gastroenterologist and a colonoscopy was performed the following month. The colonoscopy revealed a partially obstructing mass in her sigmoid colon, 20 cm from the anal verge (Figure 1). Further imaging revealed metastasis to her liver as well. Eventual biopsy confirmed colon cancer with metastasis to the liver; she was also told she most likely had colon cancer for the last 7-10 years. She was immediately placed on a Folfoxiri treatment regimen preceding her liver resection surgery in December 2019. At diagnosis her CEA level was 77 and was monitored throughout treatment, and reduced to 3.1 prior to surgery. She completed 12 cycles of Folfoxiri and underwent a partial colectomy in May 2020.

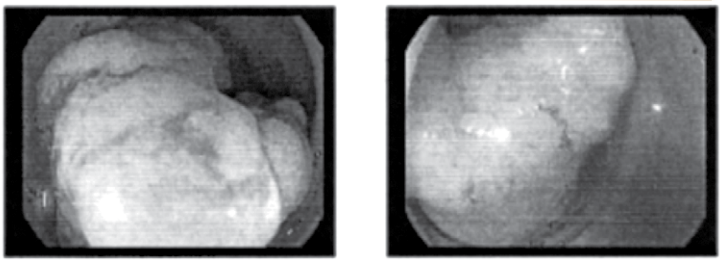


Figure 1: (A) and (B) show colonoscopy imaging of tumor in the sigmoid colon.

Conclusion

Currently there are only a handful of studies trying to identify certain risk factors associated with early onset colorectal cancer. A prospective study evaluating sedentary behaviors, associated prolonged sedentary TV viewing time with an increased risk of young onset CRC independent of exercise and obesity.[4] Another study evaluated the differences in risk factors between early onset and late onset CRC diagnoses, establishing male sex, race, irritable bowel disease, and family history as important non modifiable risk factors.[5]

The occurrence of CRC in young adults poses a tremendous risk to the healthcare system; however, the challenge to stem the trend of increasing rates is multifaceted. More modeling and data is needed to determine how screening approaches can be modified, and whether treatment options should be different for young onset CRCs. Tumors in the young population appear to be more aggressive, present at later stages and with a more advanced disease at diagnosis. Cases such as the one discussed here emphasize the need for healthcare providers to develop a heightened awareness of this change in trend to correctly evaluate any gastrointestinal problems in a young patient population.

continued on page 18

continued from page 16

Conclusion

The management of this patient illustrates the complex conditions that family physicians encounter daily. Here, a patient with burning mouth symptoms was also diagnosed and treated for celiac disease masquerading as GERD, incidental H. pylori infection and intraductal breast cancer. It is important for family physicians to remember to incorporate recommended screenings and health care maintenance into routine office visits when time permits as they can be lifesaving.

Endnotes

1. Klein B, Thoppay JR, DeRossi SS, Ciarrocca K. Burning mouth syndrome. Dermatol Clin NA 2020:38(4):477-483.
2. Klasser GD, Grushka M, Su N. Burning mouth syndrome. Oral Maxiollofacial Surg Clin NA 2016; 28(3):381-396.
3. Moghadam-Kia S, Fazel N. A diagnostic and therapeutic approach to primary burning mouth syndrome (BMS). Clin Dermatol 2017; 35(5):453-460.
4. Gurvits GE, Tan A. Burning mouth syndrome. World J Gastroenterol 2013;19(5):665-672.
5. Zakrzewska J, Buchanan JA. Burning mouth Syndrome. BMJ Clin Evid 2016;2016:1301
6. McMillan R, Forssell H, Buchanan JAG, Glenny AM, Weldon JC, Zakrzewska JM. Interventions for treating burning mouth syndrome. Cochrane Database of Systematic Reviews 2016, Issue 11. Art. No.: CD002779. DOI: 10.1002/14651858.CD002779.pub3.
7. Bender SD. Burning mouth syndrome. Dent Clin N Am 2018 (62): 585-596.
8. Alqahtani SS. The efficiency of alpha-lipoic acid in the treatment of burning mouth syndrome: a systematic review. Eur Rev Med Pharmacol Sci 2021;25(21):6585-6591.
9. Aggarwal A, Panat SR. Burning mouth syndrome: A diagnostic and therapeutic dilemma. J Clin Exp Dent 2012;4(3):180-185.

continued from page 17

Colorectal cancer can no longer be seen as a disease primarily affecting the elderly. Symptoms at time of diagnosis for young onset patients include: rectal bleeding (51%), change in bowel habits (18%), abdominal pain (32%), and weight loss (13%).[6] Patients in one study said they were often misdiagnosed with more benign ailments such as hemorrhoids upon complaining of rectal bleeding to their primary care doctor.[7] If symptoms of rectal bleeding were presented in a patient over the age of 50, a more definitive evaluation would take place. In young patients almost two-thirds of rectal cancers are found in the distal colon, indicating screening may be in reach of a flexible sigmoidoscopy.[7] Referrals for sigmoidoscopies and colonoscopies should be used as appropriate for patients with unexplained persistent bleeding, and changes of bowel habits.[8] A cost effective analysis showed evaluation of the colon with asymptomatic rectal bleeding using a flexible sigmoidoscopy plus barium enema yielded the greatest life expectancy at costs comparable to that of a colon cancer screening.[9]

Our case study emphasizes the necessity of modifying future screening policies and increasing awareness of the changing trends in CRC. Younger people need to be made aware of the symptoms of colorectal cancer, and conversations about risks and prevention strategies should be discussed with primary care doctors during routine visits to better accommodate these changing trends.

Endnotes

1. American Cancer Society. *Cancer Facts & Figures 2020*. Atlanta, Ga: American Cancer Society; 2020.
2. Dana-farber.org. 2020. *Young-Onset Colorectal Cancer Center - Dana-Farber Cancer Institute | Boston, MA*. [online] Available at: <https://www.dana-farber.org/young-onset-colorectal-cancer-center/> [Accessed 26 May 2020].
3. Sultan I, Rodriguez-Galindo C, El-Taani H, et al. Distinct features of colorectal cancer in children and adolescents: a population-based study of 159 cases. *Cancer*. 2010;116(3):758-765. doi:10.1002/cncr.24777
4. Nguyen LH, Liu PH, Zheng X, et al. Sedentary Behaviors, TV Viewing Time, and Risk of Young-Onset Colorectal Cancer. JNCI Cancer Spectr. 2018;2(4):pky073. doi:10.1093/jncics/pky073
5. Gausman V, Dornblaser D, Anand S, et al. Risk Factors Associated With Early-Onset Colorectal Cancer [published online ahead of print, 2019 Oct 14]. Clin Gastroenterol Hepatol. 2019;S1542-3565(19)31108-5. doi:10.1016/j.cgh.2019.10.009
6. Dozois EJ, Boardman LA, Suwanthanma W, et al. Young-onset colorectal cancer in patients with no known genetic predisposition: can we increase early recognition and improve outcome?. *Medicine (Baltimore)*. 2008;87(5):259-263. doi:10.1097/MD.0b013e3181881354
7. Bosworth, Ted. "Https://Www.Gastroendonews.Com/In-the-News/Article/03-18/Researchers-Seek-Early-Signs-of-Young-Onset-C RC/47114." *www.gastroendonews.com*, 9 Mar. 2018, www.gastroendonews.com/In-the-News/Article/03-18/Researchers-Seek-Early-Signs-of-Young-Onset-CRC/47114 . Accessed 26 May 2020.
8. Ahnen DJ, Wade SW, Jones WF, et al. The increasing incidence of young-onset colorectal cancer: a call to action. *Mayo Clin Proc*. 2014;89(2):216-224. doi:10.1016/j.mayocp.2013.09.006
9. Lewis JD, Brown A, Localio AR, Schwartz JS. Initial evaluation of rectal bleeding in young persons: a cost-effectiveness analysis. *Ann Intern Med*. 2002;136(2):99-110. doi:10.7326/0003-4819-136-2-200201150-00007

Esophageal Duplication Cyst, a Rare Cause of Dysphagia in an Adult

By Preetha Phillips, DO and Sami Almaskeen, MD

Abstract

A 32-year-old male with past medical history of diabetes mellitus and bipolar disorder presented with worsening dysphagia. CTA chest was significant for 2.1 x 1.8 x 2 cm soft tissue nodule abutting the distal esophagus, superior to the gastroesophageal junction. In clinical practice, a mass with worsening dysphagia is concerning for malignancy. Endoscopic ultrasound revealed an anechoic lesion suggestive of an esophageal duplication cyst. Esophageal duplication cysts are thought to occur due to defects in embryogenesis.[8] Symptoms caused by duplication cysts initially presenting in adulthood are rare, however duplication cysts should be in the differential in a patient with dysphagia.

Introduction

Gastrointestinal tract duplication cysts are rare congenital malformations. Esophageal duplication cysts account for approximately 10-15% of gastrointestinal duplication cysts.[8] They are very rare with a prevalence of 0.0122% and incidence of approximately 1/8000 live births.[4] Clinical symptoms are secondary to the compression of the cyst to adjacent structures. Dysphagia is the most common presenting symptom along with epigastric and retrosternal pain. However respiratory symptoms such as cough, stridor and wheezing can be present. It is also possible to see a mass on imaging. Although dysphagia along with mass on imaging is concerning for malignancy, it is important to have a thorough evaluation to delineate the true etiology of a patient's dysphagia.

Case Presentation

A 32-year-old male with past medical history significant for type 2 diabetes mellitus and bipolar disorder presented to the emergency department (ED) with worsening dysphagia and crampy, generalized abdominal pain. The patient reported that the dysphagia started a few days prior and was acute in onset. He described the dysphagia as difficulty with swallowing solids, noting that drinking liquids made it easier to swallow solid food. He denied a slow progression of dysphagia, any difficulty in swallowing liquids or weight loss. The patient denied alcohol, tobacco and illicit drug use.

One month prior, the patient was evaluated in the ED for chest pain. A CT angiogram of the chest was significant for a 2.1 x 1.8 x 2 cm soft tissue nodule abutting the distal esophagus just superior to the gastroesophageal (GE) junction. The patient denied abdominal pain and dysphagia at that visit. He was discharged from the ED with outpatient follow up with gastroenterology (GI) and was ultimately scheduled for Esophagogastroduodenoscopy (EGD). However, he developed a sudden onset of dysphagia and presented to the ED for re-evaluation before his scheduled outpatient EGD.

Physical exam was significant for mildly distended abdomen without tenderness to palpation. Ultrasound of the abdomen and laboratory findings were unremarkable. Endoscopy revealed mild gastritis noted in the antrum, but was otherwise unremarkable. Biopsies were taken, which revealed benign squamous mucosa. Endoscopic ultrasound (EUS) was significant for a paraesophageal, anechoic fluid filled lesion with well-defined margins measuring 20 x 16 mm at the GE junction at 40 cm with no intraluminal extension that appeared to arise from the 5th layer. No vascular pattern was seen on doppler examination. The appearance of the lesion was highly suggestive of esophageal duplication cyst. The lesion was not aspirated to avoid possibility of infection. The patient was treated with an oral proton pump inhibitor (PPI). At his follow up visit with GI, the patient reported significant improvement in dysphagia and abdominal pain. PPI was discontinued after a one-month course, with plan for further evaluation and possible surgical removal of esophageal cyst if symptoms recurred.

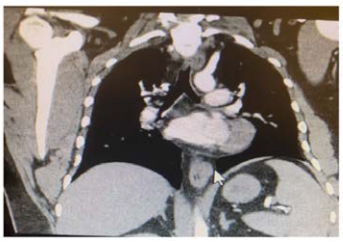


Figure 1: CTA chest displaying soft tissue nodule at GE junction.

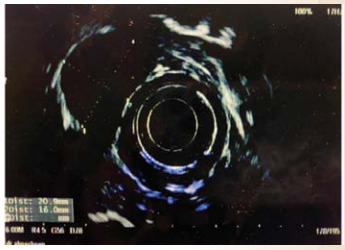


Figure 2: EUS image of 20 x16 mm anechoic lesion

Discussion

Dysphagia most commonly occurs due to an impaction of the esophagus by a foreign entity. Having certain associated symptoms such as heartburn, weight loss, anemia, regurgitation of food products, emesis and respiratory symptoms can narrow the differential diagnosis. The initial step to diagnosis after a history and physical is a barium esophagram. An upper endoscopy should also be done to exclude malignancy.[6] Although a mass may be concerning for malignancy, benign lesions such as schwannomas, polyps and cysts are also possible. Cystic lesions are rare and were found to be present in 1 in 8200 patients.[1] The utilization of endoscopic ultrasound provides a further study to delineate a possible mass, and is essential to accurately differentiate various luminal, intramural and extraluminal lesions.[6] Esophageal manometry can be done in patients when motility disorder is suspected or when endoscopy is unrevealing.[6]

Gastrointestinal tract duplication cysts are rare congenital malformations that are often seen in young patients.[2] In most cases these cysts are diagnosed in young children, approximately 60% diagnosed before the age of 2.[3] Duplication cysts are categorized based on their embryonic origin. A true duplication cyst is secondary to the duplication of the submucosa and the muscularis without the duplication of the epithelium.[8] Bronchogenic and esophageal duplication cysts are thought to form from abnormal budding of the embryonic foregut at 5-8 weeks of gestation.[8]

continued on page 20

continued from page 19

Imaging studies such as CT or MRI may reveal a mediastinal mass with esophageal compression. Endoscopy should be considered especially in symptomatic patients, with EUS being the modality of choice, due to the ability to be able to characterize the echogenicity of the lesion.[5] Typically, during EUS, an anechoic homogenous lesion with smooth margins is seen in the submucosal wall.[5] Biopsy is typically reserved in cases where the nature of the lesion is undetermined and there is high suspicion for malignancy.[5] This is due to an increased risk of infecting the cyst with aspiration to obtain biopsy. If biopsy is necessary, antibiotic coverage must be administered pre and post procedure.[9]

Esophageal duplication cysts are managed based on symptoms. Complication of esophageal cysts can include reflux esophagitis, rupture, hemorrhage, perforation, and pseudodiverticulum development. In very rare occurrences, duplication cysts can undergo malignant transformation.[7] Studies have shown due to the rarity of malignant transformation, it is not cost effective to have surveillance endoscopy in patients with symptoms that are well controlled.[7] Surgical removal is the treatment of choice in most symptomatic cases. Surgical resection can be done through video assisted thoracoscopic surgery (VATS) and robotic assisted thoracoscopic surgery (RATS).[5] Less invasively, endoscopic submucosal tunnel dissection (ESTD) can be done to excise the cyst in select cases.[5] Patients seem to do well after treatment with full surgical enucleation or excision in both short and long term. Recurrence is rare.

Conclusion

A patient presenting with dysphagia and esophageal mass on imaging is concerning for possible malignancy. It is important to acquire a detailed history and physical examination to determine the progression of dysphagia. Endoscopy is the primary mode of diagnosis. Other tests and modalities such as endoscopic ultrasound and manometry can help further delineate the etiology of dysphagia depending on clinical picture. It is important to keep in mind the other benign lesions that can cause dysphagia such as esophageal duplication cyst as seen in this patient. As seen in this patient, management of GERD with lifestyle changes and PPI were sufficient to manage his symptoms, and surgical intervention was avoided.

Endnotes

1. Arbona JL, Fazzi JG, Mayoral J. Congenital esophageal cysts: case report and review of literature. Am J Gastroenterol. 1984 Mar;79(3):177-82. PMID: 6702802.
2. Berrocal T, Torres I, Gutiérrez J, Prieto C, del Hoyo ML, Lamas M. Congenital anomalies of the upper gastrointestinal tract. Radiographics. 1999 Jul-Aug;19(4):855-72. doi: 10.1148/radiographics.19.4.g99jl05855. PMID: 10464795.
3. Ildstad ST, Tollerud DJ, Weiss RG, Ryan DP, McGowan MA, Martin LW. Duplications of the alimentary tract. Clinical characteristics, preferred treatment, and associated malformations. Ann Surg. 1988 Aug;208(2):184-9. doi: 10.1097/00000658-198808000-00009. PMID: 3401062; PMCID: PMC1493602.
4. Geller A, Wang KK, DiMagno EP. Diagnosis of foregut duplication cysts by endoscopic ultrasonography. Gastroenterology. 1995 Sep;109(3):838-42. doi: 10.1016/0016-5085(95)90392-5. PMID: 7657112.
5. Obasi PC, Hebra A, Varela JC. Excision of esophageal duplication cysts with robotic-assisted thoracoscopic surgery. JSLS. 2011 Apr-Jun;15(2):244-7. doi: 10.4293/108680811X13071180406961. PMID: 21902985; PMCID: PMC3148881.
6. Malagelada JR, Bazzoli F, Boeckxstaens G, De Looze D, Fried M, Kahrilas P, Lindberg G, Malfertheiner P, Salis G, Sharma P, Sifrim D, Vakil N, Le Mair A. World gastroenterology organisation global guidelines: dysphagia--global guidelines and cascades update September 2014. J Clin Gastroenterol. 2015 May-Jun;49(5):370-8. doi: 10.1097/MCG.0000000000000307. PMID: 25853874.
7. Singh S, Lal P, Sikora SS, Datta NR. Squamous cell carcinoma arising from a congenital duplication cyst of the esophagus in a young adult. Dis Esophagus. 2001;14(3-4):258-61. doi: 10.1046/j.1442-2050.2001.00204.x. PMID: 11869334.
8. Sonthalia N, Jain SS, Surude RG, Mohite AR, Rathi PM. Congenital Esophageal Duplication Cyst: A Rare Cause of Dysphagia in an Adult. *Gastroenterology Res*. 2016;9(4-5):79-82. doi:10.14740/gr711w
9. Valli PV, Gubler C, Bauerfeind P. Severe Infectious Complications after Endoscopic Ultrasound-Guided Fine Needle Aspiration of Suspected Mediastinal Duplication Cysts: A Case Series. Inflamm Intest Dis. 2017 Apr;1(4):165-171. doi: 10.1159/000464327. Epub 2017 Mar 30. PMID: 29922673; PMCID: PMC5988119.

An Uncommon Cause of Chest Pain: Multiloculated Hepatic Abscess in a Patient with Strongyloides Stercoralis Infection

By Yaritza Serrano Gomez, MD; Ijeoma Aneke, MD; Farideh Zonouzi- Zadeh, MD and James Mumford, MD

Abstract

Liver abscess, the most common type of visceral abscess, is an uncommon but potentially life-threatening infection. In a report of 540 cases of intraabdominal abscesses, which also included intra and retro-peritoneal abscesses, pyogenic liver abscesses accounted for 48 percent of visceral abscesses and 13 percent of intraabdominal abscesses overall. The annual incidence of liver abscess has been estimated at 2.3 cases per 100,000 people and is higher among men than women.[1] We report a case of a 53-year-old male who presented to an ambulatory clinic for evaluation of substernal chest pain. He reported associated palpitations, chills, shortness of breath, and diaphoresis. An abdominal CT revealed a mass with multiple locations in the right and caudate hepatic lobe consistent with an abscess. Blood cultures grew *Klebsiella pneumoniae*, and ova and parasite exams showed *Strongyloides stercoralis* larvae. After four weeks of antibiotic treatment, a repeat abdominal CT demonstrated complete resolution of the hepatic abscess. This case is an unusual presentation of a hepatic abscess, where the patient did not report abdominal symptoms.

Introduction

Liver abscesses are purulent collections in the liver parenchyma that result from bacterial, fungal, or parasitic infections. Infection can spread to the liver through the biliary tree, hepatic vein, or portal vein, by extension of an adjacent infection or trauma. As a result of its dominant vasculature, the right hepatic lobe is most affected in 91% of cases. Most pyogenic liver abscesses are polymicrobial, reflecting the variability among patients and geographic areas. Traditionally, *Escherichia coli* has been reported as the most common isolated microbe; however, recent data show that *Klebsiella pneumoniae* is the most common pathogen in pyogenic liver abscesses.[2] The majority of aspirated fluid cultures are positive, whereas blood cultures are positive in only 50% of cases.[3] It is noteworthy that about 40% of cases of liver abscesses develop local or systemic complications, the most common being generalized sepsis and pleural effusion. We report a case of liver abscess in a patient with strongyloidiasis infection complicated by bacteremia with *Klebsiella pneumoniae* who presented with chest pain to our institution.

Case Presentation

A 53-year-old male with a past medical history of hypertension who recently emigrated from El Salvador presented to our ambulatory clinic for evaluation of substernal chest pain that started eight days earlier. He described the chest pain as intermittent, non-radiating, and associated with palpitations, chills, shortness of breath, and diaphoresis. He denied fever, abdominal pain, nausea, or vomiting. On physical examination the abdomen was soft, non-tender and non-distended. The ECG showed junctional tachycardia and T wave inversion in lead III and aVF. The patient was sent to the emergency department to rule out acute coronary syndrome. At the emergency department laboratory tests were performed and significant for WBC 12.63 K/uL, lactate 10 mmol/L, ALK 193 U/L, AST 88 U/L, ALT 94 U/L, T Bili 0.4 mg/dL, D-dimer 878 ng/mL, troponin elevated to 0.82 ng/mL, and A1c 8.6%. CTA of the chest was negative for pulmonary embolism but revealed an incidental heterogenous mass within the liver. An abdominal CT confirmed a 5.5 x 8.9 x 9.8 cm mass with multiple loculations in the right and caudate hepatic lobe that was suspected of being an abscess (figure 1).

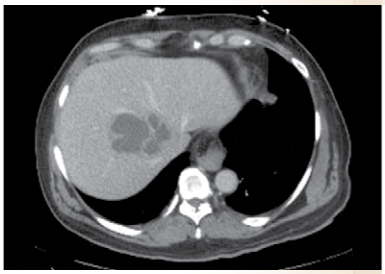


Figure 1: CT of abdomen and pelvis with IV contrast showing a 5.5 x 8.9 x 9.8 cm mass with multiple loculations in the right and caudate hepatic lobe compatible with an abscess.

The patient was admitted for the management of septic shock secondary to a hepatic abscess. Blood cultures grew *Klebsiella pneumonia*. Ova and parasite exams showed *Strongyloides stercoralis* larvae. Antibodies for Entamoeba histolytica, HIV, and HTLV-1 were negative. Interventional radiology recommended against abscess drainage due to its complexity and location. The patient initially received treatment with ceftriaxone 2g intravenous daily and metronidazole 500 mg intravenous three times daily for five days with some improvement in presenting symptoms and subsequently was discharged home on cefuroxime 500 mg oral two times daily with a recommended total therapy of six weeks. Approximately two weeks after discharge, the patient started to experience new episodes of fever and came back to the emergency department for evaluation. Abdominal CT was repeated and showed an increase in the size of the hepatic abscess. MRI of abdomen was subsequently performed and showed multiple right hepatic lobe abscesses with a dominant 4.4-cm abscess in segment eight that was new from CT two weeks prior (figure 2). Interventional radiology was consulted for the second time

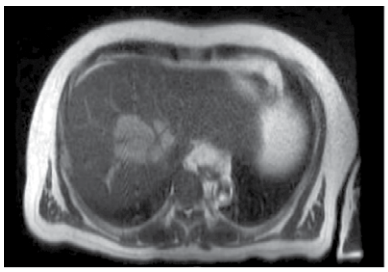


Figure 2: MRI of the abdomen w/wo IV contrast showing multiple peripheral enhancing diffusion restricting masses within segments 8,1, and 6. The dominant mass measures 4.4 x4.3cm in segment 8.

continued on page 22

continued from page 21

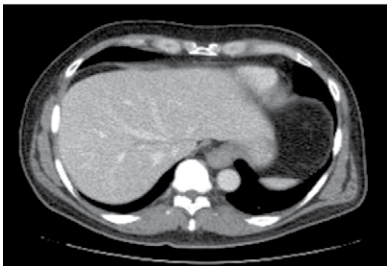


Figure 3: CT of the abdomen and pelvis with IV contrast after four weeks of antibiotic treatment showing resolution of the hepatic abscess.

and deferred percutaneous drainage of the abscess due to difficult access. Infectious disease recommended discontinuing oral cefuroxime due to inadequate response and initiating treatment with ceftriaxone 2g intravenous daily and metronidazole 500 mg orally three times daily for four weeks, and oral ivermectin for two weeks to manage strongyloidiasis. After four weeks of treatment, a repeat abdominal CT demonstrated complete resolution of the hepatic abscess (figure 3).

Discussion

A liver abscess is an uncommon but potentially life-threatening infection. Common predisposing causes include biliary disease, gastrointestinal malignancies, congenital anomaly of the biliary tree, portal vein seeding, and penetrating trauma.[2] Approximately 18%–66% are cryptogenic, with no underlying cause identified.[2] Liver abscesses are commonly caused by polymicrobial infections, with the most common organisms being *E. coli, Streptococcus, Enterococcus*, and anaerobes.[4] Recent data show that *Klebsiella pneumoniae* is the most common pathogen in pyogenic liver abscesses, with higher susceptibility in diabetic patients.[4]

The clinical manifestations are variable and depend on the abscess's size, the patient's general health, associated comorbidities, and complications.[5] Clinical manifestations often include right upper quadrant pain, high fever, nausea, and vomiting.[5] Loss of appetite, jaundice, ascites, pleural effusion, and respiratory symptoms may also occur, though less frequently. Laboratory findings include leukocytosis, elevated CRP, and sometimes liver function test abnormalities.[6] Given that neither symptoms nor laboratory testing are specific, diagnosis relies mainly on imaging. Ultrasound and CT abdomen carry a sensitivity for the diagnosis of liver abscess of 85% and 97%, respectively. However, they are not able to definitively differentiate the microbiological etiology.[6]

Management includes image-guided drainage and antibiotic therapy.[3] Most aspirated fluid cultures are positive, whereas blood cultures are positive in only 50% of cases. Some studies suggest that small abscesses (3–5 cm) can be treated by antibiotics alone. Indications for percutaneous drainage include if the abscess is large (>10 cm in diameter), subcapsular, high risk for rupture, superinfected, or if there is poor response to medical treatment.[6] There is considerable variation in clinical practice regarding total antibiotic duration. It is recommended that antibiotic treatment be continued for at least four to six weeks, but the optimal time is still unclear.[3]

Hepatic abscesses can result from bacterial, fungal, or parasitic infections. The parasite most associated with hepatic abscess is *Entamoeba histolytica*, but little has been documented of other parasites such as *Strongyloides stercoralis* in developing a hepatic abscess. *Strongyloides stercoralis* is an intestinal nematode that infects 3 million to 100 million people worldwide.[7] While strongyloidiasis is mainly asymptomatic in 50% of patients, it may present as disseminated disease with the spread of larvae to tissues outside of the autoinfection cycle, including the liver.[8] Chronic and disseminated strongyloidiasis increases the risk of severe enterobacteria infection, especially *Klebsiella pneumoniae* infection. It has been suggested that the strongyloidiasis larvae can cause intense inflammation of the cecum, which allows *Klebsiella pneumoniae* to invade the intestine. This indicates that strongyloidiasis causes severe disease with enterobacteria via induction of mucosal rupture, leading to dissemination.[9] Penetration of the larvae through the intestinal wall can be associated with gram-negative sepsis, as larvae carry enteric microorganisms into the bloodstream. However, this relationship should be further studied.

Conclusion

This case is an unusual presentation of a hepatic abscess, where the patient did not report abdominal symptoms. He presented with chest pain and tachyarrhythmia, which were found to be secondary to demand ischemia due to the septic process. There could be a relationship between new onset diabetes with strongyloidiasis and *Klebsiella pneumoniae* infections in the development of hepatic abscess. A liver abscess can have a mortality of 100% without treatment, which drops to between 2.5% and 14% when treated. For this reason, early diagnosis and treatment are essential for decreasing mortality.[5]

Endnotes

1. Altemeier WA, Culbertson WR, Fullen WD, Shook CD. Intra-abdominal abscesses. Am J surge 1973; 125:70
2. Longworth, S., & Han, J. (2015). Pyogenic liver abscess. *Clinical liver disease, 6*(2), 51–54. https://doi.org/10.1002/cld.487
3. Bachler P, Baladron M, Menias C, Beddings I, Loch R, Zalaquet E, Vargas M, Connolly S, Bhalla S, Huete A. Multimodality Imaging of Liver Infections: Differential Diagnosis and Potential Pitfalls. *RadioGraphics 2016*; 36:1001–1023
4. Akhondi H, Sabih DE. Liver Abscess. [Updated 2021 Jul 6]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK538230/
5. Sotto Mayor, J., Robalo, M. M., Pacheco, A. P., & Esperança, S. (2016). Pyogenic liver abscess: uncommon presentation. *BMJ case reports, 2016*, bcr2016214841. https://doi.org/10.1136/bcr-2016-214841
6. Shull H, Bejjani A, Kahn D. Hepatic Abscess in a Returning Traveler with Crohn's Disease: Differentiating Amebic from Pyogenic Liver Abscess. *Hindawi Case Reports in Medicine*. Vol2018. https://doi.org/10.1155/2018/9593865
7. Ramanathan, R., & Nutman, T. (2008). Strongyloides stercoralis infection in the immunocompromised host. *Current infectious disease reports, 10*(2), 105–110. https://doi.org/10.1007/s11908-008-0019-6
8. Braun, David S. MD1; John, Elizabeth MD1; Mehta, Ashwini DO1 S2606 The First Documented Case of Strongyloides Liver Abscess with Superimposed Methicillin-Resistant Staph aureus Infection, The American Journal of Gastroenterology: October 2020 - Volume 115 - Issue - p S1369 doi: 10.14309/01.ajg.0000712472.83978.c0
9. Hosoda, T., Sakamoto, M., Orikasa, H., Kubomura, A., Misaki, T., & Okabe, N. (2020). Septic Meningitis and Liver Abscess due to Hypermucoviscous Klebsiella pneumoniae Complicated with Chronic Strongyloidiasis in a Human T-lymphotropic Virus 1 Carrier. *Internal medicine (Tokyo, Japan), 59*(1), 129–133. https://doi.org/10.2169/internalmedicine.3403-19

25- year-old Female Refugee with Severe Microcytic Anemia

By Christine Krol, MD and Elizabeth Harding, MD

Abstract

Patient is a 25- year-old Swahili-speaking female who presented with anemia (hemoglobin 4) with massive splenomegaly. After a wide differential was explored, the underlying cause of her presentation was determined to be chronic hepatitis B. Unfortunately, the chart review uncovered that she was known to have this disease by her former physicians. However, due to improper translation, she was never informed of her diagnosis and therefore did not seek or receive appropriate treatment for herself (or her young children). This case illustrates the importance of utilizing proper resources, such as quality medical translation, when working with non-English speaking patients.

Introduction

This case is unique for a few reasons. First, it is unusual in the United States to see disease of this severity, especially in a young patient, due to chronic hepatitis B. Second, it illustrates grossly inappropriate care in a vulnerable patient who was not properly informed of her condition due to not obtaining adequate translation. Third, it demonstrates the difficulties many patients face in obtaining proper health care, even in a society with so many resources, due to things many of us take for granted such as reliable transportation.

Case Report

The patient is a 25-year-old Swahili-speaking female refugee from Congo who presented to clinic for a routine annual wellness visit. She complained of abdominal pain that had been occurring intermittently for many years, as well as an abdominal mass. On exam, she was found to have left upper quadrant tenderness to palpation, as well as a tender left upper quadrant mass. Labs were drawn (CBC, CMP), an abdominal ultrasound was ordered and she was scheduled for a follow-up visit the following month. The next day, she was called and advised to go to the nearest emergency department when her lab results revealed a severe microcytic anemia with hemoglobin 4.1, hematocrit 16.7, and MCV 57.2, as well as leukopenia with WBC 2.3. In the emergency room, the labs were repeated and confirmed the clinic's findings. They ordered two units of packed red blood cells to be transfused and called our team for the admission. We ordered additional labs prior to the transfusion being given, which revealed severe iron deficiency (iron 9, TIBC 495, transferrin 396, ferritin < 1), normal B12 and folate, as well as reticulocyte count of 0.6 and absolute reticulocytes of 20.

Upon admission, she confirmed her initial clinic complaints and additionally complained of fatigue. She also remembered experiencing cyclical abdominal pain with fevers and anorexia during her childhood. In addition to her abdominal findings, she was found to have conjunctival and oral mucosal pallor, and a II/VI systolic ejection murmur. Based on her exam, labs, and history, we had a wide-ranging differential diagnosis, including recurrent malaria, sickle cell disease, viral hepatitis, GI bleed, and autoimmune hemolytic anemia. A CT of the abdomen and pelvis confirmed her LUQ mass to be massive splenomegaly with craniocaudal extent of 21cm, as well as revealed hepatic nodularity with prominent caudate and left lobes suggestive of cirrhosis, with periesophageal and perigastric varices. Additional lab investigation revealed normal hemolysis markers, negative blood parasite testing, normal hemoglobin electrophoresis, negative HIV and hepatitis C testing; however, the hepatitis B surface and core antigens, as well as hepatitis Be antibody, were all positive with a hepatitis B DNA level of 66, consistent with chronic hepatitis B.

We also performed an extensive chart review. It was discovered that she did have microcytic anemia at her new patient visit (hemoglobin 8.2, hematocrit 29.3, MCV 78.8), but was also pregnant at the time under the care of a separate OBGYN, and so did not follow-up at our clinic again for a few years. Her pregnancy records were found, which contained labs consistent with chronic hepatitis B (positive surface antigen and core antigen). It was documented that this diagnosis was discussed, but did not specify that a translator was used and also stated "patient seems confused," "patient again having difficulty understanding certain terms" and "will need further birth control counselling, not understanding of this conversation either." Furthermore, we found records of her delivering this child and at the hospital labs were repeated which also revealed chronic hepatitis B. Her daughter was only given the hepatitis B vaccine (no hepatitis B immunoglobulin) and there was no documentation that this diagnosis was discussed with the patient. Her children were patients at our clinic, and we found that neither her son (who was born in Congo) nor this newborn daughter were ever tested for hepatitis B, as we were unaware of her diagnosis. We promptly informed our clinic of this and both children were tested and fortunately were negative.

During the remainder of her 5-day admission, she was seen in consult by GI, who recommended an abdominal ultrasound with doppler, which did show normal hepatic venous flow. She was also seen by hematology, who felt that her severe anemia was multifactorial due to severe iron deficiency, chronic disease, as well as possible ongoing GI loss from varices. The patient received IV iron, as well as a third unit of packed red blood cells, and at the time of discharge was stable with hemoglobin 8, hematocrit 28.1, MCV 64.6, however was now pancytopenic with WBC 3.1 and platelets 139. She was sent home with oral iron supplementation and was to have close outpatient follow-up.

continued on page 24

continued from page 23

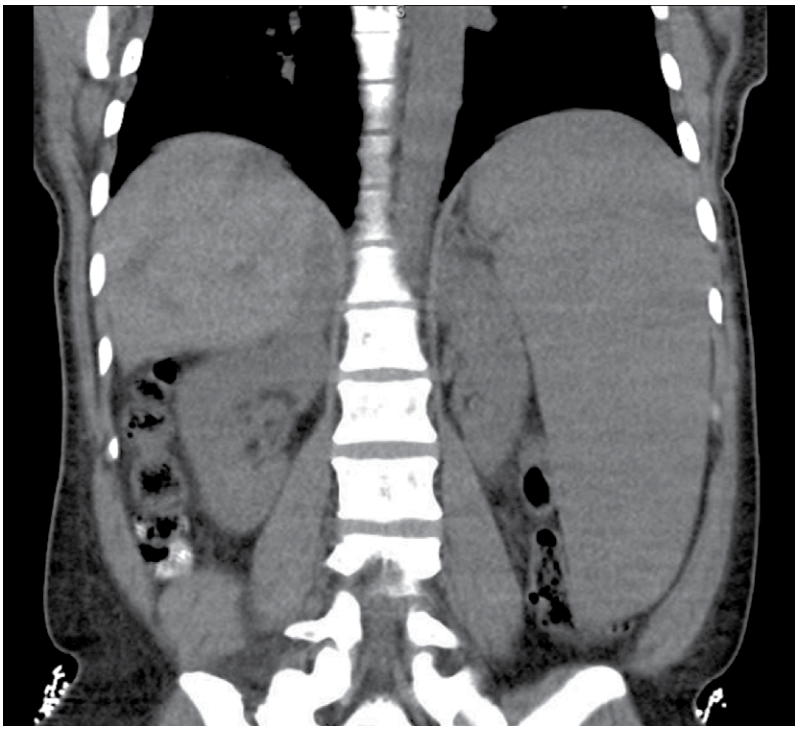
Our patient has continued to follow with our clinic, as well as with GI/hepatology and hematology. Due to transportation issues, her follow-up has unfortunately been inconsistent; she does not qualify for Medicaid transportation, cannot navigate the bus system well due to the language barrier, and her large family shares one unreliable vehicle. GI did perform a liver biopsy, which did show chronic hepatitis and patchy periportal fibrosis, but surprisingly no cirrhosis. She has not required antiviral treatment due to this, as well as low viral DNA levels. Unfortunately, due to poor follow-up attendance, EGD and colonoscopy to rule out GI bleed have not yet been performed. Hematology performed a bone marrow biopsy, which ruled out an acute leukemia or other lymphoproliferative neoplasm. They have administered multiple courses of IV iron as well as continued oral iron supplementation. Due to missing many appointments, she was found to be markedly pancytopenic once again on her most recent labs in January 2022 with WBC 1.4, hemoglobin 8, hematocrit 28.1, and platelets 54. She has been referred back to hematology and we are attempting to help find a way for her transportation to be more reliable.

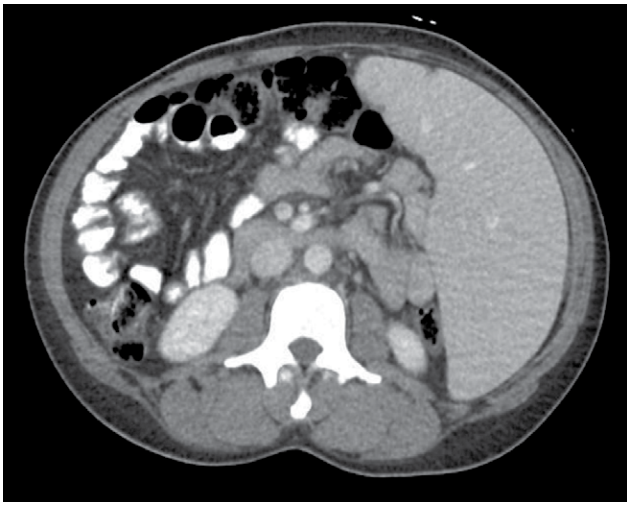
Discussion

There were multiple troubling aspects about this case. From the start, certain results, especially the reticulocyte count being significantly lower than expected with an anemia of this severity, were highly unusual. Even the end diagnosis itself was not what was expected; being an intern at the time, it was a reminder to me to never make assumptions and to always have a broad differential to consider. We were further clued in that this would be a memorable case when we informed our patient of our findings using a Swahili translator, and explained what this diagnosis means for her now, as well as for her future. She seemed shocked and had many questions; it was clear that she was hearing this information for the first time. Despite her improvement during her hospitalization, we found ourselves troubled by how a young patient could have such a severe presentation of chronic hepatitis B with no previous diagnosis especially given her previous pregnancies and extensive work-up all new refugees receive.

These factors spurred our extensive chart review, which was completed using a website on which medical records from any physicians in our geographical area can be viewed with the patient's written consent. This allowed us to access her new refugee records, as well as her pregnancy and delivery records. While we were relieved to see that her diagnosis had not been missed, we were shocked to see that she was never properly informed of this diagnosis. To not secure a translator in this situation was unacceptable. As a clinic who sees 60%+ patients who are not English speaking we recognize that working through a translator does add time to the visit. However, this case very clearly illustrates why that extra step is so necessary; especially as it concerned not only the patient, but her children as well. We encourage readers to always have a broad differential, as well as to take the time to ensure your patients, especially those facing additional challenges such as a language barrier, fully understand their health concerns.

Images from patient's CT abdomen and pelvis illustrating massive splenomegaly





Hungry Bone Syndrome

By Minakshi Shukla, MD and Alla Akivis, MD

Abstract

We describe a case of a 38-year-old female patient with past medical history of end-stage renal disease (ESRD) who developed hungry bone syndrome (HBS) post parathyroidectomy. HBS is defined as profound and prolonged hypocalcemia, serum calcium of < 8.4mg/dl persistent for more than four days post-operatively. Hypocalcemia occurs due to a shift from net bone resorption to net bone formation after the drop in levels of parathyroid hormone (PTH) post-operatively. Patients with risk factors for the development of HBS should be closely monitored with serial serum calcium, magnesium, and phosphorous levels.

Introduction

HBS is defined as profound and prolonged hypocalcemia serum calcium of < 8.4mg/dl persistent for more than four days post-operatively. HBS can be seen as a complication of parathyroidectomy done for primary secondary and tertiary hyperparathyroidism, or in some cases of total thyroidectomy.

Case Report

Our patient was a 38-year-old female with a past medical history of ESRD who presented to the emergency room complaining of numbness and twitching of her face and hands for two days.

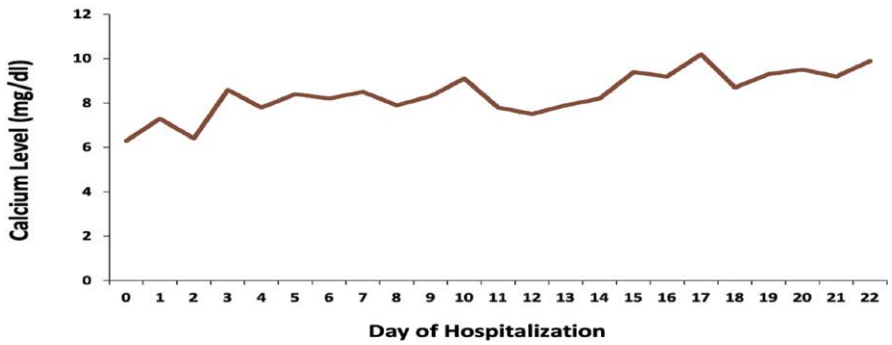
She was doing well ten days before the presentation when she underwent a parathyroidectomy with parathyroid auto-transplantation due to bone mineral disease secondary to ESRD. The patient signed out against medical advice from the hospital and was lost to follow up with no monitoring of her calcium levels and presented to the hospital ten days after surgery with complaints of numbness and twitching of her face and hands. Physical examination was positive for carpopedal spasm. Initial blood work in the emergency room revealed a calcium level of 6.3mg/dL, a magnesium level of 2.1mg/dL, and a phosphorous level of 3.3mg/dL. Before surgery, the patient had a PTH-i of 2227.5pg/mL, a calcium level of 10.4 pg/mL, and alkaline phosphatase of 1315 (IU/L). EKG showed normal sinus rhythm with a prolonged QTc interval of 527ms. The patient was admitted for severe hypocalcemia with cardiac manifestation resulting from HBS.

Course of Treatment: The patient was initially started on 4 grams of intravenous calcium gluconate repeated every 24 hours. After initial stabilization, 1250 grams of oral calcium carbonate three times a day was added. Despite aggressive calcium replacement after 36 hours, her corrected calcium was 7.4mg/dl. The calcium level was finally stabilized after 20 days on an up-titrating dose of calcium carbonate, calcitriol, and intravenous calcium. She was discharged on 12g of calcium to be taken in four divided doses on non-dialysis days and 24g of calcium every 6 hours on dialysis days. The patient was also instructed to take 2 µg of calcitriol twice daily. On follow up at 6 weeks the patient was able to maintain her calcium level on oral calcium and calcitriol.

Discussion/Conclusion

HBS is a severe complication of parathyroidectomy, with greater prevalence in patients with secondary hyperparathyroidism.[1] Parathyroidectomy is required in about 20% of patients after 3-10 years of dialysis and in up to 40% after 20 years.[2] The incidence of HBS post-parathyroidectomy in patients with primary hyperparathyroidism is reported to be 13%, compared to 28-88% in patients with secondary hyperparathyroidism.[1,3]

Predictors for developing HBS are ESRD on dialysis, age ≤ 45 years, high preoperative serum ALP (>420 IU/L), iPTH of >1000 pg/mL, and absence of preoperative hypercalcemia (>10.2 mg/dL).[3] The hypocalcemia seen in HBS can vary from mild to morbidly severe. Severe hypocalcemia can have high mortality and morbidity including neurological manifestation like seizures, cardiac manifestation like arrhythmias, QTc prolongation and cardiomyopathy and laryngeal spasm. Hypocalcemia with neurological manifestation or QTc prolongation warrants treatment with intravenous calcium for stabilization followed by oral calcium once the patient can tolerate. Patients with risk factors may benefit from preoperative vitamin D administration or its active metabolites.[4] Other authors have proposed that administering bisphosphonates pre-operatively may reduce the incidence of post-operative HBS.[5] Management of HBS requires a multidisciplinary approach and optimization of oral calcium and vitamin D dosing. Development of HBS should always be kept in mind in a post-operative patient undergoing parathyroidectomy.



Endnotes

1. Jakubauskas M, Beiša V, Strupas K. Risk factors of developing the hungry bone syndrome after parathyroidectomy for primary hyperparathyroidism. *Acta Med Litu*. 2018;25(1):45-51. doi:10.6001/actamedica.v25i1.3703
2. Tominaga Y, Uchida K, Haba T, Katayama A, Sato T, Hibi Y, et al. More than 1,000 cases of total parathyroidectomy with forearm autograft for renal hyperparathyroidism. *Am J Kidney Dis*. 2001;38:168–71.
3. Kritmetapak K, Kongpetch S, Chotmongkol W, Raruenrom Y, Sangkhamanon S, Pongchaiyakul C. Incidence of and risk factors for post-parathyroidectomy hungry bone syndrome in patients with secondary hyperparathyroidism. *Ren Fail*. 2020;42(1):1118-1126. doi:10.1080/0886022X.2020.1841655
4. Boyle IT, Fogelman I, Boyce B, Thomson JE, Beastall GH, McIntosh WB, McLennan I. 1alpha-hydroxyvitamin D3 in primary hyperparathyroidism. Clin Endocrinol (Oxf). 1977 Dec;7 Suppl:215s-222s. doi: 10.1111/j.1365-2265.1977.tb03384.x. PMID: 606419.
5. Lee IT, Sheu WH, Tu ST, Kuo SW, Pei D. Bisphosphonate pretreatment attenuates hungry bone syndrome postoperatively in subjects with primary hyperparathyroidism. J Bone Miner Metab. 2006;24(3):255-8. doi: 10.1007/s00774-005-0680-x. PMID: 16622740.

Two Guiding Principles:
(1) If it is Painless, Harmless and Cheap, Just Try It!

By Philip Kaplan, MD, FAAFP

We learn some skills from formal training, formal CME, reading, our colleagues, or as follows, through experience. "We don't learn from experience; we learn from reflecting on the experience"- *John Dewey*.[1] As I enter my 50th year of practice I reflect on the sources of what I know. Innovation and listening to advice from patients and others have augmented more formal sources of CME. I share three examples of such insights.

Sliver Under a Finger Nail

If the free edge of the sliver is too short to grab, classic treatment is to anesthetize the digit and lift the nail. Digit anesthesia is at best unpleasant. Also be aware that the thumb nail is not anesthetized by blocking the digital nerves on the lateral sides of the proximal phalanx. The proximal phalanx of the thumb is different from the fingers - it appears to be embryologically the middle phalanx, and the digital nerves are just lateral to the flexor tendons at the MP joint.[2]

Recipe: Soak the digit tip in warm soapy water for 15 minutes. (Note: you trim your nails after bathing because they are softer then). The sliver has separated the nail bed from the nail, so working over the sliver is painless and no anesthesia is needed. Incise a V into the nail over the sliver with a #11 blade with the apex of the V about half way down the nail. (Figure 1) This is a slow careful task, but rewarding. Lift the free edge of the excised portion of nail and remove. Then grasp the exposed free edge of the sliver and pull. Done.

Case report: I was hurrying through a task involving a scrap of shelving. It slipped, and I had a piece of lumber under my right (dominant) thumb nail. Expletive. I was not going to let a partner lift my nail.

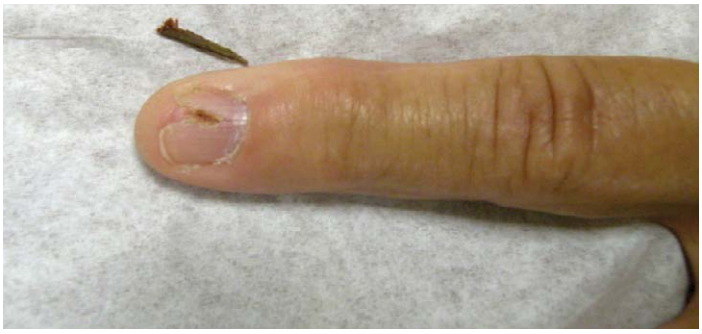


Figure 1

I soaked, positioned my thumb under bright light and magnification, held the #11 blade vertical and immobile in my left hand, and used my right hand dexterity to move my right thumb nail under the stationary blade. V removed, sliver lifted out. Absolutely painless.

Cerumen Impaction

Cerumen is fascinating stuff. There are ethnic, age and illness differences in cerumen physical characteristics. Sometimes this stuff blocks hearing, prevents visualization of the tympanic membrane, or is part of the syndrome of external otitis, trapping water. I started using a Water Pik ™ decades ago after seeing a journal article suggesting this, with some success, but some cerumen just resisted removal.[3] A patient came home from a trip excited about an ear drop given him in Boston for external otitis – Vosol HC. Decades ago it was glycerin, hydrocortisone and glacial acetic acid. The current product uses propylene glycol in place of glycerin. Either vehicle is hygroscopic; it absorbs water without cooling the tympanic membrane as does evaporating propanol. The steroid likely reduces inflammatory adhesion of sick skin to cerumen. The non-aqueous acetic acid is known to inhibit pseudomonas growth. I send patients home to use this daily a few days before retrying to remove cerumen. The drops also seem helpful if used after swimming to prevent external otitis in those prone to recurrence. The brand is no longer available, but the generic is listed in Surescripts as "hydrocortisone acetic acid otic." An additional tool I found useful for decerumination is seen in figure 2, resembling a weapon from Star Wars.

Case report: My wife was about to have back surgery. We were called at 5 AM to come in early, as preceding cases were cancelled because of a snow storm north of us. I realized my right ear was plugged, I couldn't awaken a partner at that hour, and I was going to have to hear instructions all day long. I ran to the fridge, placed two drops of Vosol HC in my ear, went to the office, stuck a WaterPik in my ear, released a gob, and I could hear again. Surgery went well too.

Figure 2

Poison Ivy

The dermatitis starts about two days after exposure and lasts for two weeks. Folklore suggests agents for sap removal to prevent or treat the malady: kerosene, gasoline, laundry detergent. Systemic steroids help some, topical steroids not much. It seems to me that the prolonged dermatitis may be related to incomplete removal of the adhesive invisible plant sap, as well as absorption described in reference 4.

continued on page 50

Management of Lower Extremity Chronic Venous Insufficiency

By Angelo Materia and Theodore J. Blum, MD, FAAFP

Abstract

Chronic venous Insufficiency (CVI) is a common vascular disease with debilitating effects on the lower extremities. In particular, ulcerations and venous stasis accompanied by pain are bothersome and can limit movement. Treatment for lower extremity CVI is a multi-layered approach and key aspects include symptomatic management as well as prevention of recurrence. The initial step of managing lower extremity CVI is through conservative methods such as compression stockings and exercise programs. Surgical methods are pursued if conservative methods fail or symptoms worsen.

Introduction

Chronic venous insufficiency encompasses a wide variety of longstanding abnormalities related to venous hypertension and effects roughly 50% of individuals. Causes of venous hypertension include but are not limited to; inadequate muscle pump function, incompetent venous valves resulting in reflux, venous thrombosis, and non-thrombotic venous obstruction.[1,2] Risk factors include advancing age, prolonged standing, elevated BMI, and smoking.[3]

In this case report, we discuss the management of a patient where conservative management was successful in treating ulceration of one lower extremity but this same management was not effective when ulceration occurred on the other lower extremity. The case report will aim to evaluate whether there is a benefit of treating the unaffected extremity as well as the affected extremity in order to prevent the development of complications especially in those where exercise and compression stockings are not viable for the patient.

Case Report

Our patient presented in an outpatient setting to her PCP. Our patient was a 65-year-old female w/ PMH of multiple sclerosis, osteopenia, chronic lymphedema, and bilateral venous stasis ulcers with edema who came in for a 1 week follow up of an ulceration on her left lower extremity (LLE). The patient was previously seen by vascular surgery for an ulceration on her RLE and at the time conservative management was recommended. She was treated for the ulcers on her RLE via an Unna's boot, which helped clear the ulceration. The patient then developed an ulceration in the LLE months later and returned to her PCP. The patient's visit from one week prior showed no signs of ulceration on her initially affected right leg. The patient demonstrated tenderness and a 2cm x 1.5cm ulcer was observed on her left leg. There were no signs of surrounding erythema or infection. The patient was treated with an Unna's boot and was to follow up in a week. Her return for follow up was as such:

During the patient's most recent visit her vitals were WNL, however she continued to endorse pain with movement and the Unna's boot was removed. Physical exam of the ulcer on the left leg continued to measure 2cm x 1.5cm and was visibly deeper with surrounding erythema and signs of infection. The patient's leg was wrapped again in an Unna's boot to provide further compression of the leg until she could meet with the vascular surgeon. The patient was given a 7-day course of Clindamycin 300 mg BID to cover for Gram (+) organisms and MRSA.

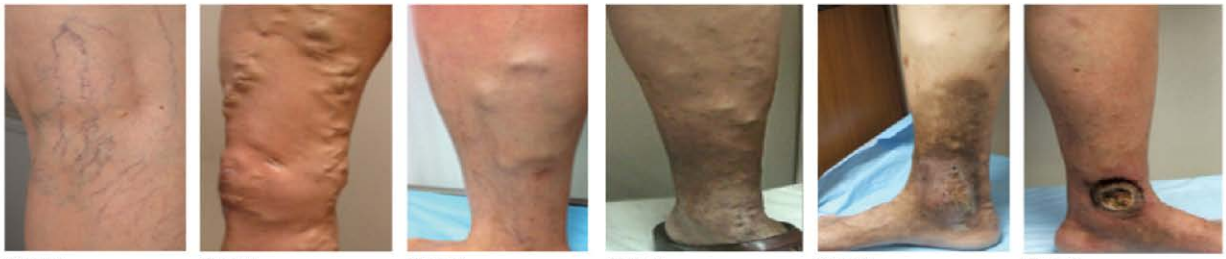
continued on page 28

Table 1: CEAP categories for clinical classification of chronic venous disorders[12]

Table 1. CEAP Classification - Clinical, Etiologic, Anatomic, Pathophysiologic

C-Clinical Class	Characteristics*	
0	No clinical findings or symptoms	E-Etiology**
1	Telangiectasia or reticular veins	C Congenital
2	Varicose veins	S Secondary
3	Edema, only due to a venous etiology	P Primary
4	(a) Pigmentation and/or eczema	A-Anatomy**
	(b) Lipodermatosclerosis, atrophie blanché	S Superficial (Great and short saphenous systems as well as any branch varices)
5	Prior ulceration, dermatitis	P Perforator (Veins that communicate between the superficial and deep systems)
6	Active ulceration	D Deep (Calf veins and sinuses, popliteal, femoral, iliac veins and vena cava)
A, S	Subscript: Asymptomatic, Symptomatic	P-Pathophysiology**
Date	Date of investigation	R Reflux
Level	Level of investigation (I, II, III)	O Obstruction
		R-O Both
		N** No evident disease**

*Complaints are expected to be related to venous insufficiency and are not classified if another etiology is present (i.e. edema secondary to heart failure).
**The N subscript indicates no evidence of disease. It is applicable to E, A, and/or P of CEAP.



Class 1: Telangiectasia. Class 2: Varicose vein. Class 3: Edema. Class 4: Pigmentation / Eczema. Class 5: Healed Ulcer. Class 6: Venous Ulcer.

continued from page 27

Given the development of this ulcer on the other lower extremity refractory to Unna's boot treatment and new onset infection, the patient was advised to follow up with the vascular surgeon. The vascular surgeon conducted an extremity venous duplex of the affected extremity to locate the location of venous reflux, which would help guide further management. Results identified venous reflux in the deep veins of the LLE and was negative for superficial reflux. It is unclear what the patient and vascular surgeon decided for the next step in management due to loss of follow up. However, in the discussion section of this report we discuss guidelines for treatment based on our patient's findings from the extremity venous duplex.

Discussion

Chronic venous insufficiency is common in the general population with a higher prevalence in older individuals. One of the most common clinical manifestations of chronic venous disease are lower extremity changes including stasis dermatitis and ulcers. Severity of chronic venous disease is based on the variety of the presentation of these lower extremity changes.[1] Classification of CVI is categorized using the Clinical-Etiology-Anatomy-Pathophysiology (CEAP) system. This classification system implements characteristics of the disease ranging from mild (C0) to severe (C6).[11] The severity of disease appears to be related to the degree of venous valve incompetence.[4] The diagnosis of CVD is suggested by the presence of clinical symptoms and physical exam findings (see Table 1).

Management differs depending on the severity of clinical features and degree of debilitation on everyday life. Diagnosis is typically made clinically by looking for characteristic features such as varicose veins and stasis dermatitis. However, to confirm the diagnosis, a venous Doppler ultrasound is obtained and typically shows bidirectional flow of venous blood across the valves.[5]

Valve incompetence is the primary anatomic abnormality associated with CVI. CVI is associated with fewer valves per unit length when compared to normal anatomic variation. This ultimately results in higher venous pressure due to maximally distended superficial veins, preventing adequate venous return to the right side of the heart.[9] The incompetent valves cause an absence of reflex vasoconstriction upon standing which is normally seen in unaffected individuals. This resultant pressure increase is responsible for the classic skin characteristics seen in patients with CVI.[10]

Treatment for asymptomatic patients depends on the degree of stress the patient experiences due to the appearance of their veins. Cosmetic treatments such as sclerotherapy are not covered by insurance and typically require several sessions in order to be effective.[6] These treatments do not prevent the future development of venous reflux.

For patients that are symptomatic, non-operative measures are recommended. An exercise program is the first conservative measure taken. However, patients who are unable to adequately fulfill an exercise program should attempt compression therapy.[1,2] Studies suggest Unna's boot treatment to be superior to hydro-active dressing treatments for compressive therapy.[7] In addition, there are fewer complications associated with Unna's boot therapy compared to hydro-active dressing.[7] Other recommended conservative measures include bed rest with leg elevation to allow blood to flow back to the heart. This provides a more mechanical approach for blood return which is typically impeded by valve incompetence in these patients.

Pneumatic compression pumps, which had initially been used for the prevention of DVT and PE, have also been used in the management of wound healing and limb volume reduction in CVI.[13] This has been especially beneficial in patients that cannot tolerate sustained compression (ex: compression stockings).

For patients with chronic and recurrent symptoms the next step in management is dependent on factors such as response to conservative measures, ongoing symptoms, extent of disease, presence of venous reflux and patient expectations. Therefore, patients with recurrent ulcers can receive superficial venous ablation treatment.[8] However, our patient had no evidence of superficial venous reflux so would not qualify for this treatment. Since the patient had evidence of deep venous reflux she would be a candidate for endovascular approaches such as angioplasty/stenting which would improve venous outflow and reduce symptoms. Other options for deep venous valve incompetence include the implantation of mechanical valves or the creation of deep vein valves.[14] Any patient with lower extremity changes secondary to CVI that is refractory to conservative treatment is advised to speak with a vascular surgeon to decide whether more invasive treatment is the right plan of action.

In our patient's case, not only did her ulcers recur, but the treatment (Unna's boot) which was once effective on her previous ulcers was no longer able to adequately treat her new ulcers. The question arises whether prophylactic treatment such as pneumatic compression pumps after successful conservative treatment for one extremity should be implemented to prevent recurrences (both in the same and/or other extremity), especially in those that cannot tolerate sustained compression for long periods of time. Further studies and trials may be needed to determine if there is significant clinical improvement with this therapeutic regimen and whether this affects the rate of surgical vascular intervention in these patients as well. These studies should also determine whether the clinical benefit is dependent on the location and type of venous reflux in the lower extremities.

Endnotes

1. Gloviczki P, Comerota AJ, Dalsing MC, et al. The care of patients with varicose veins and associated chronic venous diseases: clinical practice guidelines of the Society for Vascular Surgery and the American Venous Forum. J Vasc Surg 2011; 53:2S.
2. O'Donnell TF Jr, Passman MA. Clinical practice guidelines of the Society for Vascular Surgery (SVS) and the American Venous Forum (AVF)--Management of venous leg ulcers. Introduction. J Vasc Surg 2014; 60:1S.
3. Brand FN, Dannenberg AL, Abbott RD, Kannel WB. The epidemiology of varicose veins: the Framingham Study. Am J Prev Med 1988; 4:96.
4. Chiesa R, Marone EM, Limoni C, et al. Chronic venous disorders: correlation between visible signs, symptoms, and presence of functional disease. J Vasc Surg 2007; 46:322.
5. Coleridge-Smith P, Labropoulos N, Partsch H, et al. Duplex ultrasound investigation of the veins in chronic venous disease of the lower limbs--UIP consensus document. Part I. Basic principles. Eur J Vasc Endovasc Surg 2006; 31:83.

continued on page 30

Severe Dizziness from Unintentional Marijuana Edible Ingestion

By Patricia Wuu, DO; Sungsil Cho, MD and Cameron E. Nienaber, MD

Abstract

This is the case of a 62-year-old woman presenting with severe, sudden-onset dizziness. Although otherwise stable, she was admitted due to inability to ambulate. Further investigation revealed that the patient had unknowingly consumed her son's marijuana edibles, which was confirmed by urine toxicology positive for cannabinoids. Imaging ruled-out neurologic etiologies, and her symptoms gradually improved over several days. This case illustrates a discrete sequela of increasing marijuana use, especially of marijuana edibles that look like regular snack foods. Concerns regarding inadvertent ingestion of edibles have been broadly studied in the pediatric literature; this case demonstrates the potential for harm in the adult population.

Introduction

The use of marijuana in various forms continues to increase as many states have moved to legalize its use in recent years.[1] Marijuana edibles - food, beverage, and other snacks infused with cannabinoids - have become more widely available and consumed, increasing the possibility of unintentional ingestion in susceptible populations. Legalization of marijuana has additionally affected public perception of potential harm(s) from consumption, particularly the idea that marijuana edibles are safe.[2,3] Studies have shown distinct differences between consumption of marijuana edibles versus by smoking, such as delayed latency of drug effects with edibles that places patients at higher risk of marijuana overdose.[4,5]

The effects and risks of marijuana ingestion in children and adolescents have been well-published in the pediatric medical literature.[6] However, there are fewer case studies of accidental marijuana ingestion in adults,[7] and emerging literature suggests that there are clinically significant differences in the acute effects of marijuana in older patients.[8] Older patients are arguably at increased risk for adverse medication-marijuana interactions and marijuana toxicity, as they are more likely to have comorbid conditions requiring chronic medications.[9] Concerns about the increasing prevalence of marijuana edibles continue to be focused on the appropriately-defined pediatric population that is at risk for adverse effects from marijuana. However, we believe it is important for clinicians to apply these same considerations to all age groups, including the adult population that may not be typically considered a susceptible population. In this report, we present the case of a woman who presented to our hospital with sudden-onset, severe vertigo after accidental ingestion of marijuana edibles.

Case Presentation

Our patient is a 62-year-old woman with multiple medical conditions, including major depressive disorder and anxiety, coronary artery disease, hypertension, and non-insulin-dependent diabetes who presented to the emergency room complaining of sudden-onset dizziness and one episode of non-bloody, non-bilious emesis. She reported suddenly feeling very dizzy while at home. Prior to onset of symptoms, the patient was cleaning her home as part of her daily routine. She denied any changes in her chronic medications, and she took them as usual that morning. She noted a similar but less severe episode of dizziness in the past about one year ago due to a urinary tract infection that required intravenous antibiotics at that time.

Our patient was hemodynamically stable on initial evaluation in the emergency room. Significant exam findings included ataxia and dysdiadochokinesia without other neurologic findings. Initial work-up, including CBC, BMP, troponin, UA, COVID testing, EKG, and CXR were unremarkable. A non-contrast CT of the head did not reveal any acute findings.

Collateral information was then obtained from the patient's two sons with whom she lives. At that time, the patient's son shared a concern that his mother's symptoms may be from ingestion of marijuana edibles. He reported that he had had a full bag of "Doritos" marijuana edibles in his room that now had only crumbs left. Our patient corroborated that she had consumed her son's Doritos while cleaning his room prior to onset of symptoms. Subsequent urine toxicology returned positive for cannabinoids.

The patient required admission due to inability to ambulate and persistent dizziness and nausea. Patient received treatment addressing other possible causes of her symptoms including intravenous fluid hydration; and the patient's medications were reviewed and possible offending agents discontinued. Neurology was consulted to rule-out neurological etiologies, and non-contrast MRI of the brain showed no acute infarctions or hemorrhages but showed a chronic ischemic focus in the right thalamus. Patient was trialed on antiemetic medications, including ondansetron and meclizine, with mild improvement of nausea. The patient underwent a physical therapy evaluation as her symptoms were improving, and the patient was recommended a rolling walker for continued stability with ambulation. Patient remained hemodynamically stable with continued improvement of her symptoms, and was discharged home after 4 days.

The patient was subsequently seen at our family medicine clinic after discharge and reported complete resolution of her symptoms.

Discussion

Concerns regarding unintentional ingestion of cannabis edibles in the pediatric and adolescent populations has been broadly published in recent medical literature, but while identified as a population at high risk of adverse effects and/or harm from accidental cannabis edible ingestion,[10,11] there are fewer case studies demonstrating discrete evidence of such in older adults.

continued on page 30

continued from page 28

6. American Board of Plastic Surgery www.abplsurg.org/ModDefault.aspx (Accessed on March 31, 2009).
7. A prospective, randomized trial of Unna's boots versus hydroactive dressing in the treatment of venous stasis ulcers. M J Kikta 1, J J Schuler, J P Meyer, J R Durham, J Eldrup-Jorgensen, T H Schwarcz, D P Flanigan
8. Gohel MS, Heatley F, Liu X, et al. A Randomized Trial of Early Endovenous Ablation in Venous Ulceration. N Engl J Med 2018; 378:2105.
9. Sales CM, Rosenthal D, Petrillo KA, et al. The valvular apparatus in venous insufficiency: a problem of quantity? Ann Vasc Surg 1998; 12:153.
10. Browse NL. The etiology of venous ulceration. World J Surg 1986; 10:938.
11. Kabnick, Lowell S. "Overview of Lower Extremity Chronic Venous Disease." *UpToDate*, https://www.uptodate.com/login.
12. Esteem Surgical Care. (n.d.). Veno Leg Vein Clinic. Retrieved February 12, 2022, from https://veno.com.hk/varicose-vein.html
13. White-Chu, E. F., & Conner-Kerr, T. A. (2014, February 11). *Overview of guidelines for the prevention and treatment of venous leg ulcers: A US perspective*. Journal of multidisciplinary healthcare. Retrieved January 29, 2022, from https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3930479/
14. Weber B, Hafner J, Willenberg T, Hoerstrup SP. Bioengineered valves for the venous circulation. Expert Rev Med Devices 2016; 13:1005.

continued from page 29

The resemblance of cannabis edibles to non-cannabis food and drink items such as baked goods, candies, and beverages is strongly thought to contribute to accidental ingestion and overconsumption of cannabis edibles in the pediatric population.[11,12,13] A study done in 2018 examining THC content in commercially available cannabis edibles further cited the lack of product constraints in flavor, packaging, and form as an additional concern for the pediatric population. While these statements and precautions are traditionally and appropriately geared towards the pediatric population that is at high-risk for accidental cannabis ingestion, as demonstrated by this case study, it is important to apply such precautions to all patients, such as in the case of a non-discerning adult.[12] This case illustrates the risk posed by marijuana edibles to all patients, regardless of age, and highlights a potentially overlooked diagnosis in patients presenting with acute neurological or cognitive changes. This case also emphasizes the need for education and advocacy by healthcare providers around marijuana regulation and appropriate labeling by manufacturers and distributors, as well as for responsible storage of marijuana products by individual users.

Endnotes

1. Hasin DS, Saha TD, Kerridge BT, Goldstein RB, Chou SP, Zhang H, Jung J, Pickering RP, Ruan WJ, Smith SM, Huang B, Grant BF. Prevalence of Marijuana Use Disorders in the United States Between 2001-2002 and 2012-2013. JAMA Psychiatry. 2015 Dec;72(12):1235-42. doi: 10.1001/jamapsychiatry.2015.1858. PMID: 26502112; PMCID: PMC5037576.
2. Miech RA, Johnston L, O'Malley PM, Bachman JG, Schulenberg J, Patrick ME. Trends in use of marijuana and attitudes toward marijuana among youth before and after decriminalization: the case of California 2007-2013. Int J Drug Policy. 2015 Apr;26(4):336-44. doi: 10.1016/j.drugpo.2015.01.009. Epub 2015 Jan 14. PMID: 25662893; PMCID: PMC4469374.
3. Okaneku J, Vearrier D, McKeever RG, LaSala GS, Greenberg MI. Change in perceived risk associated with marijuana use in the United States from 2002 to 2012. Clin Toxicol (Phila). 2015 Mar;53(3):151-5. doi: 10.3109/15563650.2015.1004581. Epub 2015 Feb 3. PMID: 25646638.
4. Grotenhermen F. The toxicology of cannabis and cannabis prohibition. Chem Biodivers. 2007 Aug;4(8):1744-69. doi: 10.1002/cbdv.200790151. PMID: 17712818.
5. Barrus DG, Capogrossi KL, Cates SC, Gourdet CK, Peiper NC, Novak SP, Lefever TW, Wiley JL. Tasty THC: Promises and Challenges of Cannabis Edibles. Methods Rep RTI Press. 2016 Nov;2016:10.3768/rtipress.2016.op.0035.1611. doi: 10.3768/rtipress.2016.op.0035.1611. PMID: 28127591; PMCID: PMC5260817.
6. Richards JR, Smith NE, Moulin AK. Unintentional Cannabis Ingestion in Children: A Systematic Review. J Pediatr. 2017 Nov;190:142-152. doi: 10.1016/j.jpeds.2017.07.005. Epub 2017 Sep 6. PMID: 28888560.
7. Centers for Disease Control and Prevention (CDC). Inadvertent ingestion of marijuana - Los Angeles, California, 2009. MMWR Morb Mortal Wkly Rep. 2009 Sep 4;58(34):947-50. PMID: 19730407.
8. Mueller RL, Ellingson JM, Bidwell LC, Bryan AD, Hutchison KE. Are the Acute Effects of THC Different in Aging Adults? Brain Sci. 2021 May 1;11(5):590. doi: 10.3390/brainsci11050590. PMID: 34062795; PMCID: PMC8147270.
9. Hall W, Solowij N. Adverse effects of cannabis. Lancet. 1998 Nov 14;352(9140):1611-6. doi: 10.1016/S0140-6736(98)05021-1. PMID: 9843121.
10. Ammerman, Seth. Counseling Parents and Adolescents About Marijuana. Am Fam Physician. 2018 Jul 15;98(2):80-81.
11. Grewal JK, Loh LC. Health considerations of the legalization of cannabis edibles. CMAJ. 2020;192(1):E1-E2. doi:10.1503/cmaj.191217
12. Steigerwald S, Wong PO, Khorasani A, Keyhani S. The Form and Content of Cannabis Products in the United States. J Gen Intern Med. 2018;33(9):1426-1428. doi:10.1007/s11606-018-4480-0
13. Zipursky JS, Bogler OD, Stall NM. Edible cannabis. CMAJ. 2020;192(7):E162. doi:10.1503/cmaj.191305

Necrotic Pulmonary *Mycobacterium Tuberculosis* Infection Masquerading as Covid-19

By Elizabeth Harding, MD and Anthony Burdo, MD

Abstract

Since the beginning of the Covid-19 pandemic, there was a marked decrease in tuberculosis incidence in the United States, concerning for under-testing.[1] A 40-year-old Burmese immigrant with a history of treated latent tuberculosis infection (LTBI) and alcohol use was admitted for suspected COVID-19 pneumonia, with consistent CT findings and anosmia/ageusia. He was discharged with antibiotics for community-acquired pneumonia (CAP). Eighteen days later he was readmitted for 124 days with necrotic *Mycoplasma tuberculosis* pneumonia, complicated by lung abscess, bronchopleural fistula, hydropneumothorax, exudative effusion, SIADH and recurrent SVTs. The low index of suspicion for tuberculosis resulted in a regrettable delay in diagnosis and treatment.

Introduction

The global prevalence of tuberculosis based on a meta-analysis of studies from 2005-2018, is estimated to be around 24.8%.[2] From 2019 to 2020, it is estimated that globally the number of people who received medical care for TB declined by 1.4 million people, or 21%.[3] One study of 13,805 foreign-born individuals in the US found a prevalence of 31%,[4] though this varied widely by country of birth.

This case emphasizes that tuberculosis (TB) reactivation, or even re-infection can occur in the US. Although all refugees are screened for active TB disease overseas, latent tuberculosis infection (LTBI) testing is often left to local health departments or family doctors. This case illustrates the need for continued surveillance and follow-up with patients with LTBI, and for heightened awareness of community transmission of TB, as local rates may vary widely.

Given that our patient's isolate was pan-sensitive to first line antibiotics, it is quite possible he was re-infected in the United States.

Case Report

A 40-year-old Burmese refugee to the United States, had a positive Interferon-gamma Release Assay (IGRA) in April 2013. Subsequently, he was adequately treated for latent tuberculosis infection (LTBI) with 9-months of isoniazid in 2014.

In June 2020, He presented to our family medicine clinic with approximately two-to-three weeks of non-productive cough, chest pain, loss of taste, fevers at night and generalized weakness. His past medical history was significant for alcohol use and seizures. He had not traveled internationally since his immigration to the US. He was sent to the emergency department where he had diminished lung sounds with basilar crackles. BP was 90/60, heart rate 130 bpm. Blood cultures were negative. He had a leukocytosis (17.1 x 10^9/L) with lymphopenia (0.6 x 10^9/L). CT chest showed: *"Extensive organizing pneumonia on the left with volume loss. Early abscess formation not excluded. Multifocal pneumonia on the right."* No acid-fast bacilli sputum stain or culture was done. Respiratory viral panel, including COVID-19 PCR was negative.

Based on these findings and his length of symptoms, he was treated empirically for community-acquired pneumonia. He received 2 days of ceftriaxone and azithromycin inpatient with improvement in leukocytosis, fever, and dyspnea. He was discharged with a further 3 days of Augmentin and 7 days of PO dexamethasone, 6 mg daily. Respiratory viral panel, including COVID-19 PCR was negative, but at the time this was thought to be a false negative given his clinical and radiologic picture.

He presented again 18 days later (July 2020) with a recurrence of weakness, chest congestion, and cough which began after his antibiotics finished, as well as two episodes of near-syncope on standing. In the emergency department at the time of readmission, his blood pressure was 91/64, and his chest x-ray showed: *"Slight overall progressive mild right extensive left chest airspace lung opacity multifocal pneumonia. Development of minor cavitation or abscess at the left upper lobe."*

He was admitted and vancomycin and piperacillin-tazobactam were started. Sputum acid-fast stain was positive the next day, confirmed as *Mycoplasma tuberculosis* by PCR, and found to be pan-susceptible to first-line therapies: rifampin (R), isoniazid (INH) pyrazinamide, and ethambutol (collectively termed RIPE therapy in the US) and streptomycin. Vancomycin and piperacillin-tazobactam were stopped. Rifampin, levofloxacin, pyrazinamide and ethambutol were started, given prior LTBI treatment with INH. Fungal cultures returned negative.

He was admitted for a continuous 124 days (7/6-11/7), with a new diagnosis of necrotic *M. tuberculosis* pneumonia complicated by lung abscess, bronchopulmonary fistula, hydropneumothorax, exudative effusion, SIADH, sinus tachycardia and recurrent SVTs. He developed a persistent left hydropneumothorax, with areas of lung abscess on CT. Bronchopulmonary fistula was presumed, due to slow response of the hydropneumothorax to multiple chest tube placements. As a result, cardiothoracic surgery was consulted to consider partial pneumonectomy of the necrotic left upper lobe, however the mortality associated with the surgery was considered too high, and this was not pursued.

continued on page 32

continued from page 31

The patient was also confirmed to have hyponatremia (sodium of 124) due to syndrome of inappropriate ADH (SIADH) with urine electrolytes, and normal cortisol studies. There was no improvement when levetiracetam switched to phenytoin.

Recurrent SVTs, sinus tachycardia, and SIADH were worked up but showed no clear etiology, however they could certainly be explained by the patient's extensive intrathoracic disease.

Additionally, echocardiogram was unremarkable, and there were no signs of constrictive pericarditis.

Further, the patient was evaluated for causes of immunosuppression, which included negative HIV, HCV, and HBV tests. Ultimately, the patient's pre-albumin nadir of 6 mg/dL (normal range is 18-38) implied malnutrition. Further investigation of his social history revealed that he had escalated his alcohol intake in the months immediately prior to his illness onset, from 2-20 beer cans daily.

Due to the patient's prolonged hospitalization, he lost his housing and therefore was unable to live anywhere without a private entrance. Under regional public health policy, this necessitated that he remain in the hospital until there were three consecutive negative AFB stains (or cultures). He was discharged in November 2020 with six months of daily RIPE, levofloxacin, and pyridoxine, with the local health department performing direct-observed-therapy.

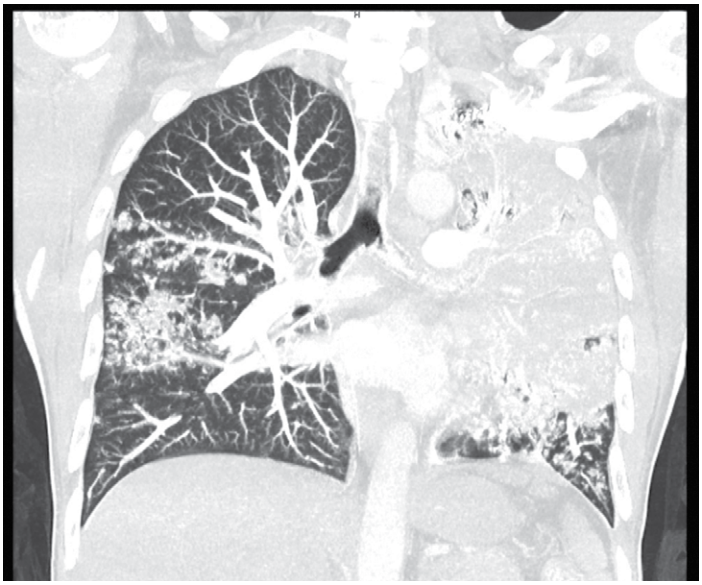
Discussion

The incidence of tuberculosis in the U.S. has been declining steadily since 1993, however Between 2019 and 2020, newly report cases decreased sharply by 20%.[1] This decrease likely largely reflected the impact of the COVID-19 pandemic on testing, community surveillance methods, and index of suspicion for COVID infection versus tuberculosis among providers in higher risk communities.

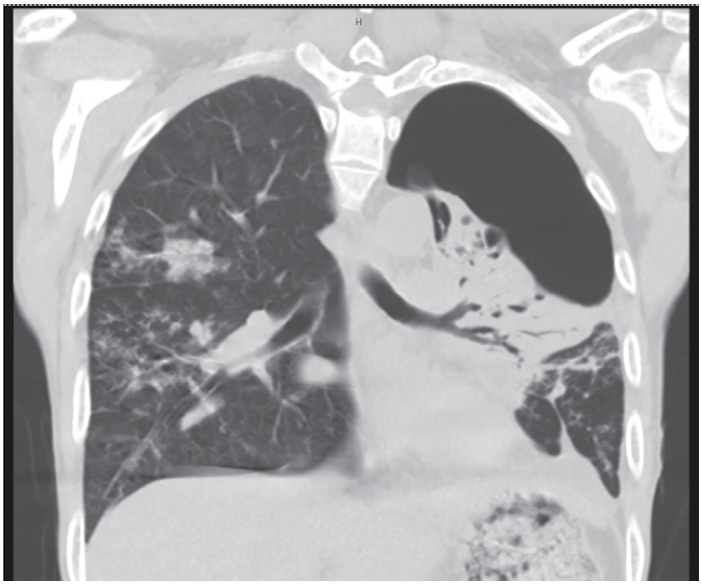
Under the present CDC/IDSA guidelines, tuberculosis screening is indicated for individuals from higher risk groups, e.g., immigrants from high prevalence countries, people who live or work in healthcare or institutional settings, and those with a known household exposure. Testing is not recommended for people who have been fully treated for TB, unless they have an additional risk (for example, frequent travel to a high-risk area or work in a high-risk setting).[5]

Since the start of the Covid-19 pandemic, there have been a multitude of papers written to caution against under diagnosis of other pulmonary diseases, particularly given that initial symptoms and risk factors are similar for Covid-19 and tuberculosis pneumonia.[6] Our case is remarkable because this patient had been adequately treated for LTBI, and initially had no risk factors for immunodeficiency, and appeared to respond to initial treatment with low-dose dexamethasone or ceftriaxone, azithromycin and amoxicillin-clavanulate. One might wonder whether either the steroid or antibiotic administration on his first admission masked his tuberculosis symptoms. This patient did not receive levofloxacin, which is notorious for masking TB symptoms and delaying proper diagnosis, and is not recommended for empiric CAP treatment in TB-endemic settings.[7] Although there is not clear evidence, it is generally thought that less than 2 weeks duration of oral corticosteroids has a low risk for LTBI reactivation.[8] Steroids should be judiciously prescribed, particularly in areas of higher TB prevalence.

Though testing for HIV, Hepatitis B and C were all negative, the patient was malnourished with a pre-albumin of 6 mg/dL and albumin of 2.5 g/dL. The patient's alcohol history was not completely understood initially. We learned after his tuberculosis diagnosis that he had a significant alcohol intake of 15-20 drinks daily in the weeks preceding his presentation. An accurate social, substance use, and nutritional history at the time of presentation is indispensable in



Coronal CT from 7/7/2020, read as: "Extensive organizing pneumonia on the left with some overall volume loss left hemithorax. Early abscess formation not excluded and close follow up advised. Multifocal pneumonia in the right lung. Prominent mediastinal lymph nodes"



This CT was obtained 7 weeks into patient's second admission, demonstrating hydropneumothorax, left lung necrosis and atelectasis due to bronchopleural fistula.

guiding the testing and diagnosis of infectious disease. Although he was seemingly immunocompetent on presentation, the combined biochemical evidence of malnutrition and history of heavy ethanol use support the conclusion that the patient was relatively immunocompromised prior to the onset of his active tuberculosis.

Conclusion

The patient did not work or live in what would be considered a high-risk setting. He did not have a history of chronic disease, except for alcohol abuse. The index of suspicion for tuberculosis, in the context of his COVID-19-like symptoms, was therefore low, resulting in a regrettable delay in diagnosis and treatment initiation. He represented an unexpected presentation of a common disease, which illustrated the potential for serious adverse outcomes resulting from our current surveillance guidelines for tuberculosis. More importantly, this case serves as a cautionary tale about the need for an index of suspicion for active tuberculosis in patients with protracted, treatment-resistant, or severe pneumonias, despite the relatively high incidence of COVID-19 related respiratory illness during the COVID-19 pandemic.

Endnotes

1. Deutsch-Feldman M, Pratt RH, Price SF, Tsang CA, Self JL. Tuberculosis — United States, 2020. MMWR Morb Mortal Wkly Rep 2021;70:409–414. DOI: http://dx.doi.org/10.15585/mmwr.mm7012a1external icon.
2. Cohen, A., Mathiasen, V. D., Schön, T., & Wejse, C. (2019). The global prevalence of latent tuberculosis: a systematic review and meta-analysis. *The European respiratory journal, 54*(3), 1900655. https://doi.org/10.1183/13993003.00655-2019
3. World Health Organization. Impact of the COVID-19 pandemic on TB detection and mortality in 2020, 2021. Available: https://www.who.int/publications/m/item/impact-of-the-covid-19-pandemic-on-tb-detection-and-mortality-in-2020 [Accessed 13 February, 2022].
4. Collins, J. M., Stout, J. E., Ayers, T., Hill, A. N., Katz, D. J., Ho, C. S., Blumberg, H. M., Winglee, K., & Tuberculosis Epidemiologic Studies Consortium (2021). Prevalence of Latent Tuberculosis Infection Among Non-US-Born Persons by Country of Birth-United States, 2012-2017. *Clinical infectious diseases : an official publication of the Infectious Diseases Society of America, 73*(9), e3468–e3475. https://doi.org/10.1093/cid/ciaa1662
5. Lewinsohn, D. M., Leonard, M. K., LoBue, P. A., Cohn, D. L., Daley, C. L., Desmond, E., Keane, J., Lewinsohn, D. A., Loeffler, A. M., Mazurek, G. H., O'Brien, R. J., Pai, M., Richeldi, L., Salfinger, M., Shinnick, T. M., Sterling, T. R., Warshauer, D. M., & Woods, G. L. (2017). Official American Thoracic Society/Infectious Diseases Society of America/Centers for Disease Control and Prevention Clinical Practice Guidelines: Diagnosis of tuberculosis in adults and children. *Clinical Infectious Diseases, 64*(2), 111–115. https://doi.org/10.1093/cid/ciw778
6. Keddy, K. H., Migliori, G. B., & Van Der Walt, M. (2020). Developing health policies in patients presenting with SARS-CoV-2: consider tuberculosis. *The Lancet. Global health, 8*(11), e1357–e1358. https://doi.org/10.1016/S2214-109X(20)30413-7
7. Wei, M., Yongjie Zhao, Zhuoyu Qian, Biao Yang, Xi, J., Wei, J., & Tang, B. (2020). Pneumonia caused by Mycobacterium tuberculosis. *Microbes and infection, 22*(6-7), 278–284. https://doi.org/10.1016/j.micinf.2020.05.020
8. Gopalaswamy, Radha, and Selvakumar Subbian. "Corticosteroids for COVID-19 Therapy: Potential Implications on Tuberculosis." *International journal of molecular sciences* vol. 22,7 3773. 6 Apr. 2021, doi:10.3390/ijms22073773

Upcoming Events

2022

May 21-22
Congress of Delegates
Desmond Hotel
Albany

August 6-7
Summer Cluster
Buffalo Marriott at
LECOM HARBORCENTER

Nov 6
Fall Cluster Board Meeting
(Commissions meet prior)
Albany Renaissance

2023

January 13-15
Winter Weekend and Scientific Assembly

February 26
Winter Cluster
Renaissance Hotel
Albany

February 27
Advocacy Day
Albany

August 5-6
Summer Cluster
Edith Macy Center
Briarcliff Manor

For updates or registration information for these events go to www.nysafp.org

What's that Discharge?

By Jasdeep Singh Bajwa, DO and Dominick Sam DeFelice, MD

Case Study

Abstract

A 36-year-old male presents to clinic for several weeks of persistent penile discharge. He was tested for gonorrhea, chlamydia, trichomonas, HIV and syphilis, and all were negative. On follow-up he was retested for these infections but also was tested for Mycoplasma genitalium (M. genitalium), which returned positive. He was treated with a course of doxycycline and azithromycin with no improvement in his symptoms. He was then given a course of moxifloxacin which completely resolved his symptoms. Our case not only highlights the importance of screening for M. genitalium in symptomatic patients but also sheds light on growing resistance to first line treatments.

Introduction

This case serves to alert family medicine clinicians to consider M. genitalium in men with persistent urethritis and highlight the possibility of increasing resistance, especially after treatment with doxycycline.

Case Report

A 36-year-old male with no relevant past medical history presented to his primary care physician's office for persistent penile discharge for four weeks. He described the discharge as cloudy but denied any yellowish or green discharge. He admitted to occasional dysuria. He denied any issues with completely emptying his bladder or pain with a long sitting. He denied noticing the penile discharge shortly after intercourse. The discharge happened randomly but most commonly occurred just prior to urination. Otherwise, he denied hematuria, fevers, chills, abdominal pain, nausea, vomiting or any type of rashes anywhere on his body. He stated that he had multiple sexual partners, specifically three female partners in the last six months. He did not use condoms regularly. His physical exam and vitals were completely normal. Milking of the urethra did not yield any discharge during the visit. Initial work up was completely negative (Table 1). His urinalysis revealed 2+ leukocyte esterase, however, was negative for nitrites, blood, or protein.

Table 1: Testing from initial visit

Variable	Value	Reference Range
Gonorrhea NAAT (Urine)	Negative	Negative
Chlamydia NAAT (Urine)	Negative	Negative
Trichomonas NAAT (Urine)	Negative	Negative
HIV (Blood)	Negative	Negative
Syphilis (Blood)	Negative	Negative

He was brought back to his primary care physician's office to repeat testing for gonorrhoea, chlamydia, trichomonas. This time however, he was also tested for M. genitalium via PCR test. His gonorrhoea, chlamydia, and trichomonas screens were negative, however his M. genitalium PCR was positive. He was given a course of doxycycline 100mg twice daily for seven days and was instructed to follow up in two weeks if his symptoms did not resolve. He was also offered PREP for HIV prophylaxis, which he declined at the time. He returned in two weeks with some resolution in his discharge, however he stated that he was still having some discharge, but the frequency perhaps had reduced. He was given azithromycin 1g which also did not help his symptoms. At the next follow-up, a week later, he was given a course of moxifloxacin 400mg daily for 7 days and was instructed that if his symptoms did not improve, he should follow up again. After completing his moxifloxacin course, he reported complete resolution of his symptoms.

Discussion

Infection with M. genitalium is a leading cause of non-gonococcal urethritis in men and a common cause of urethritis and cervicitis in women.[1] M. genitalium is clearly associated with both acute and persistent/chronic urethritis in men and accounts for one-third of non-gonococcal urethritis in men.[2,3] One should also consider M. genitalium infection in persistent or recurrent cervicitis or pelvic inflammatory disease (PID). Antimicrobial resistance in M. genitalium has shown a dramatic increase over the past decade and is now becoming a serious concern.[3] In the past, macrolides were 90% effective against M. genitalium, but further resistance has emerged. It is believed that the preference of macrolide for chlamydia treatment has led to selection of macrolide resistant M. genitalium strains. Furthermore, resistance against doxycycline is even worse as there is only a 31% cure rate when doxycycline is used to treat this infection.[4] Moxifloxacin appears to be the best choice given the high cure rate; it is recommended that a test of cure is done at 3-4 weeks post treatment.[1] Once treated for M. genitalium, one should abstain from intercourse for 7 days. Currently, the CDC does not recommend screening for asymptomatic partners, however this may change in the future as other guidelines are recommending otherwise.

From this case, family physicians should appreciate a comprehensive screen for symptomatic patients to include Mycoplasma genitalium. What made our case interesting was it appeared his infection was resistant to not only tetracyclines but also macrolides. It would have been beneficial if resistance testing for M. genitalium was more readily available, but in facilities where that may not be an option, perhaps treating with macrolide followed by a fluoroquinolone may be best practice.

Endnotes

1. Manhart, L. E., Broad, J. M., & Golden, M. R. (2011). Mycoplasma genitalium: should we treat and how? *Clinical Infectious Diseases: An Official Publication of the Infectious Diseases Society of America, 53 Suppl 3*(suppl_3), S129-42.
2. Jensen, J. S. (2004). Mycoplasma genitalium: the aetiological agent of urethritis and other sexually transmitted diseases. *Journal of the European Academy of Dermatology and Venereology: JEADV, 18*(1), 1–11.
3. Mycoplasma genitalium. (2021, July 14). Cdc.Gov.
4. van der Schalk, T. E., Braam, J. F., & Kusters, J. G. (2020). Molecular basis of antimicrobial resistance in Mycoplasma genitalium. *International Journal of Antimicrobial Agents, 55*(4), 105911.
5. Manhart, L. E., Jensen, J. S., Bradshaw, C. S., Golden, M. R., & Martin, D. H. (2015). Efficacy of Antimicrobial Therapy for Mycoplasma genitalium Infections. *Clinical Infectious Diseases: An Official Publication of the Infectious Diseases Society of America, 61 Suppl 8*(suppl 8), S802-17.

Case Report: Eruptive Skin Lesions in a Young Male

By Amit Sharma, DO; Constantino Lambroussis, DO, MS and Madeeha Lughmani, DO

Abstract

Eruptive xanthomas are rare rapidly appearing cutaneous lesions most commonly located on extensor skin surfaces. These lesions are mainly asymptomatic with underlying metabolic disorders but can have serious complications. We report a case of a 33-year-old diabetic male with a sudden eruption of grouped papules on elbows, arms, thighs, and lower back. The patient was diagnosed with eruptive xanthomas with a triglyceride level of 1,397 mg/dl. Treatment of his underlying disease led to the resolution of his cutaneous lesions.

Introduction

Xanthomas are localized deposits of lipids on different areas in the skin.[1,2] Eruptive xanthomas are a rare uncommon variant that may appear rapidly. These lesions may be primary or due to underlying metabolic disorders.[3] Characteristic presentation of these lesions are rapid onset of small yellowish papules with an erythematous halo mostly noted on a patient's extensor surfaces.[3] Eruptive xanthomas are suggestive of elevated triglyceride levels, and can also be an early sign of a patient with uncontrolled diabetes mellitus.[3] It is important for family physicians to be able to quickly recognize and start appropriate treatment of eruptive xanthomas to prevent potential life-threatening complications.

Case Report

A 33-year-old male with a history of diabetes mellitus, hyperlipidemia, and gout presented to the office with generalized skin lesions. The patient reported that the lesions began on his right elbow approximately one month prior to being seen, and then spread bilaterally on the arms, thighs, and low back regions. The patient denied any itching, tenderness, or change in character of the lesions.

A few days prior to his skin lesions initially appearing, he reports that he received his first dose of the COVID vaccine. The patient does also report that he has also been recently exposed to a rat infestation at his work in wheat breeding.

Physical examination revealed multiple groups of yellow dome-shaped 2 to 5 mm papules on his elbows (*Figure 1*), low back (*Figure 2*), and thighs.

Our patient was diagnosed with eruptive xanthomas. Serum triglyceride level obtained following the office visit was 1,397 mg/dL, and review of previous records showed a prior triglyceride level of 1,280 mg/dL. Tissue biopsy was performed and showed foam cells, mixed inflammatory infiltrate, as well as extracellular lipid deposits. The tissue biopsy confirmed the diagnosis of eruptive xanthomas. The patient reported that he had stopped taking his diabetic and hyperlipidemia medications prior to the development of the eruptive xanthomas. The patient's hemoglobin A1c (HbAlc) at the office visit was elevated at 11.3. Upon treatment of the hypertriglyceridemia with statins and fibrates our patient's xanthomas resolved. With increased compliance of diabetic medications, patient's HbAlc improved to 7.7.

Discussion

Circulating plasma lipoproteins are believed to be the origin of xanthomas.[1,2] Eruptive xanthomas are a rare xanthoma presenting secondary to elevated levels of triglycerides or uncontrolled diabetes mellitus, with a further elevated risk if a patient is afflicted with both conditions.[3] Through a thorough history and physical examination, xanthomas can be diagnosed with confirmation of diagnosis obtained via biopsy. The lipid composition gives eruptive xanthomas their traditional yellowish coloration within small papules that are in the 1 to 5 mm size range typically identified on extensor surfaces as well as the buttocks of the patient.[4] Patients with impaired glucose tolerance, as well as those with diabetes, are at elevated risk for hypertriglyceridemia, which is also a known cause of pancreatitis.[5]

Regarding triglyceride levels and risk, 5 mmol/L has an increased risk, 10 mmol/L has a high risk for pancreatitis, and 20 mmol/L carries a very high risk for pancreatitis. Patients with persistent triglyceride levels greater

continued on page 37

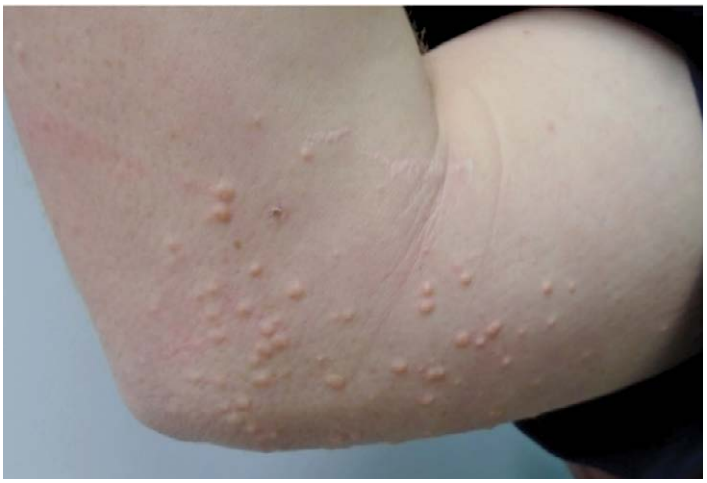


Figure 1

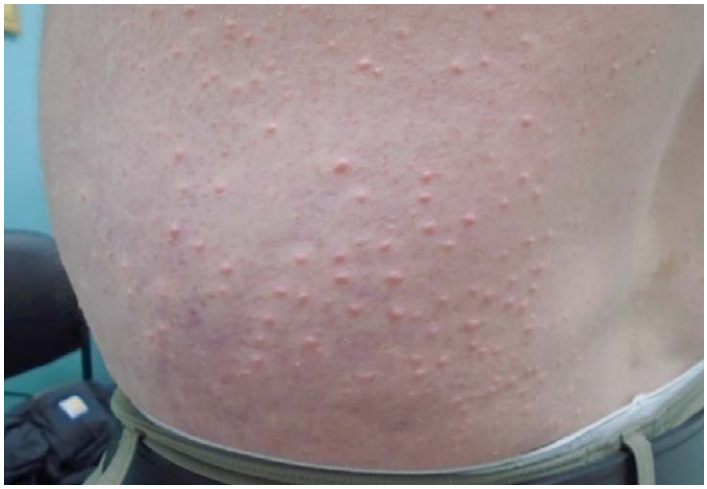


Figure 2

An Uncommon Cause of Abdominal Pain: Mesenteric Panniculitis

By Yaritza Serrano Gomez, MD; Farideh Zonouzi- Zadeh, MD, FAAFP and Sara Guevara, MD

Abstract

Mesenteric panniculitis is a rare, benign, and chronic fibrosing inflammatory disease affecting the adipose tissue of the mesentery that can lead to intractable abdominal pain. We report a case of a 74-year-old male with multiple autoimmune disorders, including paroxysmal nocturnal hemoglobinuria (PNH) and hypothyroidism, that presented to the emergency department with abdominal pain for over three weeks. Abdominal CT showed diffuse stranding of the mesenteric fat compatible with mesenteric panniculitis. The patient received treatment with steroids, which led to resolution of his abdominal pain.

Introduction

Abdominal pain is one of the most common conditions that call for prompt diagnosis and treatment.[1] There are multiple etiologies of abdominal pain, from the more benign (e.g., gastroenteritis) to the more severe causes (e.g., bowel incarceration). Common causes include cholelithiasis, hepatitis, liver abscess, pancreatitis, peptic ulcer disease, acute myocardial infarction, appendicitis, nephrolithiasis, urinary tract infections, inflammatory bowel disease, celiac disease, foodborne illness or intolerance, and malignancy. Less commonly, mesenteric panniculitis may cause abdominal pain and should be considered as part of the differential diagnosis when approaching abdominal pain in patients.[2] Obtaining a thorough history and physical examination can propound a diagnosis.[1] Additionally, laboratory and radiologic workup can help to achieve an accurate diagnosis and treatment plan.

We discuss a case of a patient with abdominal pain that was initially thought to be secondary to colitis based on history, physical exam findings, and lab results. It was only after obtaining CT imaging of the abdomen that we were able to diagnose mesenteric panniculitis.

Case Presentation

A 74-year-old male with a past medical history of hypothyroidism, diabetes mellitus, congestive heart failure, and paroxysmal nocturnal hemoglobinuria (PNH) presented to the emergency department with the complaint of abdominal pain for over three weeks. He described abdominal pain as intermittent, diffuse, colicky, sharp, and non-alleviated with pain medications. He also reported anorexia, nausea, and food intolerance. Physical examination was remarkable for a distended abdomen with associated diffuse tenderness to palpation. Initial laboratory tests were significant for WBC 8.0 K/uL, Hgb 12.0 g/dl, and Hct 34.8%. Inflammatory markers were mildly elevated with an ESR of 26 mm/hr and CRP of 4 mg/L. Abdominal pain was initially thought to be secondary to colitis, and the patient received treatment with intravenous ciprofloxacin and metronidazole. Subsequently, an abdominal CT was performed and showed diffuse stranding of the mesenteric fat at the root of the mesentery with perivascular sparing compatible with mesenteric panniculitis (figure 1).

Patient was admitted for further management of abdominal pain secondary to mesenteric panniculitis. He received treatment with intravenous methylprednisolone for seven days. Over the course of hospitalization, the patient showed improvement of his presenting symptoms and was discharged home on an oral prednisone taper therapy for eight days. During outpatient follow up the patient reported complete resolution of abdominal pain with treatment.

Discussion

Mesenteric panniculitis is a rare, benign, and chronic fibrosing inflammatory disease affecting the adipose tissue of the mesentery of the small intestine and colon.[3] It can be more commonly found in men than women, with a male-to-female ratio of 2-3:1. Although the exact etiology remains unknown, the disease has been associated with other conditions, including neoplasms, autoimmune disease, ischemia, and abdominal trauma. Several factors support the hypothesis that mesenteric panniculitis is due to an autoimmune disease. Other theories have been proposed to explain this disorder including post-inflammatory changes secondary to acute inflammation, infection, and decreased blood supply to the mesentery.[4]

Mesenteric panniculitis usually presents with pain in the mid-abdomen but can be present in other areas of the abdomen or pelvis. Patients can experience nausea, emesis, bloating, early satiety, loss of appetite, unintended weight loss, fatigue, fever, diarrhea or constipation.[4] Abdominal CT is the imaging modality of choice when evaluating suspected mesenteric panniculitis, but the definitive diagnosis is established by biopsy. The most common and specific findings in abdominal CT are a fat ring sign that reflects the preservation of fat around the mesenteric vessels and the presence of a tumoral pseudo capsule, which is detected in 50% of patients.[5] Nonspecific laboratory abnormalities, such as elevated inflammatory markers and reduced red blood cell counts, have been reported but are not typical, and laboratory values are usually within normal limits.[4]

Mesenteric panniculitis can be successfully treated conservatively without surgical intervention.[6] In some cases, surgery is needed for patients with small bowel obstruction.[6] The treatment goals are to reduce mesenteric inflammation and control symptoms of the disease.[4] Anti-inflammatory agents, especially corticosteroids, are the initial treatment of choice. Additional anti-inflammatory drugs that can be used to treat this condition include colchicine, azathioprine, cyclophosphamide, infliximab, and pentoxifylline. There are prospective studies that show effectiveness in regression of symptoms using the drug thalomide, an immunomodulatory.[4]

This case illustrates an uncommon cause of abdominal pain in a patient with multiple autoimmune disorders, including paroxysmal nocturnal hemoglobinuria and hypothyroidism. Paroxysmal nocturnal hemoglobinuria is an acquired disorder of hematopoiesis characterized by pancytopenia, intravascular hemolysis, and bone marrow aplasia. It is manifested by repeated episodes of hemoglobinuria and a tendency to

continued from page 35

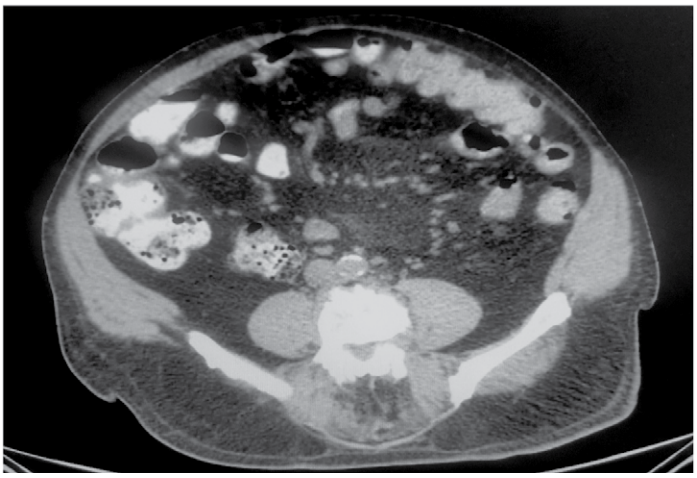


Figure 1. Axial contrast-enhanced CT image showing a diffuse stranding of the mesenteric fat at the root of the mesentery

develop widespread venous thrombosis, which may affect unusual sites, such as hepatic and mesenteric veins.[7] There is an incidence of 3% to 8% of mesenteric venous thrombosis leading to bowel ischemia in patients with paroxysmal nocturnal hemoglobinuria.[7] The mesenteric thrombosis could lead to mesenteric ischemia, which is related to one of the possible causes of mesenteric panniculitis. The association between paroxysmal nocturnal hemoglobinuria and mesenteric ischemia with the subsequent development of mesenteric panniculitis should be further studied.

Conclusion

Abdominal pain can be due to multiple diseases, and physicians should be able to recognize all causes of abdominal pain, including uncommon conditions like mesenteric panniculitis. Mesenteric panniculitis is a rare disease associated with autoimmune disorders whose symptoms can be controlled with anti-inflammatory agents like steroids. In this case, treatment with corticosteroid improved the clinical symptoms of this patient, supporting the efficacy of steroids in the treatment of mesenteric panniculitis as previously illustrated in the literature. The recognition and characterization of this disease are critical for the appropriate therapy and prevention of further complications.

Endnotes

1. Silen, W., & Cope, Z. (2010). *Cope's early diagnosis of the acute abdomen* (22nd ed.). New York: Oxford University Press.
2. Penner, R.,Fishman, M.(2022). *Causes of abdominal pain in adults*. Uptodate. Retrieved March 16, 2022, from https://www.uptodate.com/contents/causes-of-abdominal-pain-in-adults.
3. Mardi K, Wahal SP. Mesenteric panniculitis: A rare entity, report of two cases with review of the literature. Arch Med Health Sci 2016; 4:264-6.
4. Ehrenpreis, E. Mesenteric Panniculitis.NORD National Organization for Rare Disease. Retrieved on 11/23/21, from https://rarediseases.org/rare-diseases/mesenteric-panniculitis/
5. Sivrioglu, A. K., Saglam, M., Deveer, M., & Sonmez, G. (2013). Another reason for abdominal pain: mesenteric panniculitis. BMJ case reports, 2013, bcr2013009305. https://doi.org/10.1136/bcr-2013-009305
6. Kaya, C., Bozkurt, E., Yazıcı, P., diz, U. O., Tanal, M., & Mihmanlı, M. (2018). Approach to the diagnosis and treatment of mesenteric panniculitis from the surgical point of view. Turkish journal of surgery, 34(2), 121–124. https://doi.org/10.5152/turkjsurg.2018.3881
7. Ramus, J., & McPherson, G. A. (2003). Recurrent bowel infarction in paroxysmal nocturnal haemoglobinuria. Journal of the Royal Society of Medicine, 96(8), 406–407. https://doi.org/10.1258/jrsm.96.8.406

than 5 mmol/L should receive treatment.[5] Hyperlipidemia is associated with increased risk for atherosclerosis, cardiovascular disease, as well as pancreatitis. To convert triglyceride levels reported in mg/dL to mmol/L, multiply the mg/dL result by 0.0259.[6]

Treatment to lower triglyceride lipid levels may lead to the rescinding of xanthomas in some cases.[7] Additional presentations to be aware of on physical examination regarding patients with eruptive xanthomas include hepatosplenomegaly, abdominal pain from possible intestinal ischemia from lipid deposits, as well as lipemia retinalis, and potentially chronic pancreatitis.[8] In addition to the consideration of pharmacologic therapy, consideration should also be given to advising lifestyle modification including a low fat diet, incorporation of exercise, and advising for smoking cessation if the patient is a smoker.[9]

Endnotes

1. Hu CH, Ellefson RD, Winkelmann RK. Lipid synthesis in cutaneous xanthoma. *J Invest Dermatol*. 1982;79(2):80-85. doi:10.1111/1523-1747.ep12500030
2. Wilson, Jean D. Studies on the Origin of the Lipid Components of Xanthomata. *Circulation Research*. May 1963; Vol. XII, Issue 5; 472-478. Doi:10.1161/01.RES.12.5.472
3. Solak B, Kara RO, Acikgoz SB, Kosem M. First and only symptom of undiagnosed diabetes mellitus: eruptive xanthoma. *BMJ Case Rep*. 2015;2015:bcr2015212160. Published 2015 Sep 24. doi:10.1136/bcr-2015-212160.
4. Nessel, Trevor A., Kerndt, Connor C., Bills, John A., Sikorski, Lynn. Eruptive Xanthomas: A Warning Sign of Future Hyperlipidemia Complications. Int J Res Dermatol. 2020 Jul; 6(4):579-583. DOI: http://dx.doi.org/10.18203/issn.2455-4529.IntJResDermatol20202672
5. Smellie WS. Hypertriglyceridaemia in Diabetes. BMJ. 2006 Dec 16;333(7581):1257-60. doi: 10.1136/bmj.39043.398738.DE. PMID: 17170417; PMCID: PMC1702429.
6. Pierce J, Patel T, Scott C. Eruptive Xanthomas. Mayo Clin Proc. 2021 Dec; 96(12):3097-3098. doi: 10.1016/j.mayocp.2021.09.019. PMID: 34863397.
7. Zaremba J, Zaczkiewicz A, Placek W. Eruptive xanthomas. Postepy Dermatol Alergol. 2013 Dec;30(6):399-402. doi: 10.5114/pdia.2013.39439. Epub 2013 Dec 18. PMID: 24494004; PMCID: PMC3907905.
8. Naik, N.S. Eruptive Xanthomas. Dermatology Online Journal. 2001 Mar 1; 7(2):11. https://doi.org/10.5070/D37vt3m2bd
9. Ladizinski B, Lee KC. Eruptive Xanthomas In APatient With Severe Hypertriglyceridemia And Type 2 Diabetes. *Canadian Medical Association Journal*.2013;185(18):1600. doi:10.1503/cmaj.130148. PMID: 23754108; PMCID: PMC3855120.

Mycoplasma Pneumonia Presenting as Acute Coronary Syndrome

Case Study

By Emad Hanna, MD, MS, MSc; Mark Maloof, DO; Shara Feltheimer, DO and Andrew Awad, MD

Abstract

Mycoplasma pneumoniae may present with extrapulmonary symptoms. A middle-aged male presented to the emergency department with chest pain and dyspnea. Initial findings were significant for electrical alternans on electrocardiogram (EKG), an unremarkable chest X-ray, and elevated high sensitivity Troponin I (hs-TnI). Though initially evaluated as acute coronary syndrome (ACS), he was found to have left lower lobe pneumonia and myopericarditis secondary to mycoplasma pneumoniae. Mycoplasma pneumonia with cardiac involvement is uncommon, and elevated troponin secondary to mycoplasma pericarditis are absent from the literature. This case highlights the importance of considering this etiology to prevent premature narrowing of the differential diagnosis.

Introduction

Chest pain associated with non- ischemic troponin elevation has an extensive differential. After excluding myocardial infarction (MI) the differential includes infection (viral more commonly than bacterial), autoimmune disease flare, inflammatory bowel diseases, radiation therapy, uremia, toxins, or drug-induced (methyldopa, methysergide, sulphonamides, cytosine arabinoside, anthracycline derivatives, phenylbutasone, cocaine).[1]

Mycoplasma pneumonia is a common bacterial cause of respiratory infections including community acquired pneumonia. The incidence of cardiac involvement is very uncommon, with an incidence of 1%-5%.[2] In this case report we demonstrate that mycoplasma pneumonia should be considered in the differential diagnosis of myopericarditis.

Case Report

A 41- year- old male presented to the emergency room with the chief complaint of chest pain with shortness of breath. Prior to the ER visit, he was assessed at an urgent care center where he received three 81mg chewable aspirin tablets and one 0.6mg sublingual nitroglycerin and was subsequently transferred by emergency medical services to the ER.

His symptoms started 4 days prior to the ER visit and included a fever with a maximum home oral temperature of 102.7°F, chills, body aches, rhinorrhea, and sore throat consistent with viral illness. His symptoms became concerning when he developed chest pain and shortness of breath. He described his pain as substernal chest tightness, rated 4/10 in severity, alleviated by leaning forward and administration of nitroglycerin by EMS, and exacerbated by deep inspiration. He reported chest wall tenderness and pain with body movement. His immediate family had similar symptoms except chest pain.

In the ER vital signs were significant for an oral temperature of 97.7°F (36.5°C), heart rate of 82 beats per minute, a respiratory rate of 18 breaths per minute, an oxygen saturation of 100% on room air and a blood pressure of 134/91mmHg. He was 6ft 1in tall and weighed 81.64kg. The patient and his family were up to date with influenza vaccinations. He had no significant past medical or surgical history, no known allergies and was not on any medications. His social history included a remote smoking history of 12 pack years with cessation approximately 10 years ago, consumption of three alcoholic beverages 4 times per week, and no illicit substance use. In the ER, his physical exam was clinically unremarkable. An electrocardiogram (ECG) (Figure 1) demonstrated normal sinus rhythm at a rate of 91bpm, electrical alternans and left axis deviation without ST segment changes. Initial labs showed complete blood count, basic metabolic panel and coagulation studies within normal limits. Liver enzymes were twice the upper limit of normal. Cardiac enzymes were significantly elevated above normal limits; creatinine kinase-MB 10.2 (Ref 0.0-0.5ng/mL) and hs-Troponin I 9.22 (Ref 0.00 – 0.03). There was no radiographic evidence of cardiopulmonary disease. The patient was admitted to the ICU for suspicion of viral myopericarditis.

Transthoracic echocardiogram found mild left ventricular systolic dysfunction, ejection fraction of 45-50%, and moderately hypokinetic anterior septum, apical septum, and apex. CT of the chest was significant for left lower lobe infiltrate consistent with pneumonia; ESR was elevated at 62 (Ref 0-15 MM/hr); CRP was elevated at 8.1 (Ref 0.0-0.8 mg/dL); ANA ratio was normal; and urine toxicology was negative.

Further workup was required to assess whether the troponin was elevated secondary to pneumonia complicated by pericarditis versus non- ST segment elevation myocardial infarction (NSTEMI). Sixteen hours after the initial troponin, the level peaked to 15.41ng/mL. A coronary angiogram revealed no significant stenosis, borderline normal ventricular function, and mild irregularity of the coronary anatomy. These findings, in addition to the CT findings, supported the diagnosis of myopericarditis secondary to pneumonia as the cause of non- MI troponin elevation.

The patient's blood cultures and sputum cultures showed no bacterial growth. Titers for mycoplasma pneumonia IgM were <770 (Ref 770-9500). IgG titers were elevated to 416 (Ref 0-99). IgG levels were greater than 4 times the upper limit of normal; establishing the diagnosis of mycoplasma pneumonia.

Patient's management included 650mg of aspirin BID and 0.6mg of colchicine twice daily to address the myopericarditis. The patient also received empiric antibiotic therapy of 1g IV ceftriaxone qd and 500mg oral azithromycin once daily. Hs-TnI had trended down to 6.22ng/mL and repeat EKG revealed resolution of electrical alternans. The patient was discharged home on 325mg aspirin twice daily, 0.6mg colchicine twice daily for 7 days, 500mg azithromycin once daily for 2 additional days and 200mg of cefpodoxime twice daily for 10 additional days.

Discussion

In a case report published by Vijay, Aishwarya et al. in 2019, two cases of mycoplasma pneumoniae pericarditis are compared. In both cases, inflammatory markers were elevated and extensive infectious and rheumatologic evaluations were negative except for a positive M. pneumoniae IgM titer. One patient was an elderly female with cardiomyopathy, hypertension, and atrial arrhythmia while the other patient was a young female with a medical history of asthma and hypertension. Both presented with respiratory symptoms, musculoskeletal involvement, pleural effusion, normal troponins, and elevated inflammatory markers. The elder patient had an echocardiogram showing reduced left ventricular function while the younger patient had normal left ventricular function.[3] Our patient differs from these two patients in sex and in the fact that his troponin was elevated.

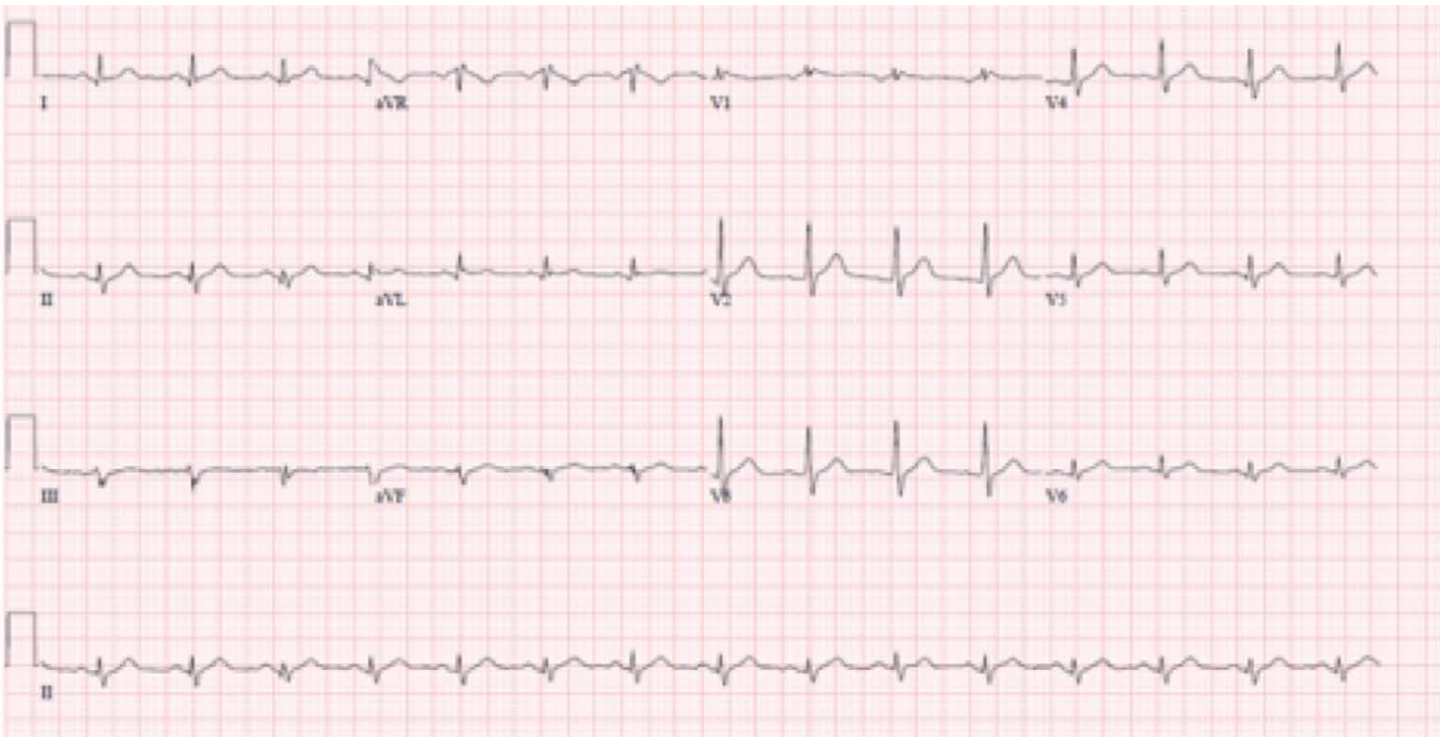
Extrapulmonary mycoplasma pneumonia usually presents as central nervous system manifestations and arthritides in children and carditis in adults. In a case report published by In Ho Park et al., a 6-year-old girl with no past medical history presented with fever, lethargy, cough, and vomiting. Cardiac enzymes were elevated in addition to the CRP. Echocardiogram showed a reduced ejection fraction and an effusion was present on chest imaging. M. pneumoniae IgM was increased.[4] Despite having elevated cardiac enzymes, this young girl did not have chest pain. Our patient presented with chest pain in the setting of elevated cardiac enzymes. This plus his age and sex led to rule out MI as first priority.

Our patient provides further evidence that the differential diagnosis should include mycoplasma induced pericarditis or myopericarditis in patients presenting with respiratory symptoms and cardiac findings. Serologic testing for M. pneumoniae should be considered in patients with pericarditis in the setting of prolonged respiratory symptoms, pneumonia, or pleural effusions. This can lead to earlier targeted treatment and avoid unnecessary invasive testing. Unlike in ACS, troponin elevation in the setting of myopericarditis is related to the extent of myocardial inflammatory involvement and is not a negative prognostic marker.[5] In the setting of elevated troponin and respiratory symptoms, it is prudent to consider non-MI troponin elevation secondary to mycoplasma pneumoniae induced myopericarditis. This case highlights the importance of considering this etiology to prevent premature narrowing of the differential diagnosis.

Endnotes

1. Imazio, Massimo, and Rita Trinchero. "Myopericarditis: Etiology, management, and prognosis." *International journal of cardiology* vol. 127,1 (2008): 17-26. doi:10.1016/j.ijcard.2007.10.053
2. Paz, Alona, and Israel Potasman. "Mycoplasma-associated carditis. Case reports and review." Cardiology vol. 97,2 (2002): 83-8. doi:10.1159/000057677
3. Vijay, Aishwarya et al. "Mycoplasma Pneumoniae Pericarditis." *The American journal of cardiology* vol. 123,8 (2019): 1383-1384. doi:10.1016/j.amjcard.2019.01.014
4. Park, In Ho et al. "A case of acute myopericarditis associated with Mycoplasma pneumoniae infection in a child." *Korean circulation journal* vol. 42,10 (2012): 709-13. doi:10.4070/kcj.2012.42.10.709
5. Imazio, Massimo et al. "Cardiac troponin I in acute pericarditis." *Journal of the American College of Cardiology* vol. 42,12 (2003): 2144-8. doi:10.1016/j.jacc.2003.02.001

Figure 1



Presentation of Acute Transverse Myelitis Leading to Diagnosis of Multiple Sclerosis

Case Study

By Bianca Diaz, MD; Tony T. Koshy, MD, MPH; Joseph Hong, MD; Mohammed Alanazi, MD; Sirisha Chirumamilla, MD; Richa Sharma, MD; Ben Lerman, MA; Jose Tiburcio, MD; Douglas Reich, MD

Abstract

Multiple sclerosis (MS), a chronic autoimmune and inflammatory condition, primarily affects the central nervous system and manifests with multiple sensory disturbances. As MS is traditionally considered a disease of Caucasian women, less than 1% of published literature is focused on minority communities. Here, we present a 37-year-old African American male with sudden onset left-sided weakness and left limp, denying other neurological deficits. Head and cervical spinal MRIs suggested a demyelinating lesion, and cerebrospinal fluid analysis was consistent with MS. This case illustrates the need for family medicine physicians, particularly those serving predominately minority communities, to be aware of atypical presentations of MS.

Introduction/Background

Multiple sclerosis is both a chronic autoimmune condition and an inflammatory neurological disease primarily affecting the central nervous system (CNS).[1] The course of MS is highly varied and unpredictable. Most patients are initially characterized by episodes of reversible neurological deficits which follow progressive neurological deteriorations over time. Twice as many women are affected as men, typically presenting in adults of 20-45 years of age.[1] Initially, the prevailing school of thought was that the African American (AA) population has a lower risk for developing MS compared to other ethnicities, but disease prognosis tends to be more severe with early disabilities (ie. Higher Expanded Disability Status Scale Score) and poor response to disease-modifying treatments.[1,2,4] However, studies have shown that MS in United States minority populations, such as African Americans and Hispanic Americans, have a higher incidence of MS compared with their ancestral countries of origin.[1] In a retrospective cohort study of 496 patients newly diagnosed with MS, the incidence rate per 100,000 person-years was higher in African Americans (10.2) than in Caucasian Americans (6.9).[2] This incidence among African Americans is also higher than Hispanic and Asian American minority groups.[1,2]

The initial MS symptom commonly reported among African Americans is limb weakness.[6] Multiple sclerosis is mainly a diagnosis of exclusion based on clinical findings, magnetic resonance imaging (MRI) of the brain, and cerebrospinal fluid (CSF) analysis.[7] It is multifactorial in etiology involving a facet of genetic, viral, metabolic, and environmental triggers. Often, MS will initially present as transverse myelitis (TM), which is an inflammatory myelopathy with rapid-onset neurological impairment. Other etiologies of TM include systemic autoimmune disorders (such as Sjogren and systemic lupus erythematosus), various infections, and idiopathic transverse myelitis. Noninflammatory myelopathies, including vascular and nutrition causes, can also present similarly. With such a broad range of etiologies and potentially nonspecific clinical presentations, it is important to promptly identify the cause, as management may be urgent. The gold standard for determining whether a patient is experiencing symptoms of MS or a different etiology of TM is a brain MRI.[8] While TM will have a singular lesion, diagnosis of multiple sclerosis is based on evidence of at least two different lesions in the white matter of CNS space (dissemination criterion) that are one month apart; at least two different episodes in the disease course where each episode last for more than 24 hours; and chronic inflammation of the CNS as determined by analysis of the CSF.[5] A majority of MS patients have moderate to severe spasticity, typically in the legs. Patients will often initially present with sensory disturbances, most commonly paresthesia, but may also present with symptoms such as dysesthesias, diplopia, ataxia, vertigo, or bladder dysfunction. A common manifestation of MS is unilateral numbness affecting one leg that spreads to involve the other leg and can rise towards the pelvis, abdomen, or thorax. Goals of therapy in this population focus on the use of disease-modifying agents, which shorten the duration of acute exacerbations, decreasing their frequency and providing symptomatic relief.[5] It is common practice to treat acute relapses of MS with a short course (3-5 days) of a corticosteroid that has a rapid onset of action and produces few adverse drug effects.[1,5]

Case Presentation

A 37-year-old African American male with a past medical history of asthma presented with sudden onset of left-sided weakness and noticeable left limp that has been persistent for four days. Patient denied facial droop, visual changes, bowel incontinence, or any other neurological deficits. Initial vital signs measured in the emergency room were unremarkable. On physical examination however, a left pronator drift of the upper extremities was present. Computed tomography (CT) of the head and CT angiogram of the brain were unremarkable, and he was subsequently discharged due to a lack of acute pathology. At the initial neurology outpatient visit, the initial working diagnosis included transverse myelitis from infections vs. inflammatory vs. neoplastic vs. demyelinating etiology. Neurology noted the decision was made to admit the patient after the outpatient visit for a more rapid completion of further imaging and possible workup needed. MRI of the head (*Figure 1*) revealed a 3mm focus with mild to moderate nonspecific enhancement in midline pons suggestive for demyelinating disease. MRI of the cervical spine (*Figure 2A,B*) revealed a well-defined lesion left of the spinal midline also suggestive of a demyelinating lesion. The patient was followed by inpatient neurology and recommended to undergo a lumbar puncture for further analysis. CSF analysis was diagnostic for the presence of more than five oligoclonal bands, and solumedrol therapy was initiated. On day two of treatment, the patient reported improvement of left-sided weakness without complete resolution of symptoms, with

a persistent left-sided limp. The patient completed the course of solumedrol and was sent home with prednisone taper. Through continued treatment by neurology, he was eventually cleared to fully return to work and resume physical activity, including sports.

Upon discharge, the patient was given outpatient follow-up with neurology and his primary care physician. The patient continued his care ~3 months after discharge with neurology and continued to endorse resolution of his symptoms. To evaluate possible progression, he was ordered a repeat MRI of the cervical spine, which illustrated similar T2 hyperintense lesion centered at the C3-C4 level. He continued to follow up with neurology as an outpatient, emphasizing the need for medical compliance.

Discussion

Acute transverse myelitis is a rare, acquired neuro-immune spinal cord disorder that can present with rapid onset of motor weakness, sensory alterations, and bowel/bladder dysfunction.[1] TM usually occurs as an independent post infectious complication, but it can also exist as a continuum of neuro-inflammatory disorders that includes multiple sclerosis.[1,4] In comparison to Caucasians, a high percentage of AA present with TM characteristics in conjunction with MS. 25-27% of AA present with TM in conjunction with the MS.[1] It is critical to note that in AA with MS, less than 20% of patients are males in comparison with females.[1,2] A research review conducted on January 13, 2014 revealed that there were nearly 60,000 published articles in total on MS, but only 113 focused on African Americans (or blacks).[1] A focused study on AA males with MS is even scarcer.

Multiple sclerosis is a progressive neurological disease with no definite cure to date. Although treatments are available to manage the disease course, they are only partially effective.[1,3] Specifically, African American patients showed poorer response to disease-modifying treatments when compared to other subgroups.[1] Further prospective studies on the response of African American patients to disease modifying agents is warranted, as well as further investigation of the presentation and management of multiple sclerosis in this population.[1,2] In general, as presented in this case, management of MS significantly depends on the acuity of clinical symptoms and signs, supporting the need for inpatient treatment or outpatient follow-up.

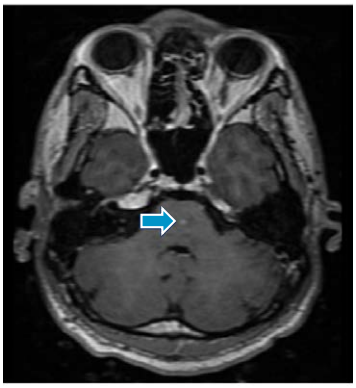
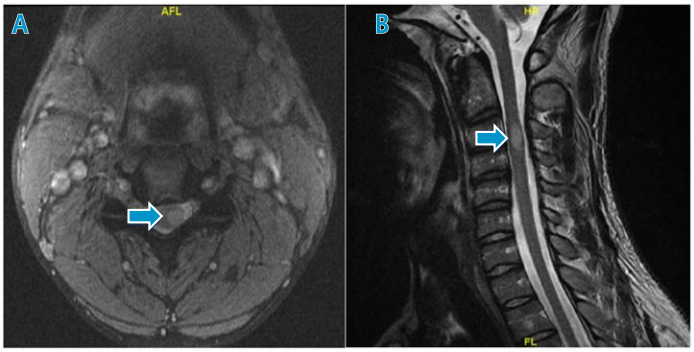


Figure 1 (left):
A 3 mm focus of mild to moderate enhancement is seen in the midline pons on postcontrast axial (arrow)

Figure 2 (below):
A homogeneous T2-hyperintense lesion is seen to the left of midline in the spinal cord at the C3-4 level. It measures 6 mm x 7 mm on axial (A, arrow) and 14mm on sagittal (B, arrow)



Conclusion

There is a misconception that MS is only found in Caucasian women. However, data shows that AA groups are at higher risk of MS and there is a lack of focused research to address the risk for AA males.[1,2,3] For this 37-year-old AA male, partial transverse myelitis was first suspected before confirmatory tests were completed to confirm new onset MS.[1] To account for diagnosis of exclusion like MS, it is critical for clinicians to have a broad differential when nonspecific symptoms arise that show neurological deficits. As the tendency would be to conduct a stroke work up to identify the etiology of the new onset neurological deficit, the differential of demyelinating diseases such as MS may be overlooked, especially for male AA populations. Our patient demonstrates the importance of not only performing a complete physical examination, but also understanding the atypical and nonspecific presentations of MS, especially in minority populations. To facilitate increased clinical awareness, it is imperative to expand research and literature as it pertains to MS in African Americans.

Endnotes

1. Khan, Omar, et al. "Multiple Sclerosis in US Minority Populations." *Neurology: Clinical Practice*, vol. 5, no. 2, 2015, pp. 132–142., doi:10.1212/cpj.0000000000000112.
2. Langer-Gould, A., Brara, S. M., Beaber, B. E., & Zhang, J. L. (2013). Incidence of multiple sclerosis in multiple racial and ethnic groups. *Neurology, 80*(19), 1734–1739. https://doi.org/10.1212/WNL.0b013e3182918cc2.
3. GEORGE W LOWIS, Ethnic Factors in Multiple Sclerosis: A Review and Critique of the Epidemiological Literature, *International Journal of Epidemiology*, Volume 17, Issue 1, March 1988, Pages 14–20, https://doi.org/10.1093/ije/17.1.14.
4. Meyer-Moock, S., Feng, YS., Maeurer, M. *et al*. Systematic literature review and validity evaluation of the Expanded Disability Status Scale (EDSS) and the Multiple Sclerosis Functional Composite (MSFC) in patients with multiple sclerosis. *BMC Neurol* 14, 58 (2014). https://doi.org/10.1186/1471-2377-14-58.
5. Crees, Zachary, The Washington Manual of Medical Therapies. Philadelphia, PA: Wolters Kluwer, 2020.Print.
6. Kaufman, Michael D. MD; Johnson, Susan K. PhD; Moyer, David MBA; Bivens, Jessica BA; Norton, H. James PhD Multiple Sclerosis, American Journal of Physical Medicine & Rehabilitation: August 2003 – Volume 82 – Issue 8 – p 582-590 doi: 10.1097/01.PHM.0000078199.99484.E2.
7. Katz Sand, I. B., & Lublin, F. D. (2013). Diagnosis and differential diagnosis of multiple sclerosis. *Continuum (Minneapolis, Minn.), 19*(4 Multiple Sclerosis), 922–943. https://doi.org/10.1212/01.CON.0000433290.15468.21.
8. Beh, S. C., Greenberg, B. M., Frohman, T., & Frohman, E. M. (2013). Transverse myelitis. *Neurologic clinics, 31*(1), 79-138.

Pancreatic Pseudoaneurysm: A Rare Cause of GI Bleeds

Case Study

By Israel Nemet, MD; Hector Reyes, MD; Dalwinder Basra, MS; Rodika Coloka-Kump, DO, FAAFP and Seth Cohen, MD

Abstract

Pancreatic pseudoaneurysms, caused by erosions of arteries that supply the pancreas, are a rare cause of GI bleeds in the US. However, individuals of Indian descent may be at higher risk for this potentially fatal condition. Detection of pancreatic pseudoaneurysms can be lifesaving, especially in patients of Indian descent, and should be considered in the differential diagnosis of severe anemia and gastrointestinal bleed.

Introduction

Pancreatitis is characterized by inflammation of pancreatic tissue that may result in permanent duct deformity and gland insufficiency. Chronic pancreatitis has an incidence of 4 to 12 people per 100,000 per year.[1] A rare cause is tropical pancreatitis, first described in 1937.[2] Further research shows that there is increased incidence of chronic pancreatitis in Asian countries and more specifically South India.[3] Pancreatic calculi are seen in over 90% of tropical pancreatitis[4] in patients of Indian descent. This cohort may be at risk for pancreatic pseudoaneurysm because of environmental and genetic causes implicated in the development of chronic pancreatitis.[5] There is no specific pathogenesis or etiology of tropical pancreatitis, however, it is hypothesized that the combination of these environmental and genetic factors play a significant role. A common food in the region is cassava and metabolic toxins and cyanide toxicity can occur from the breakdown of cassava.[6] Genetic factors associated include SPINK1 gene, CFTR gene, and CASR gene, all which causes activation of trypsin from the pancreas.[7]

Chronic pancreatitis can lead to bleeding from adjacent arteries that results from digestive enzyme erosion of vessels, most commonly the splenic, gastroduodenal, pancreaticoduodenal and hepatic arteries.[2] Undetected and untreated pancreatitis can lead to complications such as pseudocysts and pseudoaneurysm at approximately 25% and 1% respectively.[1]

Patients without treatment have a mortality rate of approximately 90% whereas the mortality rate for early diagnosis and intervention may be a little a 15%.[2]

Case Report

Patient is a 59-year-old male with a past medical history of hypercholesterolemia who presented to ED for an episode of "blacking out and fainting for 2 seconds" with no precipitating factors. The review of his systems was negative for seizures, head trauma, chest pain, dyspnea, or palpitations. The patient, one- year prior, had a hemoglobin of 13g/dl (13.5-17.5 g/dl) and unremarkable EGD and colonoscopy. A previous abdominal ultrasound showed enlarged head of pancreas without follow up. The patient is a nonsmoker and drinks 3-4 drinks per week. Additional history revealed that his hemoglobin was 7g/dl (13.5-17.5g/dl) five months prior and that he experienced a previous episode of syncope two months preceding this presentation.

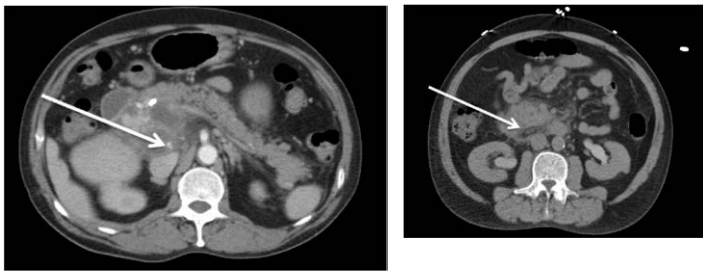
Upon initial evaluation the patient was afebrile, tachycardic, but normotensive. Remarkable laboratory findings included: hemoglobin of 3.5 g/dl (13.5-17.5g/dl) and hematocrit of 13.8 % (41.0-53.0%) with MCV 71.1 fl (80.0-100.0 fl), and MCH 18 pg (26.0-34.0 pg). Stool guaiac test was negative, and EKG showed sinus rhythm, within normal limits.

The patient was admitted for monitoring, transfusion, and further work up. While being transfused with packed red blood cells, he developed severe, band like, pulsatile and sharp epigastric pain associated with nausea followed by bloody emesis. He remained hemodynamically stable on IV hydration and pantoprazole.

A CT of the abdomen with contrast showed a 5.7 cm in greatest dimension cystic mass in the pancreatic head with adjacent inflammation. A 0.7 cm focus of increased density along the posterior aspect of this mass was extremely concerning for extravasation of contrast in the setting of hemorrhagic pancreatitis with resulting hematoma in the setting of GI bleed. (Figure1)

Figure 1 (left): CTAP with Contrast – pseudo-aneurysm
A CT A/P without contrast was completed and it revealed no increase in the size of the hematoma. The 0.7 cm focus of increased density within the posterior aspect of the collection was no longer present. The CT without contrast confirmed a pseudoaneurysm protruding into the hematoma.

Figure 2 (right): Pancreatic pseudoanerysm protruding into the hematoma



Repeat guaiac tests remained negative and no further upper GI bleeding was experienced. The patient was transferred to a tertiary care center for angiography and embolization.

The patient remained hemodynamic stable and underwent successful angiography and embolization by interventional radiology of the pancreaticoduodenal artery.

Discussion

Originally coined in 1970 by Sandblom, the term hemosuccus pancreaticus (HP) was used to describe gastrointestinal bleeding from the main pancreatic duct (PD) across the major papilla, and rarely also occurring through the minor papilla.[8,9] Considered one of the rare causes of upper gastrointestinal bleeding (UGIB), HP occurs secondary to the rupture of a pseudoaneurysm of a peripancreatic artery, or hemorrhage of a peripancreatic artery into a pseudocyst communicating with the main pancreatic duct, usually in the setting of acute or chronic pancreatitis.[10] Less common causes include iatrogenic surgical trauma, pancreatic tumors, or pancreatic cystic neoplasms. The splenic artery and its branches are near the pancreatic parenchyma and pancreatic duct. During pancreatitis, when substantial amounts of pancreatic enzymes are released, these vessels are prone to rupture forming a pseudoaneurysm. Similarly, patients with preexisting pseudocysts are also prone to rupture because they contain lytic enzymes. When a peripancreatic pseudoaneurysm ruptures into a pseudocyst, lytic enzymes, especially elastases are released that exert an elastolytic action on the vessel wall, resulting in intermittent bleeding due to clot formation.[11]

Diagnosing HP as the cause of UGIB is difficult due to its rarity and the intermittent nature of the bleeding from a peripancreatic artery. Accessibility of the UGIB site via endoscopy can be challenging which may delay an accurate and timely diagnosis.[12,13,14] The mean age of patients with HP is 32 years and it is associated with alcohol-related pancreatitis, male gender and a history of melena.[12,13,14] Patients with a history of pancreatic disease, tumors, vascular disease, malformations, and chronic pancreatitis with symptoms of epigastralgia, melena, and hyperamylasemia suggest HP. These symptoms should prompt further workup with endoscopy to differentiate other common causes of UGIB, such as peptic ulcers, esophageal and gastric fundus varices and erosive gastritis.[13]

Endoscopy is reliable for the diagnosis of either HP or haemobilia especially if active bleeding is visualized from the duodenal papilla flowing into the descending part of the duodenum.[15] However endoscopic visualization of active bleeding is only observable in 30% of patients owing to its intermittent nature. This may warrant repeat endoscopy at a later time and is supported by many authors.[16]

Endoscopic Retrograde Cholangiopancreatography (ERCP) can aid in the diagnosis of HP, through visualizing an intraductal filling defect which can visualize a blood clot that is present, or a pseudoaneurysm that is compressing the pancreatic duct lumen. In combination with papilla sphincterotomy, ERCP can be used as a total treatment.[17]

Selective arteriography of the celiac trunk and superior mesenteric artery is the optimal tool for detecting HP, as it precisely localizes the undetermined source of UGIB with a sensitivity of 96% for a definitive diagnosis.[18] However, due to the intermittent nature of the upper gastrointestinal bleeding , selective arteriography may also be limited in the detection of HP. Selective arteriography can be combined with transcatheter embolization to aid in the cessation of the bleeding and has become the mainstay of modern management. The use of CT imaging has high utility for the evaluation of the pancreas, including the presence of vascular lesions, and determination of bleeding. Conversely, magnetic resonance imaging can be useful for detecting pancreatic tumors and their relation in proximity to the pancreatic duct. Taken together, selective arteriography of the celiac trunk and superior mesenteric artery remains the gold standard.[16]

Diagnosing pseudoaneurysms early is key to successful intervention. Although a rare cause of GI bleeding, pancreatic pseudoaneurysm should be in the differential diagnosis of male patients of Indian descent.

Endnotes

1. Barry K. Chronic Pancreatitis: Diagnosis and Treatment. *Am Fam Physician*. 2018; 97:385–393
2. Kiini MG. Multiple pancreatic calculi with chronic pancreatitis. *Br J Surg*1937; 25:705.
3. Garg PK. Chronic pancreatitis in India and Asia. *Cur Gastroenterol Rep*. 2012;14(2):118–124.
4. Narendra Nathan M. Chronic calcific pancreatitis in the tropics. *Trop Gastroenterol*1981; 2:40-5
5. Garg PK, Narayana D. Changing phenotype and disease behavior of chronic pancreatitis in India: evidence for gene-environment interactions. Glob Health Epidemiol Genome. 2016 Oct 18;1: e17. Doi: 10.1017/gheg.2016.13.
6. Rajesh, G.; Balakrishnan, V. Chronic Pancreatitis. *KMJ* 2015, *8*, 47-53.
7. Pujahari AK. Chronic Pancreatitis: A Review. *Indian J Surg*. 2015;77(Suppl 3):1348-1358.
8. Sandblom P. Gastrointestinal hemorrhage through the pancreatic duct. *Ann Surg*. 1970;171(1):61-66.
9. Hemosuccus pancreatic us. Adler DG, Petersen BT, Gostout CJ. *Gastrointestinal Endosc*. 2004; 59:695
10. Toyoki Y, Hakamada K, Narumi S, Nara M, Ishido K, Sasaki M. Hemosuccus pancreaticus: problems and pitfalls in diagnosis and treatment. *World J Gastroenterol*. 2008; 14:2776–9.
11. Yu P, Gong J. Hemosuccus pancreaticus: a mini review. *Ann Med Surg (Lond)*. 2018; 28:45-48.
12. Clay RP, Farnell MB, Lancaster JR, Weiland LH, Gostout CJ. Hemosuccus pancreaticus. An unusual cause of upper gastrointestinal bleeding. *Ann Surg*. 1985; 202:75–9.
13. Han B, Song ZF, Sun B. Hemosuccus pancreaticus: a rare cause of gastrointestinal bleeding. *Hepatobiliary Pancreat Dis In*t. 2012; 11:479-488.
14. Rammohan A, Palaniappan R, Ramaswami S, et al. Hemosuccus pancreaticus: 15-year experience from a tertiary care GI bleed centre. *ISRN Radiol*. 2013; 2013:191794.
15. Sakorafas GH, Sarr MG, Farley DR, Que FG, Andrews JC, Farnell MB. Hemosuccus pancreaticus complicating chronic pancreatitis: an obscure cause of upper gastrointestinal bleeding. *Langenbecks Arch Surg*. 2000; 385:124-128.
16. Long TB, Binh NT, Dung LV, et al. Diagnosis and treatment of hemosuccus pancreaticus induced by intraductal papillary mucinous neoplasm: a case report and review of the literature. *Radiol Case Rep*. 2021;16(10): 3099-3103.
17. Lacey S.P., Chak A. Hemosuccus pancreaticus: dorsal pancreatic duct stone and gastroduodenal artery pseudoaneurysm. *Gastrointest Endosc*. 2001; 54:363.
18. Kim SS, Roberts RR, Nagy KK, et al. Hemosuccus pancreaticus after penetrating trauma to the abdomen. *J Trauma*. 2000; 49:948-950.

Diagnosis and Management of Symptomatic Polycythemia in a Well-Controlled COPD Patient

Case Study

By Kristen Kelly and Jennifer Ruh, MD

Abstract

A 67-year-old male patient presented at his physical with bloodwork showing an elevation in red cell count, hemoglobin, and hematocrit. The patient had historical elevated red cell count; he had never been worked up for polycythemia. On presentation he was complaining of new onset headache, a possible symptom of hyperviscosity. Past medical history was significant for chronic obstructive pulmonary disease, type 2 diabetes mellitus, hypertension, and mixed hyperlipidemia, all of which were controlled. The patient was sent for total iron binding capacity and erythropoietin labs which were within reference range. A hematology referral was made to evaluate for malignant processes and possible JAK2-V617F mutation.

Introduction

This clinical case report aims to guide the workup of a complex patient presenting with polycythemia. Polycythemia is often the physiologic response to various causes of hypoxia, including obstructive sleep apnea, COPD, high altitude, right to left cyanotic heart disease, and smoking. True polycythemia is categorized as primary and secondary.[1] In primary polycythemia, serum erythropoietin levels are low; in secondary polycythemia erythropoietin is elevated in a physiologic effort to elevate the body's oxygen carrying capacity. On initial evaluation of an abnormal CBC, it is important to be wary of spurious polycythemia, which is a function of volume contraction rather than true increased red cell mass. Diagnosis and management of polycythemia is critical to avoid adverse outcomes, namely thrombotic events, which occur in the setting of blood hyperviscosity.[1,2] Evidence indicates that thrombotic events in the setting of polycythemia are associated with primary polycythemia rather than secondary polycythemia or isolated erythrocytosis.[2] This demonstrates the importance of determining the cause of new onset or worsening erythrocytosis. In this case, we work up a patient following abnormal lab studies at his annual physical exam with symptomatic erythrocytosis.

Case Report

A 67-year-old Caucasian male with a past medical history of T2DM, COPD, BPH, primary HTN, mixed hyperlipidemia, and dysthymia presented for his annual exam. His COPD was stable and managed with dual LABA/LAMA therapy, and no history of long term oxygen therapy. He had a 30-pack year smoking history and quit in 2007. Laboratory studies ordered and reviewed at his annual wellness exam included a CBC w/ differential to follow his historic erythrocytosis, as well as a CMP, HbA1c, TSH, lipid panel, vitamin D, and myeloperoxidase. On presentation, the patient was well appearing. He reported new symptoms of "head fogginess," worsening headache, and dyspnea on exertion. Vitals were HR 84, BP 124/76, Spo2 97%, RR 16, temp 37.5. On physical exam the patient had no pertinent positive findings. Head was atraumatic and normocephalic; lung exam exhibited normal breath sounds with good air movement, no wheezing, no rales/crackles, or rhonchi. There was no hepatosplenomegaly or erythromelalgia. The patient's laboratory studies (figure 1) revealed elevations in RBC: 6.38, Hb: 18.3, and Hct: 55.0. The patient had historical bloodwork available from his pulmonologist which showed a steady increase in RBC, Hb, and HCT from 2017-2021. Patient's HbA1c was also mildly elevated at 7. Otherwise, patients CMP, and lipid panel were unremarkable, TSH, and vitamin D were within normal limits. Given his erythrocytosis of unknown etiology, serum erythropoietin was measured 2 weeks later and found to be 12.0 mIU/mL (normal value). Iron studies including serum ferritin and TIBC were also measured, and within normal limits. In the setting of the unexpected erythropoietin value, hematology/oncology was consulted to rule out pathologic causes of erythrocytosis. To determine the etiology of the polycythemia as primary vs. secondary, JAK2 V617F mutation analysis was ordered, and demonstrated no mutation. The patient was continued on aspirin therapy until JAK2 results were obtained. The patient followed with a pulmonologist, seen 6 months prior to this visit. Spirometry results from that time showed FEV1/FVC of 49, FEV1 of 55%, and FVC of 84%.

Figure 1- Patient laboratory study results [3,4]

Component	Patient result value	Reference range, males
RBC	6.36	4.35-5.65 x 1012/L
Hb	18.3	13.2-16.6 g/dL
HCT	55	38.3-48.6%
Iron, total	91 mcg/dL	60 to 150 mcg/dL
TIBC	416 mcg/dL	250-450 mcg/dL
Iron saturation	22%	20 to 50%
Serum ferritin	79 ng/mL	40 to 200 ng/mL
EPO	12	2.8-17.1 IU/L
HbA1c	7	4-5.6%
Jak2V617F Mutation	negative	

Discussion

The workup of polycythemia as primary or secondary is important because treatment modalities rely on treating the cause. For primary polycythemia where erythropoietin is low (primary polycythemia) expansion of red cell lines is a pathologic response and the mainstay of treatment is phlebotomy. A well-studied mutation in the JAK2 gene, V617F, produces a constitutively active protein that promotes cell proliferation and survival.[5] In secondary polycythemia, where erythropoietin is elevated, treatment is centered around treating the underlying cause.

The question in this patient case, is what is the next step diagnostically when erythrocytosis is occurring in the presence of normal erythropoietin? For this patient, it was referral to a hematology/oncology specialist to rule out malignancy. Initially, his history of COPD, cigarette smoking, and right lung lobectomy were believed to be his cause of hypoxia. However, the patient consistently had oxygen saturations above 96%, and was followed with a pulmonologist.

Additionally, this case was interesting due to the worsening of hyperviscousity symptoms in the absence of changes in respiratory status or COPD symptoms. Elevated carboxyhemoglobin in active smokers is believed to contribute to development of secondary polycythemia.[6,7] It's estimated that 6-10% of patients with COPD have secondary polycythemia with Hb >17 in males and >15 in females. In a recent study by Zhang et al, the prevalence of polycythemia in former smokers was 7.0% in males and 2.5% in females, and in current smokers was 13.6% in males and 5.5% in females.[7] Thus, active smoking status almost doubles the risk of developing polycythemia.[7]

Interestingly, aspects of this patient's history made it challenging to differentiate between primary or secondary polycythemia. He had a normal EPO value and a chronic condition that causes hypoxia, which pointed us in the direction of secondary polycythemia. Alternatively, he also had a slow onset development of erythrocytosis, was not an active smoker, and was in the demographic group where PV is most common. Mahmud et al describes a similar patient case where polycythemia had been overlooked as secondary polycythemia due to a COPD diagnosis. In their case, the patient had the JAK2617F mutation and was diagnosed with polycythemia vera which altered management.[8]

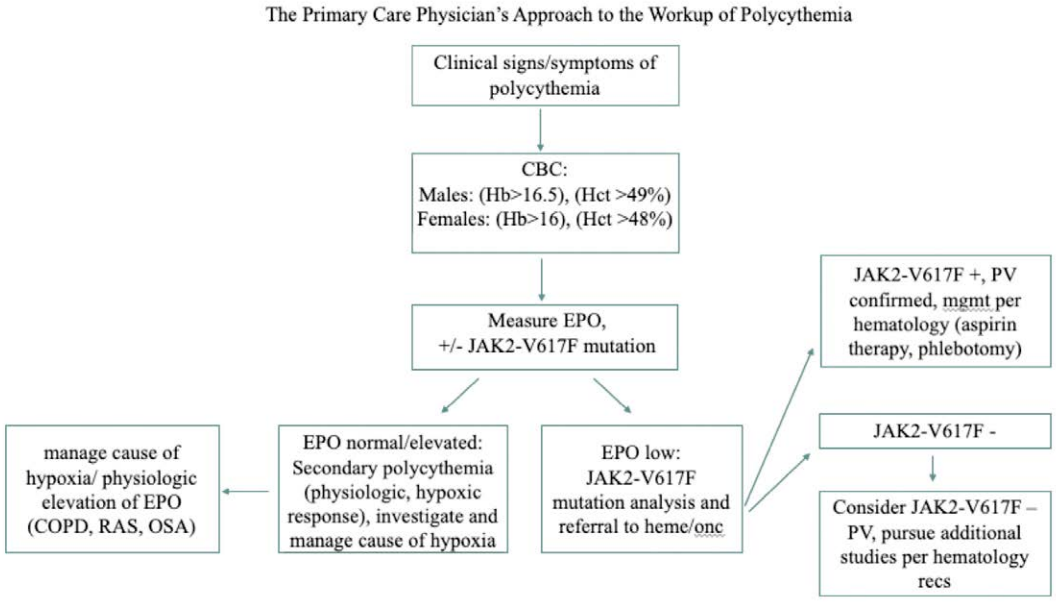
Conclusion

Erythrocytosis of unknown etiology warrants a workup, even in the context of a patient with conditions that commonly cause secondary polycythemia. The primary care physician can play a critical role in making this diagnosis, by following up abnormal CBC results with a serum erythropoietin and JAK2617F mutation analysis if warranted. The flow chart (figure 2) for the diagnostic approach allows the PCP to order the appropriate initial studies and collaborate with hematology colleagues at an appropriate point in the workup.

Endnotes

1. Pillai AA, Fazal S, Babiker HM. Polycythemia. [Updated 2021 Nov 3]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK526081/
2. Gordeuk VR, Key NS, Prchal JT. Re-evaluation of hematocrit as a determinant of thrombotic risk in erythrocytosis. *Haematologica*. 2019;104(4):653-658. doi:10.3324/haematol.2018.210732
3. Grote Beverborg N, Verweij N, Klip IT, et al. Erythropoietin in the general population: reference ranges and clinical, biochemical and genetic correlates. *PLoS One*. 2015;10(4):e0125215. Published 2015 Apr 27. doi:10.1371/journal.pone.0125215
4. Coates TD. Physiology and pathophysiology of iron in hemoglobin-associated diseases. *Free Radic Biol Med*. 2014;72:23-40. doi:10.1016/j.freeradbiomed.2014.03.039
5. James C. The JAK2V617F mutation in polycythemia vera and other myeloproliferative disorders: one mutation for three diseases? Hematology Am Soc Hematol Educ Program. 2008:69-75. doi: 10.1182/asheducation-2008.1.69. PMID: 19074061.
6. Mehta J, Wang H, Iqbal SU, Mesa R. Epidemiology of myeloproliferative neoplasms in the United States. Leuk Lymphoma. 2014 Mar;55(3):595-600. doi: 10.3109/10428194.2013.813500. Epub 2013 Jul 29. PMID: 23768070.
7. Zhang, J., DeMeo, D.L., Silverman, E.K. *et al*. Secondary polycythemia in chronic obstructive pulmonary disease: prevalence and risk factors. *BMC Pulm Med* 21, 235 (2021). https://doi.org/10.1186/s12890-021-01585-5
8. Mahmud R, Ariffin F, Shanmuganathan P. Polycythaemia Vera JAK 2 Mutation in a Patient with Underlying Chronic Obstructive Pulmonary Disease at a Primary Care Setting. *Korean J Fam Med*. 2020;41(4):263-266. doi:10.4082/kjfm.18.0161

Figure 2: Diagnostic approach



The Seven- Year Itch

By Assunta DiValentino DO, MHS and Jeff Harp, MD

Case Study

Abstract

A 32-year-old lactating female physician received a clinical diagnosis of scabies and was successfully treated, along with her infant and husband. Their visiting nanny neither developed symptoms nor completed treatment. Rash and pruritus reappeared in the family several times over the course of sixteen months, resolving with permethrin and ivermectin. Eventually the nanny, intermittently in the household, was determined to be an asymptomatic carrier. After she and the family were treated, the patient remained symptom-free. This case discusses asymptomatic carriers, demonstrates the importance of contact treatment and reviews the protean nature of scabies rash.

Introduction

The burden of scabies in the United States impacts the practice of family medicine. In an attempt to address a disease for which diagnosis is predominantly clinical, clinicians must be mindful of several potential pitfalls which delay its diagnosis and treatment. The morbidity associated with this delay puts practitioners in a position to significantly impact patients' lives.

Case Report

A 32-year-old new mother consulted her primary care physician for recurrent intense generalized pruritus and rash. She had received a clinical diagnosis of scabies sixteen months prior based on similar symptoms of pruritus, dermatographism, and papular rash of the waistline, buttocks, thigh folds, genitals, upper back, fingers, wrists, forearms and peri-areola regions (Table 1, Figures 1-3). She worked as a physician and at the time of her initial diagnosis was living with her seven-month old infant (whom she was breastfeeding) and her husband; a nanny visited intermittently to help with the infant. Her medical history for the most part was unremarkable (no medication or supplements, no allergies, C-section in the past year). She had a remote history of two isolated scabies infestations after working in Ethiopia and in Paraguay, six and twelve years prior respectively, each which had resolved with two doses of permethrin one week apart and the recommended laundering. Her infant had a similar new onset papular pruritic rash but predominantly of the scalp, face and wrists. Her husband had a new nonpruritic rash of the waistline and lower buttocks. With that initial diagnosis of scabies, all three had been treated with two rounds of permethrin one week apart; all clothing, linens, and plush toys were laundered in high heat. Symptoms abated.

Table 1. Clinical Presentation

Patient Symptoms	Infant Symptoms	Toddler Symptoms
Dermatographism Intense Pruritus Generalized Most intense: Anus Lateral feet Rash	Dermatographism Pruritus Rash	Dermatographism Pruritus Rash

Rash Locations	Rash Locations	Rash Locations
Mid-scapular/upper back Finger webspaces Side of fingers Wrist/flexor forearm Peri-aereola Periumbilical Waistline Abdomen Axillae Thigh creases Buttocks	Scalp Face Wrists Thigh creases	Face Back Feet Abdomen Thigh creases *Scalp mostly spared
Rash: extremely pruritic macules and smooth papules/plaques.		

Intense predominantly nocturnal pruritus and rash however, reappeared in the family two weeks later. Determined to be a treatment failure, the family was treated with a ten-day course of permethrin. Symptoms again completely resolved in all three family members. At that time the family moved to a new home in a nearby city with newly purchased belongings and furniture. For roughly one month the family was symptom-free.

However, similar rash and intense pruritus appeared again in the entire family one month later and did so several times over the course of the following thirteen months. The rash of the infant mostly involved the scalp, hairline, wrists and face (Table 1, Figure 4). Multiple specialists were consulted. In part due to a history of prior multiple scabies treatments, and in part because the rash had atypical features (smooth papules and plaques, atypical affected areas such as the upper back, and lack of pruritus (husband)), alternate diagnoses and treatments were explored. These included xerosis, eczema, bed bugs, dermatographism, contact dermatitis, folliculitis, allergic reaction, psychosomatic disorder, neurological disorder, and post-scabies syndrome. Over the course of many months, the patient was treated sequentially with moisturizer, topical steroid creams from hydrocortisone to triamcinolone to clobetasol, oral prednisone (which intensified the rash), bleach baths, topical and oral antibiotics, antihistamines including a combination of H2 and H1 blockers, gabapentin, and N-acetyl cysteine. The patient's quality of life suffered. High doses of gabapentin and hydroxyzine for the intense pruritus caused significant daytime drowsiness. Multiple biopsies of the lesions and one skin scraping of the upper back were performed. Biopsies revealed predominantly lymphocytic inflammatory cell infiltrate consistent with hypersensitivity reaction. The new pruritic rash of the infant was diagnosed as contact

dermatitis and eczema, unresponsive to steroid creams or bleach baths. The husband was diagnosed with waistline contact dermatitis. The family avoided contact with others when symptoms occurred. However, none of these treatments proved effective. With each episode, not until scabies treatment was again attempted did the rash and pruritus of each family members resolve, each time for several weeks to months. This treatment consisted of a combination of permethrin and oral ivermectin and hot laundering of all bedding and clothing.

With the sixth occurrence, not wanting to again treat the infant with permethrin and wanting to be spared another full-family treatment, the family sought a new family physician for advice. With careful history taking of each episode from the first onset of symptoms, this physician identified the nanny as a close contact at the time of the initial diagnosis. The nanny had never completed treatment and remained without symptoms. Following the initial outbreak, she visited intermittently to help with the child when the family had completed treatment. She later admitted to having rash which she attributed to skin sensitivity of lupus. Immunosuppression from her lupus-modulating agents was surmised to be masking her response to the scabies mite. Interestingly, the time to symptom onset after her visits was less with each subsequent exposure (Figure 5), demonstrating sensitization to scabies in the patient and family. The connection of the nanny to the episodes was made in part because the last episode of rash and pruritis occurred within one day of her last visit. With this discovery, the nanny and the family were treated and thereafter all remained symptom-free.

Discussion

This case adds to the data on asymptomatic carriers of scabies, demonstrating the importance of treatment of all close contacts. It illustrates the difficulty with treating scabies in a household with an infant and highlights, in real time, the variation of rash distribution in infants where the scalp is most heavily involved to toddler when scalp involvement is less frequent. It demonstrates longitudinally, the shortened incubation period with repeat exposures and, further calls for an appreciation of the protean nature of scabies rashes.

Regarding lessons learned and pitfalls to avoid, there are several illustrated by this case. Clinicians should be careful not to rely on what is understood as the typical scabies rash and should be aware of atypical presentations so as to limit unnecessary treatment and delay of appropriate treatment.[1,2] As Papier writes, "Atypical presentations include lesions that appear outside of the classic distribution areas of scabies…The back is usually spared…though not always." Atypical presentations lead to misdiagnosis in almost half of cases.[1] He continues, "pruritic smooth papules and plaques should lead to a consideration of scabies." When performing physical exams clinicians must be mindful of not letting concerns of over diagnosis lead to underdiagnoses and pay close attention to these atypical presentations.

Second, the incubation period of scabies can add to diagnostic and treatment errors. Scabies rash is a hypersensitivity reaction to the scabies mite. For first exposure, the reaction can be delayed four to eight weeks. If a person has already had scabies, symptoms appear sooner, even within days of exposure as seen in this case.[3] Recall

continued on page 48

Figure 1. Dermatographism

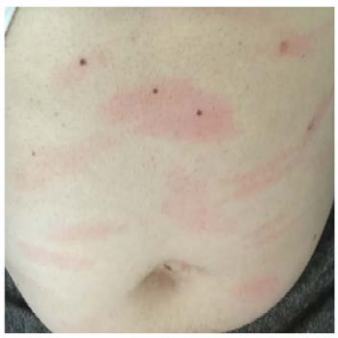


Figure 2: Upper Extremity Rash

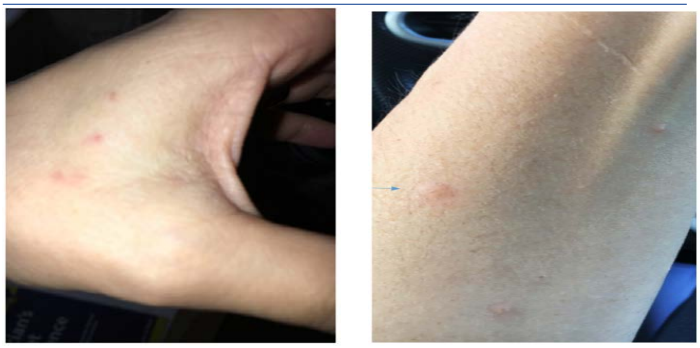


Figure 3. Rash of Waistline / Thigh Creases

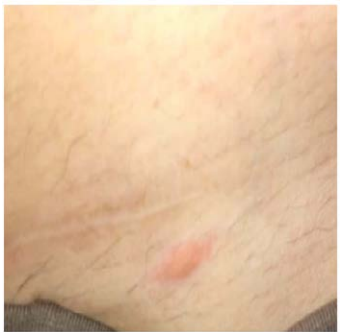
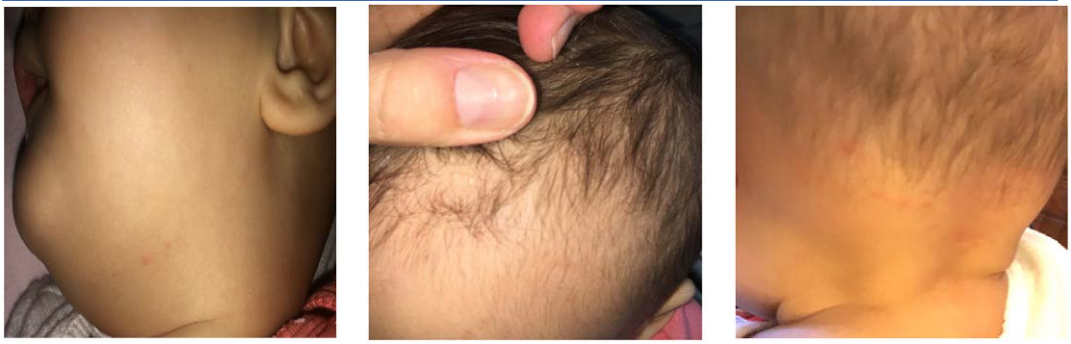
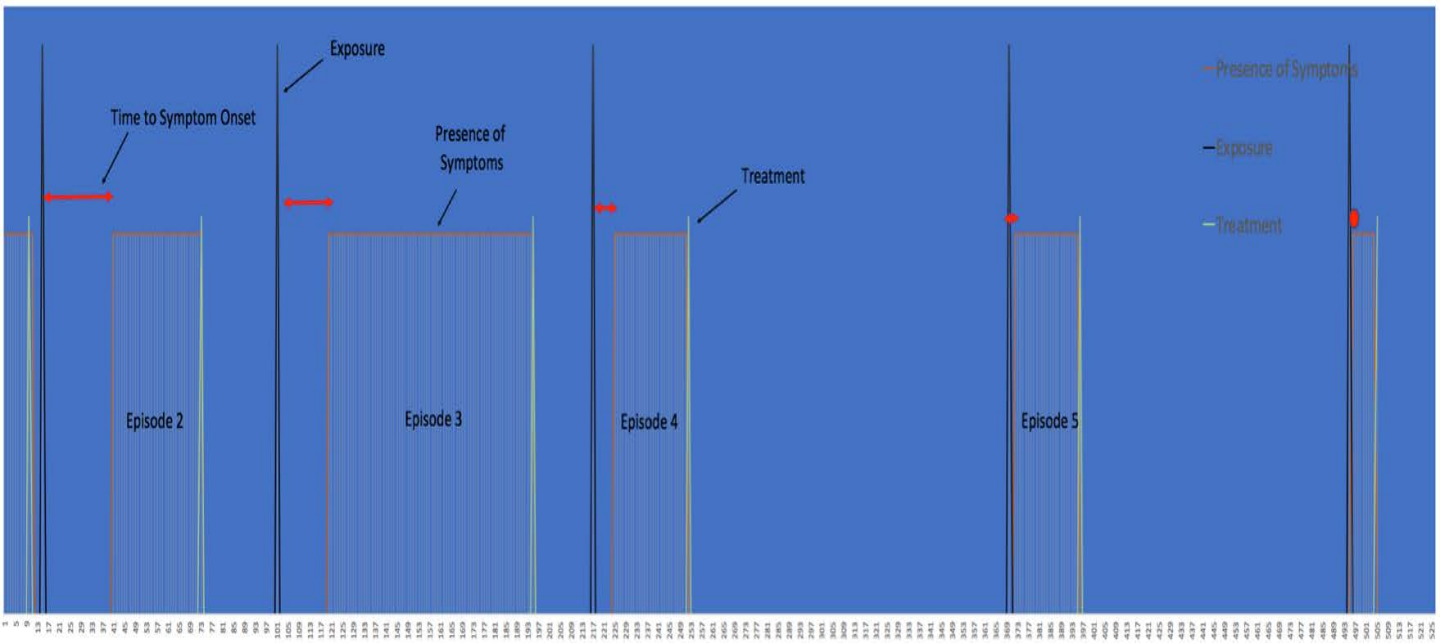


Figure 4. Rash of Infant (Ages 9 months-11months)



continued from page 47

Figure 5. Chronology of Disease and Decreasing Incubation Period



difficulty of a specific incident of exposure months prior adds to the diagnostic challenge. The incubation period of scabies can further mislead patients to not consider identifying and treating contacts who are asymptomatic, assuming that the contact's lack of symptoms implies they are not infected. With scabies rash being predominantly a hypersensitivity phenomenon, clinicians must remember that performing biopsies of the rash is of low yield for finding mites[3,4] and should not exclude a diagnosis. Rather, it is imperative that they closely assess for burrows in typical and atypical locations when looking to diagnose scabies.

Additionally, the prevalence of asymptomatic carriers of scabies must not be underestimated when considering possible diagnoses. Asymptomatic carriers are not uncommon[5] as the scabies mite can inhabit the skin without eliciting symptoms.[5,6] The CDC recommends simultaneous treatment of the patient and close contacts[3] due to variation in incubation periods from person to person, but also because some people may never develop symptoms.

Immunocompromised patients for example, may have varying degrees of rash without pruritus.[1] Additionally, visitors should be considered as potential contact even if not a household member. Caring for an infant, as in this case, involves direct skin-to skin contact so a caregiver is considered a close contact. Whether or not formally part of the household and whether or not symptomatic, all these close contacts should be treated. Because scabies is highly contagious, not doing so can lead to the "endless chain of cross contamination and reinfestation"[3] a chain which led to coining of the label "the seven- year itch."

Finally, this case reminds us of the importance of history-taking and physical exam in medicine, most especially when diagnoses are predominantly clinical. In regions where illnesses are not endemic, even the astute diagnostician may miss a diagnosis if not casting a large enough diagnostic net. Histories should include travel, childcare and visitors in addition to other exposure history. Poor dissemination of new literature on "less prestigious" illnesses such as scabies creates a further challenge to physicians. Sharing these pitfalls and data is essential for timely diagnosis and treatment of scabies, both to alleviate the morbidity associated with scabies itself and prevent further duress associated with back and forth exposure and infection.

Endnotes

1. Paul D, Papier A. "Scabies: Refine your exam, avoid these diagnostic pitfalls." *J Fam Practice*. 2020 Jan; 69(1):10-17.
2. Anderson KL, Strowd LC. "Epidemiology, Diagnosis, and Treatment of Scabies in a Dermatology Office." *J Am Board Family Medicine*. 2017 Jan; 30(1):78-84.
3. Centers for Disease Control, https://www.cdc.gov/parasites/scabies/treatment.html
4. Carr PC, Brodell RT. "Images in clinical medicine: scabies." *N Engl J Med*. 2016; 374: e13.
5. Burkhart CG et al. "An Epidemiologic and Therapeutic Reassessment of Scabies." *Cutis*. 2000 Apr; 65(4):233-40.
6. Chandler DJ, Fuller LC. "A review of Scabies: An Infestation More than Skin Deep." *Dermatology*. 2019; 235(2):79-90.

The Great Mimicker

By Ani Bodoutchian, MD, MBA, FAAFP

Introduction

Eagle syndrome is a rare condition that can be characterized by sharp pain in the jaw, the back of the throat and the base of the tongue. Moreover, it can be triggered by swallowing or moving of the jaw. The abnormal stylohyoid impinges on anatomic structures in the area resulting in a myriad of symptoms that mimic other conditions. The aforesaid causes Eagle syndrome to present clinically in different ways.

Case Report

A 60-year-old male with multiple medical problems, including diabetes, hypothyroid, hypertension, bilateral carotid disease, degenerative joint disease and arthritis, visited his family physician for persistent neck pain that lasted for several years. He had progressive pharyngeal pain with swelling in his neck for three months. He complained of dysphagia to solids as well as liquids associated with pain. Additionally, he complained of fullness in the neck and the sensation that "something is stuck there." He denied fever, chills, nausea, vomiting, coughing or weight loss. He reported being a former smoker.

The physical exam was unremarkable except for some mild hoarseness, throat pain and dryness. Additionally, there was swelling on both sides of the neck. There were no limitations on movement of the head and neck and no palpable masses. Computerized tomography (CT) scan with intravenous contrast of soft tissues of the neck and base of skull was done. (Figures 1-2)

Follow up with the patient on CT findings indicated elongated styloid process, favoring Eagle syndrome, and clinical finding of severe dysphagia. He was referred to a head and neck surgeon and was lost to follow up after his appointment with the surgeon, despite repeated contacts.

Discussion

Eagle syndrome is a rare and poorly understood condition due to the innumerable ways in which it presents. Normally, the styloid process is a cartilaginous bone approximately 20-30 mm long. Eagle syndrome is the elongation of bilateral styloid processes, with an overall length in excess of 30 mm approximating the hypopharyngeal mucosa.[1] The persistent glossopharyngeal nerve entrapment causes severe pharyngeal pain due to elongated and calcified styloid process. Eagle syndrome can be found within the range of 4% and 7.3% of the population in the United States. Moreover, Eagle syndrome is more prevalent in women and the anatomic variant of stylohyoid complex is greater amongst females who are between the ages of 60 and 79.[2]

While the underlying cause for Eagle syndrome has not yet been determined, we do have a clearer understanding of how an abnormal stylohyoid causes the complexity of symptoms. This includes numerous anatomical structures passing in the area. The elongated stylohyoid causes the compression of the surrounding nerves and vasculature. Moreover, the stylohyoid complex bifurcates the internal and external carotid arteries, so any compression or impingement of the anatomic structures in the area can potentially present clinically in an array of different ways.[3] This arduous list can be further complicated as the patient's comorbid conditions can play a significant and critical role in making a diagnosis.

Eagle syndrome's clinical manifestations often resemble other diagnoses such as:

- Neuralgias of the cranial nerves which involves disturbances of smell, taste, vision, hearing, balance, speech, swallow and muscles of the face, neck or gland regulation.[4]
- Temporomandibular disorders where there is limitation of jaw opening.
- Neoplasm of the neck which presents with loss of appetite, dysphagia, swelling or lump in neck.
- Oral and dental infections which present with adenopathy and pain on the side of the infection.
- Mastoiditis which would be preceded by a middle ear infection, pain, fever and possible hearing loss.

continued on page 50

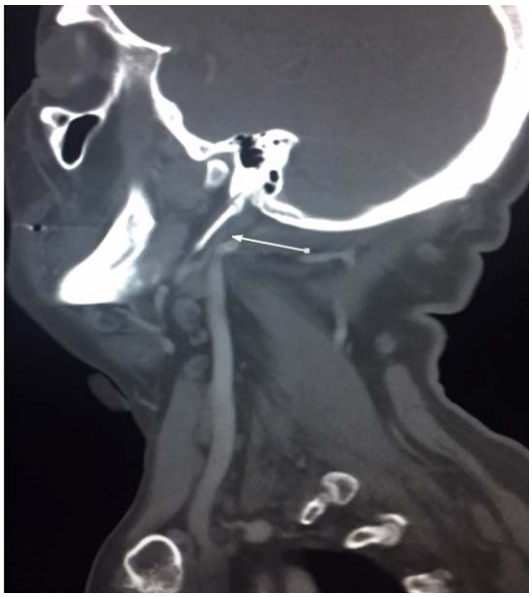


Figure 1 *(left)*: CT Scan – Sagittal view of elongated stylohyoid bone

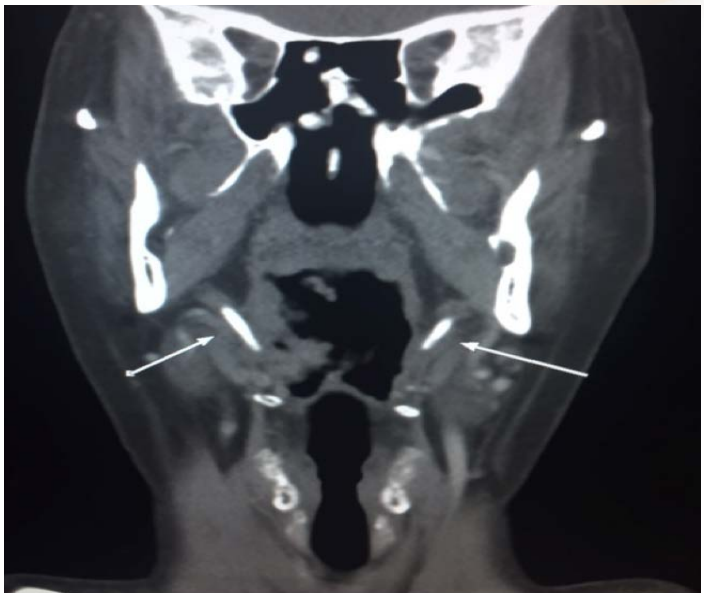


Figure 2 *(right)*: CT Scan – Coronal view of elongated stylohyoid bone

continued from page 26

Pine sap is visible. Get some on your hand or clothing, and soap and laundry detergent don't remove the sticky. Enter GoJo.™ This is a hand cleaner for auto/bike mechanics. It comes in plain and orange oil scented, and original and pumice laced. It is a cream/viscous liquid with mineral oil and petroleum distillates and removes pine sap. Perhaps it removes PI sap as well. Meanwhile, other methods of treatment and prevention have not been highly effective.[4]

I started using GoJo™ for my patients and personal exposures decades ago. Used on a small area of plant contact dermatitis it gives some immediate relief from itching, and healing is evident in a couple of days.

Case report: After an NYSAFP meeting on Long Island I wanted to camp on the shore in a park not usually accessible to this upstater. We had evolved to a civilized trailer, but towing that through Manhattan to the meeting would not make a relaxing trip, so we brought our tent. After the meeting, we arrived at the park after dark, dressed in shorts and sandals, set up our tent and went to sleep. In the morning we realized we had set up in a luxurious patch of PI. I ran to the shower house with my jar of GoJo and treated all exposed skin. Never broke out.

Case report: We drove into Southwick Beach State Park to camp on the shore of Lake Ontario. Dunes, trails and miles of beach. The agent in the contact station handed us our site assignment and a printed sheet: "We have a lot of PI in our park this summer. If you get into it, follow this route to this town, to this hardware store, and get a jar of GoJo." The park superintendent had made the same discovery. The "painless, harmless and cheap" standard is questionable here. It is unclear whether petroleum distillates applied to a large area of inflamed skin may meet the standard of harmless, but mechanics have been using this stuff on small areas, hands, for generations. And the MSDS appears benign.[5] GoJo clearly works for prevention. Early treatment of localized dermatitis has worked in my experience. Treatment of extensive dermatitis requires more study. GoJo without pumice was unavailable locally even before the pandemic. I purchased a half gallon pump container online for the office. When a patient arrives with PI, we dispense a paper cup of brew to take home and use in the shower. The instruction is to apply a generous glob, let it dissolve plant sap a few minutes, squeegee it off with a gloved hand, then shower.

"Off-label prescribing is when a physician gives you a drug that the U.S. FDA has approved to treat a condition different than your condition. This practice is legal and common, with one in five prescriptions written today for off-label use."[6] Innovation, learning from patients and even from park superintendents, being curious about how nature works – all of these enrich my life and hopefully those of my patients. The fact that one in five prescriptions is "off label" means I am not alone in this quest to learn what works.

(2) Second guiding principle: Don't argue with success.

Endnotes

1. Cover quotation "The Pharos" Autumn 2021 edition
2. you tube video: https://www.youtube.com/watch?v=i51y6t1YRNQ
3. UpToDate: There are no head-to-head trials comparing the individual methods for cerumen removal. Systematic reviews have not found superiority of one method over another
4. "Poison Ivy Dermatitis, UpToDate https://www.uptodate.com/contents/poison-ivy-toxicodendron-dermatitis?search=poison%20ivy%20treatment&source=search_result&selectedTitle=1~55&usage_type=default&display_rank=1
5. GoJo MDDS https://images.salsify.com/image/upload/s--GWL3GwAz--/kax4ry6oawkjgwmsn95a.pdf
6. AHRQ statement on off label use: https://www.ahrq.gov/patients-consumers/patient-involvement/off-label-drug-usage.html#:~:text=Off%2Dlabel%20prescribing%20is%20when,are%20for%20off%2Dlabel%20use.

continued from page 49

- Carotid vasculopathies include Takayasu's arteritis, giant cell arteritis, fibromuscular disease with nonspecific symptoms of malaise, fatigue and joint aches.[5]

Medical history, physical exam as well as meticulous interrogation of the patient's signs and symptoms are the cornerstones of the diagnosis of Eagle syndrome.[6] The astute physician needs to be mindful of this diagnosis when pain is recreated with movements of the head in flexion, extension and contralateral rotation.[6] Some more common signs and symptoms in association with the head movements are ear pain, facial pain, throat pain or fullness in the throat as if there is a foreign body, and/or difficulty swallowing (as with our patient).

However, there are also variants of Eagle syndrome. The elongated styloid that impinges the on the carotid artery can cause dissection, transient ischemic attacks or impairment of the body's fight or flight response.[7]

The jugular vein variant can manifest in headaches and vertigo due to impaired cerebral blood flow. "The jugular variant has been associated with idiopathic intracranial hypertension and peri-mesencephalic subarachnoid hemorrhage. Both as a sequala of intracranial venous hypertension due to jugular impingement." [7]

The optimal diagnostic image of choice for styloid process pathology is spiral CT scan of the neck and skull base.[8]

For the neuropathic sequelae of Eagle syndrome, noninvasive management is necessary. There is an array of different oral agents that may be taken including valproate, gabapentin and carbamazepine. If surgical intervention is necessary, that will only be considered after noninvasive therapies have not worked in an effective manner.[6]

Endnotes

1. Cumali, Gokce, Yildiray Sisman, and Murat Sipahioglu. Styloid Process Elongation or Eagle's Syndrome: Is There Any Role for Ectopic Calcification? European Journal of Dent. 2008 Jul; 2: 224–228.
2. Constantinides, F., Vidoni, G., Bodin, C., Di Lenarda, R. Eagle's syndrome: Signs and symptoms. *The Journal of Craniomandibular & Sleep Practice*. 31: 56-60.
3. Kim, E., Hansen, K, Frizzi, J. Eagle syndrome: Case report and review of the literature. *ENT Journal*. 2008; 87: 631-633.
4. Overview of the Cranial Nerves - Brain, Spinal Cord, and Nerve Disorders https://www.merckmanuals.com/home/brain,-spinal-cord,-and-nerve-disorders/cranial-nerve-disorders
5. ArijanaLovrencic-Huzjan. Diagnosis of non-atherosclerotic carotid disease, Perspectives in Medicine. Volume 1, Issues 1–12, September 2012
6. Fusco, D., Asteraki, S., Spetzler, R. Eagle's syndrome: Embryology, anatomy and clinical management. *Acta Neuropathologica*.2016; 154: 1119-1126.
7. Goomany, Anand, et al. "Eagle Syndrome: Elongated Stylohyoid-Associated Facial Pain." *BMJ Case Reports*, BMJ Specialist Journals, 1 Mar. 2020, http://dx.doi.org/10.1136/bcr-2019-234024.
8. Badhey, A. Jategaonkar, A., Kovacs, A., Kadakia, S., De Deyn, P., Ducic, Y., Schantz, S., Shin, E. Eagle syndrome: A comprehensive review. *Clinical Neurology and Neurosurgery*.2017; 159: 34- 38.

New York State Academy of Family Physicians
16 Sage Estate, Suite 202
Albany, New York 12204

PRSRT STD
US POSTAGE
PAID
PERMIT #203
ALBANY, NY

Re**MEMBER** your **BENEFITS**!

NYSAFP Membership Provides:

Advancing our Specialty, Saving Members Time, Maximizing Values of our Dues

- Representation at the AAFP
- Representation of the local county chapters at the NYSAFP Congress of Delegates
- Promotion of family medicine in the medical schools and support of student programs
- Support of family medicine residency & fellowship training programs
- Representation of family medicine in the federal & state legislatures and policy makers through the PAC

Saving Members Time

- Hosting of relevant and interactive CME workshops
- Hosting of ALSO instructor and provider courses
- Opportunity to interact with fellow family physicians throughout the state
- Reliable source of relevant and current events
- Weekly e-NewsBrief
- Quarterly peer reviewed journal – *Family Doctor*
- Timely access to current events of Academy via social media (NYSAFP Facebook | NYSAFP Twitter)

Maximizing the Values of our Dues

- Sponsorship of students and residents to Academy meetings (Winter Weekend, Regional Family Medicine) and the Congress of Delegates
- Cultivation of the next generation of family physicians by offering scholarships and awards to pre-medical students, medical students, and residents to participate in family medicine conferences and programs
- Support of residents and new physicians in development of leadership skills and practice opportunities

AAFP Member Services: http://www.aafp.org/online/en/home/membership/resources.html

- A list of the AAFP professional resources
- A list of the AAFP "Member Advantage"
- Additional Partnerships: http://www.nysafp.org/index/resources-6/partner-programs-106.html
- Jobs Board