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Number 5

2026 SDSMA Annual Leadership Conference

Advancing Patient Care Through Lifestyle Medicine

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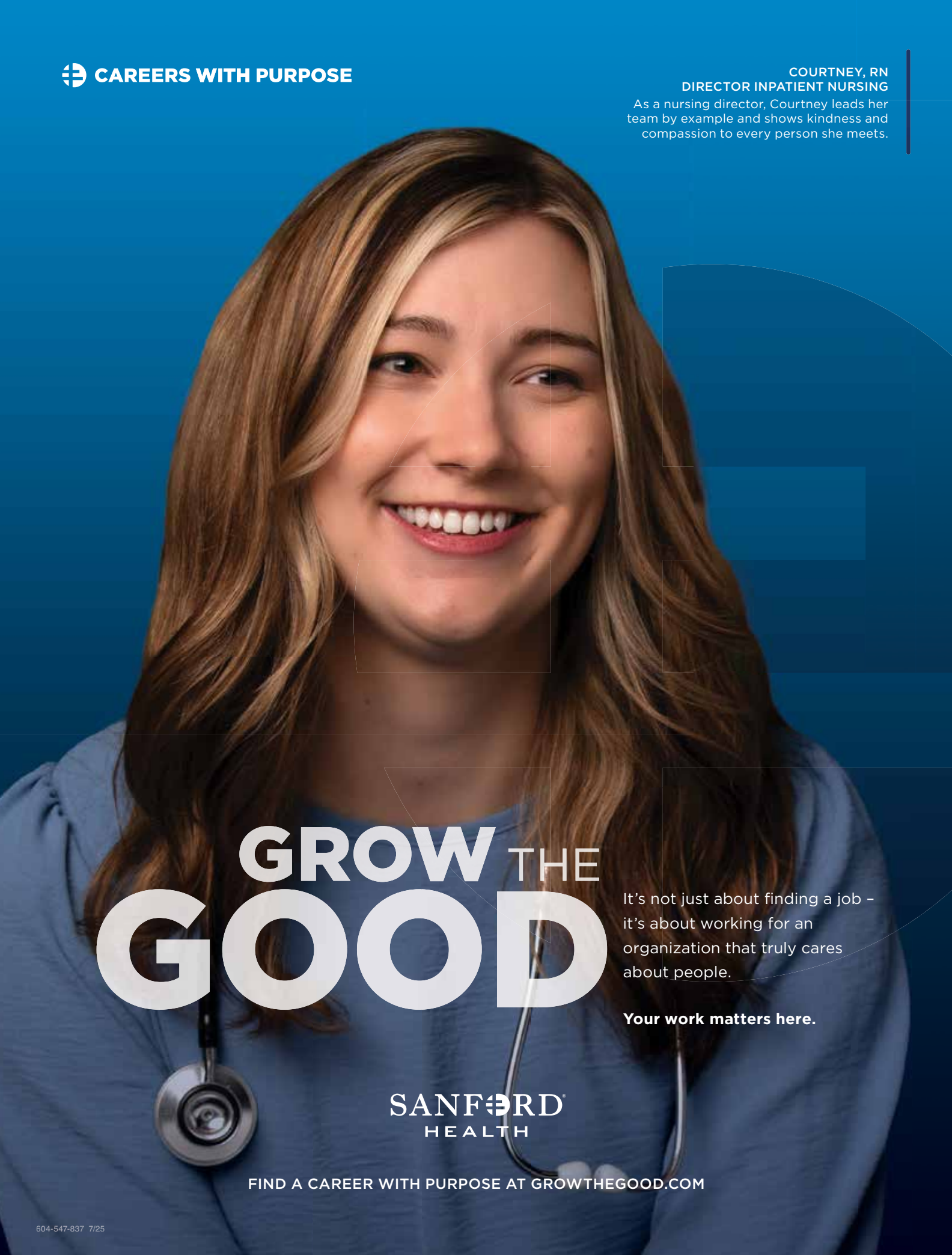

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Imagine that you're a marathon runner lining up to cover 26.2 miles as fast as possible. In the beginning, it's tempting to sprint ahead when you see other competitors charging forward. But you could burn out too quickly and not have enough gas left to finish the race.

I learned this the hard way. My first marathon was in Minneapolis, back in the early 2000's. My only strategy was to carb load the night before, with a plate of pancakes at Perkins. Other than that, I figured I would pace off the front-runners. Why not? After all, they must have known what they were doing. So, if I wanted success, chasing them made sense. I quickly realized my mistake and almost had to be carried to the car after crossing the finish line.

The same basic principle applies to investing. You might see some stocks or assets with dramatic short-term returns. The temptation to jump in and participate is overwhelming. Everyone else is doing it, right? But just because something looks like a sure bet in the moment doesn't mean that it aligns with your long-term success. FOMO is real. However, just like in a marathon, committing yourself to a solid, pre-planned strategy will help ensure you successfully cross the finish line.

Fortunately, I learned my lesson after that first race and adapted my approach. Ultimately, this landed me at the Boston Marathon a decade later. If what other investors are doing is not part of your plan, then have the discipline to tune out the noise and stick to your strategy. Chasing short-term returns might feel rewarding in the moment, but it's often a sprint that leaves you exhausted, with nothing to show for your effort.

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Receiving the Critique: What Medical Educators and Learners Both Need to Practice

Jason J. Kemnitz, EdD

In my second year of graduate school, I enrolled in a semantics course built around a linguistic discipline called E-Prime, which requires writers to eliminate all forms of the verb "to be" from their prose. The premise intrigued me: strip those passive constructions away and clearer, more accountable writing follows. For an assignment, I selected a paper I considered among my strongest work, translated it painstakingly into E-Prime, and submitted it expecting praise. My professor returned it with a B– and a single, memorable comment: "Nice job with the E-Prime portion; unfortunately, the rest reads as boring drivel."

Offended, I went to find him. He responded with two questions: "Did I assess your work incorrectly? And if not, what genuinely upsets you?" He had not assessed incorrectly. I had spent so much energy on technical compliance that I had neglected to write anything worth reading and I had arrived completely unprepared to hear that said aloud. That conversation has stayed with me for years, not because of E-Prime, but because of what it revealed: receiving critical feedback requires just as much deliberate practice as any competency we ask our learners to develop. We invest considerable effort teaching people how to give feedback. We invest far less teaching them how to receive it.

Across medical education, faculty commonly voice frustration that learners neither absorb feedback nor act on suggested improvements. Learners, in turn, could likely say something similar about certain teaching approaches. That bidirectional gap deserves our attention. Focusing exclusively on feedback delivery, while leaving reception largely untaught, means our trainees carry only half the tools they need for a professional lifetime of growth. The following framework offers a practical starting point, applicable to students, residents, and faculty alike.

Recognize the Emotional Response First

Critical feedback, whether delivered gently or bluntly, almost always triggers an emotional reaction. That reaction often feels like a threat to identity rather than a comment on performance, and understanding that distinction marks the first step toward receiving feedback productively. When defensiveness surfaces, it usually signals ego-involvement: a perception that the critique targets personal worth rather than specific actions. In most cases, that perception does not reflect the intent behind the feedback. Critical feedback identifies blind spots, surfaces gaps in performance, and charts a path forward. The discomfort a receiver feels belongs to the process. Separating self-worth from the work under review allows the feedback to function as it should: as information, not verdict.

Engage Actively During the Exchange

A five-step approach can shift the experience of receiving critical feedback from threatening to genuinely useful. First, listen to the feedback in its entirety without interruption, this allows the provider to complete their thinking and gives the receiver space to manage the initial emotional response before reacting. Second, ask clarifying questions, framed as genuine inquiries rather than veiled pushback. Third, request specific examples that illustrate the concern. Fourth, restate your understanding to confirm you have heard accurately. Fifth, acknowledge the feedback and thank the provider, regardless of whether you plan to act on every point. This sequence does not require agreement. It requires engagement, and that alone can transform a charged interaction into a structured opportunity for growth.

Reflect, Then Act

The work that follows the exchange determines whether feedback translates into meaningful change. Two questions help guide that reflection: Has another source offered similar feedback before? And does this person hold genuine expertise in the area under discussion? Affirmative answers to either question lend the feedback real weight, not as a final judgment, but as expert input worth taking seriously. From there, the task involves identifying concrete, actionable next steps. When those steps remain unclear, returning to the feedback provider for further clarification signals exactly the kind of intellectual humility and commitment to growth we try to model and cultivate throughout medical training.

Receiving critical feedback challenges even experienced clinicians and educators. The essential skill involves separating the actions under review from the emotions they stir, then translating that discomfort into forward momentum. Looking back at that graduate school encounter, my professor's bluntness improved my writing. More importantly, it improved my capacity to hear difficult truths, a skill that has served me far longer than any paper I wrote that semester. My challenge to you: the next time someone offers honest, critical feedback, resist the urge to defend first. Listen first. Ask a question. Agreement on every point does not matter. The willingness to receive feedback openly separates those who grow from those who stay stuck.

As for E-Prime: I remain uncertain whether eliminating "to be" consistently produces clearer writing. Perhaps that question, like feedback itself, ultimately depends on what the reader finds valuable. What do you think?

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Attend the SDSMA 2026 Annual Leadership Conference

Keith Hansen, MD
President, South Dakota State Medical Association

I encourage you to attend the 2026 SDSMA Annual Leadership Conference, *Advancing Patient Care Through Lifestyle Medicine*, which will be held on May 29, 2026 at The Catlin Hotel (previously the Hilton Garden Inn) downtown Sioux Falls. This meeting promises to be an interesting and educational exploration of lifestyle medicine and its incorporation into the practice of medicine. It also allows the opportunity to network with colleagues, receive an update on the University of South Dakota Sanford School of Medicine, and learn of the multitude of advocacy efforts by the SDSMA and AMA on local, regional and national stages.

The night prior to the annual meeting, District 7 Medical Society is hosting a welcome reception at the new SDSMA office at 1610 S. Minnesota Ave. Aside from seeing our new office building, please stop by to meet, if you haven't already, our new CEO Tammy Hatting, and Kayla Keeler, our new Membership and Executive Services Manager. This is also an excellent occasion to talk with our established staff members and lobbyists and allows for informal conversation with the SDSMA Board of Directors and AMA representatives.

The conference will kick off with an incredible opportunity to listen to an AMA update, delivered by Willie Underwood, MD, the AMA president-elect and practicing urologist from Buffalo, New York. Dr. Underwood is an expert on health care policies and health care disparities who has published over 120 peer-reviewed publications, chapters and abstracts. He has also co-founded KAPS Biotechnology LLC which is performing validation studies on a new prostate cancer biomarker.

Following Dr. Underwood's presentation we'll hear presentations on lifestyle medicine and its impact on health. Michelle L. Thompson, DO, will present *From Sick Care to Self-Care Transforming Self and Systems Through Lifestyle Medicine*. Dr. Thompson practices integrative family medicine at the University of Pittsburgh Medical Center. This will be followed by Allen Weiss, MD, on prevention as a cure for healthcare and our nation's economy. These two presentations will be followed by an exciting panel discussion on the progress and growth of lifestyle medicine in South Dakota. The distinguished panel, moderated by Robert Allison, MD, includes Marty Allison, MD; Michelle L. Thopson, DO; Amy Prunuske, PhD; Allen Weiss, MD, Kathryn Wermers, DNP; and Secretary of Health Melissa Magstadt.

Lunch is sponsored by the Black Hills District Medical Society and will includes a presentation by South Dakota Secretary of Health Melissa Magstadt on the Rural Health Transformation Project. With the recent federal award of $189.4 million to South Dakota to enhance rural health, it promises to be an interesting talk and conversation.

Following lunch, Tim Ridgway, MD, will give an update on the University of South Dakota Sanford School of Medicine. There are a number of exciting updates from the school of medicine including the recent announcement of the move of Pillar 1 medical education (Basic Biomedical Education) to Sioux Falls. After Dr. Ridgway's update, we will have the opportunity to attend a medical student and resident poster session. It promises to be an interesting session with many interesting cases and research activities. The students and residents have worked hard to develop these poster presentations and are looking forward to presenting to you.

The program will then move into the SDSMA Policy Council meeting and membership open forum where updates are given and policy developed for use in SDSMA's advocacy efforts. The open forum is always an interesting discussion of important concerns that impact medical practice in South Dakota and how the SDSMA can assist.

The Awards Banquet, Scholarship Recognition and Presidential Inauguration in the evening will be the finale of the meeting. I urge you to attend as this is an opportunity to recognize physicians as well as others who support our patients and the practice of medicine in South Dakota. We also have the opportunity to recognize the medical students who are SDSMA Foundation scholarship recipients. This year the SDSMA Foundation has awarded more than $400,000 in medical student scholarships, which in this time of increasing educational costs is critical for our students.

To conclude my term as president, I am honored to inaugurate our next president, Benjamin Meyerink, MD. Dr. Meyerink, a family medicine physician at Avera Medical Group in Sioux Falls, is a respected and capable leader who is well-prepared to guide our association through the coming year. Congratulations to Dr. Meyerink on this new leadership role.

I hope to see you at the conference in Sioux Falls on May 29!

HISTORICAL SERIES:

Discovery & Timeline of 65 Roses

Erin Denevan, MS II; and H. Bruce Vogt, MD

The phrase "65 Roses" was coined in 1965 by young Richard Weiss, who misheard his mother making fundraising calls for the Cystic Fibrosis Foundation. At the time, Richard and his two brothers had all been diagnosed with cystic fibrosis (CF).[1] At this time, 65 Roses was a new progressive genetic disorder with much to be discovered. Twelve years earlier, a diagnostic test was discovered, preceded twenty years by a groundbreaking discovery by Dr. Dorothy Anderson describing the condition. Dr. Dorothy Anderson laid the foundation for the future diagnosis and treatment of cystic fibrosis.

Cystic fibrosis is an autosomal recessive genetic disorder with an incidence of 1 in 2,000-3,000 live births of populations of European descent and 1 in 15,000 live births in populations of African-American descent.[2] This genetic condition mutates the cystic fibrosis transmembrane conductance regulator (CFTR) gene, which changes the protein that regulates chloride ion movement in epithelial cells.[3] The faulty protein impacts salt and water balance, enzyme production, and mucus secretions. Abnormally thick, sticky mucus is produced, leading to dysfunction primarily in breathing and digestion. While the molecular basics of CF are understood today, the recognition as a distinct pathology began decades earlier with careful clinical observations.

In 1938, Dr. Dorothy Anderson's work consisted of detailing 49 cases by their clinical symptoms, cause of death, and post-mortem findings.[4] Dr. Anderson identified pathologic changes characteristic of what she termed cystic fibrosis of the pancreas across three age-based groups of patients. The youngest infants died from intestinal obstruction; those aged one week to six months died from pulmonary complications or failure to thrive; and patients aged six months to 14.5 years died from pulmonary infections, often accompanied by symptoms consistent with celiac disease.[4] Dr. Anderson diligently examined each organ of the autopsies, looking for the pattern connecting these children's deaths. Every case of the series presented with uniform changes due to infection in the primary bronchi. The pathological report of the pancreas was the beginning of the identifiable disorder, laying the groundwork for future diagnosis and treatment. Little did Dr. Anderson know that it would be years before another advancement would be made in the clinical picture of cystic fibrosis.

Almost 20 years later, a diagnostic test was created looking to solve why patients with cystic fibrosis were subjected to vasomotor collapse in hot weather. In 1958, Dr. Paul Di Sant' Agnese, Dr. Robert C. Darling, Dr. George A. Perera, and Ethel Shae looked for the mechanism of severe dehydration observed in children as casualties of heat intolerance. Given the evidence of glandular disturbance in cystic fibrosis and the presentation of these patients with profuse sweating, they sought to relate the observed electrolyte abnormalities to the pathophysiology of the genetic disorder. Control patients and cystic fibrosis patients underwent a standard thermal stress test to collect sweat from the middle of the abdomen. Patients with cystic fibrosis had electrolyte values nearly double the chloride and triple the sodium in their sweat compared to controls. Potassium levels were also increased in the CF group, but not to the same magnitude. The team determined that the volume of sweat produced between groups stayed relatively constant, while the electrolyte differences were clearly distributed across groups. Under normal conditions, both groups' serum electrolytes were within the normal range.[5] The study was further grouped into four categories by primary pathology, from complete pancreatic deficiency to pulmonary disease. Comparing the spectrum of CF subgroups showed no significant sweat electrolyte abnormalities among them. The team concluded that while the overall volume of sweat remained relatively constant between the control and CF subgroups, significant differences in electrolyte composition highlighted a distinct physiologic difference associated with cystic fibrosis. At this time, increased sweat sodium and chloride were hypothesized to be pathognomonic of cystic fibrosis, but this was not conclusive. Instead of children presenting with a complication of malnutrition or lung disease, patients could be diagnosed proactively through a sweat chloride test in newborn screening. Fast forward over 60 years, and the sodium chloride sweat test is a gold standard for the diagnosis of cystic fibrosis.

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Cystic fibrosis's pathological findings were identified along with a diagnostic test, but the underlying genetic cause was still unknown. Adaptation of the sodium chloride sweat test was slow to be widely accepted. However, the discovery of the CTFR gene in 1989 catapulted the sphere of diagnosis and treatment. In the 1970s, the initial predictive criteria for cystic fibrosis used dried blood spot specimens looking for high immunoreactive trypsinogen levels.[6] This protocol was highly debated due to the low sensitivity with varying reliability. Starting in 1985, two cystic fibrosis centers in Wisconsin initiated a randomized clinical trial of newborn screening, which led to the discovery of the p.Phe508del (deletion of three bases coding for phenylalanine at the 508th position) CFTR gene variant on chromosome 7. The initial advancement in the field led to a two-tier screening method; first, the immunoreactive trypsinogen test, if positive, followed by the DNA analysis for the CFTR gene mutation.[6] Testing for identification of cystic fibrosis in newborns was on the rise, but not yet widely done due to concerns about contamination in the DNA PCR amplification method. After proving the dried blood spot method was not altering the results, the Madison and Milwaukee centers studied their patient population with diagnosed cystic fibrosis for the CFTR variant. 88% of the patient population were homozygous or heterozygous for the p.Phe508del variant, with the remaining population having a different CFTR mutation. Currently, over 1900 mutations have been identified in the CFTR gene. As the two-tier test was refined and more widely accepted in the 1990s, its advantages led to advocacy from the CDC and the U.S. Cystic Fibrosis Foundation for universal cystic fibrosis newborn screening in 2004.[6]

The evolution of the cystic fibrosis newborn screening test, easing its way throughout the world, led to earlier diagnosis, brought about more preventive therapies, and showed that earlier intervention benefited those suffering from the irreversible disorder. The discoveries about cystic fibrosis since 1938 have unveiled the genetics, pathophysiology, and improved treatment for patients. During Dr. Dorothy Anderson's active research years, children were likely to die in the first year of life. Presently, the median survival age of patients with cystic fibrosis is closer to 50 years of age.[2]

While there is no cure for CF, early intervention can slow the worsening condition over the patient's lifespan. Systematic antibiotic usage is utilized to treat inevitable lung infections and delay the onset of resistance. Symptomatic medications include anti-inflammatory steroids and bronchodilators.[7] A newer medication in the field of CF treatment are CFTR modulators. These modulators are specific to different CFTR gene mutations and increase lung and digestive function through improved CFTR protein function. Not all CF patients are eligible for CFTR modulators and must undergo genetic testing.[8] Breathing support involves oxygen therapy, pulmonary rehabilitation, and chest physiotherapy. Chest physiotherapy works to clear the airway, aiming to decrease inflammation and infection risk. Physiotherapy is usually performed multiple times a day and encompasses different physical techniques and mechanical devices. The advancements in treatment have nearly doubled the adult CF population. As survival improves, so too does quality of life, underscoring the importance of continued research and advocacy for those living with cystic fibrosis.

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A Rare Case of Purtscher-like Retinopathy Secondary to Anti-Synthetase Syndrome: A Case Report and Literature Review

Sam Diemer, MS II; Elizabeth Atchison, MD; and Mark Vercel, DO

Abstract

Anti-Synthetase syndrome is a rare and potentially life-threatening subtype of dermatomyositis as part of the spectrum of idiopathic inflammatory myopathy (IIM) that can affect multiple organ systems. Here, we review many of the possible ocular manifestations of this inflammatory myopathy and highlight a case with retinal involvement. A man in his 30's with a history of anti-synthetase syndrome complained of blurred vision of both eyes ongoing for two weeks. A fundoscopic examination revealed numerous cotton wool spots in both eyes without any signs of hemorrhage or Purtscher flecken. Optical coherence tomography showed no evidence of macular edema. A month prior to presentation, the patient was prescribed oral prednisone and IV methylprednisolone in adjunct to mycophenolate mofetil with a recent infusion of rituximab; the drug course was continued. Best corrected visual acuity returned to 20/20 in both eyes with accompanied improvement of cotton wool spots correlating with his course of immunosuppression.

Introduction

Anti-synthetase syndrome is a rare autoimmune-mediated condition in the dermatomyositis spectrum characterized by autoantibodies directed against an aminoacyl transfer RNA synthetase, most commonly Anti-Jo-1. Characteristic physical exam findings include weakness, cutaneous manifestations of mechanic's hands, photosensitivity dermatitis, Gottron papules and heliotrope rash, inflammatory arthritis, inflammatory lung disease, and dysphagia. Vascular changes often result in Raynaud's phenomenon, with characteristic nailfold capillaroscopy changes.[1] The cause of anti-synthetase syndrome is unknown, however, there have been multiple documented contributing factors in dermatomyositis related diseases including malignancy, several HLA haplotypes, viral infections, and certain drugs.[2] In idiopathic inflammatory myopathies (IIMs), retinal involvement occurs in nearly 33% of cases, usually manifesting as vision loss due to retinal vessel abnormalities like attenuation and AV nicking, likely due to the underlying vascular pathology of IIM. In few cases, there has been evidence of cotton wool spots or hemorrhage.[3] Further, the term Purtscher retinopathy is used to describe a fundus exhibiting cotton wool spots, hemorrhages, and confluent areas of ischemic retinal whitening termed 'Purtscher flecken', often caused by trauma. However, in absence of trauma, these clinical findings are then termed Purtscher-like retinopathy. Purtscher-like retinopathy has been associated with multiple clinical entities, including acute pancreatitis, Valsalva maneuver, thrombotic thrombocytopenic purpura, hemolytic uremic syndrome, cryoglobulinemia, pregnancy related complications, lupus, retrobulbar anesthesia, pancreatic carcinoma, thrombotic microangiopathy following antineoplastic use, and multiple myeloma.[4] Dermatomyositis subtypes have been associated with other ocular manifestations, such as dilated bulbar vessels, iritis, episcleritis, uveitis, optic disc pallor, periorbital masses, retinal artery occlusion, ophthalmoplegia, and cotton wool spots. These sequelae are caused most in part to the complement deposition in ocular muscle capillaries and membrane attack complexes depositing in vascular walls, leading to inflammation and hypoxic injury to the affected tissues. Key to this case presentation, cotton wool spots are an exam finding indicative of arteriolar obstruction or capillary damage as a result of nerve fiber layer infarction production axonal swelling and rupture.[5] In this case report we describe a man with Purtscher-like retinopathy secondary to the anti-synthetase syndrome.

Case Presentation

A man in his 30's was referred to our clinic by his local optometrist with a two-week history of worsening blurred vision in both eyes (OU). He also complained of increased

light sensitivity and mild pressure OU. He denied any floaters. He had no ocular history and no family history of ocular disease.

A thorough eye exam was performed revealing uncorrected visual acuity of 20/30 right eye (OD), 20/20 left eye (OS), intraocular pressure was normal OU, full orthotropic motility OU, full confrontational visual field, normal ocular adnexa, and sclera was white and quiet with no evidence of conjunctival vessel dilation.

Dilated examination revealed no evidence of disc edema or pallor, cup-to-disc ratio of 0.2 OU, normal caliber retinal vessels, no evidence of macular edema or subretinal fluid, and cotton wool spots. Fundoscopic photos revealed numerous cotton wool spots extending outward from the optic disk and surrounding the macula (Figure 1). Optical coherence tomography showed no signs of macular edema with further evidence of cotton wool spots encroaching the macula in OU.

The patient was diagnosed with anti-synthetase syndrome five weeks prior with symptoms associated with his progressive inflammatory myopathy, advanced pulmonary fibrosis, mechanic's hands, and weight loss, as a rapidly progressive presentation of the anti-synthetase syndrome. This diagnosis was confirmed by muscle biopsy, as well as serologic evidence of anti-Jo1 antibody positivity. He was started on a treatment regimen of IV methylprednisolone 1000mg for three days, oral prednisone 60 mg daily, mycophenolate mofetil 1500 mg twice daily, and received his first infusion of Rituxan the day prior to the initial visit. This regimen was continued with four follow-up dilated fundoscopic exams, each roughly one month apart.

Figure 1. Fundoscopic photos showing numerous cotton wool spots.

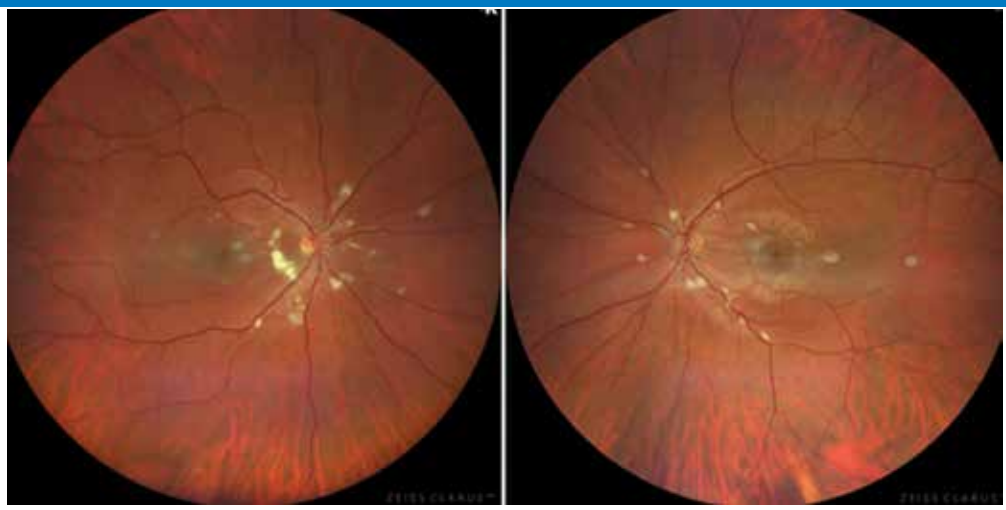
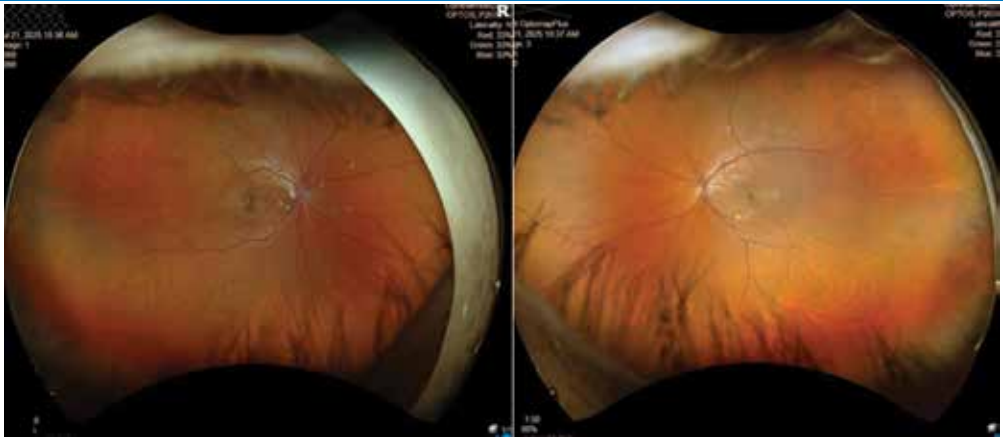


Figure 2. Fundoscopic photos showing gradual resolution of cotton wool spots.



The follow-up visits showed progressively improving appearance of cottonwool spots OU (Figure 2) and subjective improvement in the patient's vision. His final uncorrected visual acuity measured to be 20/20 OU three and a half months after initial presentation.

Discussion

Purtscher-like retinopathy secondary to IIM is an uncommon, and potentially severe complication. If left untreated, further retinal changes can occur leading to occlusion, hemorrhage, and ultimately permanent vision loss, similar to reported cases in dermatomyositis.[2] The anti-synthetase syndrome is a rare subtype of this uncommon disease spectrum, characterized by antibodies against tRNA various synthetases, the most common of which is anti-Jo1 antibody formation. Many of the manifestations of this disease, such as various cutaneous findings, muscle breakdown, interstitial lung disease, dysphagia, and predisposition to malignancy, have been well documented in literature. Retinal complications have been described in cases of IIM, however, there have been less than a dozen reported cases exhibiting Purtscher-like retinopathy. Further, there have been no reported incidences of Purtscher-like retinopathy specific to anti-synthetase syndrome. When treating this retinal condition, immunosuppressants are often chosen, however, there is no standard regimen. In similar documented cases of Purtscher-like retinopathy secondary to IIM, treatment has varied from glucocorticoids, rituximab, plasmapheresis, and cyclophosphamide. [6-11] Coincidentally, when initiating treatment for IIM, urgent use of these immunosuppressants are used to prevent progression of the disease, potentially treating and ultimately masking the incidence of this retinopathic manifestation. Providing this case, in addition to the current literature of retinal complications associated with IIM and their treatments with subsequent results, is valuable in providing more data for further investigation.

Conclusion

This case highlights a rare ocular manifestation of anti-synthetase syndrome. By detailing the clinical findings and supplemental literature review, we provide additional data and insight into this potentially severe complication seen uncommonly in IIM.

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Increased Risk of Alcohol Use Disorder Following Bariatric Surgery

Avery D. Franzen, MD, PhD; Jadah Bartow, MD; and Hannah Statz DeVries, MD

Abstract

Background: Obesity affects over 40% of adults in the U.S., and bariatric surgery, particularly Roux-en-Y gastric bypass (RYGB), is among the most effective treatments for sustained weight loss and comorbidity reduction. However, emerging evidence has linked bariatric surgery to an increased risk of alcohol use disorder (AUD), particularly in the years following surgery.

Objective: To review the evidence surrounding the incidence, timing, mechanisms, and risk factors associated with AUD following bariatric surgery, with an emphasis on RYGB.

Methods: A comprehensive review of the literature examining postoperative rates of AUD among bariatric surgery patients was conducted, highlighting trends, mechanisms, and subpopulations at risk.

Results: RYGB is associated with a significantly increased risk of developing AUD, with incidence rising notably 2–8 years postoperatively. Risk factors include younger age, male gender, preoperative substance use, poor mental health, low social support, and undergoing RYGB rather than other surgical procedures. The delayed increase in AUD appears to result from both physiological changes such as faster alcohol absorption and higher peak blood alcohol levels and psychological factors like addiction transfer. Additionally, bariatric surgery increases risks for alcohol-related liver disease and psychiatric hospitalization related to alcohol misuse.

Conclusion: AUD is a significant long-term risk following RYGB, warranting routine preoperative screening, patient education, and long-term postoperative monitoring. Use of validated screening tools and provision of behavioral health support are critical to mitigate this risk and ensure optimal long-term outcomes for bariatric surgery patients.

Introduction

Obesity is an increasingly urgent healthcare crisis with 40.3% of adults in the U.S. being classified as obese (Body Mass Index (BMI) >30).[1] One of the most effective weight loss therapies is bariatric surgery, in particular, Roux-en-Y gastric bypass (RYGB) as it functions through both a malabsorptive and restrictive mechanism. Additional weight loss surgical therapies include restrictive methods such as laparoscopic-assisted gastric band and sleeve gastrectomy. Roux-en-Y gastric bypass at the basic level involves surgically separating the stomach into a small and large portion with the small portion being connected to the esophagus and small intestine.[2] Gastric band surgery is consists of placing a small, adjustable band around the upper portion of the stomach to slow the passage of food further into the stomach, making patients feel full with less food intake.[3] Sleeve gastrectomy is accomplished by removing a large portion of the stomach creating a smaller space where food and liquid can occupy[4]. RYGB improves all-cause mortality and decreases the rate of cardiovascular disease, myocardial infarction (MI), stroke, hypertension, hyperlipidemia, diabetes, respiratory complications, cancer, liver and gallbladder disease, and memory and cognition problems.[5] Unfortunately, several studies have reported increased rates of alcohol use disorder following gastric bypass. 10% of the general population in the U.S. has alcohol use disorder (AUD),[6] but postoperative gastric bypass patients can have rates four to five times higher than the general population.[7] This is a severe outcome following bariatric surgery and careful screening for risk factors should be undertaken prior to surgery with close follow-up postoperatively.

Development of Alcohol Use Disorder After Bariatric Surgery

While various bariatric surgeries including Roux-en-Y gastric bypass (RYGB), laparoscopic gastric band (LAGB), or sleeve gastrectomy (SG) increase the risk of alcohol use disorder

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(AUD), RYGB seems to confer the greatest increase in risk for AUD.[8,9] Undergoing RYGB vs. LAGB was associated with twice the risk of incident AUD symptoms with 20% of participants reporting incident AUD symptoms within 5 years post-RYGB in one study.[10] In the Longitudinal Assessment of Bariatric Surgery study, the year 5 cumulative incidence of AUD symptoms was 20.8% after RYGB and 11.3% after LAGB. In a single-center study the overall prevalence of AUD (n = 5724) was 9.6% preoperatively, 8.5% at 1 year postoperatively, and 14.0% at 2 years postoperatively. The preoperative, 1-year, and 2-year prevalence of AUD for SG were 10.1%, 9.0%, and 14.4%, respectively. The preoperative, 1-year, and 2-year postoperative prevalence of AUD for RYGB were 7.6%, 6.3%, and 11.9%, respectively in another study.[11] This lack of increase after one year followed by a large increase after year 2 demonstrates the delayed increase in AUD. When compared to no surgical intervention, bariatric surgery patients had an AUD hazard ratio of was 7.29 [95% confidence interval (CI): 5.06-9.48] in their first year post-operation.[12] While some studies indicate that there may be a decrease in the rate of AUD after bariatric surgery,[13,14] these studies are in the minority compared to those demonstrating an increased rate. Finally, the development of AUD is not the only concern for substance abuse after bariatric surgery as the rates of non-alcohol substance use disorders also increase post-operatively.[7,15-17]

Adolescents and veterans are two populations who regularly receive bariatric surgery and have particular risks. Adolescents are at high risk for development of AUD; in one study, half of the adolescent patients who received Roux-en-Y gastric bypass or vertical sleeve gastrectomy screened positive for alcohol use disorder.[18] In the veteran community, RYBG increased AUD to the extent it decreased the all-cause mortality benefit conferred from the surgery.[19]

The risk of AUD with bariatric surgery is frequently severe enough that there is a twofold higher risk of inpatient care for alcohol abuse in patients who received gastric bypass.[20] Outside of the increased risk of AUD and its subsequent psychiatric treatment, there are hepatic medical considerations. Bariatric surgery is associated with an increased prevalence of alcohol-related liver disease.[21] RYGB was associated with a 2- to 3-fold higher hazard for alcoholic hepatitis, abuse, and poisoning.[22] When alcoholic liver disease does present to medical attention, it frequently occurs with rapid decompensation after bariatric surgery.[23]

Delayed Development of AUD After Weight Loss Surgery

Several studies consistently show that the increase in alcohol use disorder (AUD) following Roux-en-Y gastric bypass (RYGB) surgery is delayed rather than immediate. The LABS-2 consortium reported that AUD prevalence rose from approximately 7% preoperatively to 16% seven years after RYGB, while rates remained stable following laparoscopic adjustable gastric banding (LAGB).[24] A meta-analysis found no significant increase in AUD within the first two years after bariatric surgery, but the odds became significantly higher by year three, particularly among RYGB patients.[24] Similarly, a study of U.S. veterans observed a significant increase in unhealthy alcohol use 3 to 8 years post-RYGB compared to control patients.[25] Supporting this, research published in Psychology, Health & Medicine found higher rates of hazardous drinking 3–4 years after surgery compared to less than 1 year postoperatively.[26] Collectively, these findings indicate that the risk of increased alcohol consumption and AUD following RYGB typically emerges over time rather than in the immediate postoperative period.

Mechanism of AUD after weight loss surgery:

Two major prevailing theories have arisen for a mechanism explaining the increased rate of alcohol use disorder following bariatric surgery. The first of these is anatomical/physiological with faster gastric emptying times presenting alcohol to the small intestine more rapidly where it is predominantly absorbed.[27] This leads to a more rapid rise in blood alcohol concentration and a higher blood alcohol concentration. [28,29] The other explanation for the increased rates of AUD is "addiction transfer" wherein patients who have food addictions prior to surgery swap their addiction for another such as alcohol or other substances of abuse. Notably, patients do experience increased guilt with their alcohol use and this may lead to a distorted perception of their alcohol use on self-reported studies.[30] Another factor is that alcohol's effect is self-reported to be more intoxicating post-operatively in addition to functioning as an appetite stimulant, pain reliever, and food replacement.[31,32] Some of the hallmarks of highly addictive substances include a rapid onset of action, short duration, intense euphoric effects, tolerance, and withdrawal.[33] Alcohol has an enhanced effect post-RYGB in the domains of onset of action, duration, and euphoric effects potentially explaining the increased rates of AUD, but this does not explain the delayed time-course of increased rates of AUD to 2-8 years post-operatively.[34]

Risk Factors for Development of AUD After Bariatric Surgery

Important factors that increase AUD risk after bariatric surgery that can help in screening pre-surgical candidates are listed below.[11,35–41]

- Younger age
- Male gender
- Family history of substance abuse
- Higher household income
- Regular pre-surgical consumption of alcohol or other substances (nicotine, recreational drugs)
- Poor mental health
- Attention deficit and hyperactivity disorder symptoms
- Depressive symptoms
- Poor coping skills/life stressors
- Decreased social support
- Lower sense of belonging
- Maladaptive eating patterns
- Roux-en-Y gastric bypass as the specific bariatric surgery method.

Summary and Recommendations

Bariatric surgery is one of the most effective treatments for weight loss

Alcohol use disorder occurs at a higher rate 2-8 years following bariatric surgery, particularly Roux-en-Y gastric bypass

The mechanism likely involves the change in anatomy leading to a more rapid absorption of alcohol, higher blood alcohol concentration, and a greater feeling of drunkenness.

Pre-operative psychological screening is done for all mental health disorders, but The American Society for Metabolic and Bariatric surgery recommends that patients specifically be screened for AUD. Patients must be made aware of the risk of developing AUD after surgery.

Persistent clinical follow-up is needed as alcohol abuse and dependence can occur up to 2-8 years after surgery. Screening tools such as Alcohol Use Disorders Identification Test (AUDIT) or CAGE may be beneficial.

Ongoing counseling regarding risk reduction & coping skills for addictive behavior should be greatly encouraged and provided to patients.

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AMA and SDSMA Membership After Graduation

Sydney Payne, MS IV; Keith Hansen, MD; and Susan Puumala, PhD

Abstract

Background: At the University of South Dakota Sanford School of Medicine, medical students are provided with memberships to the South Dakota State Medical Association (SDSMA) and American Medical Association (AMA). In this study, the primary goal is to determine if student membership increases membership in the SDSMA and AMA after graduation.

Methods: An 18-question survey was emailed by the South Dakota Board of Medical and Osteopathic Examiners (SDBMOE) to every South Dakota licensed physician. The survey included questions about membership in AMA, SDMA, and other medical organizations.

Results: In total, 438 individuals consented and participated. Overall, 296 (67.6%) indicated membership in a medical association in medical school; 101 only AMA, 38 only SDSMA, 133 both, and 24 only other. For the 234 with a student AMA membership, 71 (30.3%) continued. Of the 171 with a student SDSMA membership, 88 (51.5%) continued. Comparing those with and without a student AMA membership, 21.0% without joined after graduation and 30.3% with continued ($p = 0.07$). For SDSMA memberships, 28.5% without joined after graduation, while 51.5% of those with continued ($p = <0.01$). Common reasons for maintaining included professional advocacy ($n = 44$) and educational opportunities ($n = 32$), for AMA and professional advocacy ($n = 58$) and networking ($n = 45$), for SDSMA.

Conclusion: Those who were members of SDSMA as students were more likely to maintain their membership. This difference was less pronounced for AMA. Networking was a common reason for maintaining SDSMA.

Introduction

According to the Liaison Committee of Medical Education and the Accreditation Council for Graduate Medical Education, professionalism, continued education, and community engagement are among the requirements for medical schools and residency programs to fulfill.[1,2] The American Medical Association's Principles of Medical Ethics also consider these values important for practicing physicians to uphold.[3] One way to foster these values as medical students, residents, and practicing physicians is by participating in medical associations.

This study focuses on two medical associations, the South Dakota State Medical Association (SDSMA) and the American Medical Association (AMA). Both organizations offer students, residents, and active and retired physician memberships. Both groups value advocacy, networking, and offer leadership and educational opportunities.[4,5] Medical students who attend the University of South Dakota Sanford School of Medicine receive donated memberships to SDSMA and AMA from SDSMA physicians. However, it is unclear if being a student member leads to continued membership as a physician. The study's primary goal is to determine if student membership increases membership in the SDSMA and AMA after graduating from medical school.

Methods

This project was a survey-based study of South Dakota licensed physicians. The project received University of South Dakota (USD) IRB approval, and the USD IRB also approved consent for the study.

An 18-question survey was created on Google Forms, consisting of questions about SDSMA, AMA, and other medical association membership status during and after medical school. A pilot survey was sent out to 10 physicians. The finalized survey was sent to SDBMOE, who then sent the survey to every South Dakota licensed physician via email. The survey was open from July 20, 2023, to August 31, 2023, and a reminder email was sent at the beginning of August. SAS software was used for data analysis. Descriptive data were assessed using counts and percentages.

Comparisons were made based on Chi-square tests and odds ratios.

Results

In total, 438 physicians consented and completed the survey, and 296 (67.6%) physicians agreed to have student memberships; 101 only AMA, 38 only SDSMA, 133 both AMA and SDSMA, and 24 other medical associations. Of the 234 physicians with AMA student memberships, 71 (30.3%) continued their membership after graduation. Of the 62 physicians who stated they did not have an AMA student membership, 13 (21.0%) joined after graduation. Comparing physicians with and without student AMA memberships, physicians with a student membership were slightly more likely to have an AMA membership (p = 0.07) (Table 1).

Of the 171 physicians who indicated having a student SDSMA membership, 88 (51.5%) physicians continued their membership after graduation. Hundred and twenty-five physicians stated they did not have a student SDSMA membership; 31 (24.8%) of these physicians joined after graduation. When comparing the two groups, physicians who had a student SDSMA membership were more likely to have a physician membership (p =< 0.01) (Table 1).

The physicians who indicated having student AMA membership were asked the reason for continuing their membership, the two most common responses were professional advocacy (n=44) and educational opportunities (n=32) (Table 2). For the physicians with student SDSMA memberships, the two most common responses for continuing their memberships were for professional advocacy (n=58) and networking (n=45) (Table 3).

Forty physicians responded as physicians who had student AMA memberships and did not continue their membership as physicians indicated the two most common reasons were for "AMA not representing my specialty well" (n=29) and being a member of their specialty medical group (n=24) with

Table 1. Summary of AMA and SDSMA membership status before and after graduation.

	Total number of memberships	Membership after graduation	No membership after graduation	Percentage of continuation or joining after graduation	p Value
Physicians with AMA student memberships	234	71	163	30.3%	p = 0.07
Physicians without AMA Student Memberships	62	13	49	21.0%	
Physicians with SDSMA student memberships	171	88	83	51.5%	p = <0.01
Physicians without SDSMA student memberships	125	31	94	24.8%	

Table 2. Summary of reasons for the continuation of AMA memberships after graduation.

Continued membership after graduation	Number of respondents
AMA	71
Reasons to continue AMA membership	**Number of respondents**
Total physicians responded	67
Patient advocacy	27
Professional advocacy	44
Networking	19
Educational opportunities	32
Wellness benefits	1
Financial benefits	1
SDSMA insurance	3
Medical student membership demonstrated value	2
Other	14

Table 3. Summary of reasons for the continuation of SDSMA memberships after graduation.

Continued membership after graduation	Number of respondents
SDSMA	88
Reasons to continue SDSMA membership	**Number of respondents**
Total physicians responded	86
Patient advocacy	40
Professional advocacy	58
Networking	45
Educational opportunities	36
Wellness benefits	4
Financial benefits	1
SDSMA insurance	0
Medical student membership demonstrated value	7
Other	15

being able to choose multiple responses (Table 4). Seventeen physicians responded as physicians who did not have student AMA memberships and did not join after graduation. The two most common reasons for not joining were being a member of their specialty group (n=13) and "AMA not representing my specialty well" (n=7), with the ability to choose multiple responses (Table 5).

There were 7 physicians who responded as physicians who had student SDSMA memberships and did not continue their membership as physicians indicated the two most common reasons were political beliefs (n=4) and "SDSMA does not represent my specialty well" (n=4) (Table 6). There were 17 physicians who responded as physicians who did not have student SDSMA memberships and did not join after graduation. The three most common reasons for not joining were being a member of their specialty group (n=11), "SDSMA does not represent my specialty well" (n=4), and cost (n=4) (Table 7).

Discussion

This study aimed to determine if having a student membership in a medical association increased the likelihood of a membership as a practicing physician among South Dakota licensed physicians. When comparing physicians with and without student AMA memberships, there was no significant

Table 4. Summary of reasons for physicians who held AMA student memberships for discontinuing AMA membership after graduation.

Student members but did not continue after graduation	Number of respondents
AMA	163
Reasons for discontinuing membership	**Number of respondents**
Total physicians responded	40
Part of a specialty medical group	23
Part of a large multi-specialty group	1
Political	17
does not represent my specialty well	29
Cost	8
Other	1

Table 5.Summary of reasons for physicians who held SDSMA student memberships for discontinuing SDSMA membership after graduation.

Continued membership after graduation	Number of respondents
SDSMA	88
Reasons to continue SDSMA membership	**Number of respondents**
Total physicians responded	86
Patient advocacy	40
Professional advocacy	58
Networking	45
Educational opportunities	36
Wellness benefits	4
Financial benefits	1
SDSMA insurance	0
Medical student membership demonstrated value	7
Other	15

Table 6. Summary of reasons for physicians who never joined AMA.

Physicians without student memberships and did not join after graduation	Number of respondents
AMA	49
Reasons for not joining AMA	**Number of respondents**
Total physicians responded	17
Part of a specialty medical group	13
Political	5
Does not represent my specialty well	7
Cost	3
Other	1

Table 7. Summary of reasons for physicians who never joined SDSMA.

Physicians without student memberships and did not join after graduation	Number of respondents
SDSMA	94
Reasons for not joining AMA	**Number of respondents**
Total physicians responded	17
Part of a specialty medical group	11
Political	1
Does not represent my specialty well	4
Cost	4
Other	0

difference in having an AMA membership as a practicing physician. The most common reasons for physicians who continued their AMA membership after medical school were professional advocacy and educational opportunities. For physicians who held a student AMA membership and discontinued it after graduation, the most common responses were due to being part of a specialty medical organization and believing their specialty was not well-represented in the AMA. Being part of a specialty medical organization and believing their specialty is not well-represented in the AMA were the two main responses for physicians who did not have a student and did not join as a physician.

When comparing physicians with and without student SDSMA membership, there was a significant difference in having an SDSMA membership as a practicing physician. This finding shows that students with SDSMA memberships are more likely to continue their membership as physicians and could assist in continuing to provide SDSMA memberships to USD SSOM students. For physicians who continued their SDSMA membership after graduation, professional advocacy and networking were the two most common reasons. The most common reasons for physicians who held a student SDSMA membership and discontinued it after graduation were political beliefs, believing SDSMA does not represent their specialty, and being part of a specialty medical organization. Being part of a specialty medical group, cost, and believing SDSMA does not represent their specialty were the most common reasons physicians did not have a student membership and did not join after graduation. SDSMA highlighting professional advocacy and networking, could promote membership retention.

Conclusion

Those who were members of SDSMA as students were more likely to maintain their membership as physicians. Networking and professional advocacy were common reasons for maintaining membership in SDSMA. This difference was less pronounced for AMA memberships. For continuing AMA memberships, professional advocacy and educational opportunities had the greatest number of respondents. Focusing on professional advocacy, networking, and educational opportunities may be beneficial for retaining members for these organizations.

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Rickets in the New Age of Food Allergies: A Case Report and Comprehensive Review

Ryan Peldo, MS II; Henry Mullaney, MS II; Emily Wilde, MS III; Cole Tessendorf, MD; Drew Erie, MD; Jared Daniel, MD; and Robert E. Van Demark Jr., MD

Abstract

Nutritional rickets, once a historical disease, has reemerged in select pediatric populations despite modern food fortification efforts. While vitamin D deficiency remains the leading cause, dietary restrictions due to food allergies present a unique and underrecognized risk factor. This case report describes a 2-year-old male with multiple severe food allergies who developed vitamin D-deficient rickets due to a highly restrictive diet. His presentation, laboratory findings, radiographic abnormalities, and clinical management are discussed. Additionally, we provide a comprehensive review of the evolving epidemiology of rickets, its resurgence in high-risk populations, and the association between food allergies and nutrient deficiencies. Given the increasing prevalence of pediatric food allergies, this report underscores the need for proactive screening, early intervention, and interdisciplinary collaboration to prevent nutritional deficiencies in at-risk children.

Introduction

Rickets is a disorder of defective bone mineralization that results in skeletal deformities, growth restriction, and increased fracture risk. Historically, it was prevalent due to widespread vitamin D deficiency, but declined significantly following the implementation of food fortification programs in the mid-20th century. However, despite these public health advancements, rickets persists in specific populations, including exclusively breastfed infants, children with malabsorptive conditions, and those with restricted diets.[1] In recent years, the rising prevalence of pediatric food allergies has introduced a new at-risk group for vitamin D deficiency and rickets.[2,3]

Many common food allergens, like dairy, eggs, and fish, are primary dietary vitamin D sources. Children with multiple food allergies are at heightened risk for micronutrient deficiencies, particularly if their diets are not adequately supplemented.[4,5] Several case reports describe rickets in children with severe dietary restrictions due to food allergies, illustrating the challenges of maintaining sufficient vitamin D intake under such constraints. This case report presents a child with multiple food allergies who developed nutritional rickets, highlighting the importance of increased awareness and tailored nutritional interventions for at-risk pediatric populations.

Case Presentation

A 2-year-old Hispanic male was referred to a pediatric orthopedic surgeon by his primary care physician due to concerns about progressive bilateral bowing of his legs. His mother first noticed the deformity six months prior and reported gradual progression. Despite this, the child remained active and did not exhibit any signs of extremity pain, tripping, or difficulty ambulating, and there were no concerns regarding balance or coordination. There was no known family history of metabolic bone disease, early childhood fractures, or skeletal dysplasia.

The child was born at full term via an uncomplicated vaginal delivery, with a birth weight and length appropriate for gestational age. He had met all early developmental milestones on time, sitting unsupported at six months of age and walking independently by ten months. His overall growth trajectory had remained within normal limits, and he had attended all well-child visits per the recommended pediatric schedule. However, his mother reported recent picky eating habits and difficulty incorporating new foods into his diet due to frequent allergic reactions.

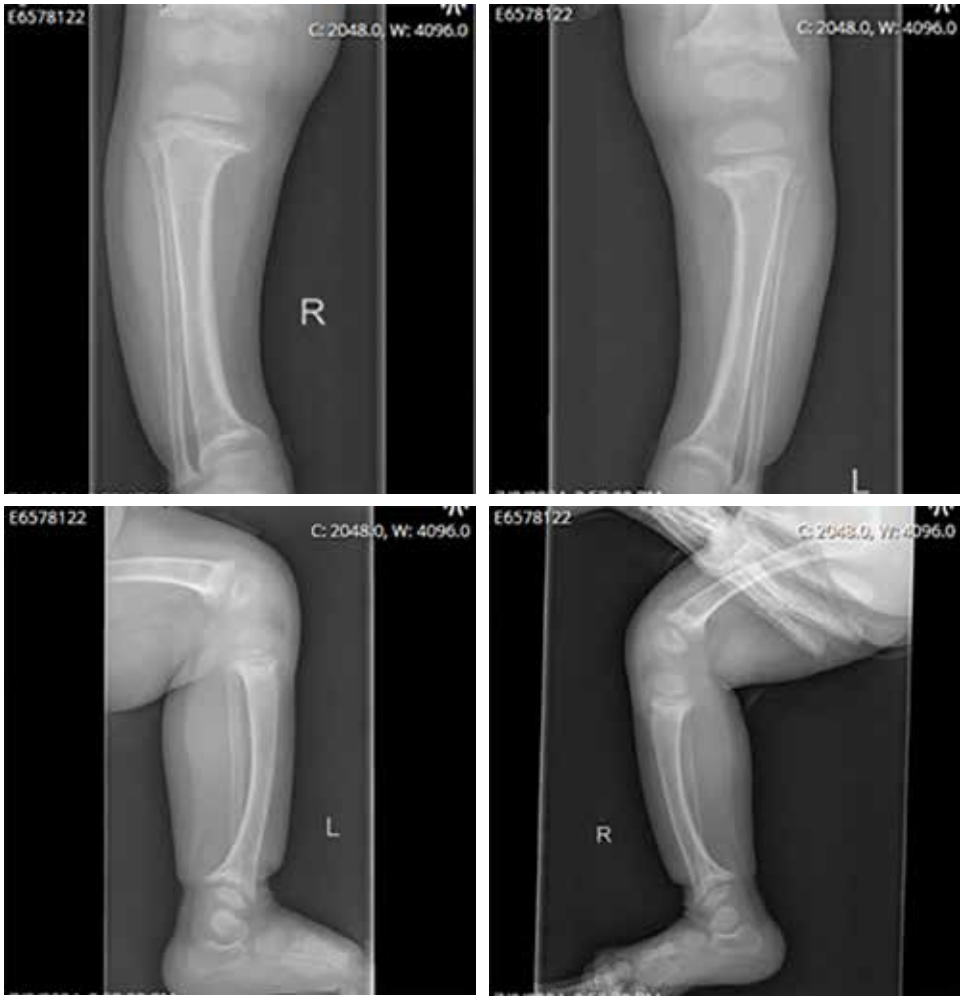
Medical history was notable for multiple severe food allergies, including chicken, shellfish-derived products, wheat husk fiber, milk, eggs, and tree nuts. These allergens had triggered reactions ranging from nausea and vomiting to facial edema,

urticaria, and anaphylaxis. As a result, he had been under the care of both an allergist and a nutritionist for the past six months. He had been prescribed an epinephrine auto-injector and mother was advised to avoid all known allergens. Attempts to introduce vitamins and other dietary supplements also resulted in allergic reactions, further limiting his nutritional intake. Consequently, his diet consisted primarily of breast milk and a limited selection of tolerated foods, without supplementation.

On physical examination, he appeared well-nourished but exhibited significant bilateral bowing of the lower extremities, which measured approximately ten degrees on each side. Despite the genu varum, he demonstrated a full, painless range of motion in the knees, with flexion measuring from zero to 130 degrees. The ankles also exhibited full, painless motion. The feet were properly aligned, with the heels well-seated within the heel pads and positioned along the midline. Hip examination revealed smooth and unrestricted abduction, with flexion reaching 60 degrees. There was no evidence of scoliosis, kyphosis, or other spinal structural deformities. Sensory function was intact bilaterally, and peripheral pulses were strong and symmetrical.

Radiographic evaluation of the lower extremities revealed characteristic findings consistent with nutritional rickets. There was irregular cupping, fraying, and flaring of the metaphyses at the distal femur, tibia, and fibula bilaterally. A mild anterolateral bowing of the tibia was also noted, though no discrete fractures were identified. Despite these skeletal abnormalities, the knee and ankle joints appeared well-maintained, with no evidence of subluxation, dislocation,

Figure 1. X-rays taken at initial presentation show irregular cupping, fraying, and flaring of the metaphyses at the distal femur, tibia, and fibula bilaterally. A mild anterolateral bowing of the tibia is present without fractures. The knee and ankle joints are well-maintained, with no evidence of subluxation, dislocation, or other joint pathology.



or other joint pathology.

Laboratory testing confirmed a biochemical profile consistent with severe vitamin D deficiency and impaired bone mineralization. The patient's serum calcium level was measured at 9.0 mg/dL (normal: 8.5–10.5 mg/dL) and serum phosphorus level was 2.9 mg/dL (normal: 3.0–6.0 mg/dL). Alkaline phosphatase level was markedly elevated at 1060 U/L (normal: 150–420 U/L for children 2 years and younger), and serum 25-hydroxyvitamin D level was critically low at 4 ng/mL (normal: 20–50 ng/mL).

Given the clinical presentation, radiographic findings, and laboratory abnormalities, the patient was diagnosed with nutritional rickets secondary to vitamin D deficiency, due to his restricted diet and multiple food allergies. Treatment was initiated with high-dose vitamin D supplementation at a dose of 6000 IU of cholecalciferol daily to replenish depleted stores. Additionally, dietary counseling and follow-up was arranged with both pediatric endocrinology and a metabolic specialist to ensure appropriate long-term management. The patient was also referred to an allergist for reassessment of his dietary restrictions and to explore potential safe supplementation strategies.

Long-term management will focus on continued vitamin D supplementation, dietary modifications, and close monitoring of skeletal development. Given the degree of bowing, careful orthopedic follow-up is ongoing to evaluate for spontaneous correction or progression of the deformity.

Discussion

Pediatric Vitamin Deficiency Disorders

Child growth and development are influenced by numerous factors, including adequate nutritional intake. Among essential micronutrients, vitamin D plays a critical role in bone mineralization and skeletal development. Vitamin D deficiency, as highlighted in this case, is a well-established cause of rickets. Current guidelines recommend vitamin D supplementation for all children and adolescents aged 1 to 18 years to prevent rickets, irrespective of individual risk factors.[6]

Beyond vitamin D, other vitamins contribute to pediatric health. Socioeconomic status and access to health education affect dietary choices and, consequently, micronutrient intake.[7] Even in resource-rich countries, dietary diversity challenges the ability to meet recommended daily vitamin intakes.[7] While vitamin supplementation is advised in cases of nutritional deficiencies or malabsorption syndromes,[7] further studies are necessary to optimize supplementation protocols.

Vitamin E, an antioxidant, has shown benefits in adult populations; however, limited evidence supports its supplementation in children.[7,8] Vitamin A, another fat-soluble vitamin, plays a crucial role in maintaining vision, supporting cellular growth, and enhancing immune function.[7,9] Unlike vitamin E, vitamin A supplementation is recommended for growing children due to its fundamental role in development.[7]

Vitamin D deficiency remains prevalent, particularly in individuals with malnutrition, malabsorption syndromes, or chronic conditions such as liver and renal disease.[7] Additional risk factors include limited sun exposure, obesity, and hyperparathyroidism, all of which can exacerbate deficiency.[7] Routine supplementation is indicated in high-risk populations, especially during the first year of life, to prevent rickets and osteopenia.[1,10]

Current Epidemiology of Rickets

Over the past century, public health initiatives, particularly food fortification, have led to a substantial decline in rickets prevalence. In the U.S., the FDA mandates vitamin D fortification in cow's milk, with additional fortification in orange juice and cereals.[11] Similar policies in Canada and Europe have also reduced rickets incidence. However, in low-income countries where standardized fortification programs are lacking, rickets remains a significant public health concern.[1]

Despite these public health efforts, rickets continues to emerge in select populations, particularly among children with food allergies and restrictive diets. Several case reports describe vitamin D deficient rickets in such children. A 16-month-old Hispanic male in northern California, for example, presented with decreased appetite, activity level, an extremely low 25-hydroxyvitamin D level of 3.5 ng/mL, elevated alkaline phosphatase, and radiographic evidence of rickets. His diet was limited due to a cow's milk allergy.[4] Another case report described a 14-month-old male of Moroccan descent living in London, who, due to parental concerns about food allergies, had been excluded from consuming both milk and eggs. He presented with biochemical and radiographic findings consistent with rickets.[2] Additionally, a 2-year-old Inuit male from northern Quebec with a severe milk allergy developed rickets with significant leg bowing and metaphyseal flaring. His condition improved with aggressive vitamin D and calcium supplementation.[5] These

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cases highlight the increasing need for heightened awareness among healthcare providers regarding the risk of nutritional rickets in children with food allergies, particularly those who avoid dairy and other fortified foods.

Diagnostic Approach to Rickets

A comprehensive approach to diagnosing rickets includes a detailed medical history, physical examination, biochemical assessment, and radiographic confirmation. Key historical considerations include vitamin D supplementation history, dietary calcium intake, and risk factors for malabsorption.[12] Clinically, rickets often presents with failure to thrive, a protuberant abdomen, dilated cardiomyopathy, and an increased risk of fractures.[13]

Certain anatomical regions exhibit characteristic findings in rickets. The cranial skeleton may display craniotabes, frontal bossing, delayed tooth eruption, and dental caries.[13] Thoracic manifestations include rachitic rosary, tachypnea, and frequent respiratory infections. Spinal abnormalities such as scoliosis, lordosis, or kyphosis may also be observed.[12,13] The key skeletal findings, however, occur in the appendicular skeleton, including bilateral enlargement of the wrists and ankles and deformities such as genu varum, genu valgum, or windswept deformity.[13]

Laboratory markers provide additional diagnostic support. Typical findings include elevated alkaline phosphatase, decreased serum phosphate, and variable calcium levels.[13] A defining feature of vitamin D-deficient rickets is a low serum 25-hydroxyvitamin D level, reflecting depleted vitamin D stores.[13] Radiographs confirm the diagnosis, revealing increased growth plate width, loss of provisional calcification, and characteristic cupping, splaying, and metaphyseal flaring.[14]

Conclusion

This case highlights the intersection of pediatric metabolic bone disease and food allergies, emphasizing the need for heightened awareness of nutritional deficiencies in children with restricted diets. Despite public health advances, vitamin D-deficient rickets continues to emerge in select populations, underscoring the necessity for early diagnosis, appropriate supplementation, and interdisciplinary management. Future research should further explore optimal strategies for preventing vitamin D deficiency in high-risk children, particularly those with multiple food allergies.

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Beyond the CHA2DS2VASc: Hypercoagulable States in Anticoagulated AF Patients

Michael Roberts, MS IV; Dawood Shehzad, MD; Tate Blankespoor, MS IV; Joe Reynen, MS IV; Mahmoud Alsaiqali, MD; Tehreem Fatima, MD; and Dawlat Khan, MD

Abstract

Despite the proven efficacy of direct oral anticoagulants (DOACs) in preventing thrombus formation in atrial fibrillation (AF), thrombus formation may rarely occur even with appropriate anticoagulation. We report the case of a 70-year-old woman with a history of AF, initially on warfarin, who underwent percutaneous left atrial appendage closure with a Watchman FLX 27 mm device due to recurrent gastrointestinal bleeding. She subsequently developed multiple unprovoked deep venous thromboses, necessitating the reinitiation of rivaroxaban therapy. She presented to the hospital with complaints of lower abdominal pain. Although the CT abdomen and pelvis was not a dedicated cardiac study, the superior slices incidentally captured the inferior and posterior left atrium, revealing a lobulated soft-tissue density suspicious for thrombus. Labs included INR 1.8, APTT of 40, and D-dimer of 0.62. Transthoracic echocardiography could not visualize thrombus. Transesophageal echocardiography revealed a large mobile thrombus in the superior cavity of LA. The watchman device was well seated with no flow noted across the device. A hypercoagulability workup showed low levels of Protein S antigen at 21. She was placed on warfarin. Follow-up TEE in 3 months showed a decrease in the thrombus size. This case highlights the importance of evaluating an underlying hypercoagulable state when thrombus develops despite DOAC therapy. While DOACs prevent thrombus formation in atrial fibrillation, their failure should not be dismissed as incidental. Clinicians should pursue a hypercoagulability workup, especially when thromboembolic events appear disproportionate to the CHA_2DS_2-VASc score. Early identification of inherited or acquired thrombophilias can guide tailored and effective long-term management.

Introduction

Protein S is an important protein that plays a role in the downregulation of blood clot formation.[1] As a result, a rare deficiency of protein S has been linked to recurrent venous thromboembolism and the formation of blood clots.[1] Protein S deficiency is both acquired and hereditary in nature.[2] Atrial fibrillation (AF) has been associated with the formation of thrombus in the left atrial appendage of the heart.[3] Thrombus formation has been described to be the result of venous stasis due to contractile abnormalities of the heart during atrial fibrillation.[3] Strategies to prevent thrombus formation in AF include anticoagulation and devices such as Watchman and Amplatzer Amulet Occluder.[3] We present a case of a 76-year-old female with a history of AF who developed a thrombus of the left atrium despite chronic anticoagulation and Watchman placement due to a protein S deficiency.

Case Presentation

A 76-year-old female with a past medical history of hypertension, hyperlipidemia, AF, rheumatoid arthritis, and deep vein thrombosis (DVT) presented to the emergency department (ED) for persistent abdominal pain over 4 weeks. She described the pain as diffuse, worsened in her upper abdominal region, and accompanied by nausea. She was initially seen at an acute care facility and diagnosed with a urinary tract infection and was prescribed a 5-day course of cephalexin. She had also been seen by her primary care provider and prescribed an H2 blocker, a proton pump inhibitor, and sucralfate for presumed gastroesophageal reflux disease. However, due to worsening pain, she sought an evaluation in the ED. Her vitals in the ER were a temperature of 98.4°F, pulse rate of 84, respiratory rate of 18, blood pressure of 160/91 mmHg, and oxygen saturation of 94% on room air. Physical exam was negative except for generalized abdominal tenderness on palpation. She denied having chest pain, shortness of breath, palpitations, or dizziness.

Lab tests, including a complete blood count, comprehensive

metabolic panel, and urinalysis, were unremarkable. Initial differential diagnoses included pancreatitis, enteritis, and diverticulitis. A computed tomography scan of the abdomen and pelvis (CT AP) with contrast did not show any acute intrabdominal pathology. Although the CT AP was not performed as a dedicated cardiac study, the superior extent of the scan included portions of the lower thorax, incidentally capturing the inferior and posterior aspects of the left atrium. Within this region, a lobulated soft-tissue density was visualized, raising concern for an intracardiac thrombus. Given the limited visualization and lack of cardiac gating, a transthoracic echocardiogram (TTE) was obtained.

TTE showed an ejection fraction of 60-65%, a normal-sized IVC, mild aortic insufficiency, mild aortic stenosis, and mild mitral regurgitation. Her left atrium was dilated; however, the suspected thrombus was still not clearly seen. A transesophageal echocardiogram (TEE) was done, which revealed a moderately to severely dilated left atrium and a large mobile thrombus in the superior cavity of the left atrium. (Figures 1, 2).

Due to suspected failure of anticoagulation, the patient was started on heparin 10 units/kg/hr and hematology was consulted. She was found to have a baseline PTT of 40, hemoglobin of 15.1, platelet count of 232,000, and heparin antiXa > 1.1. An EKG showed atrial fibrillation with a normal rate, and she was continued on her home oral diltiazem. Hematology and Oncology was consulted and discovered a lengthy history of thrombosis in the patient. She was previously on warfarin for about 20 years until she had gastrointestinal bleeding. Following this, she was given a Watchman device (3 years ago) and started on aspirin 81 mg. However, in the next two years, she experienced left upper extremity superficial venous thrombosis and a right lower extremity DVT after a hip replacement. Thus, she was started on rivaroxaban for 6 months. About 2 months after completion of therapy, she later experienced another unprovoked DVT of the right lower extremity and was started on rivaroxaban indefinitely.

Testing was negative for lupus anticoagulant, anticardiolipin antibodies, and β2-glycoprotein antibodies, making antiphospholipid syndrome unlikely. Factor V Leiden mutation, prothrombin gene mutation (G20210A), and antithrombin III deficiency were also negative. The only abnormality identified was low protein S antigen at 21% (reference range: 80–160%), consistent with protein S deficiency, which likely contributed to the patient's recurrent thromboses. Due to the failure of rivaroxaban, the use of warfarin was considered.

Due to her extensive history of thrombus formation, cardiology recommended placing her on systemic anticoagulation with warfarin (starting with 42.5 mg per week with an INR goal of 2.5-3.0) instead of utilizing invasive surgery to remove the clot. Follow-up TEE was performed 3 months later, which showed a decrease in the size of the thrombus. Her warfarin was also titrated to 47.5 mg per week to achieve her INR goal.

Discussion

This case underscores the importance of considering an underlying hypercoagulable state in patients who develop thrombus despite appropriate anticoagulation. While DOACs effectively prevent thrombus formation in atrial fibrillation, their failure should not be dismissed as incidental. Clinicians must reassess anticoagulation strategies and pursue a hypercoagulability workup, especially when thromboembolic

Figure 1. Transesophageal echocardiogram showcasing the left atrial thrombus.

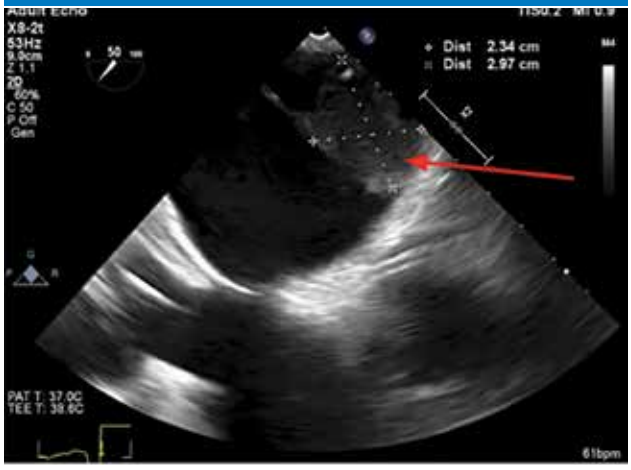
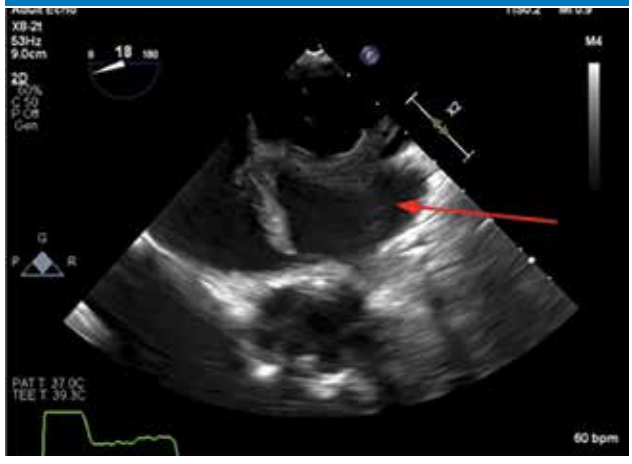


Figure 2. Another view of the left atrial thrombus as seen on transesophageal echocardiogram.



events appear disproportionate to the patient's CHA_2DS_2-VASc score. The recommendations for evaluating hypercoagulability in patients with unexplained thromboembolism are presented in Table 1. Early identification of inherited or acquired thrombophilias can guide more tailored and effective long-term management.

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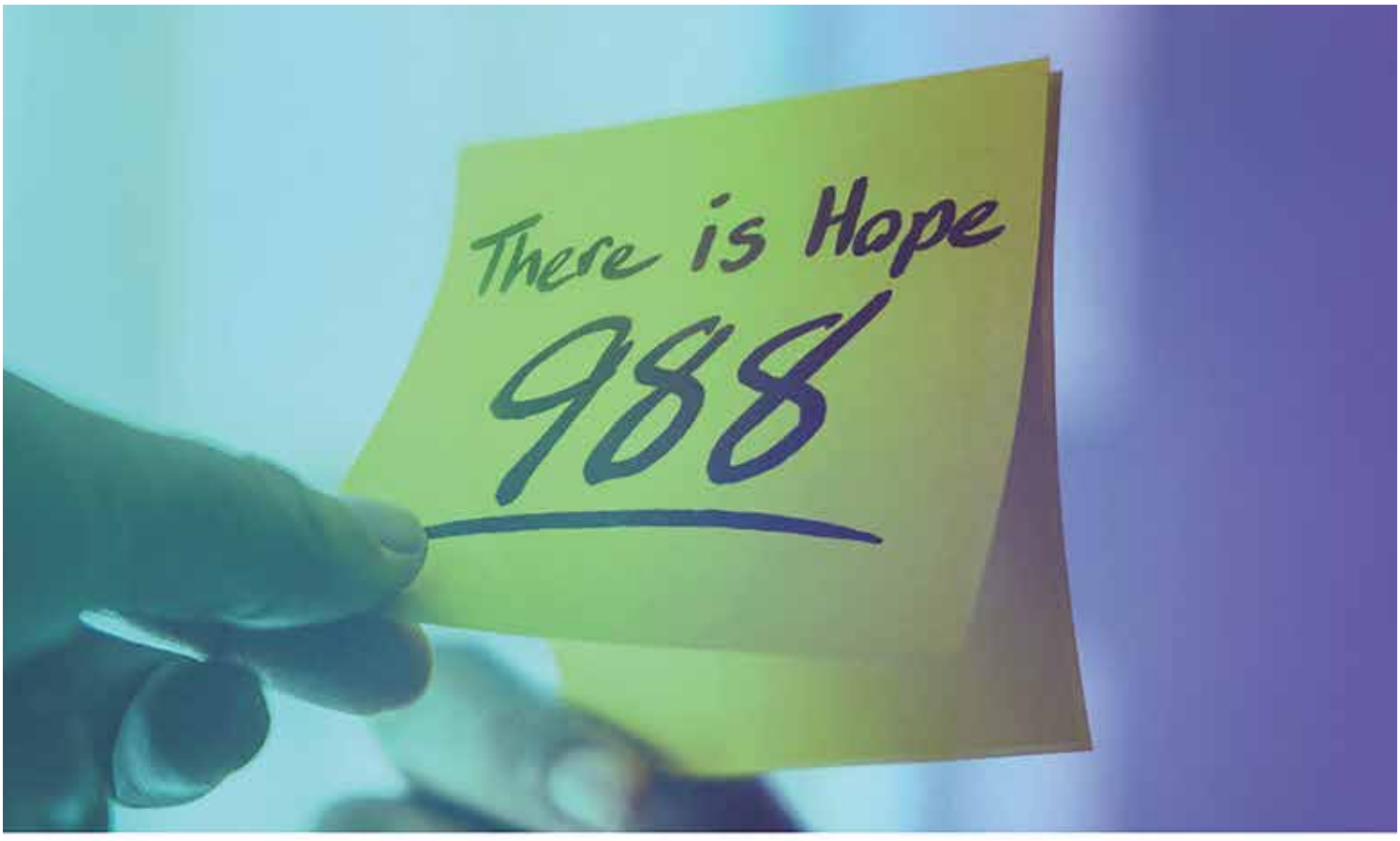
Table 1. Evaluation of hypercoagulability in patients with unexplained arterial thromboembolism.

Category	Conditions & key examples	When to test/comments
Systemic conditions	**Cancer:** Especially adenocarcinomas and hematologic malignancies. **Autoimmune or auto-inflammatory disorders:** SLE, rheumatoid arthritis, vasculitis, Behcet's disease. **Infections:** COVID-19, HIV, endocarditis, chronic inflammatory infections. **Substance use:** Anabolic steroids, cocaine, amphetamines. **Medications:** Hormone therapy (OCPs, HRT), erythropoietin, thalidomide.	Restrict testing to conditions where identifying a hypercoagulable state alters management (e.g., stopping an offending drug, initiating cancer-directed therapy). These are often secondary and potentially reversible causes.
Acquired hypercoagulable disorders	**Antiphospholipid syndrome (APS):** aCL IgG/IgM, aβ2GPI IgG/IgM, lupus anticoagulant. **Myeloproliferative neoplasms (MPN):** CBC, JAK2 V617F mutation testing. **Heparin-induced thrombocytopenia (HIT):** HIT EIA, serotonin release assay (SRA). **Thrombotic microangiopathies (TMA):** LDH, haptoglobin, peripheral smear for schistocytes. **Paroxysmal nocturnal hemoglobinuria (PNH):** Flow cytometry for CD55/CD59 deficiency.	Perform in younger patients or those with refractory/recurrent arterial thrombosis despite therapy. APS and MPNs are among the most common acquired causes. Diagnosing these entities has direct therapeutic implications (e.g., anticoagulation duration, cytoreductive therapy).
Inherited thrombophilia	**Genetic mutations:** Factor V Leiden (F5 G1691A), Prothrombin G20210A. **Protein deficiencies:** Protein C, Protein S, Antithrombin (AT). **Less common:** Dysfibrinogenemia, elevated factor VIII, MTHFR polymorphisms (limited clinical utility).	Consider testing in young patients (<50 years) or those with a strong family history of venous or arterial thrombosis. High-risk thrombophilias (AT, PC, or PS deficiency; compound heterozygosity) may warrant lifelong anticoagulation. Avoid testing during acute thrombosis or anticoagulation therapy, as levels may be transiently abnormal.

Abbreviations: aβ2GPI = anti–β2 glycoprotein I; aCL = anticardiolipin antibody; aPL = antiphospholipid; AT = antithrombin; CBC = complete blood count; EIA = enzyme immunoassay; HIT = heparin-induced thrombocytopenia; JAK2 = Janus kinase 2; LDH = lactate dehydrogenase; MPN = myeloproliferative neoplasm; OCP = oral contraceptive pill; PC = protein C; PNH = paroxysmal nocturnal hemoglobinuria; PS = protein S; SLE = systemic lupus erythematosus; SRA = serotonin release assay; TMA = thrombotic microangiopathy.



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A Case of Q fever: What Does the South Dakota Clinician Need to Know?

Angela Roberts, MD; Michael Roberts, MS IV; Ibrahim Ahmed, MD; and Randall Lamfers, MD

Abstract

Q fever is a reportable, zoonotic infection caused by *Coxiella burnetii*. It can present as either an acute or chronic infection. Acute Q fever is frequently asymptomatic or presents with nonspecific symptoms, making the diagnosis challenging. In the chronic state it will manifest as localized infection such as endocarditis, which is the most common. South Dakota has the highest incidence of Q fever in the U.S. with 12.4 cases per million people. We present a case of a patient in their 50s with mild hepatitis and persistent fevers and eventually was diagnosed with Q fever. The patient had no known exposures. The Q fever was diagnosed using Karius testing, liver biopsy, and serology. The patient was treated with doxycycline and improved. This case highlights diagnostic challenges and management considerations for South Dakota clinicians.

Introduction

Coxiella burnetii is an obligate intracellular bacterium that is the causative agent of Q fever.[1] This ubiquitous bacterium affects a variety of mammals, which often act as a reservoir for infection in humans.[2] Common modes of transmission include direct contact or inhalation of contaminated aerosols, leaving veterinarians and farmers with an increased risk of exposure.[3] Thus, described risk factors include living within 10 miles of farms with livestock, contact with livestock and use of unpasteurized dairy products.[4] Despite this, CDC data has shown that many patients do not report exposure to livestock or high risk occupations.[4] Q fever can manifest as either an acute or chronic infection following an incubation period of 2-3 weeks.[3] Acute Q fever often presents with nonspecific symptoms including fever, headaches, myalgia, fatigue, nausea, vomiting, and diarrhea.[5] Chronic Q fever occurs in 5% or less of cases and can manifest as a persistent localized infection such as endocarditis, vascular infection, or hepatitis.[3] Asymptomatic disease occurs in 60% of cases.[3] Regionally, South Dakota has reported about 4-12 cases per year from 2014-2023.[6] In this report, we discuss a patient with no clear exposure risks who was found to have Q fever.

Case Presentation

The patient was a male in his 50s with an unremarkable past medical history who was admitted with 10 days of persistent fevers, body aches, headaches, and anorexia. He denied cough, congestion, sore throat, abdominal pain, nausea, vomiting, diarrhea, or rashes. He was seen about a week prior for fever and hyponatremia that was managed outpatient. He works an office job and denies recent travel, known ill contacts, illicit drug history, and contact with animals.

In the ED, vitals included a blood pressure of 143/98 mmHg, pulse rate of 97 bpm, temperature of 36.7°C, and SpO of 97%. The physical exam was unremarkable. A broad workup was initiated. complete blood count, urinalysis, lactic acid, and thyroid stimulating hormone were normal. Comprehensive metabolic panel was notable for a mild hyponatremia (132), and elevated liver function tests (alkaline phosphatase 332 U/L, alanine aminotransferase 143 U/L, and aspartate aminotransferase was 85 U/L). C-reactive protein was 49.7 mg/L and procalcitonin was 0.57 ng/ml. Computed tomography (CT) angiogram chest and CT abdomen and pelvis with contrast were unremarkable. On right upper quadrant ultrasound, examination of the gallbladder showed the presence of pericholecystic fluid and thickening of the gallbladder wall. Gallstones were not seen.

In the hospital, the patient experienced cyclical fevers ranging from 36.3°C to 39.3°C, antibiotics were held, and Infectious Disease and Hepatology were consulted. Infectious and non-infectious etiologies were considered. Initial return of testing had positive IgM and IgG serology for Epstein Barr virus (EBV) and parvovirus B19 however polymerase chain reaction (PCR) testing for both was unremarkable. The other positive testing was slightly elevated anti-smooth muscle antibody, anti-mitochondrial antibodies, and antistreptolysin O testing. The decision was made to proceed with positron

emission tomography CT and there was abnormal uptake in the liver. Hepatology recommended liver biopsy. Cell-free DNA (Karius) testing returned positive for *Coxiella burnetii* at 8021 molecules per microliter on the day liver biopsy was completed. Liver pathology showed fibrin ring granulomas.

The patient was diagnosed with acute Q fever hepatitis and started on doxycycline 100 mg twice daily for 2 weeks. He was improving and discharged home. The *Coxiella burnetii* serology returned positive (phase II IgM >1:2048 and IgG 1:16) after discharge confirming the diagnosis. At his 4-week follow-up ID appointment, his fevers resolved but he reported occasional epigastric abdominal and fatigue but planned to return to work. Repeat liver function tests showed improvement. Repeat Q fever serology at follow-up Q fever phase I IgM > 1:2048, Q fever phase I IgG <1:16, Q fever phase II IgM >= 1:2048, and Q fever phase II IgG of 1:8192. Plan was to return to the clinic for repeat lab tests in 8 weeks; however, he was lost to follow-up.

Discussion

Acute Q fever often presents with nonspecific symptoms such as fever, nausea, vomiting, diarrhea, and fatigue, leaving the diagnosis mistaken for other illnesses.[2,5] High fever can appear as a flu-like illness due to a duration of more than 10 days and the presence of myalgia and debilitating headaches.[3] Common risk factors for Q fever infections include clear exposure risks, such as contact with livestock, unpasteurized dairy products, and living in rural regions or within 10 miles of farms that include livestock.[4] The lack of clarity in Q fever infections has been seen in data from CDC on Q fever infections between 2000 and 2010 where 79% of 405 patients identified did not have occupations with high risk of exposure to *Coxiella*.[4] Furthermore, 60% of those patients identified did not report contact with livestock.[4] This ambiguity is also seen in laboratory findings as 90% of patients appear to have a normal WBC count, including the presented patient.[4] However, laboratory abnormalities commonly seen, as evidenced by the patient, include elevated transaminases, anti-smooth muscle antibodies and increased alkaline phosphate.[4] An increased incidence of infection in people above the age of 40 and increased exhibition of symptomatic infection in males have also been reported.[4] Suspected cases of Q Fever should be reported to both local and state health department authorities for help with diagnostic testing and for national surveillance purposes.[4]

Diagnoses of Q fever are made with the help of tools such as serology, PCR, and Karius testing.[7] Serologic testing to detect the presence of phase I and phase II antibodies has proven popular due to its relative ease and safety.[8] Acute infections are associated with an increased predominance of phase II antibodies, while chronic infections are associated with prevalence of phase I antibodies.[4] Cut off for phase II antibodies is IgG titer of ≥ 1:128.[9] Phase II antibodies in acute infections typically are first seen between 1-2 weeks after symptoms begin and peak 8 weeks from infection.[9] PCR testing has been used to detect *Coxiella* in multiple sites such as CSF, blood, bone marrow, and tissue biopsies.[9] However, sensitivity of PCR testing has been noted to decrease after one week from symptom onset.[9] A novel approach to Q fever testing has been Karius testing, which utilizes next generation sequencing to detect cell-free DNA of microbes.[7] This testing tool can confirm the presence of *Coxiella* DNA in the setting of positive serology.[7] Q fever can also occur with co-infections with pathogens such as *Legionella*, *Borrelia burgdorferi*, and *Bartonella*.[7] However, these "co-infections" are most often false positives that are caused by cross-reactive serologies.[7] An absence of cell-free DNA of these pathogens on Karius testing can help determine this false positivity.

This patient was found to exhibit EBV and parvovirus positivity that could be due to serological cross-reactivity. In the clinical context, Karius has been valued due to its quick turnaround time. In this case, Karius resulted about 5 days earlier than serology testing. However, it should be noted that Karius testing continues to be an expensive endeavor at this time.

In adult patients with symptomatic acute Q fever infection, doxycycline 100 mg every 12 hours for 14 days is the treatment of choice.[4] Initiation of doxycycline may begin before confirmatory tests return.[4] When use of doxycycline is contraindicated, antibiotics such as trimethoprim/sulfamethoxazole, levofloxacin, or azithromycin can be considered along with ID consultation.[4] In asymptomatic cases, treatment is not recommended but can be considered in individuals felt to be high risk for chronic Q fever.[4] In cases of chronic Q fever treatment of choice is doxycycline 100 mg twice daily plus hydroxychloroquine 200 mg 3x daily with duration determined by location involved.[4]

Conclusion

With South Dakota having the highest incidence of Q fever cases in the U.S., [10] South Dakota clinicians need to have a high index of suspicion for Q fever. Acute Q fever often will present with nonspecific symptoms and lack of reported risk factors making the diagnosis challenging.[4,5] Chronic infections can present with localized infections, most commonly endocarditis.[3] Diagnostic laboratory findings

often have normal white blood cell counts and mild elevated transaminases. Diagnostic tools include serological, PCR, and Karius testing. Although serology has traditionally been the tool of choice, Karius has grown in popularity due to its quick turnaround time and ability to rule out false positive co-infections. The treatment of symptomatic acute Q fever is doxycycline.[4] Although Q fever can present as a diagnostic puzzle, a prompt and careful workup remains important in treatment of patients.

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Helicobacter Pylori Infection in the Primary Care Setting: Recognition, Diagnosis, and Updated Treatment Regimens

Kennedy Forest, MD; Melissa Pham, MS IV; Sydney Lovrien, MD; and Deepan Panneerselvam, MD

Abstract

Helicobacter pylori (H. pylori) is recognized as one of the most widespread infections globally and is associated with damaging gastric sequelae, including dyspepsia, peptic ulcers, and even malignancy. It is crucial for primary care physicians, in addition to gastroenterology specialists, to be well-informed about the typical clinical presentation, diagnostic modalities, and treatment protocols for *H. pylori* infections. The updated treatment guidelines from the American College of Gastroenterology (ACG), which address the nuances of antibiotic resistance trends, provide valuable direction for healthcare providers in managing patient infections effectively and mitigating the risk of complications related to chronic *H. pylori* infection.

Introduction

Helicobacter pylori (H. pylori) is a gram-negative, flagellated bacterium that colonizes the mucous layer of the gastric epithelium. It is responsible for one of the most prevalent chronic bacterial infections globally, affecting nearly 40% of the world's population.[1-3] An *H. pylori* infection can lead to several severe clinical outcomes, including dyspepsia, peptic ulcer disease, gastric adenocarcinoma, and mucosa-associated lymphoid tissue (MALT) lymphomas.[2,3] The combination of its wide prevalence and potential long-term health complications emphasizes why this infection poses a significant global health challenge. Given the variable and often non-specific symptoms associated with *H. pylori* infection, primary care physicians play a crucial role in its diagnosis and treatment. Moreover, the importance of conducting post-treatment test-of-cure is frequently overlooked.[2] This article aims to highlight and disseminate the updated treatment guidelines for H. pylori, while also providing a concise overview and a useful resource for primary care providers.

Discussion

Epidemiology

Despite a decline in global prevalence, *H. pylori* remains one of the most prevalent chronic bacterial infections worldwide, with significant variability in prevalence rates attributed to geographic, socioeconomic, and age-related factors.[4] In North America, the prevalence of *H. pylori* is estimated at approximately 35%; however, this rate is notably higher among immigrant populations, older adults, and individuals belonging to racial and ethnic minorities.[2] Research indicates that low socioeconomic status correlates with increased rates of *H. pylori* infection. In contrast, lower prevalence is observed in high-income countries and those with robust universal healthcare systems, though infection rates can still reach as high as 60-70% in resource-limited areas.[1] While the precise mode of transmission for *H. pylori* remains uncertain, it is hypothesized that person-to-person transmission occurs via saliva and fecal-oral routes, a theory supported by instances of intrafamilial clustering.[5,6]

Indications for Testing- Symptoms & Risk Factors

Healthcare providers should be cognizant that dyspepsia constitutes the most prevalent clinical manifestation of *H. pylori* infection, typically characterized by symptoms such as epigastric discomfort, bloating, early satiety, and nausea.[4] Nonetheless, it is important to note that *H. pylori* infection often remains asymptomatic and, in many instances, exhibits symptom overlap with other gastrointestinal pathologies. For example, heartburn, a common symptom frequently linked to gastroesophageal reflux disease (GERD), may also be present with *H. pylori* infection. In patients under the age of 60 who do not present with alarm symptoms, it is permissible for individuals exhibiting appropriate clinical indications to undergo non-invasive testing and treatment for *H. pylori* without the necessity for endoscopic evaluation. Conversely, when alarm symptoms such as unintentional weight loss, persistent vomiting, dysphagia, gastrointestinal bleeding, or iron-deficiency anemia manifest, prompt referral to a gastroenterologist is imperative to assess for potential complications, including the development of peptic ulcers

or underlying malignancy.[4] Therefore, recognizing these symptomatic presentations in conjunction with patient-specific risk factors is essential for the timely diagnosis and effective management.

In addition to *H. pylori* testing prompted by symptom development, specific risk factors warrant testing in certain populations. It is advised that adult household members of *H. pylori*-positive individuals, patients with idiopathic thrombocytopenic purpura, unexplained iron deficiency anemia, and those with a current or past history of peptic ulcer disease undergo testing for *H. pylori*. Furthermore, individuals with long-term use of NSAIDs should also be tested, as *H. pylori* infection significantly elevates the risk of ulcer formation.[2]

The identification of *H. pylori* is difficult due to its insidious nature, contributing to 80-90% of gastric cancers worldwide, and asymptomatic patients would only be identified if screened.[7] Overall, broad screening for *H. pylori* in the United States population for primary prevention of gastric adenocarcinoma is not recommended, but testing for patients classified as high-risk for gastric cancer should be considered, especially in patients under 50 years of age.[2] High-risk factors include a first-degree relative with gastric cancer, gastric adenomas, a history of gastric adenocarcinoma or resection, pre-malignant conditions (atrophic gastritis, intestinal metaplasia, dysplasia), or autoimmune gastritis.[2] Moreover, individuals from non-White racial/ethnic backgrounds and immigrants from high gastric cancer regions (e.g., Western Pacific, South and Central America, Eastern Europe) should also be tested.[2,7]

Diagnosis

The diagnosis of *H. pylori* infection is guided by clinical suspicion and subsequent non-invasive or endoscopic testing, although concern for *H. pylori* infection alone does not warrant endoscopy. Before non-serologic testing, it is essential to withhold antibiotics and bismuth for four weeks and proton pump inhibitors (PPIs) or potassium-competitive acid blockers (PCABs) for two weeks, as these can reduce test sensitivity.[2] The urea breath test (UBT) and stool antigen test are the most accurate non-invasive methods and are widely recommended.[2,8] Serologic testing for immunoglobulin G antibodies is less reliable, as it cannot differentiate between active and past infections; thus, it is generally discouraged unless other non-invasive options are unavailable.[2,8] For patients undergoing upper endoscopy for gastrointestinal symptoms or surveillance, *H. pylori* testing can involve biopsy-based methods, including rapid urease testing, histological examination, or culture from gastric biopsies.[2] However, bacterial culture is primarily used to assess antibiotic sensitivity rather than for initial diagnosis.[8] *H. pylori* testing should only be pursued if there is an intention to treat any positive result.

Treatment Recommendations

The American College of Gastroenterology (ACG) published updated guidelines for the treatment of *Helicobacter pylori* infection in October 2024. While the guidelines address salvage therapy for refractory infection, this review focuses on first-line regimens for treatment-naive patients (see Table 1).[2] The ACG strongly recommends a 14-day optimized bismuth quadruple therapy—proton pump inhibitor (PPI), bismuth subcitrate or subsalicylate, tetracycline, and metronidazole—for patients without known antibiotic susceptibility (moderate quality evidence).[2] Rifabutin triple therapy (rifabutin, omeprazole, and amoxicillin) is also recommended, though conditionally and based on low-quality evidence.[2] Additionally, two regimens incorporating potassium-competitive acid blockers (PCABs) are conditionally recommended with moderate-quality evidence: PCAB dual therapy (vonoprazan and amoxicillin) and PCAB triple therapy (vonoprazan, clarithromycin, and amoxicillin).[2]

Table 1. *Heliobacter pylori* treatment regimens for treatment-naïve patients.[2]

Regimen	Medication (dose)	Frequency
Bismuth quadruple therapy	PPI (standard dose)	Twice daily (BID)
	Bismuth Subcitrate (120-300 mg) or Subsalicylate (300 mg)	Four times daily (QID)
	Tetracycline (500 mg)	Four times daily (QID)
	Metronidazole (500 mg)	Three times daily (TID) or Four times daily (QID)
Rifabutin triple (Talicia)	Omeprazole (10 mg)	Three times daily (TID)
	Amoxicillin (250 mg)	
	Rifabutin (12.5 mg)	
PCAB dual (Voquezna DualPak)	Vonoprazan (20 mg)	Twice daily (BID)
	Amoxicillin (1,000 mg)	Three times daily (TID)
PCAB triple (Voquezna TriplePak)	Vonoprazan (20 mg)	Twice daily (BID)
	Amoxicillin (1,000 mg)	
	Clarithromycin (500 mg)	

The 2024 guidelines introduce regimens that differ significantly from previous recommendations, the clarithromycin triple therapy (PPI, amoxicillin, and clarithromycin) and levofloxacin-based treatments. The American College of Gastroenterology (ACG) now advises against these approaches unless susceptibility is demonstrated,

primarily due to the rising prevalence of antibiotic resistance, particularly to macrolides and fluoroquinolones, which has diminished the efficacy of former regimens. Furthermore, changes in treatment recommendations are informed by recent studies on novel antibiotics, such as Rifabutin, and advancements in gastric acid-suppressing medications, including proton-coupled antiacids (PCABs).[2]

Post-Treatment Test-of-Cure

Upon completion of the 14-day treatment course, a test-of-cure is necessary for all patients, irrespective of symptoms, to confirm bacterial eradication. This requirement is due to the complexity of the treatment regimen, which heightens the risk of incomplete or improper adherence, potentially resulting in persistent infection. Post-treatment testing options include urea breath tests, fecal antigen tests, or biopsy-based assessments, conducted at least four weeks after the conclusion of the treatment.

Conclusion

H. pylori infection is an insidious infection and, left untreated, leads to significant sequelae. Primary care physicians must recognize at-risk patients and associated symptoms to prevent persistent infection. Adhering to updated treatment regimens optimizes eradication efforts and ensures long-term management, thereby mitigating the risk of serious clinical outcomes.

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Life and Hope After Stroke

ADVERTISEMENT

Sioux Falls man shares his inspiring story of stroke recovery

On February 21, 2024, Randy Preston was getting ready to take his family on a short trip out of town. He was at the car wash when he noticed he couldn't use his left hand. He was confused as to what was happening, so he called his wife, who immediately recognized the signs of stroke from the way Randy was speaking.

Randy's wife Nancy and their son Preston raced to the parking lot where Randy had parked his vehicle. They got him to the hospital as quickly as possible.

When Randy arrived, doctors asked Nancy how long it had been since the onset of his symptoms, and Nancy replied that it hadn't been 25 minutes. The doctors told Nancy that because she got him to the hospital so quickly, Randy would have a "really good outcome." Nancy credits her quick actions to a card she'd gotten in the mail that shared BE FAST and the signs and symptoms of stroke.

As a hockey announcer, Randy says he thought his stroke would take away one of his favorite things in life. "I love doing PA work. Announcing hockey games… that's really where I want to be. That's therapy for me," Randy said. "When I woke up from the stroke, I felt like this could all be over."

Stroke can happen to anyone, at any age. But there is life – and hope – after stroke. As in Randy's case, survival and recovery for those who experience stroke depends on the quick actions of those around.

South Dakota's Mission: Lifeline Stroke project is working across the state of South Dakota to strengthen the full spectrum of stroke care for patients like Randy in all corners of the state. The $5.05 million project, a collaboration between the American Heart Association and The Leona M. And Harry B. Helmsley Charitable Trust, was launched in October 2024. Today, 28 acute sites and 25 post-acute sites are participating in the project.

Mission: Lifeline Stroke focuses on connecting all components of acute stroke care into a smoothly integrated system that reinforces the use of evidence-based guidelines to timely and effectively treat stroke patients. It brings together hospitals, emergency medical services and first responders, rehabilitation facilities, communications and regulatory agencies, and state and local government to forge a proactive system of stroke care that saves and improves lives.

For stroke survivors like Randy, Mission: Lifeline Stroke is working to ensure the best possible outcomes for everyone, everywhere.

"Your zip code shouldn't determine whether you survive a stroke," said Joani Guzman, Mission: Lifeline Stroke Program Manager for South Dakota. "No matter where you live in South Dakota, you should have the opportunity for your fullest potential recovery from stroke."

Randy had his stroke on a Wednesday morning, and by Friday night he was calling a hockey game. "I'm an incredibly lucky person," Randy said. "The fact that my wife was able to get to me so quickly, that she knew exactly what was going on, they got me to the hospital quickly, and the doctors and the facilities took care of me quickly, I'm able to do what I love."

To learn more about the Mission: Lifeline Stroke SD project, visit **www.ActFASTSD.org**.

To see more of Randy's story, scan the QR code to view his video online.

Congratulations to the following South Dakota facilities who have joined the American Heart Association and American Stroke Association's Mission: Lifeline Stroke acute and post-acute initiatives.

These providers have agreed to follow the Association's treatment guidelines and have made a commitment to providing care that is based on standards aligned with American Stroke Association science: *Sites listed as of 3/3/2026*

Post-Acute Sites

- Avantara Groton
- Avantara Mountain View
- Avantara North
- Avantara Redfield
- Avantara Watertown
- Avera Eureka Health Care Center
- Avera McKennan Inpatient Rehabilitation
- Avera Prince of Peace
- Avera St. Benedict Health Center
- Bethany Home Brandon
- Bethany Home Sioux Falls
- Bowdle Healthcare Center
- Community Memorial Hospital
- Douglas County Memorial Hospital
- Encompass Health Rehabilitation Hospital of Sioux Falls
- Faulkton Area Medical Center
- Madison Regional Health System
- Monument Health Sturgis Hospital
- Platte Health Center Avera
- Sanford Chamberlain
- Sanford Clear Lake Medical Center
- Sanford Medical Center Inpatient Rehab
- Sanford Vermillion
- St. Michael's Hospital Avera
- Sunset Manor Avera
- Wakonda Heritage Manor

Acute Sites

- Avera McKennan
- Avera Queen of Peace
- Avera Sacred Heart
- Avera St. Luke's
- Avera St. Mary's
- Avera St. Michael's
- Bowdle Healthcare Center
- Brookings Health System
- Community Memorial Hospital
- Douglas County Memorial Hospital
- Huron Regional
- Mobridge Regional
- Monument Health Custer
- Monument Health Lead/Deadwood
- Monument Health Rapid City
- Monument Health Spearfish
- Monument Health Sturgis Hospital
- Pioneer Memorial Hospital & Health Services
- Platte Health Center Avera
- Prairie Lakes Healthcare System
- Sanford Aberdeen
- Sanford Health Canton
- Sanford Health Chamberlain
- Sanford Health Clearlake
- Sanford Health Vermillion
- Sanford Health Webster
- Sanford USD Medical Center
- Winner Regional Health

For more information on Mission: Lifeline Stroke, email Joani.Guzman@heart.org or Caitlin.Schlosser@heart.org

Learn more about this initiative at heart.org/PostAcuteStroke

American Heart Association.
Mission:Lifeline®
Stroke

Member News

Visit www.sdsma.org for the latest news and information.

Keynote Speaker: Dr. Michelle Thompson

Michelle Thompson, DO, AOBFP, ABOIM, DipABLM, FACLM will be the keynote speaker at the SDSMA 2026 Annual Leadership Conference on May 29. Dr. Thompson is a leading expert on lifestyle medicine who will present "From Sick Care to Self-Care: Transforming Self and Systems Through Lifestyle Medicine" at 8:40 a.m. She is employed by the University of Pittsburgh Medical Center and has a full-time integrative family medicine practice, seeing infants through the end of life. Learn more about Dr. Thompson and register for the conference at sdsma.org. See all of the conference's experts on lifestyle medicine, including speakers and panelists at sdsma.org.

Announcing Annual Conference Speaker Dr. Allen Weiss of Blue Zones

Allen Weiss, MD, FACP, FACR, MBA is Senior Executive Consultant for Blue Zones. Having practiced rheumatology, internal medicine, and geriatrics for 23 years and been president and CEO for 18 years of a 716-bed, two-hospital integrated system in Florida, Dr. Weiss now has a national scope focused on prevention. He will give the presentation, "Prevention as a Cure for Healthcare and Our Nation's Economy" at 9:30 am.

Sign up for the 2026 SDSMA Annual Leadership Conference

Registration Deadline Approaching!

Advancing Patient Care Through Lifestyle Medicine

Get ready for the 2026 SDSMA Annual Leadership Conference on Friday, May 29 at The Catlin (formerly Hilton Garden Inn) Downtown Sioux Falls. Join us for a day of learning and engagement as we talk about the transformative role of lifestyle medicine! This year's conference will focus on a comprehensive, evidence-based approach to applying lifestyle medicine to today's health challenges. Physicians will explore practical methods for integrating lifestyle medicine fundamentals into daily clinical practice to improve patient outcomes. The information will provide physicians with actionable strategies to enhance long-term outcomes and elevate their approach to preventive care.

The conference is a benefit of your SDSMA membership – there is no conference registration fee for members (purchase required for evening Awards & Scholarship Recognition Dinner). This year, lunch is included with registration thanks to the sponsorship of the Black Hills District Medical Society. Registration, details and the schedule for the day can be found at sdsma.org. Please register by May 21.

Pre-conference Reception – Thursday, May 28

All members and conference attendees and are invited to the new SDSMA office from 6:00-8:00 pm on Thursday, May 28 for a pre-conference welcome reception. The SDSMA thanks the District 7 Medical Society for sponsoring this event! Spouse/guest are welcome to attend.

Poster Session Highlights the Work of Medical Students & Residents

Be sure to join us for the Medical Student and Resident Poster Session from 1:45-2:30 pm on May 29 during the conference. Following the poster session, Matthew Schmitz, MS III, the #1 abstract winner, will present "Conducting a cadaveric suturing laboratory to increase clinical readiness in medical students – an education research project" by Matthew Schmitz, MS III; Gary Timmerman, MD; William Spanos, MD; Amy Prunuske, PhD; and Taylor Soderling, PhD.

Social Hour is Networking Opportunity

A social hour will be held from 6:00-7:00 pm prior to dinner on Friday, May 29. Enjoy live music and an opportunity to meet SDSMA leaders and network with your peers. Meet other physicians in the

state to expand your professional network. The event is sure to be an all-around great time! Spouses/guest are welcome to attend. The social hour is free of charge. Tickets are required for the Awards Banquet and Scholarship Recognition following the social hour. See the schedule of the day's events and purchase banquet tickets at sdsma.org.

Awards Banquet, Scholarship Recognition & Presidential Inauguratoin

Enjoy a fun-filled celebration on Friday, May 29! Start the evening with the social hour at 6:00 pm and continue the celebration with the Awards Banquet, Scholarship Recognition & Presidential Inauguration, where Ben Meyerink, MD will be sworn in as SDSMA president, awards will be bestowed upon fellow colleagues, outgoing and incoming officers will be celebrated and welcomed, and medical student scholarship recipients will be recognized. Dinner tickets ($75) must be purchased in advance at sdsma.org.

No Surprises Act Needs Enforcement: SDSMA Signs Letter With AMA, State and Specialty Societies

The SDSMA signed on to an AMA Federation letter calling for enhanced oversight of the No Surprises Act implementation. The letter details strategies health plans have used to circumvent the law and shift costs onto patients and independent practices. The letter calls on administration officials and federal regulators to increase enforcement efforts and require greater transparency in the IDR process, offering several specific recommendations to do so.

Specifically, the letter pointed to:

- **Inappropriate Cost-Shifting onto Patients.** Physicians have heard reports that payers are increasing patient cost-sharing amounts after an Independent Dispute Resolution (IDR) decision in the physicians' favor—a practice that is clearly in violation of the spirit, if not the language, of the No Surprises Act.
- **Misusing Technical Guidance to Reopen Closed IDR Cases.** Some health plans appear to be exploiting June 2025 technical guidance intended to allow IDR cases to be reopened in a narrow set of circumstances to reopen final IDR decisions as a way to withhold payment from physicians.
- **Ineligible Claims in IDR.** Eligibility is often challenging for physicians to determine, especially when it comes to determining the appropriate regulator. Payers possess plan information that makes them best suited to identify eligibility for the process yet often fail to provide necessary information to physicians.
- **Lack of Transparency into Qualified Payment Amount Calculations.** Physicians report that the calculations do not reflect the market rates for services, as intended under the law. More transparency in how rates are calculated should be required.
- **Lack of Timely and Complete Payments.** Health plans are failing to reconcile payment outside of the statutory 30-day window, paying only a portion of what they owe, or are not paying at all, often repeatedly and without consequence.

Don't forget to send in your favorite scenic photo for

South Dakota Medicine

front cover consideration. Send photos to ereiss@sdsma.org.

2026 SDSMA Annual Leadership Conference Sessions, Activities & Events

Thursday, May 28

Time	Activity
12:00-4:00 pm	**SDSMA Board of Directors Meeting** SDSMA office, 1610 S Minnesota Ave
6:00-8:00 pm	**Pre-Conference Welcome Reception** SDSMA office, 1610 S Minnesota Ave *Sponsored by the District 7 Medical Society*

Friday, May 29

All activities are in the Catlin Hotel Four Winds rooms unless otherwise noted

Time	Activity
7:00-8:00 am	**Past Presidents' Breakfast** - Bar Clara Room (reserved for past SDSMA presidents)
8:00-8:30 am	**American Medical Association Update: Physician Advocacy** **Willie Underwood III, MD**, President-Elect, American Medical Association
8:40-9:30 am	**Keynote: From Sick Care to Self-Care: Transforming Self and Systems Through Lifestyle Medicine** **Michelle L. Thompson, DO, AOBFP, ABOIM, DipABLM, FACLM**, Medical Director, UPMC Lifestyle Medicine Program
9:30-10:30 am	**Prevention as a Cure for Healthcare and Our Nation's Economy** **Allen Weiss, MD, FACP, FACR, MBA**, Senior Executive Consultant, Blue Zones
10:30-10:40 am	**Break**
10:40-11:45 am	**Panel Discussion: The Growth of Lifestyle Medicine in South Dakota** **Marty Allison, MD, FAAP, DipABLM**, Avera Pierre Pediatrics; **Michelle L. Thompson, DO, AOBFP, ABOIM, DipABLM, FACLM**, Medical Directory, UPMC Lifestyle Medicine Program; **Amy Prunuske, PhD**, Associate Dean of Medical Education, University of South Dakota Sanford School of Medicine; **Allen Weiss, MD, FACP, FACR, MBA**, Senior Executive Consultant, Blue Zones; **Kathryn Wermers, DNP, FNP-C, DipACLM**, Cancer Care Institute, Monument Health; **Melissa Magstadt**, MS, MBA, APRN, South Dakota Secretary of Health Moderator: Rob Allison, MD, MACP, Avera Internal Medicine Clinic
12:00-1:00 pm	**Lunch: Rural Health Transformation Project** Melissa Magstadt, MS, MBA, APRN, South Dakota Secretary of Health; Clarissa Barnes, MD, Chief Medical Officer, South Dakota Medicaid *Lunch sponsored by the Black Hills District Medical Society*
1:15-1:45 pm	**Update from the University of South Dakota Sanford School of Medicine** Tim Ridgway, MD, MACP, FASGE, Dean, University of South Dakota Sanford School of Medicine
1:45-2:30 pm	**Medical Student and Resident Poster Session**
2:30-5:00 pm	**SDSMA Policy Council Meeting** • **Presentation of #1 Abstract -** "Conducting a Cadaveric Suturing Laboratory to Increase Clinical Readiness in Medical Students – An Education Research Project" - Matthew Schmitz, MS III • **Medical Liability Insurance Update** - Copic • **Membership Open Forum**
5:00-5:30 pm	**SDSMA Board of Directors Meeting** - Bar Clara Room
6:00-7:00 pm	**Social Hour** Live music with Jeremy DeWall starting at 5:30 pm
7:00 pm	**Awards Banquet, Scholarship Recognition & Presidential Inauguration**

SOUTH DAKOTA
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Board News is a monthly feature sponsored by the South Dakota Board of Medical and Osteopathic Examiners. For more information, contact the Board at SDBMOE@state.sd.us or write to SDBMOE, 101 N. Main Avenue, Suite 301, Sioux Falls, SD 57104.

Help Shape the Future of Medicine in South Dakota

The South Dakota State Medical Association Foundation, the philanthropic arm of the South Dakota State Medical Association, is a tax-exempt 501(C)(3) non-profit corporation, was established to assist and support medical research, medical teaching and medical education at the Sanford School of Medicine.

On average, medical students graduate with $130,000 in debt. Contributions to the South Dakota State Medical Association Foundation provide financial assistance to students at the Sanford School of Medicine and are all designated for scholarships, grants and low-interest loans for students.

Any amount can be donated at any time throughout the year. If you have questions or want more information, please call 605.336.1965.

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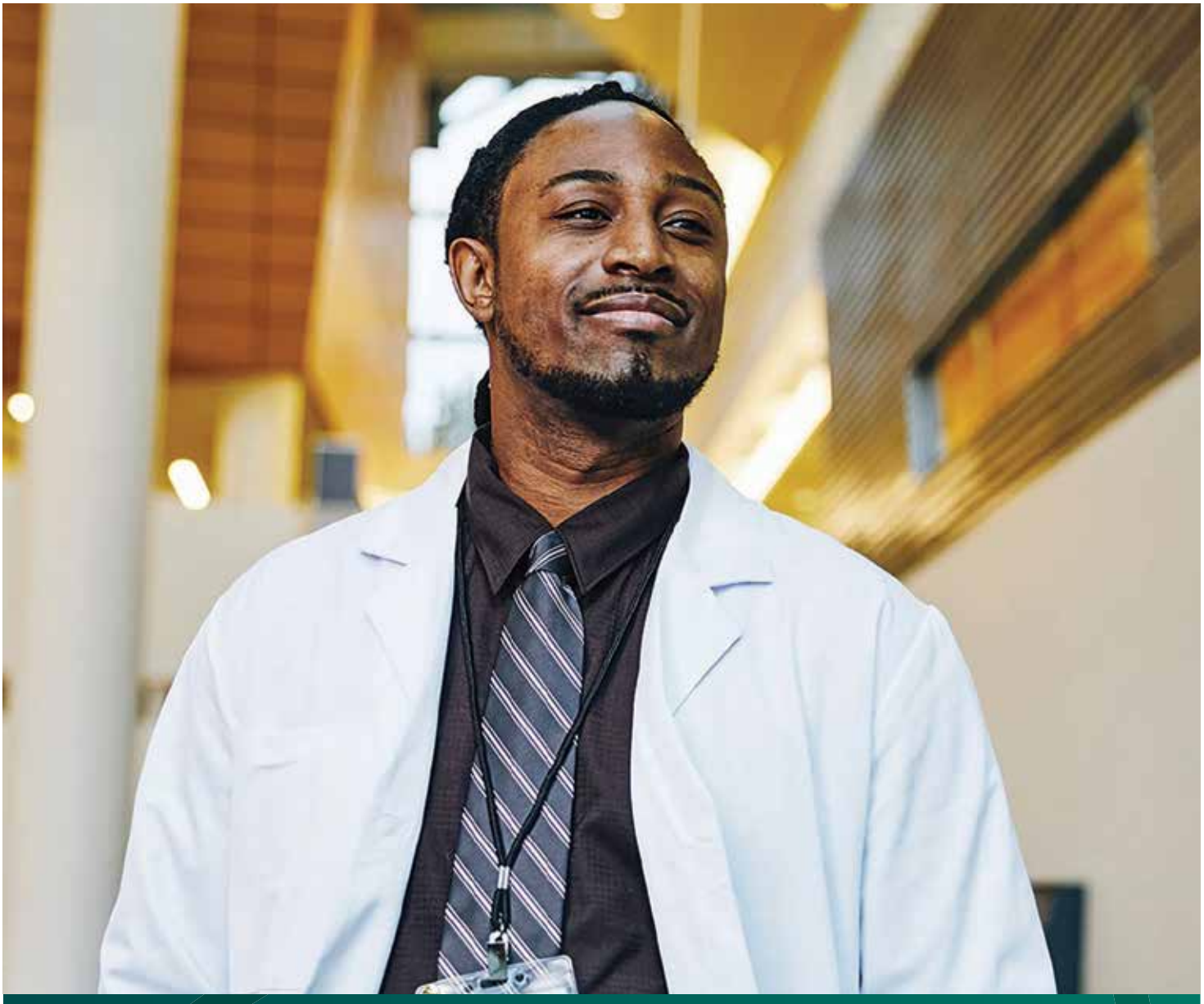

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