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South Dakota medicine

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A FINANCIAL ADVISOR'S MORNING ROUTINE: WHY I BROKE UP WITH "BREAKING NEWS"

ASHLEE VIEREGGER, JD, CFP®, CFTA

You might be surprised by my morning office routine. In the 12+ years that I've been an advisor, the first half hour of my day has been pretty consistent:

1. Say hi: greet everyone I see in the halls on the way to my office
2. Energy boost: grab a Diet Coke and a granola bar
3. Headlines: glance at Yahoo!Finance, The Wall Street Journal, and Google News online
4. Correspondence: open Microsoft Outlook and Teams, begin working on new messages and replies

The rest of my day might include meetings with clients or our team, meeting prep or follow-up, or connecting with fellow professionals who also serve the clients I work with (attorneys, accountants).

Back to the morning – you'll notice that my daily routine does not include spending hours poring over economic data, trying to read financial tea leaves, or watching market movements like a hawk. I don't avoid these things by accident; it's a deliberate choice on my part to stay high-level, to avoid getting tangled up in the weeds. Why?

The short answer is that it helps me stay calm and level-headed, which makes me a better advisor. One of my key roles as an advisor is to help clients make sense of and plan for uncertainty, not to add fuel to a panic-driven fire. Though I need to be informed, I can't get drawn into every breaking news story that touches the economy, markets, or business. The sheer volume is overwhelming, and the tone often has a pessimistic slant, because, let's face it, bad news sells. Instead, I get a daily email of business updates from Morning Brew, which presents the top stories with a dash of humor.

In addition to my role as an advisor, I'm also a long-term investor, adding dollars to a brokerage account and 401(k) plan each month to help achieve my financial goals. Research suggests that my "news diet" also has portfolio benefits, in addition to keeping me calm and composed in my work. Simply put, most of us would do better in the investment markets if we traded less often. And it follows that consuming less doom-and-gloom economic news could make us less inclined to make a reactionary portfolio adjustment, which, at best, may give us the illusion of control in response to the latest source of uncertainty.

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Addressing the Healthcare Needs of Adolescents and Young Adults

Denise Hanisch, MD
President, South Dakota State Medical Association

Does anyone really ever experience the "lazy days of summer" anymore? Life is busier than ever for families, especially if teenagers are involved. Softball, baseball, church camps and family reunions are just a few of the activities that are crammed into the summer. If you are a primary care provider, your schedule starts to fill up with sports physical exams in late July and it feels like summer is over. Sport physical exams are often the only time we get to have conversations with our adolescent patients.

Teenagers and young adults are rarely seen for yearly physicals and these are missed opportunities to identify young people at risk for drug and alcohol abuse and mental health issues. According to the South Dakota Department of Health, suicide is the leading cause of death in the age group of 10-29. Twenty-three percent of South Dakota high school students have considered suicide and 12.3% have attempted suicide. Compare this to the percentage of adults who have Type II Diabetes in our state which is 9.2%. As physicians, we devote a significant amount of our time identifying and treating diabetes and other chronic illnesses but so many barriers exist when attempting to care for our young patients.

Even during a limited visit of a sports physical it can be difficult to ascertain if a child is struggling. The South Dakota High School Association added "suggested" questions regarding possible drug and alcohol use, depression and anxiety to the standard form several years ago. It is helpful when a parent is present, but, it can be difficult to ask these personal questions with a parent in the room and I am surprised at how often they are offended by the questions I may ask. Several days ago, a mother aggressively voiced her thoughts about my conversation with her daughter. She did accept my explanation that, even though these questions may not be relevant for her daughter, they were important because they may help identify other kids that need a safe place to open up and I want my exam room to be that safe place for every person.

Another roadblock to provide care for our young adults is lack of insurance coverage. Hopefully this barrier will be mitigated by the expansion of Medicaid that went into effect July 1. South Dakota Medicaid will now include all individuals aged 19-64 who have an income that is 138% of the federal poverty level for 2023. The Department of Health predicts that an additional 50,000-60,000 South Dakotans will now have access to healthcare. This is encouraging and an obvious step in the right direction.

The new Medicaid program also includes expansion of pregnancy coverage. This will provide care to young mothers that was previously limited to postpartum care for 60 days after delivery and for issues that were related to the pregnancy. Now, postpartum women will have full Medicaid coverage, to include any diagnosis, for a time period of 12 months. Hopefully, this will help physicians identify young mothers at risk for postpartum depression and give us the time and resources to provide adequate care so that both the mother and baby can thrive.

Young people are the best resource we have. Even with improved access to care and great strides made in public awareness regarding mental health, we are still failing. Over the past 10 years, suicide rates have increased by 50 percent in our younger population. We need to encourage them to seek care. When you see a teenager for a simple upper respiratory infection or injury, schedule them for a yearly exam on their way out. Have discussions with them during sports physicals, even if it is uncomfortable at times with parents in the room. Encourage young mothers to care for their own health as they are trying to navigate the world of caring for a newborn.

I hope all of you take some time these last weeks of summer to relax and recharge.

Editorial

Embracing the ~~Soft~~ Essential Skills

Jason J. Kemnitz, EdD

One of the fastest growing genres of literature is self-help. The self-help category grows by 20% annually and had a net worth of $10.5 billion in 2020. Names like Covey, Brown, Kondo and Gladwell have been common parlance for years. But this phenomenon is not new. I recall as a child looking at the family bookshelf and seeing titles by Dale Carnegie, Napoleon Hill, and Norman Vincent Peale. As a precocious high school debater (shoutout Arrow Forensics). I read titles like "Speak and Grow Rich" chasing the dream of using a skill that came easy to me to become what I though was happy and, while that idea didn't pan out, I still find a number of these authors on the bookshelf in my office. At their heart, each of these authors is advocating to increase the readers comprehension and use of what many call "soft skills." In medical education we deal with specific task-oriented skills, accepted hard science and evidence-based medicine and, while not unchanging, doctrine pretty much stick to those realms. Items like soft skills we often under teach and overlook. I contend that by increasing our knowledge of soft skills we can not only complement but increase the effectiveness of medical education.

The U.S. Department of Labor lays out the following "competitive" soft skills: including ideas like enthusiasm, communication skills, professionalism and many others. Although not listed, emotional intelligence is often considered a soft skill as well. Each one of the skills complements what we are teaching our students as medical students.

Harvard Medical School relates "Physician leaders who are able to exhibit high degrees of emotional intelligence (EI),... demonstrate better clinical outcomes, greater professional satisfaction, increased empathy and improved teamwork within health care organizations." The process of teaching students the concepts of self-awareness, self-management, empathy and social skills can make them more proficient physicians and team members. In turn, your ability as faculty to understand and be comfortable enough to speak to these skills will improve your ability as a teacher.

In the last five years the concept of work life balance has dominated conversations about medical education. Further, I feel that we have seen a specific change in the work demeanor in medical education as seen in the shift from the concept of live to work to work to live. This manifests itself in many ways such as students expressing their hesitance to work weekends, or evenings, or hyper focusing on their chosen specialty instead of all specialties they were studying. While this attitude is not necessarily bad, it is often viewed as a lack of enthusiasm for their profession. As faculty, we should model and help our students understand, that by showing enthusiasm, they will also receive more ownership of their education. Faculty are much more willing to work with a student that appears to want to be there.

Not surprisingly, both communication and professionalism are addressed in medical education. These two skills are competencies in our curriculum and are graded as such. Both carry enormous weight in patient care. Communication, including interpersonal, small group and interviews should be stressed for their obvious needs in medicine. Unfortunately, the art of intrapersonal communication is a valuable tool often not taught. Helping students become aware for their own self-awareness, perception and self-expectations helps them grasp the understanding for other situations. This empathy is cited by many as setting a good physician apart from a great physician. Professionalism takes on many forms and is often hard to quantify in medical education. In some regards professionalism can be broken into the concepts of responsibility, maturity, and respect.[1] Explaining specific responsibilities,(arriving on time and accepting feedback) helping students grow to became a mature physicians (discretion in times of stress) and demonstrating respect (sensitive to teams needs) are all valuable lessons. The simplest way to achieve this is to model professional behavior. Remember, our students are constantly watching you and gleaning what they consider acceptable information, ideas and behaviors from you.

While these skills are often hard to teach, by simple adjustments in stressing to students how you incorporate these skills as a physician we can help them grow in these skills. Will it be easy? Not as easy as teaching your specialty, but with a little thought you can find ways to incorporate both into a teaching session. Is it a comfortable process? Probably not, but it is important. Dale Carnegie would remind you to "do the hard jobs first. The easy jobs will take care of themselves.

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Mediastinal Presentation of Testicular Embryonal Carcinoma

Sanyogita Chandra, MD, MS; Heidi McKean, MD

Abstract

Testicular embryonal carcinoma is a type of nonseminomatous germ cell tumor (NSGCT) that commonly presents with scrotal swelling due to testicular mass. About half of patients with NSGCTs will present with metastases at initial diagnosis. Rarely, testicular embryonal carcinoma can present primarily in the mediastinum. Treatment is well-studied and effective: chemotherapy with bleomycin, etoposide, and cisplatin. Post-chemotherapy retroperitoneal lymph node dissection (RPLND) is common adjuvant therapy. In this report we present a case of testicular embryonal carcinoma in a 32-year-old Caucasian man. The rarity of the case resides in its presentation: supraclavicular lymphadenopathy and no testicular mass on palpation or scrotal ultrasound.

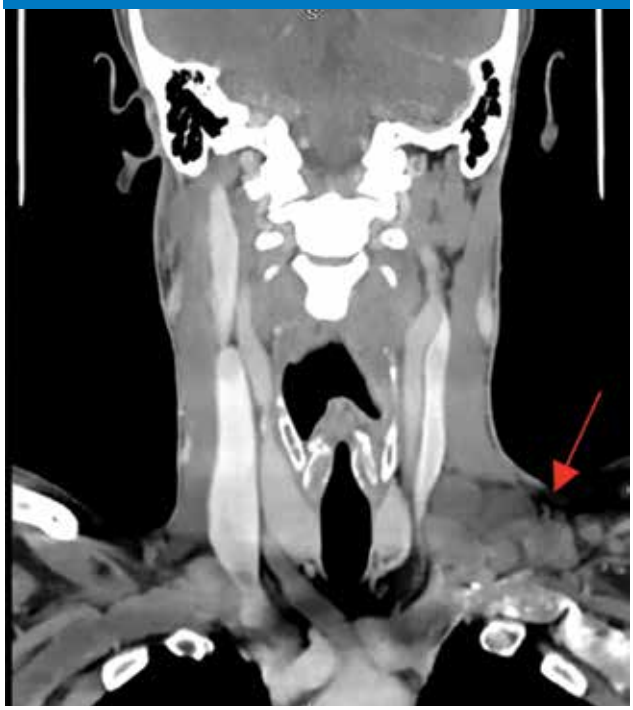
Introduction

Testicular cancer is the most common malignancy in men aged 20-35 years.[1] More than 90% of germ cell tumors develop from multipotent germ cells and arise in the testes, presenting with testicular swelling that may or may not be painful. Rarely, the tumors can arise from the mediastinum, retroperitoneum, or suprasellar area. Elevations of alpha-fetoprotein (AFP), a major serum protein of the fetus, and human chorionic gonadotropin (hCG), a placental hormone, are commonly found elevated in NSGCT.[2] Chemotherapy includes bleomycin, etoposide, and cisplatin. RPLND is common adjuvant therapy in patients with negative tumor markers after chemotherapy.[3] The purpose of this report is to highlight a rare presentation of testicular embryonal carcinoma.

Case Report

A 32-year-old Caucasian male presented to his primary care provider with complaints of a lump on the left side of his neck that he palpated three weeks ago. A firm 4 cm supraclavicular nodular mass is palpated without axillary adenopathy with a normal thyroid and neck that is supple without adenopathy. There was no inguinal adenopathy. A thorough genitourinary exam was not done at this initial visit, given that the patient's main complaint was the supraclavicular nodular mass. Patient denied weight loss and fevers but did endorse night sweats. A chest X-ray was unremarkable. A CT scan of the neck showed a large cluster of enlarged lymph nodes in the left inferolateral neck involving level IV (deep cervical chain) and level VB (transverse cervical and supraclavicular) lymph nodes (Figure 1). These CT scan findings are concerning for malignant lymphadenopathy.

Figure 1. CT scan of neck shows a large cluster of enlarged lymph nodes in the left inferolateral neck involving level IV (deep cervical chain) and level VB (transverse cervical and supraclavicular) lymph nodes.



Lymph node excisional biopsy reveals metastatic embryonal carcinoma as well as lymphovascular space invasion and extranodal extension. Flow cytometry analysis found mixed population of T- and polyclonal B-lymphocytes with no

monoclonal B-lymphoid population identified, ruling out lymphoma. The pathology report also found the tumor cells are positive for AE1/AE3, CD30 and placental alkaline phosphatase (PLAP). Maximum intensity projection (MIP) PET scan finds abnormal fluorodeoxyglucose (FDG) uptake in the medial and lateral left supraclavicular lymph nodes, as well as enlarged para-aortic, bilateral common iliac and internal iliac lymph nodes. The scrotal ultrasound did not show a testicular mass. MRI of the brain with and without contrast did not show metastatic disease. Lab work showed alkaline phosphatase elevated to 163 U/L (34-104 U/L), lactate dehydrogenase (LDH) elevated to 464 U/L (140-271 U/L), tumor marker AFP elevated to 110 ng/mL (0-15 ng/mL), and tumor marker hCG elevated to 6,218 mIU/mL (0-5 mIU/mL).

The patient was started on IV cisplatin, bleomycin, and etoposide therapy. The patient endorsed occasional nausea, fatigue, and constipation after chemotherapy. He also endorsed tinnitus in both ears, but it is infrequent and does not bother him. He denied a decline in his hearing ability. Three months after treatment was started, PET scan showed that patient has excellent treatment response with only small residual nodes along the peri-aortic and left common iliac level. Lab work showed tumor marker AFP normal at 3.7 ng/mL (0-15 ng/mL) and tumor marker hCG normal at 1 mIU/mL (0-5 mIU/mL). CT scan of abdomen and pelvis five months after the initial diagnosis stated, "mild retroperitoneal adenopathy with several nodes demonstrating peripheral enhancement and central low attenuation. Mild mesenteric and retroperitoneal edema. These findings could be consistent with the patient's reported history of treated metastatic embryonal cell [carcinoma]. No evidence of solid organ metastatic disease." Repeat scrotal ultrasound was unremarkable.

The patient underwent RPLND after chemotherapy treatment. The following lymph nodes were dissected: para-aortic, left inter-iliac, paracaval, interaortocaval, right common and external iliac, and intra-iliac. The final diagnosis was found to be stage IIIB intermediate risk embryonal NSGCT. New pathology report stated, "approximately 1% viable tumor in a para-aortic lymph node mass measuring 9.5 cm in greatest dimension and in a single lymph node in the left interiliac region with just 1% viable tumor. Other areas of nodal tissue show no evidence of viable tumor, consistent with necrosis and treatment effect." Further chemotherapy was not recommended at this time.

A year later, the patient returned to oncology clinic for follow-up and stated he is doing well. However, a month later, he returned with complaints of palpating a lump in his left groin. On physical exam, no firm masses or abnormalities are palpated. There was a hernia noted in the inguinal region that was soft and reducible. The patient was advised to avoid heavy lifting and let his physician know if the area becomes firm, larger, or painful. A CT scan of the abdomen and pelvis with IV contrast stated, "no acute changes in the chest, abdomen, or pelvis. Stable postsurgical changes from retroperitoneal lymph node dissection. No new lymphadenopathy." A repeat CT scan of the abdomen and pelvis with IV contrast six months later stated, "no residual or recurrent disease." Repeat scrotal ultrasound shows bilateral testicular microlithiasis without masses. Repeat imaging has remained unchanged for three years. The patient continues to follow with oncology.

Discussion

NSGCT include embryonal carcinomas. Almost half of patients with NSGCT will present with metastatic disease at diagnosis. According to the International Germ Cell Cancer Collaborative Group (IGCCCG), NSCGT are split into good, intermediate, and poor prognostic categories based on levels of tumor markers AFP, hCG, LDH, as well as presence of non-pulmonary visceral metastases.[4] Patients presenting with a testicular mass or with persistent testicular pain or swelling should receive a testicular ultrasound examination and bloodwork for tumor markers. Furthermore, an ultrasound suspicious for cancer should proceed to surgical exploration and radical inguinal orchidectomy. Needle biopsy is contraindicated due to risk of tumor cell seeding.[1] Ultrasonography is the primary imaging modality for testicular masses. Ultrasound imaging has nearly 100% sensitivity in the differentiation between intra-testicular and extra-testicular lesions, and extra-testicular lesions tend to be benign, while intra-testicular lesions tend to be malignant.[5]

One to four percent of mediastinal tumors are primary mediastinal malignant germ cell tumors. Of these, 50-70% are NSGCTs. These occur exclusively in young males and grow rapidly, often leading to compression and invasion into mediastinal structures such as the trachea and nearby major vessels.[6] Mediastinal germ cell tumors are a heterogenous group of benign and malignant neoplasms thought to originate from primitive germ cells that are "misplaced" in the mediastinum during early embryogenesis. In a series of patients with extragonadal germ cell tumors, 71% of patients with NSGCT had elevated AFP levels and 54% had elevated hCG levels.[7]

NSGCT can metastasize via vascular or lymphatic pathways or both. Common sites are retroperitoneal nodes, mediastinal and supraclavicular nodes, as well as the lungs. Testicular cancer staging is based on tumor, node, metastasis (TNM) staging system, where assessments of the primary tumor (T), lymph nodes (N), and distant metastases (M) are combined with serum tumor marker values (S) for beta-HCG, AFP and LDH to define prognostic stages from groups I-III.[8] Treatment for patients with stage IS, IIA-B with persistent serum tumor marker elevation, IIC, or IIIA who are at low risk for advanced disease, offer etoposide plus cisplatin for four cycles or bleomycin, etoposide, and cisplatin for three cycles. For patients with stage IIIB who are at intermediate risk for advanced disease, offer bleomycin, etoposide, and cisplatin for four cycles.[3] With chemotherapy, about 70% of patients with metastatic NSGCTs will obtain a complete clinical response with normalization of serum tumor markers and resolution of metastatic disease. Furthermore, patients can undergo post-chemotherapy RPLND. Research suggests that pathology of a residual mass can represent necrosis in 45% of patients, as seen with the presented patient, teratoma in 45% of patients, and recurrence of NSCGT in 10% of patients.[9]

Conclusion

Testicular embryonal carcinoma is a type NSGCT that usually presents with primarily testicular symptoms, such as scrotal swelling and/or pain. Rarely, it will present in the mediastinum, likely due to mislocated primitive germ cells. NSGCTs present with metastases at initial presentation in about 50% of cases. Tumor markers AFP and hCG are elevated, as well as LDH. Fortunately, first-line treatment with bleomycin, etoposide, and cisplatin is effective to improve prognosis in most patients with testicular embryonal carcinoma, including those with metastatic disease. In patients who undergo post-chemotherapy RPLND and the pathology results show necrosis, such as the patient presented in this report, less than 5% will have tumor recurrence.[9]

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Colorectal Adenocarcinoma in a 35-year-old Female with No Germline Mutations

Annika van Oosbree, MD; Lindsay Leigh, MD; Michael Burt, MD; and Ryan Garry, MD, FACS

Abstract

Colorectal cancer (CRC) is the third most common cancer to cause death in the world. Despite the recent change in age from 50 to 45 in some societies' recommendations for CRC screening, its incidence is increasing in patients under 50 years. We present a case of a 35-year-old female with no family history of CRC who presented to the emergency department with abdominal pain, vomiting, and obstipation and was found to have sporadic obstructing transverse colon adenocarcinoma.

Introduction

Colorectal cancer (CRC) is the third most common cancer to cause death in the world.[1] Although over 90% of those diagnosed are over age 55, the incidence of diagnoses in younger patients has been gradually increasing over the last 20 years, from 2% to 8%. Now, CRC has risen to the status of one of the top 10 most common causes of death in patients between ages 20 and 49.[2]

Different societies offer various recommendations regarding when to begin CRC screening via colonoscopy. The American Cancer Society and American Society of Colon and Rectal Surgeons now both recommend screening beginning at age 45,[3,4] whereas the U.S. Multi-Society Task Force (USPSTF) on Colorectal Cancer and American Academy of Family Physicians recommend starting at age 50.[5,6] The USPSTF does give a grade B recommendation to beginning at age 45. Similarly, the American College of Gastroenterology gives a conditional recommendation for age 45 as well as a strong recommendation for age 50.[7]

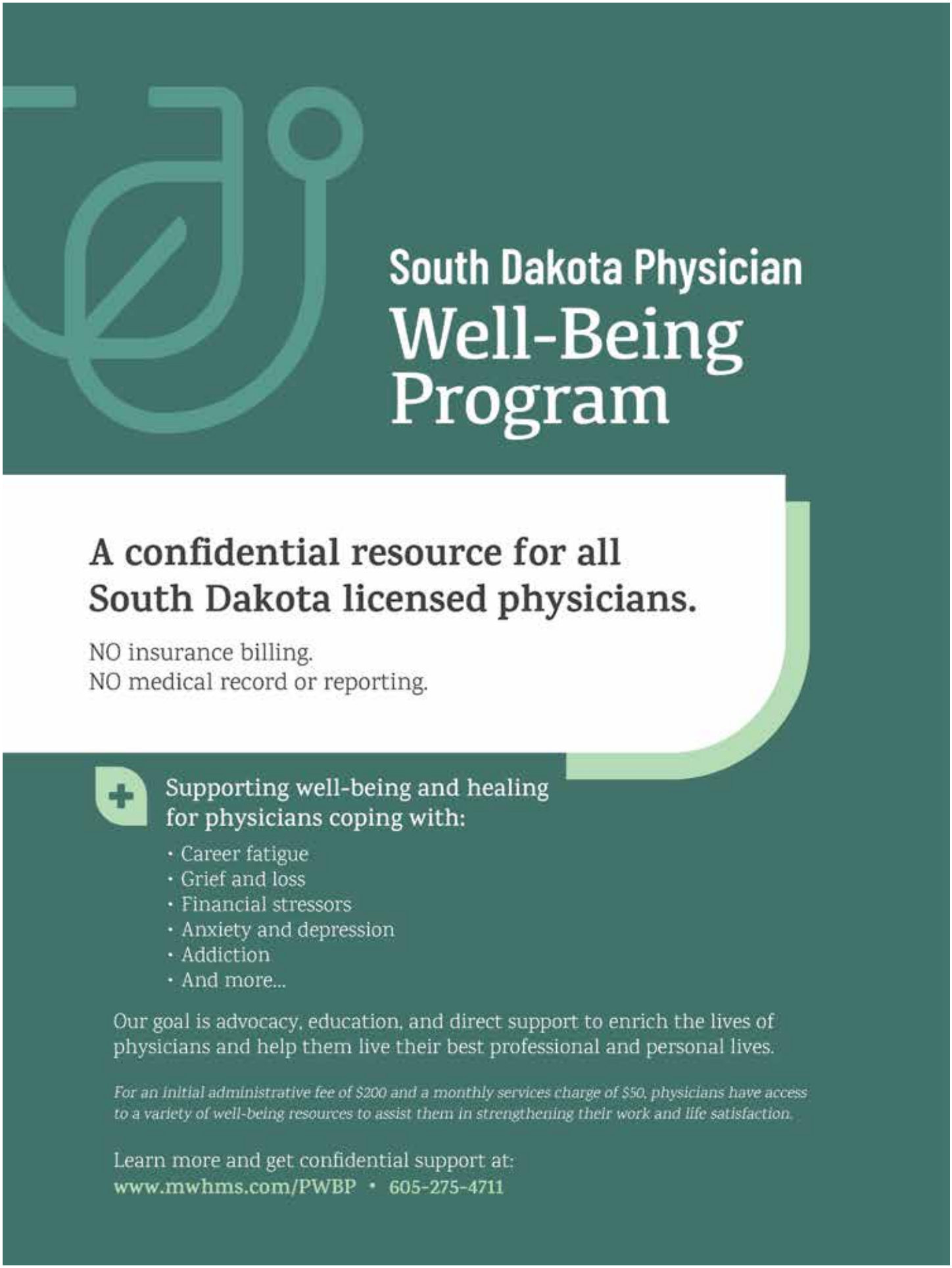
CRC in younger patients often presents secondary to a hereditary cancer syndrome, including Lynch syndrome (HNPCC), familial adenomatous polyposis (FAP), MYH-associated polyposis (MAP), and the hamartomatous polyposis syndromes (Peutz-Jeghers, juvenile polyposis, and Cowden disease). Additionally, a history of Crohn's disease and ulcerative colitis both confer increased risk.[8] Although new data supports multiple pathological mutations, sporadic CRC classically arises via the adenoma-carcinoma sequence. In this pathway, an adenomatous polyposis coli gene (APC) inactivation increases the risk for formation of an adenoma, and a subsequent KRAS or BRAF mutation leads to the formation of an intermediate adenoma polyp. A TP53 mutation and loss then increases risk of transformation to carcinoma.[9]

The incidence of CRC in patients under 50 is increasing without corresponding increase in patients over 50.[10] Currently, 4.8% of all colon and 9.5% of all rectal cancers are diagnosed in those under age 50. Population-based analyses predict that, by 2030, these numbers will rise to 10.9% and 22.9%, respectively.[10] This report will discuss a case of obstructing sporadic colon cancer in a 35-year-old without family history or other risk factors.

Case Presentation

This patient is a 35-year-old Caucasian female with a history of asthma, gastroesophageal reflux disease (GERD), and an appendectomy/cholecystectomy as a teenager who presented to the emergency department with a six-day history of intermittent abdominal pain, nausea, and vomiting that progressed to obstipation. On presentation, she had not passed flatus or had a bowel movement in 3 days. Prior bowel habits included passing three to four loose stools a day; she attributed this to her cholecystectomy. She denied melena and hematochezia. In the past few months, she had experienced several bouts of constipation. She also had intermittent night sweats over the past few months but denied unintentional weight loss or low-grade fevers.

Social history was notable for a 17-pack year smoking history and occasional alcohol intake. She had never undergone a colonoscopy. She had no family history of inflammatory bowel disease including Crohn's and ulcerative colitis. She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also had no family history of colorectal cancer. Her family history of cancer included breast cancer in her mother at age 27 or 28, maternal grandfather with leukemia, a maternal aunt with skin cancer, a maternal great uncle with brain cancer, and a maternal great uncle with throat cancer.

CT abdomen/pelvis (with IV contrast) was obtained in the ED which demonstrated focal enhancement and wall thickening of the transverse colon with luminal narrowing and proximal colonic dilation concerning for colonic obstruction. Etiology at that time was believed to be colonic stricture. Initial labs were notable for a mild leukocytosis of 13.2 cells/µL (4,400-11,000) and a potassium of 3.4 mEq/L (3.5-5.0). Carcinoembryonic antigen (CEA) was elevated at 5.8 µg/L (2.5-5). Creatinine was normal at 0.7. She was admitted with bowel rest and crystalloid resuscitation.

On hospital day one, a colonoscopy was performed and demonstrated a stricture versus inflammatory mass in the mid transverse colon that was unable to be traversed by the colonoscope. No biopsies were obtained due to planned resection given inability to pass the colonoscope. The following day she underwent open extended right hemicolectomy with primary stapled ileocolonic anastomosis. She had a return of bowel function on post-operative day one, and her post-op course was unremarkable. She was discharged home on post-op day three.

Pathologic review demonstrated a 3.9 cm moderately differentiated adenocarcinoma with invasion through the muscularis propria into pericolorectal tissue and two of 48 lymph nodes were positive for invasion.

Immunohistochemical staining was performed on the specimen for mismatch repair proteins, including MLH1, MSH2, MSH6, and PMS2. All were normal. Given her age, she met revised Bethesda guidelines for genetic testing; her mother's personal history of breast cancer at age 27 also met National Comprehensive Cancer Network (NCCN) criteria for genetic testing. Testing for MSH2, MSH6, MLH1, PMS2, EPCAM, BRCA1, BRCA2, CHEK2, ATM, PALB2, and 26 additional genes were all negative.

Staging was completed with non-contrast CT chest which did not demonstrate metastatic lesions. Final staging resulted in diagnosis of Stage III (pT3, pN1, M0) moderately differentiated adenocarcinoma. She elected to start adjuvant treatment with leucovorin calcium, 5-fluorouracil, and oxaliplatin (FOLFOX) for six months. According to the American Cancer Society (ACS), surveillance for this patient should include follow-up visits with a history and physical examination and CEA antigen testing every three to six months for two years and then every six months up to five years. A CT scan of the chest, abdomen, and pelvis is recommended annually for five years. A colonoscopy should be performed one year post-surgery. Colonoscopy should be repeated in one year if positive for advanced adenoma or in three years if negative. If no advanced adenoma is found on the second interval colonoscopy, screening then can be repeated every five years.[11] Screening for other cancers, such as breast cancer, continue according to regular guidelines.

Although genetic testing was negative in this patient, first-degree relatives of an individual with CRC are at increased risk of acquiring familial CRC. Risk increases with multiple first-degree relatives with CRC or a first-degree relative who developed CRC younger than 50 years.[12] The National Comprehensive Cancer Network (NCCN) recommends that first-degree relatives of patients with CRC or advanced adenoma be offered screening with colonoscopy at age 40 or 10 years prior to the first-degree relative's diagnosis and repeat screening every five years.[13] Thus, this patient's first-degree relatives should be offered screening colonoscopy at age 25.

Discussion

CRC incidence in individuals younger than 50 is projected to double by 2030.[10,14-16] Currently, 10% of all CRC cases are diagnosed in those younger than 50. Common underlying etiologies of CRC in patients under 50 are hereditary cancer syndromes, including Lynch syndrome, familial adenomatous polyposis (FAP), Peutz-Jeghers syndrome, juvenile polyposis, Cowden disease, and Li-Fraumeni syndrome.[17]

The Amsterdam or Modified Amsterdam criteria are the gold standard guidelines when deciding whether to test a patient who does not yet have CRC for Lynch syndrome, the most common of the hereditary syndromes.[18] The Amsterdam I criteria include family history of young-onset (less than age 50) CRC plus three cases of CRC in two successive generations.[19,20] One of these three cases must be in a first generation relative of the other two cases.[21] The Modified Amsterdam criteria require three or more relatives on the same side of the family with histologically verified Lynch-associated tumors, Lynch-associated cancers involving at least two generations, and a minimum of one cancer diagnosed before the age of 50 years. The sensitivity of the Modified Amsterdam criteria is 22%; the specificity is 98%.[22] The revised Bethesda criteria (Table 1) are used for patients who present with earlier-onset CRC; these criteria advise when to test tumors of individuals for microsatellite instability (MSI).

Table 1. The revised Bethesda guidelines for testing colorectal tumors for microsatellite instability (MSI).

Tumors should be tested for MSI in the following situations:	
1.	Colorectal cancer diagnosed in a patient who is less than 50 years of age.
2.	Presence of synchronous, metachronous colorectal, or other HNPCC-associated tumors, regardless of age.
3.	Colorectal cancer with MSI-H-like histology diagnosed in a patient who is less than 60 years of age.
4.	Colorectal cancer diagnosed in a patient with one or more first-degree relatives with an HNPCC-related tumor, with one of the cancers being diagnosed under 50 years.
5.	Colorectal cancer diagnosed in a patient with two or more first- or second-degree relatives with HNPCC-related tumors, regardless of age.

The sensitivity of one of the revised Bethesda guidelines for diagnosis of Lynch syndrome is 82%; the specificity is 77%.[23-25]

In patients with Lynch syndrome, routine screening colonoscopies are recommended beginning at age 20 to 25 years, or two to five years before the earliest CRC diagnosis in the family — whichever comes sooner.[22,26,27] In patients with FAP, colonoscopies should begin at age 10 to 15 years. This should be done annually until colectomy is performed. Timing of colectomy should be based on size, shape, and distribution of polyps plus polyp histology found during the first colonoscopy.[28,29]

Yet, prevalence of mutations associated with hereditary cancer syndromes range from only 5% to 35% in CRC patients under 50.[30-36] A recent study involving 430 patients under age 50 with CRC found that only 20% (85 patients) had germline mutations associated with a cancer syndrome.[18] Another study found that in patients less than 35 with CRC, only 35% tested positive for hereditary cancer syndromes.[35] Hypothesized reasons for increased sporadic CRC in young patients include increased prevalence of a sedentary lifestyle,[16] a diet high in red meat or low fiber, and smoking/alcohol use.[37] One study found that introducing a high-fat, low-fiber diet in rural South Africans triggered inflammation and proliferation in colonic mucosa within just 2 weeks; conversely, introducing a high-fiber, low-fat diet to African Americans demonstrated decreased bile acid synthesis and decreased inflammation.[38] Other hypotheses include an increase in antibiotic use and Cesarean delivery, both of which alter gut flora.[39]

In response to this increase in cases in younger individuals, the American Cancer Society decreased its recommended age to begin screening in patients with average CRC risk via colonoscopy from 50 to 45 in 2018.[6] The USPSTF on Colorectal Cancer still gives screening from age 50 to 75 years old a grade A recommendation, whereas screening from ages 45 to 49 is a grade B recommendation.[8] However, one study showed a mean age of diagnosis of 41 in patients who were diagnosed before age 50. This would mean that, even with this change in recommendation, a large number of CRC diagnoses will still be missed.[40] At diagnosis, the majority of these cases are already stage III or IV.[40,41] Lymph node invasion, which represents a significant factor affecting both staging and prognosis, has a higher incidence in patients less than 40 years old with early-stage CRC.[42] Similarly, CRC tumors in this same age group tend to be more aggressive and present with a higher histological grade.[43]

This case highlights an example of sporadic CRC in a patient under the age of recommended routine screening, per all society guidelines. Despite the association between hereditary cancer syndromes and early-onset CRC, many — perhaps even the majority — of patients with CRC under age 50 are sporadic cases.

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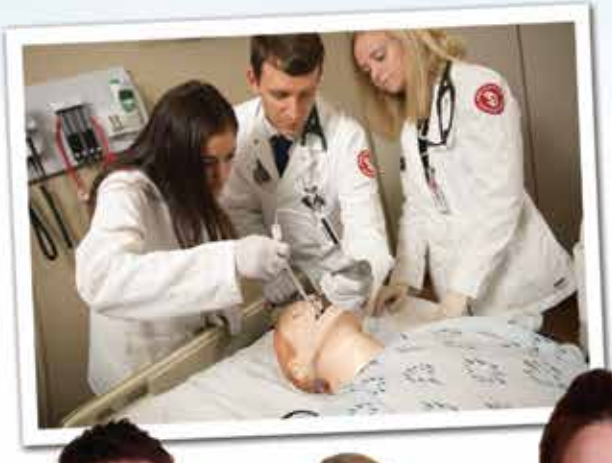

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A Case of Neurocysticercosis in Rural South Dakota

Luke Merrill, MS IV; Autumn Hurd, MS IV; Layne Hohn, MS IV; and Peter Reynen, MD

Abstract

Neurocysticercosis (NCC) is a rare, potentially life-threatening parasitic infection endemic in many developing countries where pig farming and pork consumption are popular. The rates of neurocysticercosis could increase in the U.S. due to the influx of immigration from Central and South America, sub-Saharan Africa, and parts of Asia. Careful evaluation, diagnosis, and treatment is needed to prevent complications from the disease. We present a case of neurocysticercosis which presented as an unresponsive adult female in a rural South Dakota healthcare facility.

Introduction

Neurocysticercosis is a helminthic infection of the nervous system. Infection begins during the larval stage of the pork tapeworm *Taenia solium*, the most common nervous system helminthic infection in humans.[1] *Taenia solium* is endemic in most developing countries, but cases of NCC can also be seen in developed countries due to travel and immigration.[2]

The life cycle of *Taenia solium* involves an intermediate host harboring the parasitic larvae in its tissues, and a definitive host carrying the adult tapeworm in its intestines. Pigs are the most common intermediate host and humans as the sole definitive host.[3,4] If consumed, the parasite can penetrate through the intestinal wall and lodge in any organ, including the brain.[3,4]

The clinical manifestations of NCC are widely variable and depend on the number, location, size, and stage of the infecting parasitic larvae, as well as the immune/inflammatory response of the infected host.[1] Of importance, parasite location inside the brain parenchyma (intraparenchymal NCC) has shown to be less aggressive and is associated with headaches, seizures and epilepsy, while infections locating in the ventricles or subarachnoid spaces (extraparenchymal NCC) are associated with intracranial hypertension and constitute a major driver of mortality and morbidity.[5-13] Parenchymal NCC accounts for one-third of all epilepsy cases within endemic regions, therefore representing the most important cause of acquired epilepsy worldwide.[14] Extraparenchymal NCC may cause mass effect and hydrocephalus due to blockage (ventricular cysts) or direct compression of the structures related to cerebral spinal fluid (CSF) flow.[15-18]

This report examines the presentation, treatment, and outcome of a patient that was diagnosed with neurocysticercosis in rural South Dakota.

Case Presentation

A 33-year-old female from Nicaragua living in the U.S. for the past year presented to the emergency department unresponsive. With the help of a translator, family was able to report that she had a cough, headache, and an episode of emesis the day prior to arrival. The following morning, she was unable to be aroused from sleep and so the family alerted EMS. She had a history of migraines treated with ergotamine and caffeine. No other past medical history or diet was known.

The patient was initially treated en route by EMS with 0.4 mg narcan. Upon arrival to the facility, additional narcan was given with no response. Physical exam revealed no signs of trauma. Spontaneous, coarse breath sounds were auscultated bilaterally with no apparent wheezing. Heart rate was mildly accelerated at 110 beats per minute but auscultation of the heart revealed a regular rhythm with no significant murmurs, rubs, or gallops. Distal pulses were palpable and strong in all four extremities. No posturing or focal neurological deficits were noted. Pupils were constricted and nonreactive She moaned with stimulation, did not open her eyes, and localized pain resulting in a Glasgow coma scale (GCS) of 7. Labs showed a white blood cell count of 16.5 (n: 4-11) with an absolute neutrophil count of 17.5 (n: <7.7) and no eosinophilia. She had a mildly elevated CRP at 2.1 (n:< 0.5) with normal serum creatinine, LFTs, lactate, and urinalysis. Toxicology screen was negative. Arterial blood gases showed a pH of 7.27 (n: 7.35-7.45), pCO2 of 54 (n: 35-45), pO2 of 83 (n: 75-100), and HCO3 of 25 (n: 21-27). Chest X-ray revealed mild bilateral upper lobe infiltrates. Due to the patient's

encephalopathy, leukocytosis, and infiltrates noted on chest X-ray, she was started on metronidazole and cefepime for suspected aspiration pneumonia. A head CT was performed and showed mild to moderate obstructive hydrocephalus with dilation of the lateral and third ventricle and periventricular edema. It also showed multiple scattered bilateral cerebral cortical and gray-white matter junction hyperdenisities (Figure 1). After the head CT, antibiotics were switched to ampicillin and vancomycin with the addition of 10 mg dexamethasone for meningitis coverage. The patient was intubated to secure an airway and noted to have otherwise stable vital signs.

The patient was transferred by air to a tertiary care center where she was further evaluated and treated. An external ventricular drain placed with an opening pressure of 27 cmH2O (n: <20). An MRI of the head was completed and showed approximately 13 mixed intensity and peripherally enhancing lesions scattered within portions of the cerebral hemispheres and the subependymoma within the temporal horn of the right lateral ventricle. A scolex within a cyst was identified in the right temporal lobe making the diagnosis of NCC highly likely. Two cysts remained viable, while the other eleven were likely old/calcified (Figure 2). MRI of the spine was completed and found no lesions. Cerebral spinal fluid was taken and analysis showed neutrophilic pleocytosis, normal glucose, and normal protein. CSF cultures were negative for acid fast bacilli, fungus, and no gram stain organisms were identified. Diagnosis was confirmed when CSF tested positive for IgG to cysticercus via Western Blot. Complete meningitis panel was negative. The following day, the patient became responsive and was successfully extubated. A repeat head CT at this time showed resolved hydrocephalus.

The patient's initial presentation was thought to be due to elevated ICP from obstructive hydrocephalus or seizures. She was started on levetiracetam and continued steroids for 72 hours. After meningitis was ruled out, antibiotics were switched to ceftriaxone and doxycycline for continued treatment of the suspected aspiration pneumonia. Additional testing for tuberculosis and strongyloides was completed due to the high prevalence of these diseases in Nicaragua. Once the patient was stabilized and initial testing was complete, ophthalmology was consulted to assess ocular involvement. After no ocular involvement was found, the patient was started on albendazole and praziquantel. She remained in critical care for monitoring while taking these medications for ten days where she remained stable. While on the antiparasitic therapy, her liver enzymes became elevated peaking at day ten of therapy with an alanine aminotransferase of 1202 (n: < 25) and aspartate aminotransferase of 271 (n: <39). She developed a drug induced liver injury during her treatment but at this point had finished the full regimen. She was discharged on a steroid taper and will stay on anti-seizure medications. Follow-up labs three weeks later showed normal liver enzyme levels.

Figure 1. Head CT showing mild to moderate obstructive hydrocephalus with dilation of the lateral and third ventricle and periventricular edema. It also showed multiple scattered bilateral cerebral cortical and gray-white matter junction hyperdenisities.

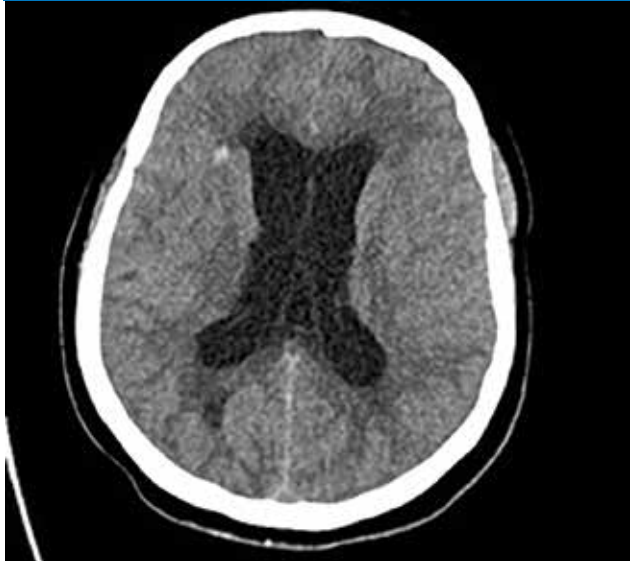
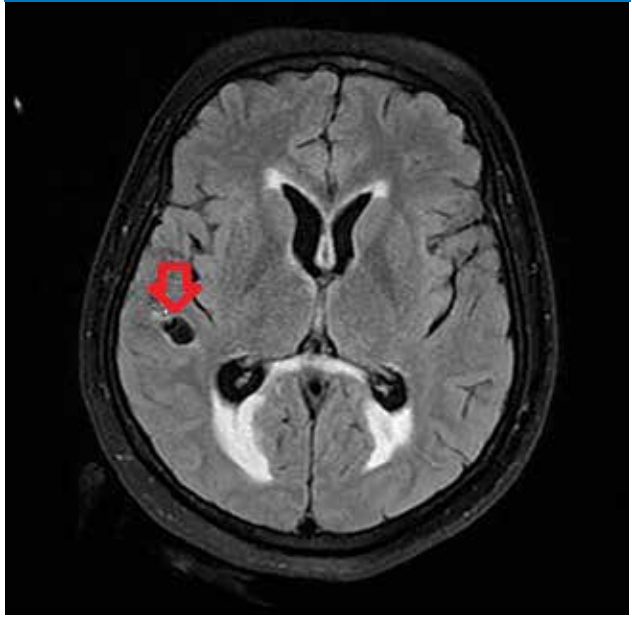


Figure 2. MRI showing a scolex (see arrow) within a cyst identified in the right temporal lobe.



Two months after discharge, the patient followed up with her primary care physician. She had difficulty remembering the incident and reported increased anxiety and headaches since but no alarm symptoms. She also followed up with infectious disease at this time who recommended an MRI six months after discharge and to continue anti-seizure medications for two years.

Discussion

Taenia solium has a world wide distribution but is endemic in many low-income countries that commonly raise pigs.[19] It is not considered endemic in the U.S. However, a rise in immigration from endemic countries could increase the prevalence of disease and carriers of the parasite in the U.S.[9] In a large-scale study looking at NCC cases in the U.S. between the years of 1991-2008, 92% of cases were found in young adult Latinos.[20] Because of this potential rise in cases, it is important to review the clinical manifestations and management of the disease.

Clinical manifestations are nonspecific and can vary from case to case with some cases being asymptomatic while others have life-threatening disease. Most commonly, parenchymal occupying cysts present with focal or generalized seizures (66%), hydrocephalus (16%), and signs of increased intracranial pressure such as headaches, dizziness, emesis, and altered mental status (15%).[21] Extraparenchymal occupying cysts may present similarly but with a higher mortality, up to 20% without appropriate treatment.[21] Ophthalmic cysticercosis may present with vision loss, uveitis, retinitis, and extraocular muscle weakness.[22]

Neuroimaging is the primary method of diagnosing neurocysticercosis. CT and MRI can be used to identify inflammation, number, location, and stage of the cysts.[19] Visualization of scolices within cysts and visualization of subretinal parasites on fundoscopic exam are considered absolute criteria for diagnosis.[1] Major neuroimaging criteria findings include cystic lesions without a scolex, enhancing lesions, multilobulated cysts, and calcifications.[23] Confirmation of the diagnosis from these findings can be established by resolution of the cysts after cysticidal drug treatment, spontaneous resolution of a single enhancing lesion, and migrating ventricular cysts on subsequent imaging.[23] Minor neuroimaging criteria include leptomeningeal enhancement and obstructive hydrocephalus.[19,23]

Clinical manifestations and epidemiological exposure can also aid in the diagnosis of neurocystercercosis. Major criteria for this include detection of anticysticercal antibodies or cysticercal antigens, cysticercosis outside of the nervous system, and household contact with known to have it. Minor criteria signs and symptoms of neurocyscercosis and current or prior residence in an endemic region. A definitive diagnosis is made with any absolute criteria, two major neuroimaging criteria plus clinical/epidemiologic criteria, or one major neuroimaging criteria plus two clinical/epidemiologic criteria, and excluding other pathology.[1]

Symptomatic treatment for seizures, headaches, and increased intracranial pressure is an important initial step in management. Especially with this patient's concomitant pneumonia, seizure prophylaxis becomes more important as her seizure threshold may be lowered. Seizure management depends on clinical presentation, types/degree of involved tissue, and stages of cysts.[9] Patients who present with seizures must first be treated with anti-seizure medications for both single seizure episodes and recurrent seizures. Monotherapy is commonly started with levetiracetam as this has less severe adverse effects, but phenytoin and carbamazepine are commonly used as well.[8] This treatment needs to be given at for at least two years after the last seizure with slow tapering after that.[18] Headaches are treated with standard analgesics as first line.[8] However, most of the symptoms are caused by the host inflammatory response so steroids such as dexamethasone are critical.[8,18]

Hydrocephalus, as in this patient, can occur in extraparenchymal NCC and less commonly in parenchymal disease. Intracranial hypertension is a life-threatening symptom as well. Both can be due to obstruction but inflammation in itself can cause hydrocephalus if not promptly treated.[9] Extraparenchymal NCC are more difficult to treat and generally have a worse prognosis.[8,9] Due to the obstructive nature, treatment requires removal of the obstructing cysticerci via neuroendoscopy with the potential for shunt placement.[8] Neurosurgery did not believe this patient needed cyst removal to relieve the obstruction so a drain was placed instead.

Anthelmintic treatment is not an emergent treatment and is held until the patient is stable and their symptoms are under control. Definitive treatment may exacerbate symptoms and cause harm to the patient if not properly monitored for inflammatory responses. The antiparasitic regime depends on the location of the viable cyst. Albendazole and praziquantel are the antiparasitics used.[9,18] The length of time for treatment varies on form as well, from three days (for those with one viable cyst) up to two weeks.[18]

Conclusion

The rate of neurocysticercosis cases have the potential to rise with increasing immigration to the U.S. Recognition and careful evaluation are necessary to guide appropriate treatment and prevent worsening of the disease. Thankfully, this parasite is not transmitted from person to person. The patient presented in this case was quickly and accurately diagnosed and underwent treatment that minimized the risk of complications due to neurocysticercosis. She is scheduled for follow up brain imaging at a later date.

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A Case Presentation of Metastatic Hepatic Epithelioid Hemangioendothelioma: A Rare Vascular Tumor

Sanyogita Chandra, MD, MS; and Heidi McKean, MD

Abstract

Epithelioid hemangioendothelioma (EHE) is an uncommon vascular tumor that can present in various organs, including the liver. Hepatic EHE (HEHE) may showcase with metastases at initial presentation, as patients have vague symptoms such as right upper quadrant pain leading to the risk of delayed diagnosis. There is no standard treatment. Fortunately, prognosis is good. This remains true for some patients with metastatic disease who are not being actively treated. In this report, we present a unique case of metastatic HEHE in a 65-year-old Caucasian male who has not received treatment and continues to remain in stable condition after his initial diagnosis three years ago.

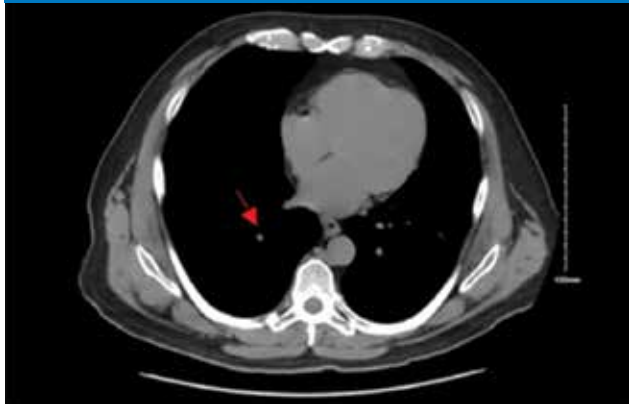
Introduction

Epithelioid hemangioendothelioma (EHE) is a rare vascular tumor that is composed of epithelioid and histiocytoid vascular endothelial cells that are wrapped in a myxoid or fibrotic stroma.[1] EHE can occur in the lungs, soft tissue, head and neck, bones, and many other organs. Hepatic EHE (HEHE) is an exceedingly rare vascular tumor with an estimated incidence of one to two cases in every 1 million people. It most commonly occurs between the ages of 30-50 years old with a predominance in females, with the female to male ratio being 3:2.[2] Patients can present with right upper quadrant pain, fatigue, nausea, vomiting, and weight loss; however, 25-40% of patients are asymptomatic. Unfortunately, 37% of patients will have distant metastases at time of diagnosis.[2]

Case Report

A 65-year-old Caucasian male presented to his local emergency department with abdominal pain and left-sided tenderness. Physical exam found mild tenderness to deep palpation in the mid-epigastric region without rebound or guarding. Thorough work-up including a computed tomography (CT) scan of the abdomen with and without contrast was completed, which found mild pancreatitis and diverticulosis without diverticulitis. He was managed appropriately for pancreatitis. Unfortunately, the patient continued to have abdominal pain and was referred to gastroenterology a month following his initial visit to the emergency department. An esophagogastroduodenoscopy (EGD) showed evidence of Barrett's esophagus, which the patient had been diagnosed with years prior, as well as mild gastritis in the antrum. A colonoscopy found tubular and tubulovillous adenomas but was otherwise unremarkable. To rule out pancreatitis, a repeat CT scan of the abdomen with and without contrast was conducted. Findings included multiple pulmonary nodules measuring up to 6 mm (Figure 1). There was also a <5 mm flash filling hemangioma in the left lobe of the liver. A six-month follow-up CT scan of the chest without contrast was recommended and showed multiple pulmonary nodules, some calcified, favoring the diagnosis of granulomatous disease. There was no evident mediastinal lymphadenopathy. The patient continued to have occasional abdominal pain. Repeat CT scan of the chest a year later showed an increase in the size of a pulmonary nodule to 10 mm, as well as other enlarged pulmonary nodules, now concerning for possible malignancy. Pulmonology was consulted and a positron emission tomography (PET) CT

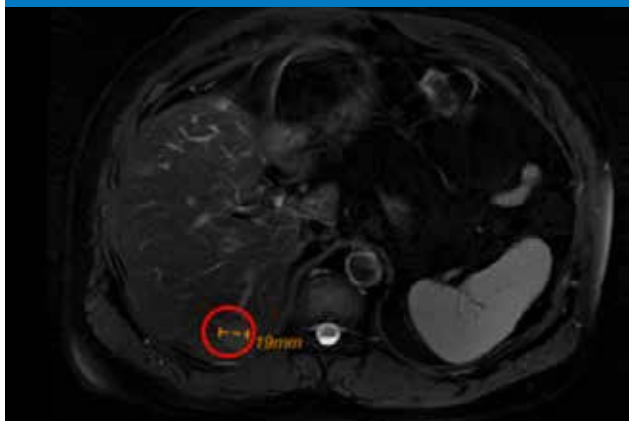
Figure 1. CT scan of the abdomen showcasing multiple pulmonary nodules. The red arrow points to a 6 mm nodule.



scan and pulmonary function tests were recommended. PET CT scan displayed mild hypermetabolic activity to some of the pulmonary nodules. PET CT scan also exhibited mild hypermetabolic activity to the base of the tongue and parts of the upper to mid esophagus. EGD was conducted and biopsied tissue was negative for dysplasia. Pulmonary function testing was unremarkable. Pulmonology noted that the 10 mm pulmonary nodule was too small for fine-needle aspiration. Follow-up CT scans of the chest every six months to every year were recommended.

The patient received a CT scan of the abdomen with and without contrast five years later after presenting to his local emergency department with altered mental status and concern for a complicated urinary tract infection (UTI). The CT scan noted enlargement of multiple pulmonary nodules, as well as an indeterminate hypoattenuating mass in the posterior segment of the right hepatic lobe measuring approximately 18 mm in size. Ultrasound of the right upper quadrant showed several small hypodensities as well as one indeterminate hypoattenuating mass in the posterior segment of the right hepatic lobe that measured 18 mm in size. CT scan of the chest showed that the largest nodule in the right middle lobe of the lung grew to 12 mm after previous CT scan showed it at 10 mm. Repeat pulmonary function testing was normal. Lab work showed normal complete blood counts, normal rheumatoid factor, normal angiotensin-converting enzyme (ACE), and normal fungal serologies at that time and thus, follow-up was recommended. Three-month follow-up ultrasound of the right upper quadrant showed relatively similar appearance of the hypoechoic masses in the liver as seen previously. Repeat CT scan of the chest six months later showed slightly increased size of pulmonary nodules and two rim-enhancing lesions in the liver that are suspicious for metastatic disease. An MRI of the abdomen and pelvis showed several mildly T2 hyperintense hepatic lesions with the largest lesion being 1.9 cm in size (Figure 2). Liver is not cirrhotic but hepatic steatosis was noted. Liver biopsy results stated, "proliferation of atypical cells sometimes forming small lumens in a myxoid background." The tumor cells were positive for CD31, CD34 and ERG. There was also focal weak staining for CK7, while CDX2 and TTF1 were negative. These findings point to the diagnosis of epithelioid hemangioendothelioma. CT-guided biopsy of liver confirmed the diagnosis of epithelioid hemangioendothelioma. Needle core biopsy of the 12 mm right pulmonary nodule was consistent with metastatic epithelioid hemangioendothelioma.

Figure 2. MRI of the abdomen and pelvis encircled 1.9 cm hepatic lesion.



The patient is feeling well except for fatigue which he attributes to his age of 73. He denies shortness of breath, cough, weight loss, or abdominal pain. The patient's pertinent laboratory values are normal except for a slightly elevated alanine aminotransferase (ALT) to 34 U/L (4-33 U/L). Given that the patient is asymptomatic, he continues to undergo imaging studies every three months to monitor lung and hepatic lesions and follow-up with medical oncology. The lesions remain stable for almost a year after initial diagnosis, when repeat CT scan of the abdomen and pelvis show a new area of low attenuation in the posterior right hepatic lobe concerning for disease progression. He remains asymptomatic and the new lesion is measured at only 7 mm, so the patient is simply monitored. His laboratory values continue to be normal except for a slightly elevated alanine aminotransferase (ALT) to 38 U/L (4-33 U/L). Repeat CT scan six months later shows stable pulmonary nodules and a slight decrease in a previously noted hepatic lesion. Six months later, CT scan shows one new hepatic lesion measuring 0.9 x 0.7 cm and patient continued to be asymptomatic. Pertinent laboratory values were normal. Repeat six-month imaging for the last year has been stable. Given slow progression of the disease, the patient has not been treated.

Discussion

HEHE showcases an infiltrative growth pattern of epithelioid, dendritic, and intermediate cells interspersed in a hyaluronic acid-rich myxoid matrix. These neoplastic cells grow along vascular structures and infiltrate hepatic sinusoids. A dense, mucopolysaccharide, extracellular matrix is often present.[1] Older lesions look sclerotic and calcified. Most patients with HEHE make their initial presentation with disseminated disease, usually in both lobes.[3] Histologically, the tumor

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can be mistaken for metastatic carcinoma, angiosarcoma, hepatocellular carcinoma, or cholangiocarcinoma. Immunohistochemical staining for HEHE is positive for CD31, CD34, and Factor VIII. This staining pattern is similar to angiosarcoma. However, HEHE is generally less aggressive than angiosarcoma.[1] Furthermore, HEHE also stains positive for vimentin, type IV collagen and D2-40 to differentiate it from other angiomatous lesions. ERG is a vascular marker that is often also expressed in HEHE.[2]

The most common abnormal laboratory value is an elevation in alkaline phosphatase. However, other liver enzymes, including γglutamyl transpeptidase, aspartate aminotransferase (AST), and ALT can also be elevated.[1] One study finds that the most common presenting symptoms include right upper abdominal pain (48.6%), hepatomegaly (20.4%), and weight loss (15.6%).[4] Some patients are asymptomatic. HEHE on magnetic resonance imaging (MRI) or computed tomography (CT) features include peripheral location of the nodules, the contraction of the capsule, and the tendency of multiple foci to merge.[2]

There is no gold standard therapy for treatment of HEHE. Liver transplantation has been found to be effective; however, this remains a treatment option for patients in critical condition. A study of 149 patients with HEHE that received a liver transplant were analyzed for post-liver transplant HEHE reoccurrence. This study calculated a HEHE-liver transplant score based on macrovascular invasion, transplant wait time of 120 days or less, and pathological invasion of hilar lymph nodes. These values were pertinent because of their calculated hazard ratios (HR). The HR for macrovascular invasion was 4.8 ($P < 0.001$), 2.6 for liver transplant wait time of 120 days or less ($P = 0.01$), and 2.2 for hilar lymph node invasion ($P = 0.03$). These elevated hazard ratios indicate significant risk factors for reoccurrence. Disease-free survival (DFS) rates were then found using the Kaplan-Meier method. Patients with a score of two or less had DFS of 93.9% as compared to a DFS of 38.5% for patients with a score of 6 or higher ($P < 0.001$).[5] Surgical resection, although a treatment option, was found to be less useful in HEHE. Patients with tumors greater than 10cm who received a liver resection had a worse prognosis as compared to those who received a liver transplant.[3] Two small studies on patients with surgical resection for HEHE found the following: in the first study of six patients, three had disease relapse. In the second study of seven patients, five had disease relapse. It is hypothesized that the disease reoccurrence is due to the upregulation of hepatic growth factors after surgical resection.[6] Observation for a median duration of 15.5 months with routine examination was observed by 18 patients and 88.9% of them had progressive disease.[6] Another study notes that given the vascular origin of HEHE, treatment with vascular endothelial growth factor (VEGF) inhibitors, including sorafenib, pazopanib, bevacizumab and others can play a role. A study found that patients taking pazopanib, a VEGF inhibitor, had intratumoral changes on follow-up CT and although there is no evident tumor contraction or calcification, there is a change in the tumor density. This should be regarded as a positive treatment response. Furthermore, another study notes that the combination of bevacizumab and cell-cycle inhibitor capecitabine achieved a curative effect in one patient.[2]

HEHE has an overall good prognosis when compared to other malignant liver tumors. A study found that 50% of the patients survived greater than five years without treatment, and the presence of metastasis did not change the survival rate.[2] This is also shown with the presented patient in this case report.

Conclusion

HEHE is a vascular tumor that can present with right upper quadrant pain, fatigue, and other vague symptoms. It can also arise in asymptomatic patients. The tumor itself is rare and oftentimes, patients simply require close follow-up rather than treatment. Patients with rapid progression of the tumor can be treated with liver resection, liver transplant, and medications such as VEGF inhibitors. Overall, it has a good prognosis, regardless of metastases present.

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Clinical Practice Update: Achilles Tendon Rupture

Hunter A. O'Connor, MS IV; Luke W. Adams, MD; and Nathan Wm. Skelley, MD

Abstract

Achilles tendon rupture is a common injury. It most often occurs in middle aged men who participate in recreational sports. The injury classically presents with a loud popping noise and immediate pain and weakness of the lower extremity during actions such as jumping or running. The diagnosis is made clinically, but an MRI is often obtained for confirmation of rupture and to aid in surgical planning. Treatment is either operative, with open or minimally invasive approaches, or non-operative, with functional bracing or plaster casting. Surgical treatment was preferred for much of the 20th century, but non-operative treatment has gained significant favor in the past 15 years as new evidence has demonstrated similar long-term outcomes to surgery. Neither treatment option is currently considered superior to the other in all cases. Surgery is associated with a risk for surgical complications and is, therefore, often a poor option for the elderly and those with significant comorbidities. Non-operative management is associated with an increased risk for re-injury which is often undesirable for young and highly active patients. Ultimately, the goals and priorities of each individual patient should guide the decision of which treatment option to pursue.

Epidemiology and Pathophysiology

Achilles tendon rupture is one of the most common tendon injuries in the world, with an incidence between five and 35 per 100,000 people.[1,2] This injury is usually caused by rapid tensioning of the Achilles tendon with movements such as jumping, turning, or sprinting, and most commonly occurs in men aged 30 to 40 during participation in recreational sports.[3,4] Men are up to eight times more likely than women to develop an Achilles rupture.[2] Rupture is especially common in patients with a history of competitive athletics, with around 8.3 percent experiencing this injury over the course of their lifetime.[5] The current understanding of the pathophysiologic mechanism of Achilles tendon rupture is related to the hypovascular nature of the tendon, especially in the location 2-6 cm proximal to the calcaneus.[6] Poor blood supply in this area leads to decreased healing capacity and allows repetitive microtrauma to weaken the tendon over time.[6] The chronically damaged tendon is then susceptible to rupture during an acute increase in physical activity.

Diagnosis

The diagnosis can be made clinically based on history and physical examination. The classic presentation involves an audible popping sound during a sport that involves jumping or turning such as basketball or soccer. There is usually associated pain, and weakness of the affected leg, leading to difficulty ambulating. Patients often report a sensation that something had struck the back of their lower leg.[6] Characteristic physical examination findings include a palpable defect in the tendon, plantar flexion weakness, and a positive Thompson test. The Thompson test, or squeeze test, is the most sensitive and specific examination finding for complete Achilles tendon rupture.[7] The test is performed with the patient in the prone position with their feet hanging off the end of the bed. The physician squeezes the posterior calf and watches for plantar flexion of the foot. A lack of plantar flexion with this maneuver is considered a positive test and is indicative of tendon rupture. A common misconception is that patients will be unable to plantar flex the foot if a complete rupture is present. Although the motion is weakened, most patients retain their ability to plantar flex the foot through the action of accessory muscles such as the flexor digitorum and peroneal muscles. Rarely, patients are even able to ambulate. These findings can lead physicians to mistakenly conclude that no rupture is present and are likely a significant contributor to the greater than 20% of tendon ruptures that are misdiagnosed.[7] Therefore, a high level of clinical suspicion in high-risk individuals reporting lower leg pain and a thorough physical examination with a Thompson test is required. MRI is usually also obtained to confirm tendon rupture and guide surgical planning.

Treatment

The treatment of acute Achilles tendon rupture is controversial. An Achilles rupture can either be treated

operatively or non-operatively, and there is ongoing discussion and research on which method is superior.[8] Achilles tendon ruptures have been treated non-operatively for hundreds of years. Successful non-operative techniques using soft bandages to hold the foot in plantar flexion were described as early as 1781 by Alexander Monro.[9] The first surgical repair was performed in 1888, but operative management did not gain popularity until the 1920s. The first study comparing the two treatment options was published in 1929 by Quenu and Stoinovitch.[9,10] They found that both options were equally effective, but they favored the surgical option. Their study was followed by nearly a century of debate and hundreds of studies on the topic.

Surgical repair involves reapproximating the severed ends of the tendon with sutures. Traditionally, this is performed through an open incision at the level of the tendon defect. Recently, several less invasive techniques incorporating multiple smaller incisions or percutaneous repair have been developed. Studies have demonstrated that minimally invasive techniques may have lower incidence of infections and other wound complications than open repair.[8,11,12] However, there is some evidence that percutaneous repair leads to higher risk of sural nerve injury.[8]

Non-operative management traditionally involves immobilizing the foot in plantar flexion with a solid plaster cast. The cast is initially placed with the foot in maximal plantar flexion. By replacing the cast every few weeks, the degree of plantar flexion is progressively decreased, and the foot is returned to a neutral position. Patients are usually non-weight-bearing on the foot for around 8 weeks.[13] Prolonged non-weight-bearing can interfere with activities of daily living, and may also lead to calf atrophy, joint stiffness, and increased risk of deep venous thrombosis. In response to these perceived problems, newer functional bracing techniques have been developed that allow patients to begin weight bearing immediately after injury. These functional bracing techniques utilize a removable walking boot with adjustable wedges beneath the heel that hold the foot in plantar flexion while allowing the patient to ambulate nearly normally. Functional bracing has gained significant popularity in recent years. However, a recent large randomized controlled trial found no long-term difference in functional outcomes, quality of life, or risk of re-rupture between functional bracing and traditional plaster casting.[13] Interestingly, the trial did find that functional bracing had a statistically significant but clinically insignificant functional and quality of life advantage at eight weeks post-injury.[13]

For much of the 20th century, surgery was favored over non-operative treatment because it was believed to have better functional outcomes and lower re-rupture rates. However, non-operative treatment has gained increasing favor over the past 15 years as newer evidence demonstrating similar outcomes between the two options has emerged. Following the publication of several large randomized controlled trials in the mid 2000's, the number of surgical repairs performed dropped by nearly 50%.[2,14-16] As a result, less than 15-20% of all ruptures are currently treated operatively.[2, 14-16]

The advantages and disadvantages of each treatment option have been further clarified with the publication of several large meta-analyses in the past 10 years. The main benefit of surgery is a 1.5-8.5% lower risk of re-rupture than non-operative treatment.[3,8,11,17-19] The main disadvantage of surgery is the risk of surgical complications including infection and sural nerve injury.[3,8,11,17-19] According to a recent large randomized controlled trial, the functional outcomes of both treatment options appear to be equal 12 months after surgery.[20] However, two meta-analyses have found that surgical treatment may allow slightly faster recovery of function and return to pre-injury level of activity.[17,19] A few small randomized controlled trials have also found that surgical treatment has faster and greater isokinetic strength recovery than non-operative treatment.[21,22]

As neither operative nor non-operative management have been demonstrated to be superior to the other, the currently accepted best practice is to tailor treatment decisions based on the unique goals and risk factors of each individual patient. Elite athletes and young, highly active individuals wishing to return to competitive sports will likely benefit most from surgical intervention, as there is decreased risk of re-injury as well as slightly faster and greater strength recovery.[21] Other specific indications for surgery include prolonged tendon rupture for greater than six to eight weeks, re-rupture, and a large tendon gap.[23] Non-operative management is more likely to be the superior treatment option for patients who are older, and less active, and for those with significant comorbidities. Avoiding surgical complications is of greater importance in this group and there are similar long term functional outcomes between patients treated operatively and non-operatively. As most Achilles ruptures occur in non-professional athletes above the age of 30, the majority of ruptures are treated non-operatively.[2,16] Ultimately, individual patient goals and tolerance for certain risks should be considered when deciding which treatment option to pursue.

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Conclusion and Takeaways

Achilles tendon rupture is a common injury that can be diagnosed clinically based on history and a positive Thompson test. Treatment is either operative, with open or minimally invasive approaches, or non-operative, with functional bracing or plaster casting. Minimally invasive surgical techniques may have fewer wound complications than open techniques. Non-operative management with functional bracing allows for early ambulation with equivalent long-term outcomes to traditional plaster casting. Both operative and non-operative management are viable treatment options. The goals and priorities of each individual patient should guide the decision of which treatment option to pursue.

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Anemia In Pregnancy: Examining Missed Diagnoses and Racial Disparities in the Upper Midwest

Tess L. Weber, MPH; Aarabhi Gurumoorthy, MPH; Kari A. Bates, MSc; and Ashley Briggs, MD

Abstract

Anemia in pregnancy (AIP) is associated with poor maternal/fetal outcomes. The prevalence of AIP globally ranges from 44-53% and varies drastically depending on maternal race/ethnicity and other factors. Screening and treatment of AIP is disputed. This study is a retrospective review of electronic medical records (EMR) of pregnant adults over three years (2018-2020, inclusive) of Sanford Health, a large healthcare system in the upper Midwest. AIP was determined by either diagnosis or lab values (hemoglobin, hematocrit, and ferritin) overlapping with pregnancy. A missed diagnosis was characterized by confirmed anemia through lab values but lacking a diagnosis of anemia within EMR. A total of 35,498 patients were included in this study, 42.9% were determined to have AIP. Of AI/AN (American Indian/Alaska Native) patients, 58.3% were anemic and 55.1% of Black/African American patients were anemic compared to 40.0% of anemic white patients. Of anemic patients, 81.1% did not have an anemia diagnosis listed in EMR. This study identifies racial and ethnic disparities of AIP among patients in the upper Midwest. In addition, this study highlights the need for improved data integrity within EMR.

Background

Anemia in pregnancy (AIP) is associated with many adverse outcomes for both the mother and the fetus, including peripartum hemorrhage, perinatal mortality, preterm delivery, increased risk of maternal infections, low birthweight, and brain development.[1–3]

According to the U.S. Preventive Services Task Force (USPSTF), there is insufficient evidence to recommend routine screening and treatment for iron deficiency during pregnancy.[4] There are significant gaps in research in the benefits of screening and treating AIP and harms of untreated anemia during pregnancy for both maternal and fetal outcomes.[5,6] Although the stakes of AIP are high for both maternal and fetal outcomes, there is still uncertainty as to whether screening for AIP should be part of routine care. This may be due, in part, to lacking data of the prevalence of AIP in recent years.

The prevalence of AIP globally is estimated to be 38.2%.[7] Rates of AIP in the U.S. range between 44-53%[8] but may be considerably higher depending on maternal race/ethnicity, health history, and sociodemographic characteristics.[9,10]

This descriptive study utilizes retrospective electronic medical records (EMR) over three years (2018-2020, inclusive) to examine the prevalence of anemia in pregnancy in Sanford Health's pregnant population. Sanford Health is one of the nation's largest rural health systems, primarily based in the Upper Midwest, including South Dakota, North Dakota, Minnesota, and five other states. The Sanford Health footprint includes 47 medical centers and 224 clinics. In addition, this study also examines differences in clinician-diagnosed AIP compared to anemia determined retrospectively by EMR lab values.

Methods

This is a retrospective electronic medical record (EMR) review of Sanford Health, a large healthcare system primarily in the upper Midwest. Included in this study were pregnant women (18+ years) during calendar years 2018-2020. Approval was received through Sanford's Institutional Review Board and only study staff had access to the data. Data was pulled through the established process at Sanford Research using an honest broker to ensure accuracy of data and to maintain data confidentiality. Only complete data was used and missing information was removed from the final dataset used for analyses.

Included in this study were all females aged 18 years and older who were pregnant during the three-year study period (2018-2020, inclusive). Patients had at least one Sanford visit during the pregnancy but did not necessarily seek all or any prenatal care at Sanford facilities. Trimesters were

determined using the estimated date of conception: First trimester: 0-93 days; second trimester: 94-186 days; third trimester: 187+ days.

Diagnosis of anemia was determined by a Diagnosis Name or Problem List containing "anemia" (excluding "history of anemia" and "family history of anemia") overlapping with the time from the estimated date of conception to the estimated date of delivery. Anemia based on lab values were also determined using hemoglobin, hematocrit and ferritin (First trimester: hemoglobin <11 g/dL, hematocrit <33 percent; second trimester: hemoglobin <10.5 g/dL, hematocrit <31 percent; third trimester: hemoglobin <10.5 g/dL, hematocrit <33 percent; ferritin <30 at any time point in conjunction with low hemoglobin or hematocrit). A missed diagnosis was characterized by confirmed anemia through lab values but lacking a diagnosis of anemia within EMR.

Chi square was used to determine statistical differences.

Results

Total Population

A total of 35,498 patients were included in this study (Table 1). A total of 15,237 patients were determined to have AIP by either diagnosis or lab values. Most anemia cases were determined through lab values (n = 15,022; 98.6% of total anemic) while 486 (3.2% of total anemic) were determined through diagnosis of anemia alone. There were a total of 12,354 missed diagnoses which accounted for 81.1% of all anemic patients.

The overall median age was 28.7 (SD = 5.2; Table 2) and did not differ significantly between anemic and non-anemic patients.

The majority of patients self-identified as white (28,400; 80.0%). Of anemic patients, 1,870 (12.3%) were American Indian/Alaska Native (AI/AN); however, AI/AN patients only accounted for 9.0% of the entire population. A similar pattern was seen for Black/African American patients. Of anemic patients, 7.6% identified as Black/African American while Black/African American patients only accounted for 5.9% of the entire population.

There was a significant difference in distribution of anemic and non-anemic patients between ethnic groups. Most patients self-identified as non-Hispanic (33,004; 93.0%). Of anemic patients, 14,126 (92.7%) were non-Hispanic and 1010 (6.6%) were Hispanic/Latino.

Missed Diagnoses

Of anemic patients, only 486 had an anemia diagnosis charted within EMR (Table 3). An additional 12,354 were

Table 1. Anemia status.

Anemia status	n=35,498	Percent of anemic n=15,237	Percent of total population (N=35,498)
Anemia (labs)	15,022	98.6%	42.3%
Anemia (diagnosis)	486	3.2%	1.4%
Total anemic (labs or diagnosis)	15,237	100.0%	42.9%
Missed anemia diagnosis	12,354	81.1%	34.8%

Table 2. Demographic variables comparing non-anemic versus anemic patients.

Demographics	**Non-Anemic** n=20,261	**Anemic** n=15,237	**Total** n=35,498	P-value
Age (mean, SD)	28.6 (5.2)	28.5 (5.5)	28.6 (5.3)	
Race, n (%)				<0.0001
American Indian/Alaska Native	1,336 (6.6%)	1,870 (12.3%)	3,206 (9.0%)	
Asian/Native Hawaiian/Pacific Islander	471 (2.3%)	449 (3.0%)	920 (2.3%)	
Black/African American	940 (4.6%)	1,155 (7.6%)	2,095 (5.9%)	
White	17,041 (84.1%)	11,359 (74.6%)	28,400 (80.0%)	
Unknown/missing	473 (2.3%)	404 (2.7%)	877 (2.5%)	
Ethnicity, n (%)				<0.0001
Hispanic/Latino	1,180 (5.8%)	1,010 (6.6%)	2,190 (6.2%)	
Non-Hispanic	18,878 (93.2%)	14,126 (92.7%)	33,004 (93.0%)	
Unknown/missing	203 (1.0%)	101 (0.7%)	304 (0.9%)	

Table 3. Demographic variables comparing diagnosed anemia versus missed anemia diagnosis.

Demographics	Diagnosed anemia n=486	Missed anemia diagnosis n=12,354	Total n=12,840	P-value
Age (mean, SD)	28.2 (5.3%)	28.5 (5.5)	28.5 (5.5)	
Race, n (%)				<0.0001
American Indian/Alaska Native	49 (10.1%)	1,454 (11.8%)	1,503 (11.7%)	
Asian/Native Hawaiian/Pacific Islander	14 (2.3%)	334 (2.7%)	348 (2.7%)	
Black/African American	59 (12.1%)	807 (6.5%)	866 (6.7%)	
White	345 (70.1%)	9,448 (76.5%)	9,793 (76.3%)	
Unknown/missing	19 (3.9%)	311 (2.5%)	330 (2.6%)	
Ethnicity, n (%)				0.76
Hispanic/Latino	34 (7.0%)	1,812 (6.6%)	846 (6.6%)	
Non-Hispanic	450 (92.6%)	11,462 (92.8%)	11,912 (92.3%)	
Unknown/missing	2 (0.4%)	80 (0.7%)	82 (0.6%)	

identified via lab values alone and were classified as a missed diagnosis. There was no significant difference in age between anemia diagnoses and missed diagnoses. White and AI/AN patients had slightly higher percentages of missed anemia diagnoses compared to diagnosed anemia (11.8% vs. 10.1% and 76.5% vs. 71.0%, respectively). Conversely, Black/African American patients had higher percentages of diagnosed anemia versus missed diagnoses (12.1% vs. 6.5%). There were no significant differences between ethnic groups when comparing diagnosed anemia and missed diagnoses.

Discussion

This study shows anemia disparities among different racial and ethnic groups of adult pregnant women within the Sanford Health footprint. AI/AN and Black/African American patients had higher percentages of anemia compared to white patients. Of AI/AN patients, 58.3% were anemic and 55.1% of Black/African American patients were anemic compared to 40.0% of anemic white patients (percentages not shown in table). The global estimate of anemia in pregnancy is 38.2%.[7] Several studies have noted racial disparities of anemia during pregnancy, including increased anemia rates among Mexican American,[9] non-Hispanic Black [9,11,12] and Asian[12] women compared to non-Hispanic white women.

Of anemic patients, 12,354 were missing a charted anemia diagnosis within EMR. While there is a large number of missed diagnoses in this population (n=12,840, 81.1%; Table 1), there is no evidence of discrepancies of missed anemia diagnoses by racial/ethnic group among this population. Future studies could examine screening rates by racial/ethnic groups to investigate potential disparities.

Within this population, anemia was broadly underdiagnosed. Our methods of determining AIP were comprehensive and included either a lab value indicating anemia or a diagnosis within the diagnosis list or problem list. Only 3.2% (n = 486) of patients had any diagnosis of anemia listed in EMR. Problem lists are a key feature within EMR and provide a useful summary of important health issues. However, in practice, many patients have incomplete problem lists which may negatively impact patient care.[13] Undiagnosed and untreated AIP may lead to adverse maternal and fetal outcomes. Furthermore, the overall data integrity issue within EMR may compromise patient care. This merits further quality improvement strategies to improve either diagnostics or charting among clinicians.

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IMAGES IN MEDICINE:

Persistent Left Superior Vena Cava Syndrome Complicating Central Dialysis Catheter Placement

Ibrahim Munaf Ahmed, MD; Muhammad Hamza, MD; and Jiannan Huang, MD

A 70-year-old male with end stage renal disease on dialysis was admitted for septic shock secondary to urinary tract infection with Klebsiella sp. Due to refractory fluid overload and worsening urine output, the decision to initiate continuous renal replacement therapy was made. A right internal jugular vein dialysis catheter was placed under sterile conditions with real time ultrasound guidance. Post placement chest X-ray demonstrated the catheter to be crossing the midline (Figure 1), raising concern for inadvertent arterial placement. Interventional radiology was alerted to the possibility. On review of a contrast enhanced chest computed tomography scan earlier from the admission (Figure 2), a persistent left sided superior vena cava was identified. Pressure measurement from the dialysis catheter was consistent with venous insertion. After discussion with radiology, the dialysis catheter was determined to be in the anomalous superior vena cava, and was cleared for use. Physicians should be aware of this rare congenital anomaly when undertaking procedures requiring central venous access.

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Figure 1. Post placement anteroposterior chest x ray demonstrating the dialysis catheter crossing the midline (arrows).

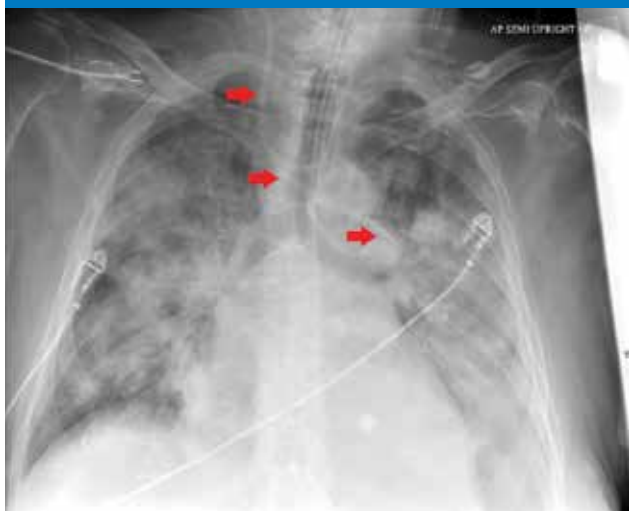
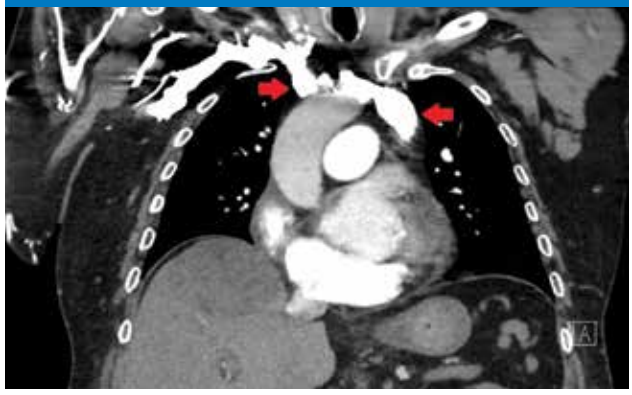


Figure 2. Contrast enhanced chest computed tomography scan demonstrating left sided superior vena cava (arrows).



Review of Retinopathy of Prematurity

Collin Olevson, MS II; and Geoffrey Tufty, MD

Abstract

Retinopathy of prematurity (ROP) is a leading cause of blindness in pre-term infants and is caused by incomplete vascularization of the retina at birth. Early diagnosis and treatment of ROP is important to reduce the risk of vision loss. The risk factors, pathogenesis, screening, diagnosis, and treatment of this disease will be reviewed.

Introduction

Retinopathy of prematurity (ROP) is a disorder that uniquely effects preterm infants. This disorder is due to incomplete vascularization of the retina at birth and subsequent abnormal vascular proliferation.[1] If left untreated, ROP can lead to retinal detachment and threaten vision.[2] The disorder was first described in the early 1940's and was likely a consequence of supplemental oxygen used to improve survivability of preterm infants.[2,3] It has been found that oxygen, gestational age, and birth weight are the three biggest risk factors for development of ROP. [1,2,4,5] In the U.S. 12% of births are preterm (before gestational age of 37 weeks) and due to advances in technology preterm infants can survive at younger ages.[6,7] With the high prevalence and increased survivability of neonates, ROP remains one of the leading causes of childhood blindness.[4]

Risk Factors

The incidence and risk of ROP is primarily dependent on gestational age, birthweight, and oxygen.[2,4] Both gestational age and birthweight are likely related to the degree of immaturity of the retina.[2] Overall, one study found 68% of premature infants will develop ROP.[8] The incidence of ROP increases with decreased gestational age and birth weight.[8] 83.4% of infants born at 27 weeks or earlier developed ROP compared to 55.3% of infants born 28-31 weeks and 29.5% of infants born at 32 weeks.[8] Additionally, 90.0% of infants born at less than 750 g developed ROP compared to 78.2% of infants born between 750 g-999 g and 46.9% of infants born between 1000 g-1250 g.[8] The severity of ROP increases with lower gestational age and birth weight. [8] These results have been consistent across multiple other studies.[4]

There have been many studies completed to assess the effect of oxygen on development of ROP as this is a risk factor that can be controlled. Higher concentrations of oxygen, longer use of oxygen, and fluctuation in blood oxygen have all been identified as risk factors for developing ROP.[4,9] Although using less oxygen in premature infants does help prevent development and progression of ROP, this needs to be considered in the context of the overall health of the infant as reduced oxygen can lead to mortality. [9] The ideal oxygenation of premature infants is not known, however 90-94% SpO_2 appears to produce favorable visual outcomes as well as limits increased mortality.[9,10]

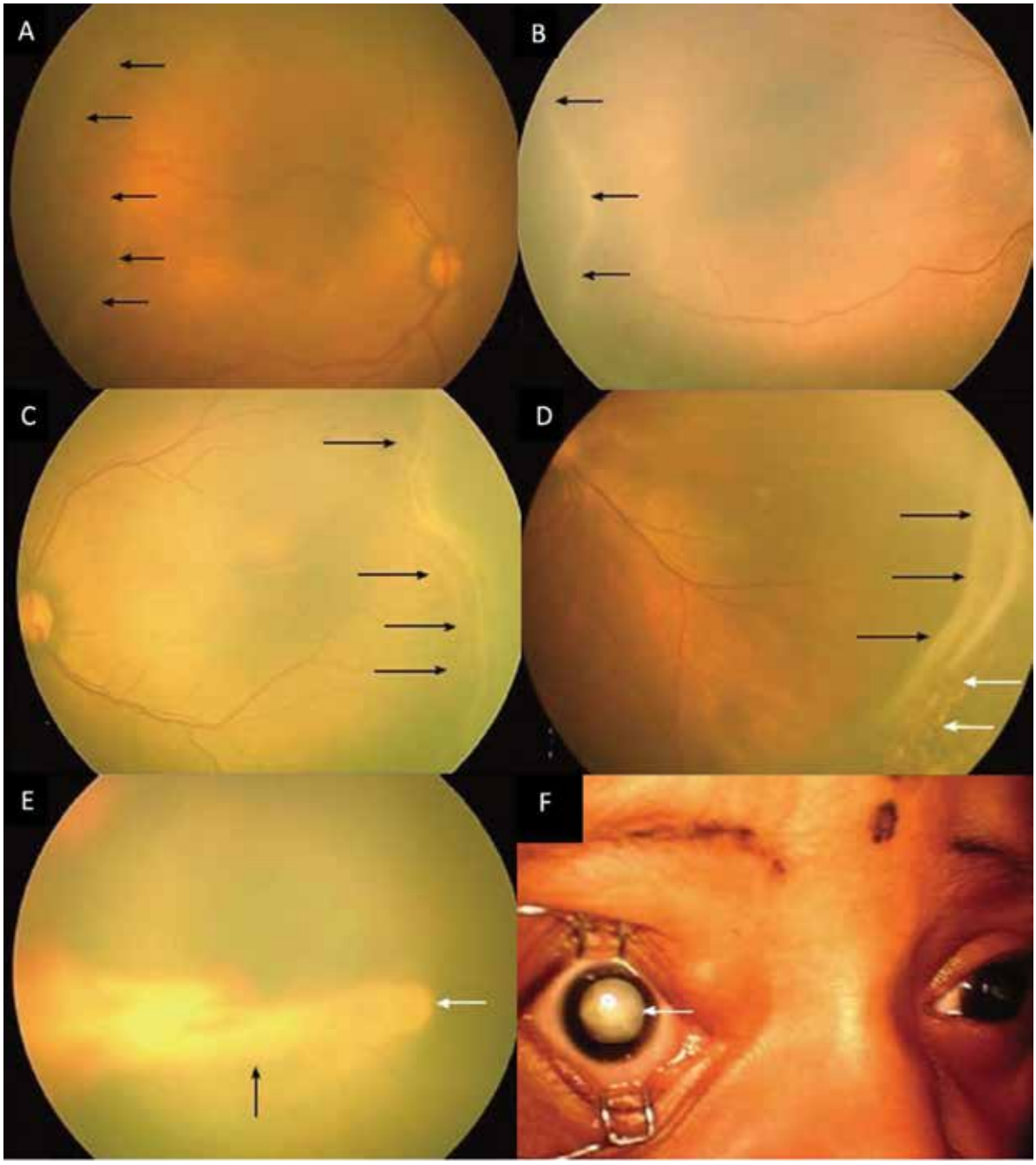
Pathogenesis

ROP is caused when normal retinal vascularization is interrupted which stimulates pathologic compensatory mechanisms.[1,2] The pathogenesis of ROP is broken into two primary phases. In phase I, premature birth exposes the retina to an environment that is hyperoxic and lacks factors, such as insulin-like growth factor 1, that are important for retinal development.[1,2] The sudden change in environment leads to suppression of important growth factors, particularly vascular endothelial growth factor (VEGF) and erythropoietin, and the retinal vasculature stops developing and even regresses.[1,2] Lack of vascularization causes hypoxia as the retina begins to become more metabolically active and this marks the transition to phase II.[2] Hypoxia drives production of factors, notably VEGF and erythropoietin, to stimulate new vessel growth.[1,2] Rather than vascularizing the hypoxic retina, the new blood vessels grow anteriorly into the vitreous.[1] The newly formed vessels are leaky, which leads to edema, fibrous scarring, and eventual retinal detachment.[2]

Screening and Diagnosis

Guidelines to screen newborn infants are primarily based upon birth weight and gestational age.[11] The American Academy of Pediatrics currently recommends that all neonates weighing less than 1,500 grams at birth or are born at a gestational age of 30 weeks or earlier should be referred for evaluation of ROP.[11] Additionally, neonates >1,500 g or >30 weeks gestational age should be referred

Figure 1. Staging of ROP. Retina images show stages 1, 2, 3, 4A, 4B, and 5. A: Stage 1 ROP. A demarcation line between vascular and avascular retina (black arrows). B: Stage 2 ROP. A demarcation line with a ridge (black arrows). C: Stage 3 ROP. A demarcation line with a vascularized ridge (black arrows). D: Stage 4A ROP: A partial detachment not involving the fovea (black arrows) and scars from a laser (white arrows). E: Stage 4B: A partial retinal detachment (black arrow) involving the fovea (white arrow). F: Stage 5: A total retinal detachment (white arrow).



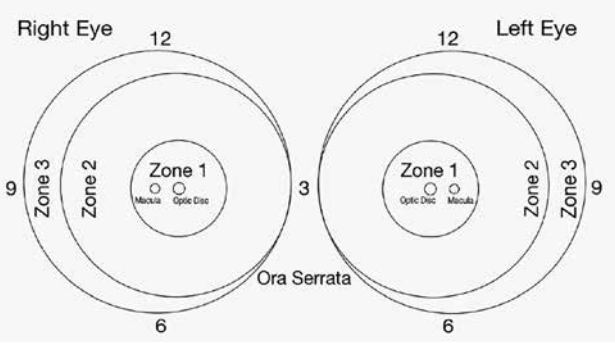
Adapted from Shah PK, Prabhu V, Karandikar SS, Ranjan R, Narendran V, Kalpana N. Retinopathy of prematurity: past, present and future. World Journal of Clinical Pediatrics. 2016;5(1):35-46.

for ROP evaluation if they have increased risk for ROP, for example, use of supplemental oxygen therapy beyond a few days.[11] However, it is argued that these current guidelines are not sufficient and result in missed diagnoses.[12] For this reason many NICUs in the United States and around the world utilize gestational ages of <31 weeks, <32 weeks, or <34 weeks, and birth weight up to 2000 g for ROP screening.[13] A study of more than 300,000 neonates found that almost 70% of NICU admissions are outside screening guidelines and will not require ROP examinations.[14]

Traditionally, ROP examinations have been done with an indirect ophthalmoscope by an ophthalmologist that has experience with ROP, often between four and nine weeks after birth.[11] Unfortunately, access to an ophthalmologist with ROP experience may not be available, particularly in rural settings. In these cases the use of a contact fundoscopic camera operated by trained healthcare professionals has yielded high specificity and sensitivity when images are graded by an ophthalmologist.[15,16] Use of a camera could be a valuable tool in facilitating detection of ROP in areas that do not have an ophthalmologist available. The timeline of these examinations can range from weekly to every third week and is determined by the severity of ROP with more severe ROP requiring more frequent follow-up examination.[11] Once ROP has regressed and the retina is vascularized frequent examinations can be terminated, however it is important to follow up with an ophthalmologist within four to six months of discharge from the NICU to ensure there is no recurrence.[11] Additionally, neonates with a history of ROP should receive routine eye care throughout their life due to increased risk for ophthalmic disorders in both the anterior and posterior segments of the eye associated with ROP.[11,17]

There is an established standardized grading system of the severity of ROP published by the American Academy of Ophthalmology.[18] ROP grading is classified primarily on the stage of the ROP (Figure 1), the zone where ROP is found (Figure 2), and the presence of pre-plus or plus disease.[18,19] Plus disease is characterized by dilated and tortuous vasculature near the optic disc and pre-plus disease is also dilation and tortuosity, however effecting fewer vessels.[18] Aggressive ROP is a severe and rapidly progressing subtype of ROP that is characterized by severe plus disease that does not progress through the normal stages of ROP and this carries the highest risk of poor visual outcomes.[18,20]

Figure 2. Zones of ROP. Zone 1 is defined as twice the distance between the center of the optic disc and center of the macula. Zone 2 is a circle with all sides being equal to the distance between the center of the optic disc to the nasal termination of the retina. Zone 3 comprises the remaining temporal retinal area.



Treatment

Fortunately, 92-96% of ROP will regress and resolve on its own.[20] The decision to treat is based upon the zone, stage, and presence of plus disease which divides ROP into type 1 or type 2. Type I ROP (table 1) requires treatment as the risk of blindness outweighs the risk of treatment.[2] This is defined as zone 1 ROP with plus disease regardless of stage, zone 1 ROP without plus disease once at stage 3, and zone 2 ROP with plus disease once at stage 2 or 3.[11] Type 2 ROP (table 1) is recommended to be followed closely as it may progress to type 1 ROP, however is not severe enough to warrant intervention.[20] This is defined as zone 1 ROP without plus disease at stage 1 or 2 or zone 2 ROP without plus disease at stage 3.[20]

Cryotherapy was used in the 1980s as a treatment for ROP to ablate part of the retina which prevented poor visual outcomes associated with ROP.[2,20] Today, cryotherapy is

Table 1. Typing of ROP. Based upon the zone, staging, and presence of or lack of plus disease ROP can be characterized into type I ROP, which treatment is recommended for, or type II ROP, which close follow up is recommended for.

Type I ROP	Type II ROP
Zone 1 ROP w/ plus disease at any stage	Zone 1 ROP w/o plus disease at stages 1 or 2
Zone 1 ROP w/o plus disease at stage 3	Zone 2 ROP w/o plus disease at stage 3
Zone 2 ROP w/ plus disease at stage 2 or 3	

rarely used as laser therapy and anti-VEGF have been shown to provide better outcomes with less risk. [2,20] Lasers are also used to ablate the peripheral avascular retina, this results in scarring that helps prevent new blood vessel growth and retinal detachment. [2,20] Drawbacks to laser therapy is that is does not address the underlying pathogenesis of neovascular tissue and carries risks such as include vitreous hemorrhage, development of cataracts, elevated intraocular pressure, and postoperative inflammation. [2,20]

Anti-VEGF is not FDA approved for ROP but has been used off-label to treat ROP. [20] This medication is injected into the vitreous and prevents neovascularization. Due to the recency of anti-VEGF being used for ROP the outcomes and long-term effects are not well established. [20] A review of randomized control trials that evaluated anti-VEGF for ROP found no reduction in risk of retinal detachment when used as a monotherapy, however when used in combination with lasers there was reduction in retinal detachment compared to lasers alone. [21] If the use of lasers and/or anti-VEGF is insufficient to treat ROP and prevent a retinal detachment surgical re-attachment of the retina may also be used. [20] Approximately 16% of infants with type I ROP that receive treatment will progress to a retinal detachment. [20]

Conclusion

ROP remains a leading cause of vision loss in premature infants. This disease is caused by pathogenic neovascularization following a premature birth. Incidence and severity of ROP increase with decreased gestational age, decreased birth weight, and excessive oxygenation. Identification of at-risk infants is important to initiate screening and treatment, when necessary, to preserve vision.

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EXTENUATING CIRCUMSTANCES:

Names Forever Remembered

Mariah M. Shafer, MS II

Growing up, I knew I was going to be a doctor. Being diagnosed with a chronic illness as a young child, I think back to my earlier years and remember a little girl infatuated with medicine who spent countless hours in hospital beds, doctor's offices, and phlebotomy labs. I challenged myself to fearlessly watch each needle-poke – envisioning what it must take to be a physician. And oh, how I drove the nurses crazy with endless questions: Why does blood flow into that tube so fast? What are you listening for with that stethoscope – can I try? From the day I was diagnosed with adrenal insufficiency, I became my own case study– every doctor visit was a learning opportunity. I didn't understand my parents' worries; to me, my diagnosis was the best thing ever – it was a VIP ticket to learn.

When the time came to apply for medical school, I spent some time considering the experiences that shaped my desire to be a physician. As I looked over the list I had made of these formative aspects of my life, I realized that rather than an exhaustive list of impressive accolades or adventures, I was staring at a list of mostly names – names of patients, strangers turned to dear friends, and teammates.

Pat

I knew that in order to get started in medicine, I needed to get involved in patient care early. So, in high school, I became a certified nursing assistant. During my clinical portion of the course, Pat was the first patient I ever had, and one I will not soon forget. Her tiny frame was engulfed by her large hospital bed as she stared back at me with wild and severe eyes. With end-stage dementia, her communication was mostly limited to "yes or no," but nevertheless, we quickly developed a mutual understanding. Though she was silenced outwardly, in her eyes it was clear that she yearned to be heard. Often, this culminated in cries of "HELP ME!"– repeated in fervent desperation for hours until she slipped into an exhausted slumber. Most of the staff was so used to this occurrence that they barely noticed it; I was told, "She is fine, she does this all the time." But I couldn't ignore it; this cry gripped something within me. Day after day, I sat with her every chance I could, to talk or listen. On days off, I visited her with my golden retriever and my guitar. Over time, I got to witness moments when her cries ceased and her urgent demeanor left her. Like everyone, she just needed to be heard and seen. She taught me the immeasurable power we all possess when we take the time to truly see those around us. This helped convince me that healthcare is far more than applied science. It encompasses caring for others when they're most vulnerable and goes beyond meeting their immediate physical needs.

Elba

A few years later, during my sophomore year of college, I had the opportunity to volunteer with a hospital in Siguatepeque, Honduras. On my first day there, I boarded the mobile clinic (a hardy 4x4) with the doctor and driver (who also happened to be the pharmacist). Heading for a mountain village, we pulled over on a dirt road and picked up Elba, the nurse. Being the only English speaker in the crew of Honduran staff, I sat quietly in the back, attempting to disguise my nerves. Elba's quiet presence melted any trace of my fear. As Elba's assistant, I helped her with charting, vitals, and injections. Although limited in our ability to communicate through speech, we quickly developed a strong friendship through our own language consisting of a little Spanish, lots of hand gestures, and mostly laughter. She asked me endless questions in Spanish that I couldn't answer– but she didn't mind. Nevertheless, I was determined to learn enough Spanish to contribute to the team. This was extremely humbling because not only was I learning a new professional role, but I was also within an unfamiliar culture. Though challenging, this experience cultivated in me an ability to adapt and interact with patients of diverse backgrounds. It affirmed my aspiration to provide care to underserved areas.

Paige

As I reflect on the outdoor track and field conference championships, the day is clearly etched into my mind. It was a particularly warm morning with no clouds in the sky. I was sitting on the edge of the track, cheering on my teammate Paige as she ran the final 10k race of her career. With two laps left, her legs buckled and her back arched just before

she hit the ground. The crowd faded from my mind as I sprinted onto the track and followed as she was carried into the medical tent. At first, what seemed like heat exhaustion quickly escalated to stress-induced delirium as my once docile friend began to scream and hit herself. It took my whole body to keep her on the table and from hurting herself. As she worsened, her agitation and fear became palpable, and her parents began to panic. Inside, I was terrified. I wondered if I ever see my best friend again. For the sake of Paige and her parents, I knew that I needed to remain calm. Holding in tears and pushing fear aside, I held on through the screams and convulsions to keep her safe. Finally, the ambulance arrived, and we followed it to the hospital where she recovered. Throughout my work in critical care at Aspirus Hospital, I've seen countless patients experience delirium. However, observing Paige as she endured this, changed my perspective on how drastically shock can alter one's demeanor. It revealed to me my ability to consider the well-being of all involved and remain calm despite uncertainty and fear. It showed me the profound impact that a reassuring presence can have on a patient and their family that goes beyond medical treatment.

*****

As I pursue the path to become a physician, I carry with me the lessons I've learned from this list of names I've shared, knowing that they've given me far more than I could repay. I'm grateful that these experiences have continued to develop my genuine passion for medicine, and I'm eager to have the opportunity to provide care informed by these experiences which have molded me since my earliest years. I will confer the insight I've gained by caring for patients holistically: being cognizant of emotional needs, adapting to communicate effectively, and reflecting stability in the face of fear to prioritize my care for others.

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EXTENUATING CIRCUMSTANCES:

Updates to Residency Applications: The Unknown Role of Program Signaling

Alaire Buchholz, MS IV; Garrett Quinn, MS IV; and Kristi Pond, MS IV

Fourth-year medical students and attending physicians from the University of South Dakota Sanford School of Medicine recently attended the American College of Obstetricians and Gynecologists (ACOG) Annual Clinical and Scientific Meeting in Baltimore, Maryland. The conference featured a variety of sessions geared towards medical students, including but not limited to discussions about obstetrics and gynecology (OB/GYN) as a career, residency applications, and skills practice stations. One question that was persistently brought up by medical students at every Q&A session was about the role of program signaling in the upcoming residency application cycle.

Program signaling was previously unheard of by most medical students and faculty. It was introduced and used by 16 specialties in the 2023 application cycle, including OB/GYN. Prior to the COVID-19 pandemic, applicants had to invest significant time and money into traveling to residency interviews. This demonstrated commitment and interest in a program on the applicant's behalf. After residency interviews switched to a virtual format during the COVID-19 pandemic, residency programs saw a significant uptick in applicants vying for interviews because applicants were no longer bound by travel and financial restraints. Comparing the electronic residency application service (ERAS) statistics from 2019 to 2023, the average number of OB/GYN programs in which applicants applied went from 56.8 to 67.0 programs – an 18% increase in four years.[1] Students could interview at more programs than ever. This made it more difficult for programs to determine who was genuinely interested in them versus who was mass applying to programs. However, program directors remarked that virtual interviews are likely here to stay even beyond the COVID-19 pandemic because they are more equitable and reduce disparities amongst applicants by eliminating the financial constraints of travel. Thus, signals were designed to be another data point for residency programs to help compare applicants and to allow applicants to express direct interest in a program to aid in the virtual interview format. In the upcoming 2024 application cycle, there are 20 listed programs that will be using signaling. The number of given signals varies by specialty, ranging from three to 30 signals.[2] In this editorial, we comment on the role of signaling in OB/GYN and suspect this advice translates to other specialties that offer a large number of signals such as otolaryngology, orthopedic surgery, dermatology, and anesthesiology. We encourage students pursuing other specialties to work closely with their advisors and department heads to strategically use their signals to strengthen their residency applications.

OB/GYN offers three "gold" signals and 15 "silver" signals to each applicant. Anecdotally, we heard from many students that applied last year that they were unsure of how to use their signals, and a couple program directors at the ACOG meeting commented that they did not use the signals to evaluate applicants last year because they too were unsure how to use them. An AAMC survey of the pilot year found over 80% of programs used signals in three main ways: as a holistic way to determine which applicants are offered interviews, a tiebreaker when deciding whom to interview, and for questions during the interview itself.[3] Several program directors at the conference stressed to students the importance of using signals wisely. This is difficult for students because data is limited on signaling since it was the first year of use. However, one program director emphasized that her data showed that applicants had less than a 5% chance of receiving an interview at a program that they did not signal. All the program directors highlighted the importance of distributing your signals across a broad range of programs. They advised against sending all of one's signals to highly competitive "reach" programs. While sending signals to "reach" programs may give an applicant a higher chance of an interview with that program, it also means they have less signals to use at programs that are more aligned with their competitiveness and where they are more likely to match. Some program directors mentioned they only offered interviews to applicants who signaled them, others said they reviewed the applications with signals first before reviewing other applications, and some stated they did not factor in the signals at all. All the program directors that did use the signals

agreed they did not treat a gold signal differently than a silver signal. Based on what we learned, we encourage applicants to signal a few "reach" schools but also, more importantly, signal an array of programs, including a mix of academic and community programs, and those whose goals and reputation align more closely with one's own application.

As mentioned, the signals are intended to be another data point to compare applicants by evaluating the applicant's interest in a specific program. They are not meant to substitute other parts of the application or replace a holistic review of a student's application. The signals are meant to be used to help residency programs decide which applicants to interview, but they were not intended to evaluate applicants post-interview when they are creating their rank list.

Other recent updates to residency applications include the option of including geographic preference. Applicants can indicate preference for up to three out of nine geographic divisions, or no preference at all, along with a corresponding essay explaining their preference. They can also indicate whether they'd prefer a rural or urban setting. This is only visible to programs that align with their preference. For example, if a student states that they prefer a residency program in the West North Central division, this preference is visible to West North Central programs, but programs outside of this region will see no geographic preference to prevent disadvantaging the applicant. Signaling and geographic preferences were part of the prior supplemental application but are now included in the primary application.

In summary, ERAS is evolving to accommodate virtual interviews and better serve students and residency programs alike by clarifying students' past experiences, preferences, and goals in a standardized format and encouraging residency programs to review applicants holistically. These changes include signaling which is still a newer portion of the residency application, affecting 20 specialties in the upcoming 2024 application cycle. Signaling offers another data point to residency programs to evaluate and compare applicants. With a majority of the 2024 applicants having taken Step 1 Pass/Fail and not having a Step 1 score as a data point, signaling may play an even larger role this year than last in the evaluation of applicants. Because data is still limited regarding how signaling affected last year's match, many students are unsure of how to use signaling and feel uneasy about the topic. In the field of OB/GYN, initial data reviewed by program directors at the ACOG Annual Clinical & Scientific Meeting suggests that students are much less likely to get an interview with a program that they did not signal. Students are encouraged to apply broadly and to use signals on programs that range in competitiveness. We do not advise using a majority of one's signals on competitive "reach" schools. We hope this information helps students feel more confident in the signaling process and increases their chance of matching at a program that they align with as well as aids faculty and mentors advising students in guiding applicants to use their signals advantageously.

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Member News

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

Are you interested in serving in a SDSMA leadership position?

The SDSMA wants to make members aware of opportunities to become more involved. With that in mind, we are seeking interested individuals to contribute to the continued growth and success of the association.

The SDSMA Policy Council and SDSMA PAC Board currently have roles available for members. These positions accomplish important work in various areas. This is an opportunity to share your expertise and experience!

- The Policy Council is the SDSMA's official policymaking body. The Council meets twice annually, including during the SDSMA annual conference. To learn more about becoming a councilor, please contact Terry Marks at tmarks@sdsma.org.
- The SDSMA PAC encourages SDSMA members to take a more active and organized part in governmental affairs and provides an effective way for members to become politically involved at both the state and national levels. To learn more about serving on the SDSMA PAC Board, please contact Justin Ohleen at johleen@sdsma.org.

We ask you to consider these opportunities to devote time, energy, and ideas to lead SDSMA policies and programs forward, and in turn, support the SDSMA's efforts on behalf of you and your patients.

Thank you to those currently serving in positions on the Policy Council and SDSMA PAC Board:

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Member News

A confidential program for all South Dakota licensed physicians and residents

South Dakota Physician Well-Being Program

South Dakota physicians seeking support for their health and well-being have a confidential and professional resource. The South Dakota Physician Well-Being Program is a responsive, high-quality program for physicians and residents that offers confidential, individualized support services and resources. The SDSMA, with funding from Avera Health, COPIC, Monument Health, and Sanford Health, developed this program as a resource for all South Dakota physicians.

The program aims to improve physician wellness and reduce burnout, and is committed to supporting the well-being and health of physicians, enabling them to continue to provide high-quality patient care, recognizing that physician well-being is essential for high-quality patient care. Burnout among physicians continues to be a major issue that was only compounded by the COVID-19 pandemic.

The program's staff have extensive expertise in providing support for health care professionals and physicians. The program is confidential and independent of the licensing board, state government and employers.

The South Dakota Physician Well-Being Program offers confidential, supportive services and seeks to empower physicians to move beyond burnout and lead more satisfying lives. It is offered via in-person and remote access. **This program is confidential – no insurance billing, no medical record.**

Individualized services may include:

- Well-being resources
- Career fatigue
- Grief and loss
- Financial stressors
- Stress and anxiety
- Depression
- Substance abuse
- Behavioral concerns
- Work/family balance
- Personal and family stressors

To learn more and get confidential support, call 605-275-4711 or visit www.mwhms.com/pwbp.

Physician leadership program now accepting participants

The SDSMA Health Leadership Institute (HLI) is now accepting participants for the 2023-24 cohort!

The HLI helps physicians balance life and work, sharpen their skills in practice management, and train for future leadership positions.

The program offers current and future leaders the opportunity to gain an in-depth knowledge of the role leadership plays in medicine. It prepares participants for roles within their practice location or health system, and provides professional development, community engagement and leadership skills training.

SDSMA CENTER FOR PHYSICIAN RESOURCES
Health Leadership Institute

Six monthly sessions will be held with courses beginning in October.

How to apply – Those interested in participating may contact Justin Ohleen at 605-336-1965 or johleen@sdsma.org. Learn more at sdsma.org.

Legal brief highlight: Brand and Generic Prescriptions

A pharmacist, under certain circumstances, may substitute a generic equivalent of a drug prescribed by its brand name. The pharmacist must notify the person receiving the drug of the substitution, and must also notify them of their right to refuse the selected drug.

The physician may prohibit the substitution of a generic equivalent by including on the prescription order the words "brand necessary" or words of similar meaning. If the prescription order is oral, the physician or authorized agent must specifically instruct the pharmacist that substitution is prohibited. The selection of an equivalent drug product does not give rise to a cause of action against the practitioner.

More information is available in the SDSMA legal brief *Brand and Generic Prescriptions* at sdsma.org. The SDSMA provides legal briefs online for member physicians on a variety of rules and regulations that apply to the medical profession. For nonmembers, legal briefs are available for purchase.

Physician peer support group meetings

Support groups are provided for South Dakota physicians through the South Dakota Physician Well-Being Program.

SIOUX FALLS AREA RETIRED PHYSICIANS GROUP

8-9:30 a.m., All Day Cafe, Sioux Falls

- September 11, 2023
- October 9, 2023

All retired or soon-to-be retired physicians are welcome to join in this informal gathering. RSVPs appreciated but not required. Email Dan Heinemann, MD at djheineman@aol.com or Tad Jacobs, DO at doctalkconsulting@gmail.com to save your spot.

PHYSICIAN MOMS GROUP - VIRTUAL

7-8 p.m. CT, Zoom

- Third Tuesday of each month

Physician moms are invited to a one-hour Zoom to share their experiences, struggles and encouragement with one another. Email Beth Jensen, DO at elizabethj@mwhms.com to be provided with the Zoom link or call 605-275-4711.

Please call the South Dakota Physician Well-Being Program at 605-275-4711 if you have questions about any of these groups.

For Your Benefit:

Your Legislative Advocate

SDSMA's legislative staff is your eyes, ears and voice in Pierre and Washington. We track hundreds of pieces of legislation that affect you, as well as coordinate opportunities for you to get involved in the process.

Do you want to be the Doctor of the Day during the South Dakota legislative session? How about a Physician Lobbyist? You can, and we can make it possible.

There are more SDSMA benefits to tell you about, and you'll hear about them in the months to come. In the meantime, if you'd like more information about our legislative services and advocacy programs, give us a call at 605.336.1965 or visit www.sdsma.org.

Thank you for your membership in SDSMA.

"For Your Benefit" is the SDSMA's monthly update on programs and services available to physicians through their affiliation with the SDSMA.

SOUTH DAKOTA BOARD OF MEDICAL & OSTEOPATHIC EXAMINERS
BOARD NEWS

The SDBMOE staff, in partnership with LakeNology, have been hard at work designing a new licensing system. This project first kicked off in early 2022, with the steering committee and subject matter experts meeting weekly to plan, analyze, design and build. The new licensing system will be ready for use early 2024. Read the below Frequently Asked Questions for more information.

FREQUENTLY ASKED QUESTIONS

1. **Do we have goals and objectives for this new licensing software project?** Yes. A Project Charter has been provided with project scope, timelines, goals, objectives, and deliverables. Here is a brief list of what we expect to accomplish:

GOALS/DELIVERABLES

- Optimize the user experience for applicants and licensees through reengineering and feedback
- Eliminate current system workarounds and tedious reconciliation processes.
- Deliver an integrated and comprehensive approach to investigation, board management and discipline tracking functions.
- Enhance the authorized agent and supervisor experience through forms and checklist automation.
- Shift to a more strategic focus by reducing manual efforts and redundant tasks for staff.
- Automate high volume processes and payment functions to improve productivity.
- Create integrated on-line work ques for automated approvals, status updates and document routing.

2. **Is there an established team working to make this project a success?** Yes. A steering committee was formed in early 2022 when the project first kicked off. Both the vendor (LakeNology) and SDBMOE have defined resources. The committee meets weekly and subject matter experts are invited to the meetings as additional guests when appropriate.

3. **How will I know what my role is?** Roles and responsibilities are defined in the Project Charter for SDBMOE and Lake Nology resources. In addition, applicants, licensees, SDBMOE staff, board members, etc. will be part of continuous communications and invited to participate in focus groups, surveys, testing and training when appropriate.

4. **Is there commitment from top level management and will we have support from managers?** Yes. The project is fully funded and approved. The SDBMOE Executive Director is the project sponsor The Steering Committee members and subject matter experts are actively involved and provide input to the overall design and process reengineering efforts. There is also an Engagement Manager from LakeNology working with SDBMOE staff to ensure product acceptance and employee readiness levels throughout the project timeline.

5. **How will we benefit from this new project?** There are a variety of expected benefits specifically for our customers, but also for the SDBMOE staff. Here is a brief list of what we believe those are:

BENEFITS

- Automated self-service features for applicants and licensees to download/upload and electronically submit their required documents with integrated email correspondence.
- One-stop application and payment processing will speed time to completion.
- Extended platform capabilities that provide customer convenience and efficiency by using their device of choice (computer, laptop, phone, tablet, etc.) to access SDBMOE services.
- Improved customer relations through system-generated contact logging and a more personalized user experience.
- Auto-generated case numbers and on-line dockets for role-based document review and approvals.

6. **Will we receive training or documentation on the new software?** Yes. There will be emails, newsletters, video tutorials and quick reference guides distributed to key stakeholders using the software to facilitate the communication of new functions and features. SDBMOE staff will also conduct face-to-face training for those who will be using the licensing software.

7. **How do I provide feedback and/or help contribute to the product you are building?** If you wish to provide feedback and/or contribute to the product, please send an email to SDBMOE@state.sd.us.

8. **What are the project timelines?** There is a very detailed project plan with timelines, tasks, and resources. This document is maintained by LakeNology's Project Manager and is divided into phases. The initial team kickoff meeting took place in early February 2022 and the planning phase was completed the end of March, 2022. We are currently in the design and build phase. The design and build phases will take us into Fall 2023 when our comprehensive testing will begin. Our planned Go-Live date is projected for early 2024.

9. **Will we get to see what the new licensing software will look like before it goes live?** Yes. We will communicate throughout the project and will be sharing snippets of information and screenshots related to the new licensing software as we get closer to the testing phase. If you are participating in the user-acceptance testing, you will have interactive and hands-on experience with the new software.

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.

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