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South Dakota medicine

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BROCK PURDY AND THE IMPORTANCE OF INCONSEQUENTIAL DECISIONS

ROSS POLKING CFP®, AIF®, MBA *Lead Advisor - Business Development*

"With the final pick in the 2022 NFL draft, the San Francisco 49ers select…Brock Purdy, quarterback, Iowa State University," boomed the voice of commissioner Roger Goodell as the multi-day event back in April came to a close. After 261 players were chosen before him, a new "Mr. Irrelevant" emerged with the undersized Purdy, donning the label given annually since 1976 to the last pick in the NFL draft. As the nickname implies, this pick is typically inconsequential, because rarely does anyone in that slot make much out of a football career.

While the 49ers didn't think this was a throw-away pick, they could not have predicted the ensuing fairy tale. After two experienced and talented quarterbacks in Jimmy Garoppolo and Trey Lance were injured for the season, Brock was handed the reins to the 49ers offense in early December. The 49ers clearly had a plan with that pick, regardless of the shoulder shrugs, even from their own fans. They admittedly did not see Purdy as a future starter, much less a star in the making but rather a competent emergency back-up who could fill in if necessary.

Mitigating risk, of which there is plenty in the course of a football season, ironically catapulted San Francisco toward a Super Bowl run in a season that, otherwise, could have gone in the tank. Mr. Irrelevant quickly became relevant, winning every game as a starter until he suffered an elbow injury in the NFC championship game and the 49ers' season came to an end. Brock racked up stats never before seen by a rookie quarterback and most likely cemented his spot as an NFL regular going forward. Even his jersey sales finished in the top 10 of all NFL players!

One seemingly small decision that no one thought would matter made a significant difference for the Bay Area team and its fans.

Think about your financial life and what, in retrospect, felt like an inconsequential decision at the time. Think about how that decision has now become incredibly rewarding. Maybe it was to start accelerating your debt repayment. Maybe it was to begin saving into a child's 529 plan the month they were born. Maybe it was to increase your 401k contribution by 1% every year. Maybe it was to adjust your investment allocation to lessen individual company risk. Maybe it was to hire an advisor.

Whatever that decision(s) was, were, and/or will be, nothing relative to money is small when compounded by time. Do not shrug your shoulders at what may appear to be a decision that carries no weight. Rather, don't wait! Make the effort to plan; ask good questions; hedge your risks in the event life blindsides you; engage someone you trust to be objective. Retirement may be right around the corner. Staying diversified is Purdy good advice (see what I did there?).

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COVID-19 Pandemic Forever Changed Our Perception of the Importance of Preventive Medicine, Global Health and Physician Well-Being

Lucio N. Margallo II, MD
President, South Dakota State Medical Association

Last month we celebrated National Doctors' Day, which provides an opportunity to thank the courageous physicians who have worked throughout the COVID-19 pandemic to save lives and continue to take on other huge challenges.

Our present expectation of the COVID-19 pandemic has proven that the virus can manifest not only as a perfect cytokine storm to infected individuals but also as a global force that has forever changed our perception of the importance of preventive medicine and global health.

Understanding the multifaceted aspect of the COVID-19 pandemic is essential to the future of healthcare and our society. Our response to COVID-19 changes as we gather new information. However, one thing that remains constant is that all of us are encouraged to learn from this disastrous experience to promote better public health and prevent future catastrophe.

Viruses like SARS-Co V-2 continuously evolve as changes in the genetic code (caused by genetic mutations or viral recombination) occur during the replication of the genome. Multiple variants of SARS COV-2 have been documented in the U.S. and globally throughout this pandemic. Currently, according to CDC data, the XBB 1.5 is now about 90% of COVID cases in the U.S. It has quickly become the dominant strain – in mid-December, the variant was only about 7% of cases.

Vaccinations and Treatments

85% of people in the U.S. have at least 1 dose of COVID-19 vaccine (age 5+ years), according to the CDC. Those with a booster total 17.5 percent.

The FDA has authorized antiviral medications to treat mild to moderate COVID-19 infected patients. Antivirals target specific parts of the virus to stop multiplication in the body to prevent severity and deaths. Treatments include: Paxlovid-Nirmatrelavir-Ritonavir (for age 12 years and above), Remdesivir (Veklury) (children and adults); and Molnupiravir (Lagevrio) (Adults), convalescent plasma (for immunocompromised patients).

Misinformation/Disinformation

The rampant spread of misinformation and disinformation about medicine and science, often found on social media, can affect vaccine confidence and vaccination rates. Disinformation about COVID-19 has frequently focused on vaccine developments, safety and effectiveness as well COVID-19 denialism. We have seen this in the questions we receive from our patients to the care and treatments that patients request or decline. The SDSMA continues to be a leading voice on vaccine science and efficacy as we have since the beginning of this pandemic.

Physician Burnout

Recently published research shows how the COVID-19 pandemic magnified long-standing issues that have accelerated the physician burnout rate. At the end of 2021, nearly 63% of physicians reported symptoms of burnout, up from 38% in 2020.

The SDSMA has led the conversation in South Dakota to help solve physician burnout, advocating for new solutions that acknowledge physicians need support. I am thrilled that the SDSMA, with funding assistance from Avera Health, COPIC, Monument Health and Sanford Health, developed the South Dakota Physician Well-Being Program as a resource for all South Dakota physicians and residents. The program offers services via in-person or remote access. 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, and personal/family stressors. The program is confidential with no insurance billing and no medical record.

It has been inspiring to see the SDSMA working to tackle the many challenges that both physicians and patients face.

Editorial

Better Teaching Through Clear Communication

Jason J. Kemnitz, EdD

Communication is part of our daily lives. The AACME core competencies for entering medical school include interpersonal and intrapersonal communication skills. Further, as faculty, you are expected to grade students on their interpersonal and intrapersonal communication, and students rank you on these skills as well. Overall our teaching faculty receive very high reviews but in the last year, the lowest-rated categories were offering timely and constructive feedback (4.69/5) and setting expectations for the learning session (4.65/5). While these results are not alarmingly low, they do warrant some discussion. At the heart of both of these issues is clear concise communication. These issues can best be addressed by offering some simple tips to help clarify the teaching environment.

Use a framework – The simplest way to ensure that you will communicate clearly with your students is to use a framework to teach. Using a framework will ensure your teaching sessions are organized and well-planned. A framework will also ensure that you communicate your expectations clearly as each one of the designs call on the educator to articulate what they plan to teach and conclude with a feedback session. A popular framework is OAS, or Outcomes, Activity, Summary. The outcome portion asks you to consider the background knowledge of the learners and set expectations based on this knowledge base. The activity portion suggests that you think about how you will actively incorporate the learner in the teaching session so they can learn by contributions. The summary portion is a process of summarizing what the students learned and setting expectations for the next teaching session. This framework can be used with one or multiple students as well as in an interprofessional manner.

Pick a teaching method – Once you have thought through your framework, the next step to clear concise teaching is to pick a teaching method. Too often we enter a teaching situation thinking we can "wing" it, or that we have done this for so long that we don't think about the best way to teach. In addition, we may think that some teaching methods take too much time, but if done right, they can make the teaching experience better. There are many teaching methods you may have heard of such as Once Minute Preceptor, Rounding Like a Ninja, The Stanford Method, and SNAPPS to name a few, of these I think SNAPPS offers the best teaching interaction. SNAPPS stands for Summarize, Narrow, Analyze, Probe, Plan, Select. This logical process follows closely clinical reasons and is easy to incorporate into the teaching session. This method also allows you to set clear expectations per session and offer specific feedback per patient.

Provide feedback – Better teaching through communication also includes clear concise feedback. The USD SSOM endorses the Reflective Feedback Conversation as a method of student feedback. This feedback is a simple conversation between you as an educator and the learner. If you chose not to use this method, please consider the following tips when giving feedback: Make a direct observation; Be constructive; Seek the learner's input; Limit to two or three areas of improvement; Create a plan to help the student learn.

Take a moment to self-reflect – Our students tell us one of the main ways they learn is through role modeling. We must take a moment of self-reflection periodically to become better educators. When thinking about your teaching ask yourself what your strengths are as a teacher, and what are your areas for improvement. Are you up to date on where your learners are in their education and are you teaching them the appropriate lesson at the appropriate level? An easy process for this is to ask the following questions: Was my teaching effective? Why? or Why not? If not, did I set the explicit expectations early? What could I improve? Where did my student struggle and excel? This simple line of questions can help you and your learner find more enjoyment in the teaching and learning process.

Finally, the easiest way to make sure that your student is learning is to simply ask them periodically throughout the session. As the Irish playwright George Bernard Shaw penned, "the single biggest problem in communication is the illusion that it has actually taken place." A periodic check will let you know you are being effective and also keep the learner engaged.

The USD SSOM has great faculty, our students rated our Pillar 2 faculty a 4.7/5 last year and our Pillar 3 faculty a 4.8/5. Any medical school would be envious to have such a strongly rated teaching faculty, and we thank you for all you do for our students and our medical school. If you would like further information on the above topics scan the QR code.

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Nasopharyngeal Lipoma: A Rare Presentation of a Common Lesion

Danielle Mack, MS IV; Brandon Rosell DO; and Ashwyna Sunassee, MD

Abstract

Lipomas are common slow-growing benign tumors composed of mature adipose tissue. They can grow in any location where fat is present. They are well encapsulated and are generally of little clinical concern. Lipomas are typically located in the subcutaneous fat of the neck and trunk. The presence of a lipoma in the nasopharynx is rare. These lesions may produce clinical symptoms or be found incidentally. Here, we present the case of a 34-year-old male who was found to have an incidental pharyngeal lipoma following a nasopharyngoscopy for unrelated tympanostomy tube placement. This case illustrates a rare presentation of an otherwise common lesion.

Introduction

Lipomas typically develop in the fifth and sixth decades of life and are slightly more prevalent in males.[1,2] These are slow-growing benign tumors composed of adipose tissue which can grow in any location where fat is present. They can be single or multiple and are typically well circumscribed with a thin, delicate capsule. Grossly, they resemble yellow lobulated fat with fine fibrous septa. Microscopically, they are composed of mature adipose tissue without cellular atypia. They are immunohistochemically indistinguishable from normal adipose tissue, staining positively for S-100 and calretinin. Lipoma variants may contain areas of fibrous, myxomatous, or capillary angiomatous tissue and areas of ischemic change, infarct, and cystic degeneration can be observed.[3]

Lipomas are generally asymptomatic but may disrupt adjacent structures causing rhinorrhea, nasal obstruction, foul odor, foreign body sensation, voice change, and cranial nerve involvement.[4] Lipomas are typically located in the subcutaneous fat of the neck, shoulders, back, and buttocks.[2,3,5] Approximately 25% of these arise in the head and neck.[6] Of these, only rare cases have been reported in the nasopharynx.[1,2,4-10]

Case Presentation

We present the case of a 34-year-old Caucasian male who presented to an ENT clinic with a complaint of a four-year history of waxing and waning fullness in his ears. He reported an occasional feeling of fluid in his ears and a popping sensation with sporadic nasal congestion. He noted decreased hearing in crowded areas and certain tones were difficult to detect. His medical history was significant for bilateral eustachian tube dysfunction with recurrent ear infections and PE tubes as a child.

He proceeded with myringotomy with little symptom relief. Two months later, he underwent a tympanostomy with bilateral tube placement. A nasopharyngoscopy was performed to assess for proper tube placement. Bilateral myringotomy tubes were noted in addition to a small fatty lesion identified in the fossa of Rosenmüller. On CT, this lesion measured 13 mm and its location was confirmed in the midline nasopharynx (Figure 1).

Nasal endoscopy with excision of the mass was performed. A polypoid pedunculated mass originating from the left fossa of Rosenmüller was identified (Figure 2). The mass was excised at the stalk and sent to pathology. Gross examination of the lesion identified a single tan-pink 2.0 x 2.0 x 1.5 cm polypoid lesion with an evident resection margin. The tissue was trisected to reveal a tan-pink to yellow parenchyma. Microscopic analysis of the lesion revealed benign sinonasal

Figure 1. Computed tomography (CT) scan revealing an 11 mm diameter homogenous mass (circle) in midline nasopharynx on sagittal cross-section.

Figure 2. Endoscopic image depicting the fossa of Rosenmüller. The fossa (dashed line) is visualized abutting the underlying mass. The Eustachian tube sits anteriorly (asterisk).

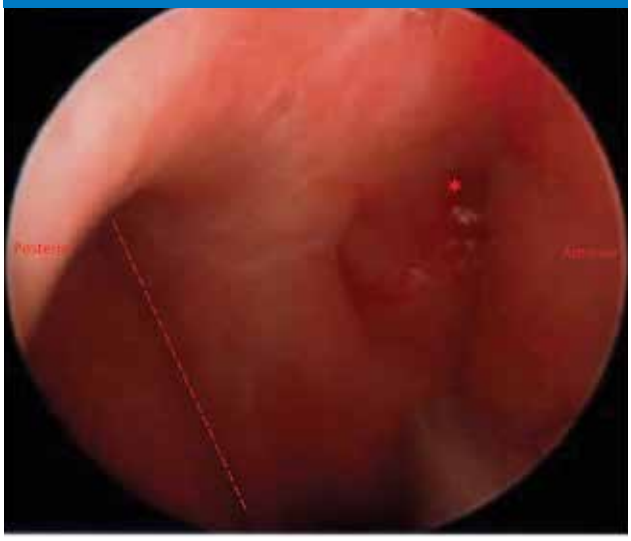
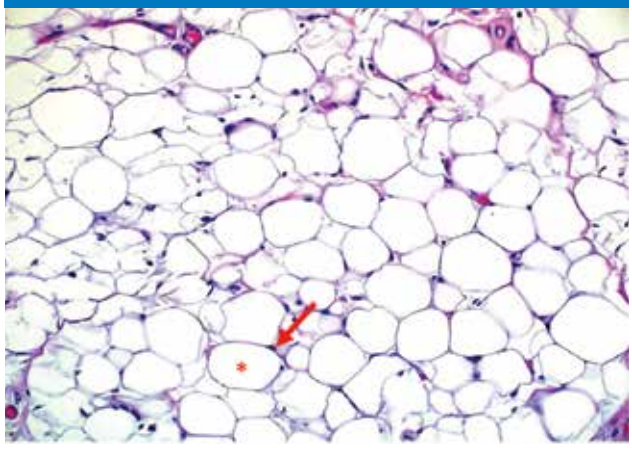


Figure 3. H&E section of the lipoma (200x magnification). The adipose cells with typical central lipid storage (asterisk) and eccentric nucleus (arrow) are mature and without atypia.



mucosa with underlying mature adipose tissue suggestive of a lipoma (Figure 3).

Concerning this patient's clinical outcome, he reported that his symptoms resolved following ear tube placement. He has yet to return for additional follow-up.

Discussion

A 34-year-old man was found to have a pharyngeal mass following a nasopharyngoscopy for bilateral tympanostomy tube placement. Surgical removal and pathological analysis of the lesion revealed it to be a lipoma. Nasopharyngeal lipomas are rare with as few as ten cases with pharyngeal involvement reported in the literature.[7] In this region, these lesions arise from the scant submucosal adipose tissue and are typically attached with a pedicle or sessile stalk.[4,5]

This lesion was found in the fossa of Rosenmüller which is located in the superolateral aspect of the nasopharynx and is bordered by the eustachian tube anteriorly. The most common lesion in the fossa of Rosenmüller is nasopharyngeal carcinoma. This is commonly seen in Southeast Asian populations in association with EBV infection. It will invade locally and has a propensity to spread to regional lymph nodes.[11] In our case, carcinoma was not suspected based on the endoscopic and CT findings as well as the patient demographics.

Nasopharyngeal lipomas are rare, even more so arising in the fossa of Rosenmüller. Another case has been reported in which the mass caused partial upper airway obstruction.[4] However, the lesion described here was not involved in our patient's symptomatology. The mass in this patient was found incidentally on an unrelated post-tympanostomy endoscopy. The bilateral dysfunction in our patient also pointed away from an obstructive cause that would typically present unilaterally.

The differential diagnosis for this mass also included liposarcoma; however, malignancy was not suspected due to the lack of supporting morphologic features which include a tumor size larger than 10 cm, presence of thick septa, and the presence of greater than or equal to 25% lesional, non-adipose tissue.[3] Liposarcomas may be well-circumscribed but are generally non-encapsulated. This patient's mass on CT was a homogenous 13 mm mass without evidence of abnormal septation or additional tissue types. Well-differentiated liposarcomas may appear much like benign lipomas on the cut surface and by low-power microscopic examination. However, high-power inspection demonstrates scattered tumor cells with large hyperchromatic nuclei. Atypical cells with features of lipoblasts may be present, though these are not required for diagnosis.[3] In contrast, the lipoma in this patient demonstrated bland, mature adipocytes without atypia, and no lipoblastic features were identified.

Endoscopy is the preferred method to visualize these lesions. CT and MRI can also provide useful information and aid in ruling out other diagnoses such as adenoid hypertrophy, liposarcoma, dermoid cysts, and salivary gland tumors. Lipomas are treated surgically with cautery or laser with good

results.[7] The risk of local recurrence is low at approximately 5%.[6]

Conclusion

This case presents a rare incidence of nasopharyngeal lipoma. Lesions such as the one presented here may not manifest themselves clinically. The differential diagnosis includes malignant entities such as nasopharyngeal carcinoma and liposarcoma, but endoscopy, CT, and MRI can provide important clues. When found incidentally, these lipomas may be surgically removed with good clinical outcomes.

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Metastatic Basal Cell Carcinoma with Axillary Lymph Node Invasion Treated with Left Axillary Node Dissection

Nicholas Wixon, MS IV; and Tyler Bergstrom, MD

Abstract

Basal cell carcinoma (BCC) is the most common skin cancer, accounting for 50% of all cancer in the U.S. Mortality is often considered negligible due to the rare rate of metastasis at 0.0028%. A 71-year-old fair-skinned male with a history of more than 34 documented skin carcinomas presented to his dermatologist with a non-painful, mobile, growing mass in his left axilla. Biopsy revealed metastatic basal cell carcinoma. Left axillary node dissection was performed in which perineural, perivascular, and perimuscular invasion was noted. Invaded structures were excised, and the patient reported no long-lasting sequelae following surgery. Patient refused referral to radiation oncology. Treatment of metastatic BCC consists of surgical excision of the primary tumor with adjuvant radiation therapy and chemotherapy if indicated for palliative measures. Chemotherapeutic regimens often include one or more of the following: 5-FU, cisplatin, vincristine, etoposide, bleomycin, cyclophosphamide, methotrexate, doxorubicin, as well as the new options of vismodegib and sonidegib. Despite surgical excision with adjuvant chemotherapy, metastatic basal cell carcinoma has been found to have a dismal 56% five-year and 27% 10-year survival rate. The best way to increase survival is through early diagnosis and wide surgical excision with clear margins followed by chemotherapy.

Background

Basal cell carcinoma (BCC) is the most common skin cancer, and it has been increasing in incidence due to an aging population and widespread sun exposure. BCC accounts for 50% of all cancers in the U.S.;[1] however, the mortality from this cancer is often considered negligible due to the rare rate of metastasis of 0.0028% or one in 1 million BCC diagnoses.[2] There are only 230 reported cases of metastatic basal cell carcinoma in world literature review.[3] Although rare, when metastatic spread does occur it usually happens in a middle-aged male who has had a BCC for a long duration.[3] When it spreads, BCC often involves lymph nodes, lungs, and bones.[4]

BCCs are slow-growing and locally invasive epidermal skin tumors. They typically appear in the head and neck region (70%) but can also appear on the trunk and extremities (30%).[5] BCC is differentiated by clinicopathological features into high- and low-risk subtypes. High-risk BCCs include morpheoic, infiltrative, and micronodular. BCC can also be considered high risk if the tumor is greater than 5 cm in size, is a recurrent lesion, located on the face, or has perineural/perivascular invasion. Low-risk BCCs are superficial/nodular and are located at a low-risk site such as the extremities.[5]

Our patient had an extensive history of BCCs and squamous cell carcinomas (SCC). Due to the sheer number of skin carcinomas in his history as well as at the time of presentation, it was impossible to ascertain the exact primary source. Our case deals with an incredibly rare metastatic disease in a fair-skinned 71-year-old male.

Case Presentation

A 71-year-old male with an extensive history of basal cell and squamous cell carcinoma visited his dermatologist for his routine three month visit. He has a history of more than 34 documented cancerous skin lesions over the last 10 years. During his dermatological visit the patient reported a mobile mass in the left axilla. Over the next several months this mass slowly grew. A left axillary lymph node biopsy was eventually performed and revealed metastatic basal cell carcinoma from an unknown exact primary source. PET/CT imaging revealed moderate hypermetabolic activity in the high left axilla consistent with the location of the mass that was palpated on exam and biopsied. (Figures 1 and 2). MRI chest with and without contrast revealed a 2.8 x 2.9 cm deep subcutaneous enhancing mass in the left upper axillary region consistent with malignancy. The mass was located immediately deep to the neurovascular structures without definite direct invasion. The mass abutted the deltoid muscle without evidence of direct invasion. After discussion with the patient's dermatologist and the radiation oncologist, it

Figure 1. CT PET scan coronal view. White arrow pointing to hypermetabolic mass in the left axilla.

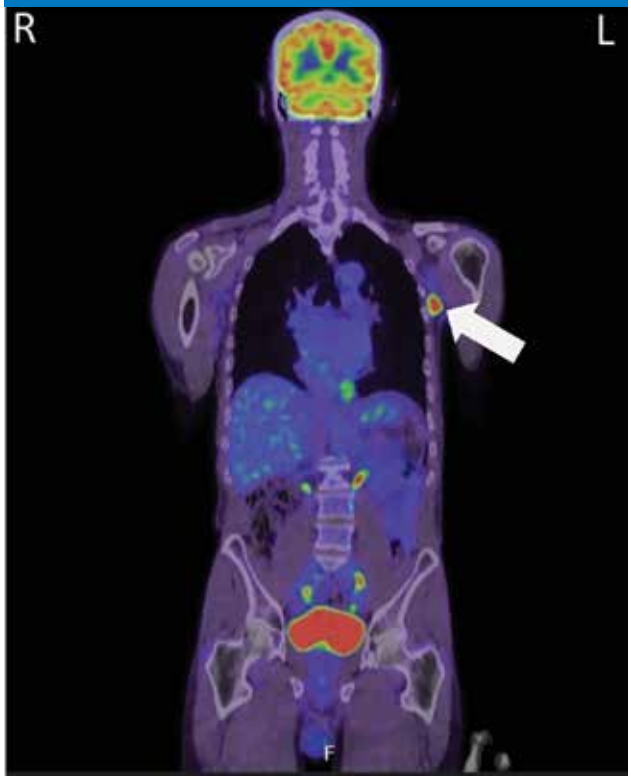
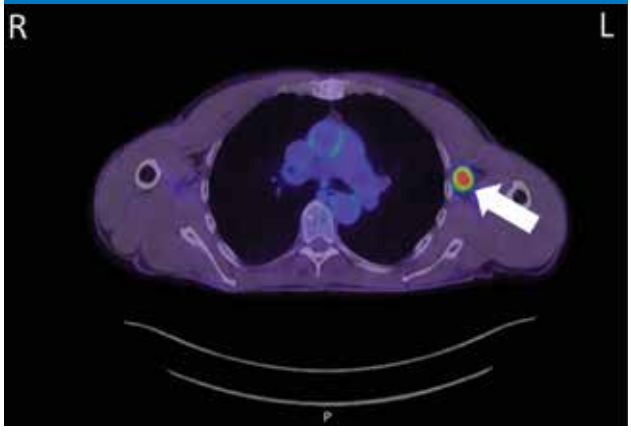


Figure 2. CT PET scan axial view. White arrow pointing to hypermetabolic mass in the left axilla.



was determined that the patient should be referred to general surgery to undergo left axillary node dissection to clear the axilla of metastatic disease.

In the operation, the mass was palpable in the left axilla. An elliptical skin incision was performed encompassing the skin directly over the mass obliquely in the left axilla. The dermis and subcutaneous tissue were dissected to axillary fatty tissue. Mass was palpable throughout the procedure. The pectoralis major muscle was exposed. We visualized a mass attached to a portion of the pectoralis along the lateral edge. We stayed outside the mass taking a margin of muscle. We then continued along the chest wall and encountered a few filmy adhesions. Multiple feeding vessels were encountered to the mass. Several vessels were clipped and divided. We continued to dissect down to the nodal and fatty tissue of the axilla. The superior aspect of the dissection was performed identifying the axillary vein superiorly. Mass was near the axillary vein but did not appear to involve the axillary vein. We cleared the mass with careful tedious dissection. Diving inferior, the thoracodorsal neurovascular bundle was preserved. Subscapular vein was directly involved in the mass, and therefore had to be excised. Several nerves crossing the axilla also appeared to be involved in the mass which were also excised. The intercostal brachial nerve was involved in the tumor. Tumor did involve pectoralis minor, and we continued to dissect to levels 2 and 3 to free up the tumor. Once the mass was freed, we continued to clean out the axilla avoiding the thoracodorsal neurovascular bundle. Several lymph nodes did appear to have potential metastatic disease and were subsequently excised. Other involved axillary contents were removed utilizing wide surgical excisions. All excised contents were sent off to pathology for analysis.

Pathology reported sclerosing/infiltrative basal cell carcinoma with perineural invasion that extended to the margins of excised tissue. Of the four collected lymph nodes, only one was positive for basal cell carcinoma by direct extension. The lymphatic involvement as well as his concerning margin findings indicated his lesion possessed a high likelihood of recurrence. Subsequently, the patient was offered a radiation oncology referral as he may be a candidate for radiation therapy (RT) of his post-surgical bed. The patient was hesitant to go this route due to fear of the development of further skin cancers. Overall, the patient had recovered well and was participating in physical therapy.

Discussion

Metastatic BCC is a rare entity that affects twice as many males as it does females.[6] Predicting metastasis is difficult, but there is a correlation with age, site of lesion, size, depth of invasion, duration and recurrence, incomplete surgical resection, number of lesions, and histological pattern.[7] Other known risk factors for BCC metastasis include fair skin, trisomy of chromosome 6, immunosuppression, perineural or blood vessel invasion, multiple recurrences, and prior radiation therapy.[8,9] Our patient has very fair skin and has had numerous recurrences of both basal cell and squamous

cell carcinoma. Perineural, perivascular, and perimuscular invasion was noted during surgical excision. He has no known history of immunosuppression, radiation therapy, or trisomy chromosome 6.

The main initial therapy for metastatic BCC is wide surgical excision of the local metastasis. It is imperative that the excised tissue has clear margins as there is only a 5% recurrence with clear margins, and 30% recurrence for positive margins. Margin clearance is usually established utilizing a Mohs procedure.[10,11] Our patient was not a candidate for a Mohs surgery due to the deep anatomical location as well as the lymphatic and vascular involvement of the metastatic lesion, thus requiring general surgery axillary dissection. Retrospective studies involving patients with lymph node metastasis have shown improved regional control rates with surgical excision followed by adjuvant post-surgical bed radiation. These findings have led the American Society for Radiation Oncology to recommend therapeutic lymphadenectomy followed by adjuvant RT for patients with clinically apparent regional lymph node metastasis.[12] Our patient was considered high risk for recurrence given the unclear margins and the lymph node involvement found on his pathology report. These findings suggest that the patient would have been a great candidate for RT of his post-surgical bed, and that is why the referral was made to radiation oncology. Although the patient has avoided RT thus far, he has continued to follow up with radiation oncology for further monitoring and management.

There is little evidence to support the use of chemotherapeutics in conjunction with RT. Chemotherapy is usually indicated for palliative matters in patients with unresectable distant metastasis.[12] Chemotherapy with 5-FU, cisplatin, vincristine, etoposide, bleomycin, cyclophosphamide, methotrexate, or doxorubicin alone or in combination was the standard regimen used when indicated.[11,13] Although there are many options, historically, cisplatin was believed to be the most effective chemotherapeutic agent.[11,13] A more recent study found that a chemotherapeutic regimen of vincristine, bleomycin, and prednisolone was the most effective.[11,14] However, the discovery of hedgehog signaling pathway inhibitors such as vismodegib and sonidegib have provided more advanced treatment regimens with more specific targets. These synthetic molecules act on the direct pathway responsible for BCC tumor growth. In theory, these agents should be nearly curative, but a recent study found that 50% of BCC cases are resistant to treatment, and another 20% had recurrence after the initial trial. This resistance can be overcome with the use of intravenous arsenic oxide and oral itraconazole conjunctively with vismodegib/sonidgeib.[15]

Although BCC metastasis is rare, historically when it does metastasize it has a relatively poor prognosis with a median survival of 10 months, despite 44% of patients receiving adjuvant chemotherapy or radiation. The historical data suggest that radiation and chemotherapy have not significantly improved metastatic BCC survival.[16] However, a more recent study performed in 2021 reviewed 22 cases and had a median survival of 66 months (95% confidence interval: 15-117). In this most recent study, all patients underwent surgical excision for management of primary and metastatic tumors. Seven received radiation therapy only, two received radiation and chemotherapy, and one received chemotherapy only. Inhibitors of the hedgehog signaling pathway were utilized as a part of the therapy in all three patients who received chemotherapy. Overall survival rates in this study were 56% at five years and 27% at 10 years.[2]

There is no definite chemotherapeutic protocol for metastatic BCC; however, there are increasing advancements with sonic hedgehog pathway inhibitors. These advancements have boasted improvements in survival. Although improved, the prognosis remains rather dismal. Despite this unfavorable outcome, it appears the data suggest the most important aspect of metastasis prevention/survival is complete wide excision of the primary tumor and local metastasis.

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Ruptured Ovarian Artery Aneurysm Complicating a Term Vaginal Delivery in the Early Puerperium Period: A Case Study

Mitchell B. Van Kalsbeek, MS III; Kahlen Morris, MS III; and Katherine Degen, MD

Abstract

Spontaneous rupture of a gonadal artery aneurysm is an extremely rare occurrence and can lead to a rapidly life-threatening retroperitoneal hemorrhage. We report a case of a pregnancy-related spontaneous rupture of a right ovarian artery aneurysm in a multiparous woman. A 37-year-old woman, gravida 4, para 4, presented to an outside facility with right flank pain roughly six hours following spontaneous vaginal delivery of full-term baby with complication of advanced maternal age (AMA). Computerized tomography at our facility revealed contrast extravasation from a right ovarian artery aneurysm and retroperitoneal hematoma. Successful management was achieved via selective angiography and embolization using both micro-coils and gel-foam. Diagnostic angiography followed by transcatheter arterial embolization is an effective and minimally invasive technique for management of ovarian artery aneurysm.

Introduction

Spontaneous rupture of an ovarian artery aneurysm (OAA) is an extremely rare complication that can occur in the peripartum and puerperium period that can lead to rapidly growing and life-threatening retroperitoneal hemorrhage. The majority of documented cases of ruptured OAA in the English literature occurred during the peripartum and early puerperium periods. They historically include the use of laparotomy for hematoma evacuation and typically achievement of hemostasis via adnexectomy.[1-4] Recently, the use of percutaneous and transcatheter arterial embolization (TAE) have become increasingly utilized treatment options for management of ruptured OAA as opposed to historical laparoscopic surgical repair methods. However, TAE can be a difficult procedure due to the tortuosity and likelihood for vasospasm of the ovarian artery.[5,6]

In this paper, we report a case of pregnancy-related rupture of an OAA in a multiparous woman of advanced maternal age, successfully managed with percutaneous TAE. We discuss proposed etiologies of this pathology as well as the surgical intervention of this pathology.

Case Presentation

A 37-year-old woman, gravida 4 para 4, birthed a healthy infant via uncomplicated spontaneous vaginal delivery at an outside hospital. Six hours later, she reported right flank pain, became hypotensive, and postpartum hemorrhage was suspected. Exploratory laparotomy revealed a retroperitoneal hematoma (Figures 1A-B) that could not be managed at the current facility. Prior to transfer to our facility, the patient experienced rapid decompensation with blood pressure in the 60/40's. Prior to arrival, she was intubated and stabilized for transport. Computerized tomography (CT) with contrast and CT angiogram (CTA) of the abdomen and pelvis revealed a 272 mm x 197 mm retroperitoneal hematoma, an adjacent right-sided ovarian artery aneurysm (OAA), and an intact inferior saccular aneurysm of the proximal left renal artery. A second OAA was also present at the take-off point from the abdominal aorta.

Following a multidisciplinary discussion, percutaneous angiography and embolization was determined for the treatment of the retroperitoneal bleed. Interventional radiology approached the bleeding site by puncturing the right common femoral artery via ultrasound guidance. Imaging visualized the characteristic tortuosity of the right ovarian artery and the presence of an aneurysm with active extravasation of contrast (Figure 1C). Embolization of the artery was conducted through the use of a 5 French catheter, to initially occlude the more distal ovarian artery via deployment of a *Gelfoam* slurry, followed by placing 3, 7 mm embolization coils more proximally to the aneurysm. The aneurysm was successfully embolized via the use of a gel-slurry composed of N-methyl cyanoacrylate, which was positioned 3-5 cm proximal to the rupture. Additional comparisons

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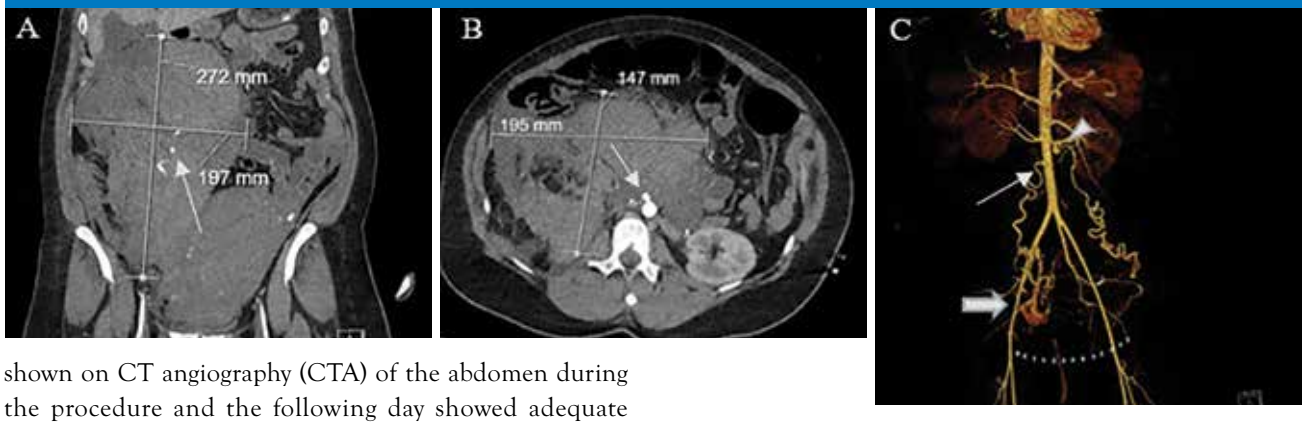
Figure 1. A) Coronal view of abdominal CT with IV contrast displaying a 272 mm x 197 mm retroperitoneal hematoma with enhancement of tortuous right ovarian artery near location of aneurysmal rupture. B) Cross-sectional view of abdominal CT with IV contrast displaying OAA at aortic take-off (thin arrow) and a 147 mm x 195 mm intra- and retroperitoneal hematoma. C) CT with volume rendering technique displaying OAA and extravasation of contrast dye (thick arrow), OAA at aortic take-off point (thin arrow), and saccular aneurysm of the left renal artery (triangle).



shown on CT angiography (CTA) of the abdomen during the procedure and the following day showed adequate occlusion with ceased retroperitoneal hematoma growth (Figures 2A-D).

Further imaging the following day and on post-op days 9 and 12 revealed no residual bleeding nor additional aneurysms throughout the body. Given the stability of the saccular aneurysm at the left renal artery, this aneurysm would require future follow-up for coil-placement after resolution of the retroperitoneal hematoma. During this time, discussion with the patient did not reveal a past personal or family history of aneurysms.

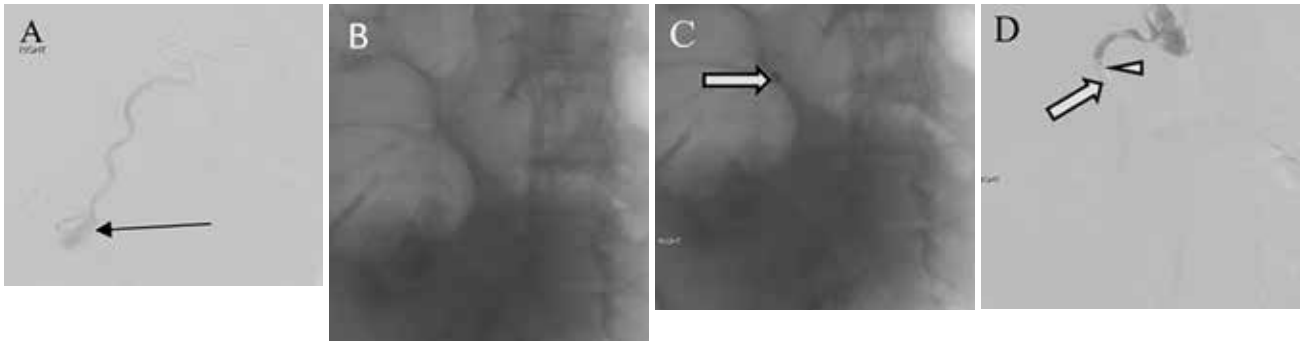
The patient's hospital stay was 12 days and was complicated on post-op day 9 when the patient complained of shortness of breath. Chest CTA revealed bilateral pulmonary emboli and venous ultrasound revealed bilateral lower extremity deep vein thromboses. The patient was started on a heparin drip due to her being a poor candidate for an inferior vena cava (IVC) filter due to compression of the IVC by the still-present retroperitoneal hematoma.

The patient later elected to cease breast-feeding and transitioned to oral apixaban on post-op day 11. The patient was discharged the following day with follow-up with pulmonology in two to four weeks.

Discussion

The mechanism of ovarian artery aneurysm formation and rupture has not been fully elucidated due in part to this pathology being silent until it progresses to clinically emergent situations. However, several important factors have been implicated in playing a role; such as hemodynamic changes associated with pregnancy and delivery as well as pregnancy-associated hormonal changes. Pregnancy-associated hemodynamic changes that have been implicated include increased cardiac output, increased total blood volume, and increased vascular demand of the uterus during gestation. These mechanical forces lead to local damage causing release of vasoactive elements which may impact vascular integrity and remodeling.[3,7,8] Hormonally regulated microvascular changes seen at the uteroplacental junction have similarly been implicated in the vulnerability of the ovarian and uterine arteries to aneurysm formation. High levels of

Figure 2. A) CTA right OAA showing active extravasation of contrast dye (thin arrow). B) CTA displaying transarterial catheter advancement into the R. ovarian artery via the R. femoral artery route. C) Release of microcoils (thick arrow) into vessel wall with partial occlusion. D) Release of microgel slurry (triangle) proximal to microcoils (thick arrow).



circulating steroid hormones such as estrogens, increase the likelihood of pathological changes in the arterial intima and media.[2,7,9] Improper involution of the uterus in the late pregnancy and immediate puerperium periods may also be a predisposing factor in arterial rupture, as the majority of cases reported in literature (20/33 of 33, or 60%) occurred between 39 weeks gestation and 5 days postpartum (21 out of 33 in one month). Burnett and Carfrae have postulated that during normal involution of the uterus, failure of a segment or segments of the ovarian artery may fail to simultaneously involute, predisposing this section of the artery to aneurysm formation in subsequent pregnancies.[2,3,10]

Multiparity may also play a role in the development of ovarian artery aneurysm.[11] Both ovarian artery and uterine artery aneurysms (UAA) may rupture in the intrapartum and immediate puerperium period. However, because distal uterine artery branches penetrate into the myometrium, uterine contraction and involution may play a protective role in reducing excess blood loss in the case of a ruptured UAA. The ovarian artery, on the other hand, traverses retroperitoneally and external to the uterus along the infundibular pelvic ligament and is not subjected to the same protective effect offered by uterine contraction.[3,9,10,12]

In addition, the clinical picture of ovarian artery rupture may also be clouded during the concurrent childbirth occurring. Epidural anesthesia and narcotics interfere with accurately identifying and localizing pain during the time of the rupture. Similarly, hemodynamic instability and blood loss may be inappropriately attributed to intrapartum and postpartum blood loss, with volume replacement via crystalloid solutions possibly masking the real cause until much later in the postpartum period.[9]

The summation of these factors likely explains why our patient was at an increased risk for an OAA rupture, as increased stress placed on arterial walls may have increased susceptibility to aneurysmal dilation, as seen at the ovarian takeoff point and renal artery.

Conclusion

Ovarian aneurysm rupture resulting in flank pain and retroperitoneal post-partum hemorrhage is a rare event with few documented cases in literature. This case illustrates important lessons to point towards rapid diagnosis and management of a ruptured OAA. First, a high index of suspicion for vascular compromise should be held for a patient complaining of atypical (right) flank pain, especially in multigravidas during or shortly following labor. Second, due to the anesthetic effects of epidural anesthesia and narcotics used during labor, spontaneous rupture of an ovarian artery should be a differential diagnosis in the acute postpartum/puerperium period when clinical symptoms do not correlate to the amount of blood loss seen at the time of delivery. Prompt diagnosis, intervention, and the use of a multidisciplinary team were crucial for successful management in this case. Furthermore, diagnostic angiography followed by percutaneous and TAE is an effective and minimally invasive technique for management of ovarian artery aneurysm.

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Cocaine Use Presenting as Concomitant Myocardial Infraction and Transient Ischemic Attack – A Case Report

Muhammad Asif, MD; and Wahab Jahangir Khan, MD

Abstract

Cocaine abuse with its complications is a common problem that presents often in the emergency room. Complications of cocaine use can involve multiple systems. These complications can arise within each system simultaneously or at different times. We treated a patient who presented with symptoms of cerebrovascular accident and was found to have concomitant non-ST segment elevation myocardial infarction (NSTEMI). A 54-year-old male with medical history significant for hypertension and prior MI presented to emergency department with left leg and arm numbness first noticed when he woke up in the morning of presentation. He admitted using cocaine the night prior to presentation. Neurological exam was remarkable for decreased sensation to left extremities. His National Institute of Health Stroke Scale (NHISS) score was 1. Blood work was significant for an elevated troponin I of 1.74 ng/ml [nl <0.80 ng/ml], and an elevated Creatinine of 2.34 mg/dl [nl 0.60-1.20 mg/dl]. CT head and MRI brain were negative for acute intracranial hemorrhage or radiological evidence of stroke. He was treated with aspirin, clopidogrel, statin and therapeutic enoxaparin for NSTEMI. His symptoms of left sided numbness resolved over the course of his stay. This case underscores why cocaine abuse should always be considered in the differential for patients presenting with symptoms suggestive of acute coronary syndrome or stroke, especially in young and middle-aged males.

Introduction

Cocaine is widely used as a drug of abuse by an estimated 18.2 million people worldwide.[1] Cocaine is the illegal drug most often associated with visits to U.S. hospital emergency departments. In 2011, it was involved in an estimated 40.3 percent of illicit drug-related emergency department visits.[2] Cocaine use can lead to a variety of complications to different organ systems such as seizures, strokes, myocardial infarctions, arrhythmias, cardiomyopathies, aortic dissections, nasal septal perforations, and gastric ulcers. We present a case of a patient with a past medical history of cocaine abuse who presented with concomitant non-ST segment elevation myocardial infarction (NSTEMI) and transient ischemic attack (TIA).

Case Presentation

A 54-year-male with medical history significant for hypertension and prior MI presented to the emergency department with left leg and arm numbness first realized upon awakening the morning of presentation. He admitted using cocaine the night prior to presentation. Blood pressure was 110/82 mmHg and heart rate 112 bpm. Stroke evaluation was performed and his National Institute of Health Stroke Scale (NHISS) score was 1 for partial loss of sensation to light touch and pin prick on left lower and upper extremities. He was evaluated by neurology and was deemed not a candidate for tissue plasminogen activator due to low NIHSS score. Blood work was significant for elevated troponin I, peaking at 1.74 ng/ml [nl <0.80 ng/ml] and elevation of creatinine of 2.34 mg/dl [nl 0.60-1.20 mg/dl]. Initial CT head was negative for acute intracranial hemorrhage or other acute cerebral disease. He was started on aspirin, clopidogrel, statin and therapeutic enoxaparin for concurrent NSTEMI and TIA. Beta blockers were held in view of recent cocaine abuse and angiotensin converting enzyme inhibitors were held initially due to acute kidney injury. The echocardiogram and carotid dopplers were negative for thombi or hemodynamically significant carotid artery stenosis. Telemetry was negative for intermittent arrythmias. Due to a mildly decreased ejection fraction 45-50% [nl 50-75%] on the echocardiogram, cardiac catheterization was performed; this revealed a normal left ventricular ejection fraction and stable coronary artery disease. MRI of the brain without contrast was done and found to be negative for acute stroke. Counselling for cessation of substance use was done. The patient was seen by physical and speech therapies. His symptoms of left sided

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numbness resolved over the course of his stay and his renal function improved with hydration. Subsequently, he was started on nitrates and discharged home to follow up as an outpatient.

Discussion

Cocaine abuse should always be considered in the differential diagnosis for patients presenting with symptoms suggestive of acute coronary syndrome (ACS) or stroke, especially in young and middle-aged males.[3-7] A dedicated history and urine toxicology screen can assist in detecting cases of cocaine induced complications. Cocaine results in tissue ischemia via multiple mechanisms including vasospasm, along with inflammation resulting in vasculitis and subsequent thrombus formation in both the coronary and cerebral vessels. Cocaine also weakens the vascular walls and increases the likelihood of vessel dissection and subsequent intracranial/subdural hemorrhage. Therefore, when evaluating a patient with symptoms of ACS and stroke; cocaine use must be considered as a possible mechanism that leads to these conditions. Patients having cocaine induced myocardial infarction generally are younger and have fewer conventional known cardiovascular risk factors than patients with acute coronary ischemia associated with a ruptured plaque. Management of cocaine mediated ACS and stroke is similar to standard care for these emergencies. One exception includes low sensitivity of electrocardiogram (EKG) to predict the magnitude of myocardial infarction in cocaine associated chest pain. Moreover, beta-blockers are contraindicated in acute cocaine toxicity at least for the initial 24 hours to avoid unopposed alpha-1 activity that can promote vasoconstriction and tissue ischemia.[5] If beta blockers have to be used; then non-selective ones are recommended and should be cautiously used following administration of nitrates, benzodiazepines and calcium channel blockers. Phentolamine, an alpha-adrenergic antagonist, can be used to reduce cocaine-induced coronary artery vasoconstriction when managing cocaine associated chest pain or hypertension that is unresponsive to benzodiazepines. Comprehensive counselling for secession of cocaine use is very important but the rate of recurrence is still significant.[6]

Conclusions

This case report identified a patient who presented with symptoms of stroke and was found to have concomitant non-ST segment elevation myocardial infarction secondary to cocaine use. Cocaine abuse with its complications is a common problem that presents often in the emergency room. Complications of cocaine use can involve multiple systems. These complications can arise within each system simultaneously or at different times. Fortunately, patients with ACS caused by cocaine abuse are relatively younger, have fewer cardiovascular risk factors and have more favorable outcomes than those who present with ACSs of more traditional etiologies.

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Ischemic Cerebellar Stroke in a Young Patient With Neurofibromatosis Type 2: A Case Report

Ariadne Trautman, MS IV; and Divyajot Sandhu, MD

Abstract

Background: Neurofibromatosis type 2 (NF2) is a multifaceted disease characterized primarily by benign CNS tumors, especially meningiomas and vestibular schwannomas. There have been several cases of brainstem ischemic stroke in young NF2 patients reported in the literature. To date, there is no unified theory about the connection between NF2 and pediatric stroke.

Methods: We present a case of ischemia in the left cerebellar peduncle of a young patient with NF2, as well as a narrative review of the literature of previous cases.

Results: Serial magnetic resonance images (MRIs) displayed an initial area of restricted diffusion in the left cerebellar peduncle with peripheral enhancement which did improve over several months, consistent with maturing infarct.

Conclusions: Our case joins several others with similar presentations and findings. The primary theory for the cause of brainstem ischemia in juvenile NF2 is a microvascular cause due to deficiency of neurofibromin 2, a regulator of endothelial development. Multicenter studies with large NF2 cohorts are needed to better characterize this syndrome.

Introduction

Neurofibromatosis type 2 (NF2), an autosomal-dominant inherited disorder, can present in a variety of ways. Hearing difficulties are a common first symptom and bilateral vestibular schwannomas are pathognomonic for the syndrome. Meningiomas and other noncancerous neural growths are also common.[1] Other manifestations include juvenile cataracts, retinal hamartomas, subcutaneous peripheral nerve tumors, scoliosis, and neuropathy.[2] Over 400 mutations in the *neurofibromin 2 (NF2)* gene can cause NF2. The *NF2* gene produces the protein *merlin* (also known as *schwannomin),* a regulator of cell shape, growth, and adhesion in the nervous system. Most mutations result in a truncated version of *merlin* which can no longer perform its regular function, leading to the growth of neural tumors. [1] While cerebrovascular disease is a known cause of morbidity and mortality in neurofibromatosis type 1 (NF1),[3] it is considered a rare manifestation of NF2 with unclear etiology. Cerebrovascular insults in young NF2 patients most often involve the brainstem, typically the midbrain or pons.[4]

Magnetic resonance imaging (MRI) in these cases usually shows restricted diffusion, while vascular imaging is normal.[5] Previous reviews of case studies have found marked similarities in the presentations of NF2-associated ischemic events.[4] This suggests a possible NF2-associated syndrome of which stroke neurologists and pediatric neurologists should be aware. At this time, there is no unified explanation for the predisposition of NF2 patients to brainstem ischemia, but some variety of vascular dysplasia is a leading theory, similar to the vasculopathy that can occur in NF1, leading to aneurysms, stenosis, and occlusions in cerebral vasculature.[6] The main goal of this report is to contribute to the knowledge base on the characteristics of cerebrovascular insults in NF2 patients. We present a case of ischemia in the left middle cerebellar peduncle of a young NF2 patient.

Case Report

A 29-year-old female patient presented to the emergency department (ED) with a seven-day history of left hemifacial paresthesia, dizziness, headache, and intermittent left upper leg weakness. The patient had a history of NF2, which was diagnosed six years prior after bilateral acoustic schwannomas were found in an investigation of sensorineural hearing loss. The patient was initially seen in acute care for her symptoms several days prior to the ED visit, where she was given a dose of intramuscular ketorolac for headache and prescribed meclizine 25 mg for dizziness. She saw her primary care provider two days later and was prescribed ibuprofen 800 mg for headache and referred for audiological and ENT evaluations. Audiological evaluation the same day as her ED visit displayed bilateral sensorineural hearing loss, worse

on the right. An ENT visit later that day led to an MRI of the internal auditory canals with and without contrast being performed. When the patient called the ENT office for results, she was referred to the ED due to the MRI report indicating possible acute/subacute infarct.

In the ED, she reported headache was improved, but dizziness and left facial paresthesia persisted. She also endorsed tinnitus and left hip weakness. Vital signs were unremarkable. Physical exam revealed normal strength of extremities and cranial nerves intact apart from reduced sensation to light touch in the left 5th cranial nerve maxillary (V2) distribution. complete blood count, comprehensive metabolic panel, and protime/international normalized ratio had no significant abnormalities.

MRI of internal auditory canals with and without contrast from earlier that day redemonstrated bilateral acoustic schwannomas, several meningiomas, an abnormal fissure at the inferior aspect of the right cerebellar hemisphere suggesting dysplasia, and an enhancing lesion in the central portion of the lower medulla suggestive of an ependymoma. It also displayed abnormal signal measuring up to 2 cm in diameter within the left middle cerebellar peduncle associated with restricted diffusion and a small amount of faint peripheral enhancement. This was deemed to be of uncertain etiology and was suspicious for an acute to subacute infarct. There was also a 1 cm focus of abnormal signal at the superolateral aspect of the left cerebellar hemisphere that was thought to represent chronic ischemic change. MRA Brain without contrast showed patent carotid siphons, patent anterior cerebral arteries, patent middle cerebral arteries, and patent posterior cerebral arteries, as well as a patent vertebrobasilar system and no aneurysm. At that time, all results were discussed with the patient in the ED, and it was determined she was stable for discharge home with close follow-up by vascular neurology within one week with repeat MRI brain. She was started on low dose aspirin (81 mg) and prednisone 10 mg with taper.

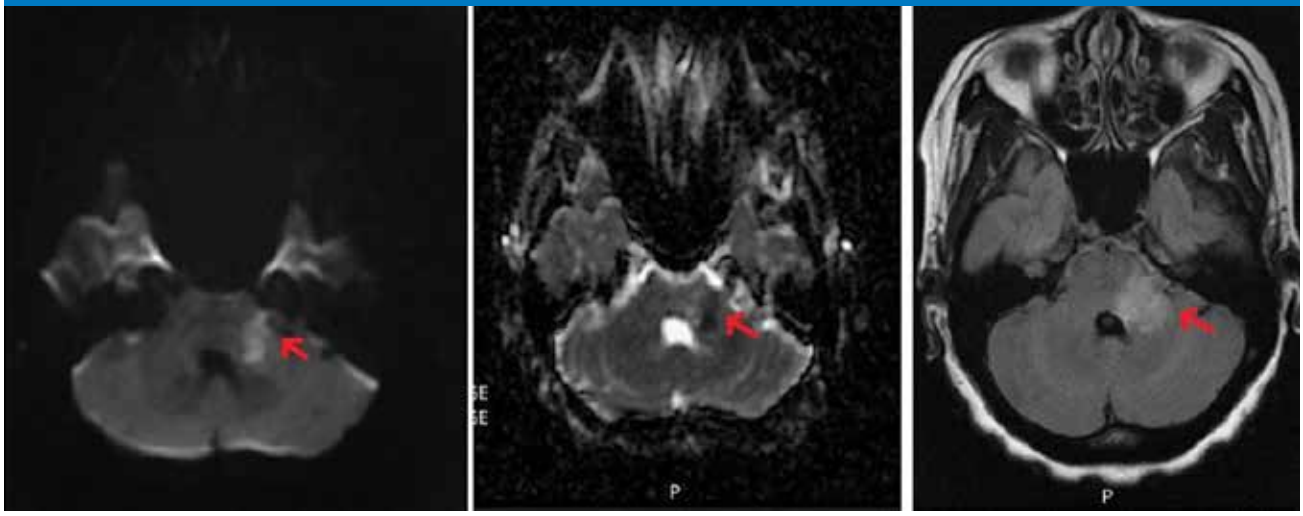
One week later, she was seen in the office by vascular neurology. Her headache was resolved, but she still had intermittent dizziness, left facial paresthesia, and intermittent left leg numbness. MRI brain with and without contrast showed stability of the restricted diffusion, fluid attenuated inversion recovery (FLAIR) hyperintensity and peripheral enhancement in the left middle cerebellar peduncle with stable sites of probable ischemic change in the left cerebellar hemisphere, as well as the chronic NF2-associated tumors (Figure 1).

At follow-up six weeks later, she reported continued left facial numbness and intermittent dizziness. MRI showed previously noted FLAIR hyperintensity in the left middle cerebellar peduncle significantly decreased in size and the restricted diffusion was diminished as well, consistent with a maturing infarct. At her most recent follow-up approximately three months after initial symptoms, patient still had intermittent dizziness and headaches with left facial numbness and left shoulder numbness. Neurologic exam was described as normal. Follow up MRI Brain showed continuing resolution of the area of restricted diffusion in the left cerebellar peduncle. Aspirin 81 mg daily was continued.

Discussion

NF2 is a complex disorder with a myriad of effects. There are many similarities between our patient's case and others in the

Figure 1. Initial MRI brain with and without contrast: DWI, ADC, and FLAIR images showing FLARE hyperintense peripherally enhancing focus in the left middle cerebellar peduncle with associated restricted diffusion stable from initial MRI.



mat
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literature. Henson et al. have proposed "brainstem ischemia in juvenile NF2" as a name for this syndrome.[4] They explored seven prior cases of brainstem ischemia in NF2 and detailed their own case with a particularly devastating outcome of locked-in syndrome in a 25-year-old female NF2 patient with pontine and left middle cerebellar peduncle infarcts. Our patient also had ischemia in the posterior circulation, but in the left middle cerebellar peduncle, not including the brainstem. The patient in the Henson study initially had left facial numbness and a "subtle, non-enhancing focus of T2-weight hyperintensity in the left middle cerebellar peduncle adjacent to a subsequent area of restricted diffusion" on their first MRI, similar to the reading on our patient. However, serial MRIs showed multifocal bilateral spreading infarction in the pons and a biopsy of the left middle cerebellar peduncle revealed white matter that "varied from near-normal to necrotic" with axonal swelling, infiltration of macrophages and "scant lymphocytes."[4] In our case, the improvement of the area on serial MRIs was consistent with a maturing infarct.

Multiple other cases of brainstem ischemia in young NF2 patients exist in the literature. Bonne et al. report on a 6-year-old for whom brainstem ischemia was the initial presentation of NF2.[7] Gugel et al. detailed three cases occurring in patients aged 7, 13, and 22 where brainstem ischemia was the presenting symptom of NF2. One patient had a biopsy that revealed reactive astrogliosis and activation of microglia. The strokes in these 3 patients were in the left ponto-medullary transition/cerebellar peduncle, in the left cerebral peduncle and in the right pons, respectively. They all experienced hemiparesis and two of them had dizziness and facial palsy, similar to our patient. The paper also alluded to three prior cases of NF2-associated juvenile ischemic strokes from the literature and noted that 5/6 of the insults were in the midbrain/brainstem area and that MRA's did not find any macroangiopathic abnormalities.[5]

No definite explanation has been found for the predisposition of young NF2 patients to brainstem ischemia. Cerebral blood flow abnormalities are known to occur in NF1, often in the posterior circulation.[8] Arteriography in the NF2-associated ischemia cases has largely been normal, although it is worth noting that vertebrobasilar abnormalities can be missed on conventional MRA.[9] Thus, a microvascular cause is a common theory for the predilection for ischemic events in NF2.[5,10] The tumor suppressor protein *neurofibromin 2*, also known as *merlin*, is thought to also play a role in vascular endothelial development,[10] likely through promoting apoptosis.[11] It has been shown that deficiency of this protein, as in neurofibromatosis 2, has been associated with neointima hyperplasia following vascular injury in animal models,[12] suggesting that blood vessels in those with NF2 may be more prone to stenosis in response to endothelial injury. Additionally, *merlin* has been shown to regulate angiogenesis of the nervous system by interacting with antiangiogenic factors such as semaphorin 3F.[13] The absence of *merlin*, therefore, is likely to disrupt vascular homeostatic mechanisms.

Another possibility is a digenic process (phenotypic effects manifested only when two genes interact), especially given the rarity of brainstem ischemia in juvenile NF2. Other cerebrovascular abnormalities such as aneurysms have been noted in NF2. One study found a prevalence of 4.4% for aneurysms in NF2 patients, compared with 3% in the general population.[14] Additionally, there have been cases of NF2 patients also experiencing moyamoya syndrome.[15] Whole genome sequencing in patients with NF2 and cerebrovascular abnormalities could provide more information. Treatment guidelines for these patients will partly depend on establishing a cause for their ischemia. However, it is reasonable to recommend typical stroke management such as in this patient with low-dose aspirin and close follow-up. Several authors have recommended avoiding the use of bevacizumab, which is sometimes prescribed in NF2 patients to reduce tumor size, as it can be associated with increased cerebrovascular accident risk.[5,10,16] Continued MRI and MRA surveillance as well as cerebral blood flow studies in patients with NF2 and acute stroke have also been proposed.[16]

Conclusion

This case contributes to the increasingly recognized syndrome of brainstem ischemia in young NF2 patients and shares many similarities with prior cases in the literature. Many theories exist for this correlation, with cerebral vasculopathy in posterior brain circulation being the dominant proposed cause. Population-based studies on large NF2 cohorts would reveal more information about the prevalence and potential causes of this condition. Understanding this syndrome of brainstem ischemia in young NF2 patients will inform the development of treatment guidelines for such patients.

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Mature Cystic Teratoma Mimics Heterotopic Pregnancy on Sonograph

Peter Wilson, MS IV; Ashley Durant, MS IV; and Katherine Degen, MD

Abstract

Introduction: Mature cystic teratomas (MCTs) present clinically with a wide host of different symptomatic manifestations. Because they are often comprised of multiple tissue types, imaging can also take on many different forms making the diagnosis of MCT difficult in certain situations. In this case, we present a patient who had a MCT that mimicked a heterotopic pregnancy on sonograph.

Case Presentation: A 21-year-old female G1P0 at approximately six weeks gestation by last menstrual period presented to the emergency department with a chief complaint of vaginal bleeding that began the previous day. A transvaginal ultrasound demonstrated a viable intrauterine pregnancy with fetal cardiac activity and an additional structure in the left adnexa resembling a gestational sac and fetal pole absent of cardiac activity. Obstetrics was consulted, and a preliminary diagnosis of heterotopic pregnancy was made. Diagnostic laparoscopy with intraoperative ultrasound revealed a 3 cm cyst in the left ovary that appeared to contain serous fluid. Aspiration and subsequent dissection of the cyst revealed material consistent with a mature cystic teratoma confirmed by pathologic analysis.

Discussion: This case highlights the importance of ultrasound in detecting adnexal masses while also exposing some of its limitations. Due to their variable presentation and potential to mimic other insidious pathologies, MCTs must always be considered when identifying adnexal structures on ultrasound.

Introduction

Heterotopic pregnancy (HP) refers to the presence of two or more concurrent pregnancies at different implantation sites. The risk of heterotopic pregnancy among women with a naturally achieved pregnancy ranges from 1 in 4,000 to 1 in 30,000, whereas the risk among women who have undergone in vitro fertilization is estimated to be as high as 1 in 100.[1]

The diagnosis of HP is made with a combination of clinical suspicion and imaging demonstrating two or more yolk sacs or fetal poles in two or more distinct implantation sites. In contrast to an ectopic pregnancy, Human chorionic gonadotropin (HcG) levels are not utilized in the diagnosis of HP as a portion of the measured levels results from the viable intrauterine pregnancy. Presentation varies, with 24% of cases being asymptomatic, 72% presenting with abdominal pain, and 54% presenting with vaginal bleeding.[2] Early diagnosis and removal of the ectopic pregnancy are crucial to preserving maternal and intrauterine fetal health.

A high level of clinical suspicion and thorough transvaginal ultrasound are crucial to diagnosing HP as the presentation can mimic many diagnoses such as appendicitis, threatened abortion, ruptured corpus luteum cyst, ovarian hyperstimulation syndrome, normal implantation bleeding, and many others. Free fluid in the abdomen is a nonspecific finding.[2]

In contrast, mature cystic teratomas (MCTs) can present with a wide range of symptoms, with most MCTs being asymptomatic and discovered incidentally on routine pelvic examination or imaging.[3] Symptomatic MCTs are found due to complications, with the most common being ovarian torsion (16% of teratomas), malignancy (1%-2%), infection (1%), and hemolytic anemia (<1%).[4] MCTs are the most common ovarian neoplasm (20%). They are comprised of at least two mature germ cell layers (endoderm, mesoderm, ectoderm), allowing for variable tissue within the tumor.[5,6] Ultrasound is the imaging modality of choice for female pelvic imaging, especially in patients with or suspected to have an intrauterine pregnancy. Pelvic ultrasound is beneficial in diagnosing MCTs with a reported sensitivity of 58% and specificity of 99% using a classification system consisting of three different manifestations.[7] The first and most common manifestation presents as a cystic lesion containing an echogenic mass that projects into the cystic

lumen and is often described as a Rokitansky Nodule.[8] The second manifestation results from sebaceous material and hair that causes diffuse or partial echogenicity within the cystic cavity.[9] The third manifestation is caused by hair within the cyst, resulting in multiple thin echogenicity areas.[6]

The purpose of the case report is to outline one of the many manifestations of MCTs, and to stress the importance of keeping MCTs as a differential diagnosis when evaluating pelvic masses with sonograph.

Case Presentation

A 21-year-old female G1P0 at approximately six weeks gestation by last menstrual period presented to the emergency department with a chief complaint of vaginal bleeding. The patient reported minimal brown vaginal discharge that began the evening before. Concerned about possible complications of her pregnancy, she called a nursing line and was advised to go to the emergency department for evaluation. In the emergency department, she denied any abdominal or pelvic pain, fever, nausea, vomiting, or diarrhea. Her medical history was significant only for a remote history of uncomplicated chlamydia treated with antibiotics. Family and surgical history were unremarkable. Pregnancy was achieved naturally without assistive reproductive technology.

Vital signs were as follows: BP 126/60; Pulse 103; HR 18; Temp 37.2; SpO2 98%. Physical examination revealed an anxious but comfortable woman, not in acute distress. The abdomen was non-tender, no guarding or rebound tenderness, and her uterus was nonpalpable. Pelvic examination revealed minimal dried blood in the vaginal vault with a normal-appearing, long-closed cervix without effacement or cervical motion tenderness. All other exam findings were normal.

Following physical exam, a transvaginal ultrasound was performed (Figures 1A and B). The radiology report is as follows: "There is a viable intrauterine pregnancy with a fetal heart tone of 123 bpm. However, there is also what appears to be an additional gestational sac with possible fetal pole in the left adnexa without detectable fetal heart rate. There is no significant hyperemia of the wall of this structure. Adjacent abnormal material may represent blood products." Laboratory testing revealed a quantitative hcG of 64,044 $^{mlU/mL}$. No other significant laboratory findings were noted in the workup, including complete blood count, complete metabolic panel, and urinalysis. A suspected diagnosis of heterotopic pregnancy was considered, and the patient was prepared for an urgent diagnostic laparoscopy with possible ectopic localization and removal.

Therapeutic Intervention

A laparoscopic technique was performed to evaluate the pelvis and adjacent structures. Upon examination of the pelvis, no tubal ectopic pregnancy nor hemoperitoneum was found. There was a trace amount of normal pelvic free fluid. There was a prominent right-sided corpus luteum. The left ovary had a 3 cm cyst that appeared to contain another serous-

Figure 1A. Freeze frame image of transvaginal ultrasound showing a viable intrauterine pregnancy. Fetal heart tone of 123 bpm was recorded with ultrasound during examination. "FHT" was denoted by ultrasound device to mark evidence of fetal heart tone. Red arrow refers to fetal pole. White arrow refers to uterine myometrium.

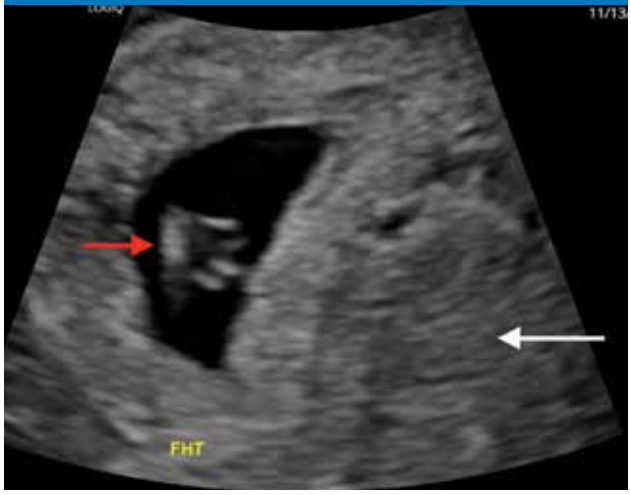
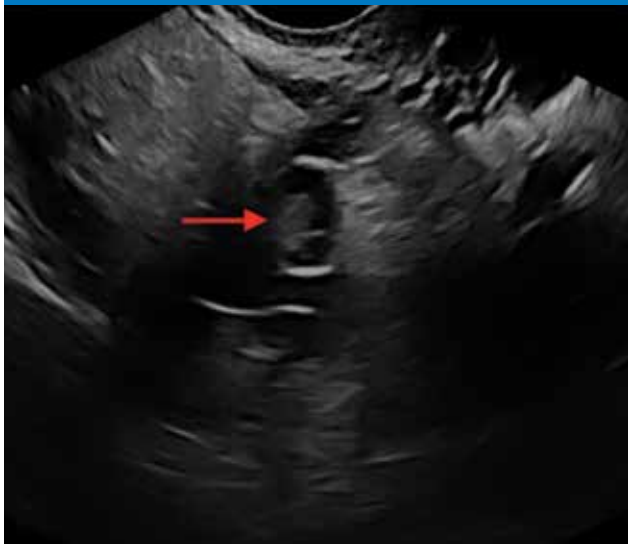


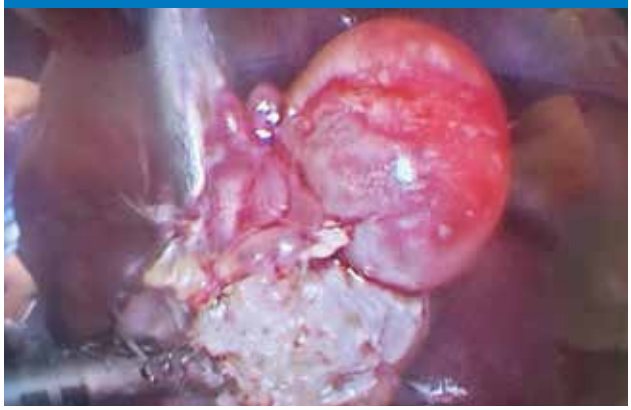
Figure 1B. Freeze frame image of transvaginal ultrasound showing structure in left adnexa. No fetal heart tones were detected on ultrasound device. Red arrow refers to suspected fetal pole.



filled cyst. There was no sign of an extrauterine pregnancy in the abdomen or pelvis. Intraoperative transabdominal pelvic ultrasound was obtained to further evaluate the left adnexal mass. The left ovary did not have any hyperemia expected with an ovarian ectopic. Transvaginal ultrasound confirmed the previously visualized ectopic structure was intraovarian. There was no heartbeat in the left fetal pole. The intrauterine pregnancy maintained a healthy heartbeat intraoperatively. A collective decision was made to incise the left ovarian cyst to look for the presence of an ectopic pregnancy within the left ovary. Of note, there was very little blood flow to the suspected ectopic gestational sac.

Aspiration of the left cyst revealed turbulent, serous fluid and hair, raising suspicion of a dermoid. The cyst wall was opened, and caseous material was retrieved. A suspected gestational sac was observed within the larger cyst (Figure 2). The cyst was excised, and the rest of the cyst contents were irrigated and suctioned. There was little spillage of the contents. The cyst wall was removed, and hemostasis was achieved. Both ovaries and tubes were left intact. The final Pathology report was consistent for benign mature cystic teratoma.

Figure 2. Contents of left ovarian cyst after excision and drainage of serous contents.



Discussion

Upon further review of the transvaginal ultrasound images, it is evident that this patient presented with common radiological findings consistent with a MCT. Figure 1B shows a cystic structure with an intracystic mass at the lateral pole. While the structure was initially suspicious for a yolk sac, confirmation of the MCT by pathology found it to be consistent with a Rokitansky nodule (dermoid plug). Figure 1A shows the intrauterine fetal pole with fetal heart tones present. In comparing these images, many similarities can be noted, prompting exploration for the suspected heterotopic pregnancy. Figure 1B closely resembles both a fetal pole and a Rokitansky Nodule. On closer inspection of Figure 1B, there is little evidence of thin or diffuse areas of echogenicity even though sebaceous fluid and hair were excised from the cyst. The adnexal structure also did not have the characteristic peripheral vascular enhancement on ultrasound that is present with intrauterine and ectopic pregnancies.

This case highlights the importance of ultrasound in detecting adnexal masses while also exposing some of its limitations. While pelvic ultrasound is an excellent tool for detecting ovarian cysts and other adnexal masses, this case illustrates these structures' clinical and radiologic ambiguity. Of all adnexal masses, MCTs possibly have the most variable presentation on ultrasound due to their often-heterogeneous composition. The patient presented without peritoneal signs, making ruptured heterotopic pregnancy less likely, but the possibility of ectopic pregnancy warranted surgical exploration. MCTs are not a surgical emergency unless accompanied by ovarian torsion but may still present as more ominous diagnoses. Due to their variable presentation and potential to mimic other potentially insidious pathologies, MCTs must always be considered when identifying adnexal structures on ultrasound.

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Statins Therapy and Intracranial Hemorrhage

Fouad Khalil, MBBCh; Mashood Badshah, MD; Swaminathan Perinkulam Sathyanarayanan, MD; and Nessim Amin, MD

Abstract

Statins have grown to become an important class of medications in the primary and secondary prevention of cardiovascular diseases. While lowering low-density lipoprotein-C (LDL-C) levels, statins also have a role in stabilization and regression of atherosclerotic plaques. In the last two decades, there is a mounting body of evidence that suggests that statins and lowering serum lipid levels may be associated with an increased risk of developing intracerebral hemorrhage (ICH). This review article provides a concise summary of the most important studies performed to date and the pathophysiology behind the association between statins and ICH.

Introduction

Stroke, characterized by neurological deficit due to acute focal injury to the central nervous system by a vascular cause including cerebral infarction, intracerebral hemorrhage (ICH), and subarachnoid hemorrhage is a major cause of death and disability worldwide.[1] While hyperlipidemia is an established risk factor for ischemic stroke and the use of high-intensity statins is advocated following ischemic stroke,[2] some studies suggested that lower levels of serum lipids and lipid-lowering therapy may be associated with ICH. The incidence of ICH varies across countries and ethnicities accounting for 10 to 20% of all strokes.[3] The case fatality of ICH is high – 40% at one month and 54% one year with only 12% to 39% of survivors having long-term functional independence after ICH. [3] This puts healthcare providers in a situation where they have to weigh the very well-established benefit of statins against atherosclerotic cardiovascular disease versus the emerging but uncertain risk of deadly and morbid ICH associated with statins. This review article summarizes the latest evidence on the association of ICH with blood lipid levels and lipid-lowering therapies along with the theorized mechanisms behind the phenomenon.

Discussion

Statins play a major role in the prevention of atherosclerotic cardiovascular diseases including acute ischemic strokes. The primary mechanism of action of statins is through competitive inhibition of HMG-CoA reductase, the rate-limiting step in cholesterol synthesis. This leads to a reduction in intrahepatic cholesterol with a subsequent increase in LDL-C and LDL precursors uptake from systemic circulation.[4]

Other mechanisms of lipid-lowering effects of statins include inhibition of hepatic synthesis of apolipoprotein B 100 and reduction in synthesis and secretion of triglyceride-rich lipoproteins. Statins also inhibit platelets activity and have a mild anticoagulation effect via multiple mechanisms such as increased tissue factor expression and enhancing endothelial thrombomodulin expression.[5-8]Available statins approved in the U.S. are divided into hydrophilic statins which include rosuvastatin, pravastatin, and fluvastatin, and lipophilic statins which include simvastatin, lovastatin, pitavastatin, and atorvastatin.

Serum Lipids and ICH

The impact of serum triglyceride (TG) levels on the incidence of ICH is not clearly defined. A large-scale study involving 21,680 patients showed an increased incidence of ICH in patients with low TG levels.[9] Another study demonstrated a more than two-fold increase in ICH risk in patients with serum TG ≤ 83.26 mg/dL (HR 2.35, 95 % CI 1.18–4.70).[10] Conversely, in a study that involved 33,346 subjects, 147 patients with primary ICH were compared to 11,029 stroke-free controls after a mean follow-up period of 14 years.[11] Patients with ICH were found to have a significantly higher TG compared to controls (1.7 mmol/l (150.4 mg/dl) vs. 1.4 mmol/l (123.8mg/dl)).

High serum total cholesterol (TC) levels seem to be associated with a decreased risk of ICH. A multi-center study that included 2190 subjects and 2127 controls demonstrated a reduced risk of ICH in patients with high serum total cholesterol (TC) levels (OR 0.62, 99 % CI 0.42–0.92).[12] A meta-analysis that included 23 prospective studies, totaling

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1,430,141 subjects with 7960 hemorrhagic strokes also demonstrated an inverse relationship between serum TC and risk of ICH.[13] The meta-analysis also demonstrated a reduced risk of ICH in patients with higher levels of LDL. Another large-scale nested case-control study from China also authenticates the above findings revealing a positive association of LDL-c levels with ischemic stroke (IS) and a negative association with ICH.[14] The study also showed a 17% higher risk of IS (rate ratio [RR]=1.17, 95% CI, 1.10-1.25) and a 14% lower risk of ICH (RR 0.86, 0.80-0.92) for each 1 mmol/L (38.6 mg/dl) increase in LDL-C value. The recurrence of ICH is also related to LDL levels with the risk aggravated when the LDL levels go below 70 mg/dL with HR of 1.65, 95% CI (1.32-2.05) for LDL-C between 50 to 69 mg/dL and HR of 2.69, 95% CI (2.03-3.57) for LDL-C <50 mg/dL compared to LDL-C of 70 to 99 mg/dL.[15] Although the studies indicate an inverse relationship between serum TC, LDL-C, and ICH risk, the mechanism remains elusive. Proposed mechanisms include increased fragility of red blood cells, the endothelium, and arterial medial smooth muscle necrosis by low cholesterol levels.[13,16] The arterial medial smooth muscle necrosis might contribute to the development of cerebral vessel microaneurysms which is the primary driving pathology for ICH.[17]

Statin Therapy and ICH

The clinical utility of statins after ischemic stroke for secondary prevention has been well established. However, the association of statins with intracerebral hemorrhage (ICH) is [HR 1.66 (95 % CI, 1.08 to 2.55)] controversial. As of now, there are no specific guidelines regarding the use of statins after ICH. Per the landmark Stroke Prevention by Aggressive Reduction in Cholesterol (SPARCL) trial, statins were effective for secondary prevention of ischemic strokes [adjusted hazard ratio (HR) 0.78 CI, 0.66-0.94]. However, the SPARCL trial also showed statins associated with increased risk of secondary ICH after previous cerebrovascular accident [HR 1.66 (95 % CI, 1.08 to 2.55)].[18] Another observational study from Italy demonstrated a similar increased risk of ICH in statins users [odds ratio (OR), 1.54; 95% CI 1.31 to 1.81)].[19] The study also indicated an increased risk of regional lobar hemorrhages in statins users (OR, 1.58; 95% CI 1.28 to 1.96 for lobar ICH). The above findings were corroborated by another study revealing higher rates of lobar ICH in statins users (odds ratio, 1.57 [1.03-2.40]; *P*=0.038).[20] Statins also had a detrimental impact on ICH volume revealing the increased volume of hemorrhage in statins users with a median volume of 31.2ml, CI: 10-82.1 ml vs 16ml, CI: 4-43.8 ml; p=0.006).[21]

Nevertheless, other studies showed no difference in the risk of ICH recurrence between statins users and non-users and indicated improved all-cause mortality in patients who were started on statins after ICH with adjusted HR of 0.742 (95% CI 0.598-0.919).[22,23] A large scale study from Israel even showed a dose-response relationship between statins use and decreased incidence of ICH with adjusted HR for ICH 0.68 (95% CI 0.58-0.79) in those on average atorvastatin equivalent daily dose (AAEDD) of 10-19.9 mg/d, and 0.62 (95% CI 0.47-0.81) in those with AAEDD ≥20 mg/d compared to AAEDD <10 mg/d.[24] The effect of statins on ICH recurrence is also related to the type of statins being used with decreased risk of ICH recurrence in patients using hydrophilic as compared to lipophilic statins with adjusted HR of 0.69, 95% CI = 0.48-0.99.[25] Furthermore, meta-analyses of large scale studies failed to show any association between statins therapy and ICH.[26-28] Since the duration of most statins follow-up trials did not exceed 5 years, it is unclear if a longer duration of statins exposure is required to impair cerebral vessel integrity and increase the risk of ICH.

Statins therapy after ICH

Statins use after ICH is a common dilemma and a decision that is usually surrounded by uncertainty due to the lack of clinical guidelines. An important aspect that needs to be considered in this context is the risk of ICH recurrence. Patients with lobar ICH have a higher annual recurrence rate (15%) compared to patients with deep ICH (2.1%).[29] This is likely due to the difference in ICH pathophysiology between the 2 groups; cerebral amyloid angiopathy (CAA) is the main mechanism of hemorrhage in lobar ICH while hypertensive vasculopathy is responsible for the deep ICH.[30,31] While hypertension is easily controllable, there is no effective preventive therapy for CAA. Cardiovascular benefits of statins need to be balanced against the risk of ICH recurrence. Therefore, it might be reasonable to avoid statins in patients with lobar ICH as the risk of ICH recurrence might offset the cardiovascular benefits.

Another clinical aspect to consider is the acuity of ICH. Since experimental studies indicate decreased hematoma volume, cell death, and improvement in recovery with statins use, the use of statins in the acute phase of ICH may be beneficial.[32,33] Many clinical studies have also supported these findings. A study has illustrated that statins use after ICH improved short term survival and long-term outcome with OR to be alive at 30 days after ICH of 4.25 (95% CI, 3.46-5.23, P < .001) and OR for discharge to home or rehabilitation facility of 2.57 (95% CI, 2.16-3.06; P < .001).[34]

Another large-scale cohort study from South Korea also validated the beneficial impact of statins use on mortality with adjusted HR for all-cause mortality in statins users of 0.61 (95% CI 0.57-0.64).[35] Statins use after ICH was found to be associated with more generalized positive outcomes with decrease in overall mortality [HR 0.54 (95% CI 0.45-0.65)], cardiovascular death [HR, 0.54 (95% CI, 0.39-0.75)] and ICH [HR, 0.62 (95% CI, 0.46-0.83)].[36] A meta-analysis that included 17 studies investigating statins use and ICH outcomes reported improved short-term mortality outcomes in statins users and illustrated a decreased mortality with the continuation of statins after ICH (OR, .14; 95% CI, .1-.20).[37] Although there is growing evidence supporting statins use in the acute phase of ICH, it is important to highlight that robust controlled clinical trials are lacking and most of the clinical data is derived from observational studies.

Conclusion

The relationship between statins and ICH is not clearly defined. In the light of the current evidence, it is reasonable to continue statins therapy in the acute phase of ICH. Following the acute phase, the decision to continue statins therapy should be individualized. The cardiovascular benefits of statins have to be balanced with the risks of ICH recurrence. It is reasonable to discontinue statins therapy in lobar ICH patients following the acute phase. Further studies are required to delineate the relationship between statins ad ICH and guide statins therapy in this patient population.

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Tranexamic Acid in the Prevention and Treatment of Postpartum Hemorrhage: A Review

Alaire Buchholz, MS III; Keith Hansen, MD; and Rachel Rodel, MD

Abstract

Postpartum hemorrhage (PPH) continues to be one of the leading causes of maternal morbidity and mortality worldwide. The four main causes of PPH are uterine atony, lacerations, retained placenta, and bleeding diathesis. In the patient with PPH, immediate evaluation is needed to diagnose and treat the underlying cause of hemorrhage. Uterotonic agents such as oxytocin remain first line for prevention and treatment of uterine atony. Studies have evaluated the antifibrinolytic tranexamic acid (TXA) as an adjunctive therapy in the prevention and treatment of PPH. TXA has been shown to reduce blood loss, bleeding-associated mortality, and transfusion rates in a variety of clinical settings and thus may serve a role in treating PPH. Current studies have demonstrated that TXA is an effective treatment option with limited risk of adverse events in appropriately selected patients; however, additional studies are needed to further clarify the role of TXA in the prevention of PPH.

Introduction

Due to inconsistencies in the definition of postpartum hemorrhage (PPH) across countries and organizations, the incidence rate of PPH is not clearly defined, with estimates ranging from 1-10% of pregnancies around the world.[1] It has been estimated to be the cause of 33% of pregnancy-related mortalities worldwide, and between 2016-2018, hemorrhage was the cause of 11% of pregnancy-related mortalities in the U.S.[2,3] The National Center for Health Statistics reported the maternal mortality rate in the U.S. as 17.4 per 100,000 live births in 2018, with 2.1 deaths per 1,000,000 live births attributed to PPH.[4] South Dakota's maternal mortality has ranged between 6-10 pregnancy-associated deaths annually from 2011-2020 with 1.9% of these pregnancy-associated deaths due to hemorrhage.[5] It should be noted that pregnancy-associated deaths are defined as deaths within one year of pregnancy from any cause; whereas, pregnancy-related deaths are defined as deaths within one year of pregnancy as a result of a complication of pregnancy or a cause initiated or worsened by pregnancy.[5] Serious adverse effects of PPH include other important morbidities such as blood transfusions, hysterectomies, and the resulting complications of infections, infertility, and psychological stress.[6]

Several risk factors have been identified for PPH. One study published in 2013 analyzed over 8.5 million hospital births and used sequential logistic regression models to assess risk factors and trends of PPH.[7] Some of the risk factors with the highest adjusted odds ratios were cervical laceration (adjusted odds ratio [aOR], 94.0; 95% confidence interval [CI], 87.3–101.2) and uterine rupture (aOR, 11.6; 95% CI, 9.7–13.8), but these two risk factors could arguably be better labeled as causes of PPH.[7] Other strong risk factors include: placenta previa or abruption (aOR, 7.0; 95% CI, 6.6–7.3), preeclampsia (aOR, 3.1; 95% CI, 2.9–3.3), amnionitis (aOR, 2.9; 95% CI, 2.5–3.4), multiple pregnancy (aOR, 2.8; 95% CI, 2.6–3.0), fibroids (aOR, 2.0; 95% CI, 1.8–2.2), and advanced maternal age (aOR, 1.5; 95% CI, 1.5–1.6).[7] While risk factors have been identified, our ability to predict PPH is low.[8] We must therefore closely monitor all patients for PPH.

PPH was redefined by the American College of Obstetricians and Gynecologists (ACOG) in 2014 to a total blood loss of greater than 1000 milliliters (mL) or blood loss with symptoms of hypovolemia in the first 24 hours of the birthing process.[9] PPH was traditionally defined as more than 500 mL of blood lost after a vaginal delivery or 1000 mL following a cesarean delivery (CD).[10]

There are four main causes of PPH: uterine atony, trauma/lacerations, retained placental tissue, and coagulopathy. Management is dependent on the underlying cause.[11] Uterine atony accounts for nearly 70% of cases of PPH.[11] For uterine atony, the first step in management is bimanual uterine massage while simultaneously administering the uterotonic agent oxytocin.[11] Second-line medication

options include methylergonovine maleate, 15-methyl-PGF2-alpha, misoprostol, and dinoprostone. Failed medical management requires surgical intervention which may include uterine packing, balloon tamponade, uterine or hypogastric artery ligation, uterine compression sutures and hysterectomy should be considered.[11] Trauma in the form of lacerations to the lower genital tract makes up roughly 20% of cases of PPH.[11] Providers must carefully inspect and repair any lacerations with absorbable sutures.[11] Retained placental tissue accounts for about 10% of cases, and the placenta must be carefully inspected to evaluate for possible retention.[11] When there is retention of placental tissue, it is recommended that surgical evacuation under ultrasonographic guidance be performed.[11] Deficiencies of clotting factors make up less than 1% of cases of PPH, and treatment may involve replacement of clotting factors with blood products.[11] Transfusions are used to manage an acute coagulopathy caused by placental abruption or an amniotic fluid embolism where disseminated intravascular coagulation can occur.[10] Depending on the severity of PPH, transfusions may play a critical role no matter what the underlying cause of hemorrhage. It is vital to have blood that is type specific or O negative in an emergency of PPH.[10] Therefore, consider type and screen for all pregnant women and type and cross for women with an even higher risk of PPH to ensure that compatible blood is readily available. Women at increased risk of PPH should deliver at a hospital with the appropriate level of care to ensure availability of blood products.

A newer addition to treatment options is tranexamic acid (TXA). In 1962, Drs. Utako and Shosuke Okamoto discovered 1-(aminomethyl)-cyclohexane-4-carboxylic acid (AMCHA)[2], which is now known as TXA, while trying to develop a solution to reduce the rates of PPH in Japan.[12] It is an indirect antifibrinolytic agent that is a synthetic analog of lysine which works by binding and blocking lysine-binding sites on plasminogen molecules, thus reducing plasminogen's affinity to bind fibrin.[12,13] Therefore, the activation of plasminogen to plasmin is decreased, and plasmin cannot degrade fibrin into its degradation products.[12,13] Since its discovery, TXA has been demonstrated to reduce blood loss, transfusion rates, and bleeding-associated mortality in various clinical settings.[14] It was first used in the treatment of heavy menstrual bleeding and hereditary bleeding disorders.[13] It has since been used in a variety of fields such as dermatology, obstetrics, and orthopedic surgery.[14,15] However, the use of TXA in the treatment of PPH, the problem for which it was originally created, was not studied widely until more recently.

Tranexamic Acid as a Treatment for PPH

After the reported efficacy of TXA in reducing deaths due to bleeding in fields such as trauma and orthopedic surgery,[14] the World Maternal Antifibrinolytic (WOMAN) trial was conducted to determine if the drug could have the same result for reducing deaths due to PPH.[14,16] In this trial, PPH was defined as clinically estimated blood loss of greater than 500 mL following vaginal birth, 1000 mL following a cesarean delivery (CD), or any amount of blood loss that compromised hemodynamic stability.[16] Over 20,000 participants across 21 countries were enrolled in the trial from 2010-2016.[16] Participants with PPH received 1 gram of IV TXA or placebo following either a vaginal birth or CD in addition to usual management.[16] A second dose was given if the participant was still bleeding after 30 minutes or if the bleeding had resumed within 24 hours of the first dose.[16] The primary outcome of the study was deaths from any cause or a hysterectomy within 42 days of randomization.[16] Secondary outcomes of the study were thromboembolic events, surgical interventions, adverse events, quality of life, and thromboembolic events in breastfed babies.[16] The WOMAN trial demonstrated a 31% decrease in death from bleeding when TXA was given within 3 hours of delivery for the treatment of PPH.[2,16] In addition, the trial demonstrated there was no evidence of an increase in thromboembolic events or other adverse events from the use of TXA for treatment compared to placebo.[2,16] While the WOMAN trial demonstrated that TXA decreased the number of deaths due to bleeding, it did not demonstrate a decrease in the number of patients that needed a hysterectomy following PPH.[16]

A smaller study was conducted in 2011 that also set out to assess the use of TXA in patients with a diagnosis of PPH in 8 French obstetric centers.[17] PPH was defined in this study as greater than 800 mL following a vaginal delivery.[17] Nearly 150 women were randomly assigned to receive TXA in the form of a 4 gram loading dose followed by maintenance of 1 gram/hour for 6 hours or to not receive TXA at all.[17] The primary outcome of the study was blood loss, and secondary outcomes included: PPH duration, anemia, transfusion, and the need for invasive procedures.[17] The study was not powered to address safety issues, but adverse events were reported to only be mild and transient.[17] The study demonstrated a significant difference in blood loss between the groups as well as decreased maternal morbidity, further supporting the use of TXA in the treatment of PPH.[17] From the time of diagnosis of PPH to six hours later, the group that received TXA lost a median of 173 ml of blood, and the group that did not receive TXA lost a median of 221 ml.[17]

Tranexamic Acid as a Prevention for PPH

Inspired by the success of TXA in the treatment of PPH, the Tranexamic Acid for Preventing Postpartum Hemorrhage Following a Vaginal Delivery (TRAAP) trial was created to assess the efficacy of prophylactic TXA in addition to oxytocin in preventing PPH in women undergoing vaginal delivery.[18] In this trial, PPH was defined as blood loss of 500 ml or greater.[18] Approximately 4,000 participants from 15 maternity units across France were enrolled in the study from 2015-2016.[18] Participants were randomized to receive either 1 gram of TXA intravenously or placebo within two minutes after delivery in addition to prophylactic oxytocin.[18] The primary outcome of the study was PPH.[18] The results demonstrated that the rate of PPH in women undergoing vaginal delivery who received TXA was not significantly lower than the rate in those that received the placebo, and the data was not statistically significant.[18] A notable reported outcome was that there was no increase in thromboembolic events in the TXA group although nausea and vomiting were more common than in the placebo group.[18]

The World Maternal Antifibrinolytic-2 (WOMAN-2) trial is currently underway to determine the usefulness of prophylactic TXA in the prevention of PPH in patients with anemia who have undergone vaginal delivery.[19] Patients identified with anemia will either receive 1 gram of TXA or placebo within 15 minutes of the umbilical cord being clamped.[19] The primary outcome will be the diagnosis of PPH.[19] While the results of the TRAAP trial failed to demonstrate a decreased incidence of PPH in the general population, the results of the WOMAN-2 study will provide valuable insight into the possibility of using prophylactic TXA in targeted populations such as those with anemia.[18,19]

The Tranexamic Acid for Preventing Postpartum Hemorrhage Following a Cesarean Delivery (TRAAP2) trial sought to assess the efficacy of prophylactic TXA in addition to oxytocin in preventing PPH in women undergoing CD.[20] In this study, PPH was defined as blood loss greater than 1,000 ml or a red-cell transfusion within two days of delivery.[20] Between 2018 and 2020, 4,551 participants were enrolled and randomized to receive either 1 g of TXA or placebo IV in the three minutes following birth in addition to a prophylactic uterotonic agent.[20] The primary outcome of the study was PPH.[20] The study demonstrated a significantly lower incidence of PPH and blood transfusions within two days of delivery for the group that received the prophylactic TXA.[20] PPH occurred in 26.7% of women that received TXA and 31.6% that received a placebo with an adjusted risk ratio of 0.84 (95% CI 0.75-0.94; p=0.003).[20] However, the incidence of secondary outcomes related to PPH was not significantly lower than the placebo group.[20] The secondary outcomes that were assessed were gravimetrically estimated blood loss, provider-assessed significant PPH, use of additional uterotonic agents, and postpartum blood transfusion.[20]

A recent meta-analysis published in the *American Journal of Obstetrics and Gynecology* assessed the use of prophylactic TXA in women who had a CD.[1] The meta-analysis included 36 studies with 10,659 participants.[1] The outcomes demonstrated lower postpartum blood loss, less of a hemoglobin drop, and a lower transfusion requirement with the use of TXA in addition to a standard prophylactic uterotonic agent.[1] The administration of TXA resulted in a mean difference (MD) of -189.44 mL (95% CI, -218.63 to -160.25) when comparing total blood loss with TXA administration to placebo.[1] The MD for lower hemoglobin drop with prophylactic TXA was 8.22% (95% CI, 5.52-10.90).[1] When comparing the need for transfusion in women receiving prophylactic TXA compared to placebo, the OR was 0.41 (95% CI, 0.26-0.65).[1] The authors stated that further research should be done to further assess the safety of TXA long-term and the drug's efficacy in high risk populations.[1]

Discussion

While treating the cause of PPH is the first line of therapy, TXA should be added to our armamentarium. For the treatment of PPH, the World Health Organization (WHO) recommended that patients diagnosed with PPH receive 1 gram of TXA intravenously within three hours of delivery in addition to standard care, followed by an additional dose if the bleeding continues after 30 minutes or resumes within 24 hours of the first dose.[21] This guideline was given following the successful outcome of the aforementioned WOMAN trial that demonstrated a lower number of deaths due to bleeding in women with PPH that received TXA.[16,21] ACOG also recommends the use of TXA but only recommends that TXA be considered for the treatment of PPH when uterotonic agents first fail to control it.[10]

While there is consensus for the use of TXA in the treatment of PPH, the results of studies of prophylactic TXA have been mixed, and thus the recommendation for the use of prophylactic TXA for the prevention of PPH is actively being debated. Some claim that more research is needed before a recommendation for prophylactic TXA can be given.[22] Meanwhile, others argue that the recommendation is long overdue and lament the fact that it has taken 50 years for the

drug to be studied this extensively in the setting of PPH, the problem it was originally designed to treat.[23] As previously mentioned, a recent meta-analysis demonstrated that TXA is a safe and effective prophylactic option for PPH in women undergoing a cesarean section.[1] However, the TRAAP2 trial did not demonstrate a significant decrease in secondary outcomes related to postpartum hemorrhage, which calls into question the relevancy of this data.[20] In the setting of vaginal deliveries, the TRAAP study found no significant decrease in the incidence of PPH when TXA was given prophylactically before vaginal deliveries.[18] Meanwhile, current studies such as the WOMAN-2 trial are underway to provide better insight into the efficacy of TXA for the prevention of PPH in patients with anemia after vaginal deliveries.[19]

For the prevention of PPH, there is consensus for the recommendation of active management of the third stage of labor.[10] This active management includes the administration of oxytocin or another uterotonic agent in the immediate postpartum period, uterine massage, and umbilical cord traction.[10] In addition, obstetric providers must evaluate the clinical circumstances at the time of delivery to determine whether the addition of prophylactic TXA may be appropriate.

Conclusion

PPH is one of the leading causes of maternal mortality and morbidity. Early identification of the cause of the PPH is necessary to determine directed therapy. Studies have demonstrated that TXA decreases death due to blood loss by 31% when given for the treatment of PPH[14] with minimal risks of thromboembolic events or other safety risks.[16] Therefore, we recommend the addition of TXA for the treatment of PPH in addition to standard care. However, when a patient does not respond to the initial directed therapy, it is vital to re-evaluate to ensure the correct cause of hemorrhage is identified and treated appropriately. We also emphasize that TXA is not a replacement for other medication therapies for treating PPH but rather an adjunct medication. Management of PPH is still dependent on the underlying cause.[11] In patients identified with PPH that are not responding to first-line uterotonic agents, 1 gram (100 milligrams/milliliter) of IV TXA can be given over 10 minutes at a rate of 1 milliliter/minute.[24] Early therapy is recommended, particularly within three hours of birth.[24] A second dose could be considered if bleeding continues after 30 minutes, in accordance with WHO recommendations.[24] Do not use TXA if the patient has a known hypersensitivity to the drug or has had a known thromboembolic event during pregnancy.[24] While there is some promise for using TXA for the prevention of PPH, we do not routinely recommend using TXA prophylactically due to mixed results of the drug's efficacy in the currently published literature. However, obstetric providers in areas of limited resources should consider the potential benefit of prophylactic TXA. We eagerly await the outcomes of further research, such as the WOMAN-2 trial, to further guide our care and provide us insight into managing patients from specific populations, such as those with anemia.[19]

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Isthmoceles: A Growing Concern

Brian Bishop, MS IV; and Keith Hansen, MD

Abstract

An isthmocele is described as a pouch-like defect in the uterus with a thin roof formed by inadequate healing of the smooth muscle of the anterior uterine wall at the site of a hysterotomy. With increased rates of deliveries by cesarean section, isthmoceles are becoming a more common cause of gynecologic and obstetric complications. Gynecologic issues include pelvic pain, postmenstrual bleeding, and reduced fertility or infertility. Therefore, it is important that providers consider these defects in their differential diagnosis as their presentation can mimic other gynecologic conditions, such as endometriosis. Fortunately, many effective treatment options are available, such as conservative hormonal treatment or surgical repair by laparoscopic, hysteroscopic, or transvaginal methods.

Introduction

First described by the obstetrician gynecologist Hugh Morris in 1995,[1] isthmoceles, also known as cesarean scar defects or uterine niches, are defects in the anterior wall of the myometrium at the site of a scar from a previous caesarean section (C-section). These defects can cause a variety of symptoms, including postmenstrual bleeding, pelvic pain, dysmenorrhea, dyspareunia, and secondary infertility. During future pregnancies, isthmoceles have been associated with scar dehiscence, abnormalities in placentation, and cervical ectopic pregnancies.[2] Isthmoceles have also been associated with a decrease in live birth rates after assisted fertilization techniques.[3] Although the World Health Organization recommends an overall C-section rate of 10-15%, this rate is greatly exceeded all over the world. For example, approximately half of all deliveries performed in China over the past decade were via C-section,[4] and the rates in Europe, North America, South America, and Latin America range from 25% to 43%,[3] with approximately 30% of live births in the U.S. delivered by C-section.[4] With the rapid rise in the rate of C-sections both in the U.S. and around the world, the incidence and prevalence of isthmoceles is also rapidly increasing.

Pathophysiology

Although the exact pathophysiology of isthmocele formation is not yet well understood, inadequate healing of the C-section incision which results in a reduced thickness of the overlying myometrium is seen as the primary mechanism.[5] As these defects are more common with increasing numbers of cesarean sections, it has been proposed that preexisting scar tissue from past cesarean sections negatively affects the healing of later incisions. This scar tissue also reduces the contractility of the myometrium, leading to the retention of menstrual blood, which can result in pelvic pain, postmenstrual bleeding, inflammation, possible infection, and may affect future fertility by interfering with sperm viability and motility as well as potentially impeding embryo implantation.[1]

Treatment

While there is no standard treatment for the repair of these uterine defects, current treatment options vary from conservative treatment with combined estrogen/progesterone therapy to surgical repair via hysteroscopic, laparoscopic, or transvaginal methods. Additionally, hysterectomy is a curative option in women with large, symptomatic uterine defects who no longer wish to become pregnant. It is important to note that most currently suggest only treating symptomatic isthmoceles, and surgery should solely be done for symptoms that can be attributed to the uterine defect. Presently, abnormal uterine bleeding is the only symptom that has been strongly linked to isthmoceles and surgeries for other indications should be considered experimental as no statistically proven benefit has been shown.

While none of the surgical repair techniques have been proven to be statistically superior, the laparoscopic method is usually

preferred over a hysteroscopic or transvaginal approach as it has the added benefit of increasing the myometrial thickness after repair.[6] This is especially important in women with large isthmoceles and those who wish to become pregnant again, as a thickened myometrium will reduce the risk of the scar rupturing during pregnancy. The first documented laparoscopic repair of an isthmocele was performed in 2003 and the results have typically been very favorable, with one study noting that pain from these defects decreased after surgery by 97%.[7] Additionally, pregnancy rates after surgical repair have been reported to be as high as 92 percent in one small study (13 patients).[8] During surgery, the isthmocele is often identified using the 'Rendezvous technique," where simultaneous laparoscopy and hysteroscopy are used in order to outline the isthmocele with the light of the hysteroscope.[1] After the defect has been properly visualized, the surgeon performs laparoscopic resection of all scar tissue, as well as excision of the edges of the defect. Then it is recommended that the repair is closed with at least two layers of suture which can be facilitated with robotic assistance, and dye is used to check for any leakage. It is currently advised for the patient to wait at least three months after the repair before attempting to become pregnant, and the recommended method of delivery for a pregnancy after the repair is cesarean section, although vaginal deliveries after isthmocele repair have been reported.[1]

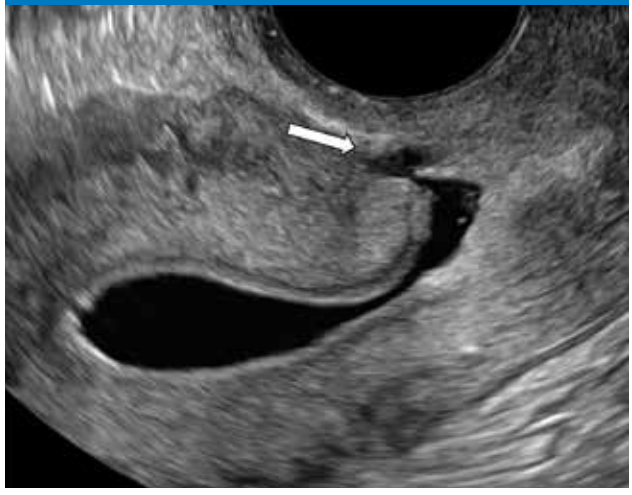
The hysteroscopic approach to isthmocele repair is another option for patients that is effective, as one study found that over 80% of patients reported resolution of their symptoms postoperatively. It is also very quick, as the average length of the resection was 8 minutes.[9] During this technique, the uterus is distended, allowing for direct visualization of the defect using the hysteroscope. The superior and inferior edges of the defect are then resected with a resectoscope loop until the myometrium is reached.[10] While effective, the hysteroscopic approach is most useful for patients who do not wish to preserve their fertility, as it does not increase the thickness of the myometrium at the site of the defect. Risks of this procedure include bladder injury and uterine perforation, especially if the myometrium thickness at the location of the defect is less than 3 mm. In these cases, and in cases where the patient wishes to preserve her fertility, laparoscopic excision and repair is the preferred approach.

The transvaginal technique to isthmocele repair has not been studied as extensively, although one retrospective study found outcomes comparable to laparoscopic repairs.[11] With this approach, the surgeon first locates the defect using hysteroscopy, and then makes an incision on the anterior wall of the vagina to access and dissect away the bladder to reach the anterior peritoneal reflection. The location and boundaries of the isthmocele are then confirmed using hysteroscopy and the scar tissue is removed. Finally, the myometrium is closed (preferably in two layers), followed by closure of the vaginal incision, and the hysteroscope is used to confirm the repair. Benefits of this approach compared to laparoscopic repair include significantly lower cost and surgical duration, as well as a faster recovery time. However, due to the higher risk of injury to adjacent structures such as the bladder, it is important that the surgeon be very proficient in vaginal surgery both to minimize this risk and to accurately locate and repair the defect through the vaginal incision.[12]

Discussion

The prevalence of isthmoceles varies widely, but a systematic review in 2014 estimated the prevalence in women with a history of at least one C-section to be between 56% and 84%.[13] While isthmoceles have been described for over 25 years, no standard definition currently exists for defining or diagnosing them. They are commonly described as "any visible filling defect in the anterior isthmus of the uterus on transvaginal ultrasound scan."[14] They can also be visualized via MRI, saline-infusion sonogram (SIS) (Figure 1), or hysteroscopy. These wedge-shaped defects can be classified as large or small, depending on the thickness of the overlying myometrium. Large isthmoceles have an overlying tissue thickness of less than 2.5-3.0 mm.[15] While small isthmoceles are often asymptomatic, larger ones can present with a multitude of symptoms, most commonly postmenstrual bleeding, which is often described as dark and mucousy, but

Figure 1. Isthmocele can be visualized using saline infusion sonohysterography (SIS).



also nonspecific pelvic pain, dysmenorrhea, dyspareunia, uterine rupture during pregnancy and labor, cesarean scar ectopic pregnancy, and secondary infertility.

Known risk factors for the development of isthmocele include: a history of multiple cesarean sections, an incision in the cervical area, the use of oxytocin, higher maternal BMI, gestational diabetes, more ischemic closures of the uterus, and retroverted uteri.[1,16] One hypothesis regarding the increased incidence of defects in retroverted uteri is the potential for increased tension on the sutured C-section incision, which could interfere with its ability to heal properly. A uterine suspension at time of laparoscopic repair is thought to be one possible solution to this problem. In addition, one study found that a double-layer uterine closure after a C-section resulted in a significantly lower risk of later developing an isthmocele (RR, 0.32; 95% CI, 0.17 to 0.61).[17]

Conclusion

Given the increasing rates of cesarean sections both in the U.S. and around the world, isthmoceles are becoming increasingly common and may be a cause of secondary infertility, irregular bleeding, pelvic pain, and complications in future pregnancies. There is also often a delay in diagnosing isthmoceles as other pathology may present with similar symptoms. By raising awareness of the increasing incidence and prevalence of these uterine wall defects, it is hoped that these diagnostic delays can be prevented.

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Talking the Walk: Our Obligation to Speak Out

Henry Travers, MD, FACP

In South Dakota today clinical practice, medical science and public health have been distorted by our state's governmental leaders in areas of abortion restriction, gender identity, vaccine use, firearms safety and even our medical school curriculum. Physicians and others in related professions, together serving the needs of patients – a calling, through one oath or another, to which we swore our stewardship – are deeply affected in our everyday practice by the consequences of these distortions. While social media as well as print and broadcast media would permit us to characterize these leaders as coming from the "radical left" or the "religious right" or to suggest their actions would "destroy families" or result in other dire consequences, the pages of *South Dakota Medicine* require of physicians a more professional standard for commentary.

It is not necessary to debate moral, religious or philosophical aspects of abortion, gender identity or the rights of parents vs the state regarding vaccination to challenge the distortion of science and the spread of misinformation. As research dating from the 1970s has shown, *all* of us are influenced by personal reactions to nearly everything in our lives that are independent of objective facts and which influence the choices we make. Our reactions come from perceptions, biases, beliefs, fears, and other highly variable elements of which we are often unaware. When public policy choices, framed to appeal to those reactions, subvert objective fact in support of manipulative framing, though, we have an obligation to speak out.

Ours is not the only state where policy choices are the result of deliberate scientific manipulation. As Caplan recently pointed out (Arthur L. Caplan. Florida heads down a dangerous covid road: ideology first, science second; *Medscape*, Dec 28, 2022), Florida Gov. DeSantis staffed his Public Health Integrity Committee[1] with COVID skeptics. Seeming to adopt a "let politics and fringe beliefs drive health policy in Florida" agenda, DeSantis, Caplan says, "has learned nothing from the misery and horror that others have caused by pioneering the idea that science is a popularity contest."

Human Life and the Heartbeat. Gov. Noem indicated recently that human life begins at conception (https://news.sd.gov/newsitem.aspx?id=28863). Some medical professional organizations extended that claim based on "the body of scientific evidence"[2] (https://acpeds.org/position-statements/when-human-life-begins) or as a philosophical viewpoint[3] (https://aaplog.org/about-us/our-mission-statement/). That "body of scientific evidence" is simply what we know about genetics, fertilization, cellular differentiation, growth, reproduction, organization and metabolism. There is no question that our beginnings are *human*: the haploid sperm and egg each contain 23 chromosomes made up of genes characteristic of humans.

Beyond this, the "body of scientific evidence," in fact, shows human embryos are self-organizing and initiate key functions of development in the absence of any maternal influence (*Nature Cell Biol* 2016; 18:700-8). An important correlative finding is the requirement of cells which are not part of the embryo itself to achieve fully expressed pluripotency of embryonic stem cells, at least until post-fertilization day 14 (*Pol Life Sci* 2016; 35:54-68). Related research has suggested events in the first three mitotic divisions "predict the success or failure of developing to the blastocyst stage," offering the provocative hypothesis that "success or failure to develop might be a *predetermined* [italics added] property of early human embryonic cells" (*Development*, 2012; 139:829-41). What begins at *conception* is penetration of the egg by a sperm with the formation of the zygote containing a polar body and pronuclei. Immediately following is cellular division, with the earliest *differentiation* [into inner cell mass (cells that will become the embryo) and trophoblast (cells that will become the placenta)] complete at the *fourth day*. Implying that this knowledge *defines human life* is beyond its capacity; there is no universally accepted biological definition of life. To philosophy, religion and whatever laws a society chooses to impose are left defining the boundaries of human life, the elements of what it is to be a human. Throughout history, those definitions have been malleable.

In accepting that laws may define such words, we must acknowledge those laws may reflect more the biases of *lawmakers* rather than a synthesis of either public opinion or learned scholarship. Such is the case with South Dakota's abortion statute (SD 34-32AA-1) which offers a definition of *human being* that serves the unstated purposes of the law and

does not address the concept of *personhood*. Asking science to determine social philosophy has been a fool's errand since Aristotle. Between the sperm's penetration of the egg and the birth of an infant are numerous steps that each play a role in determining gestation's outcome. Identifying any moment in time or any one step as *the* step initiating human life comes from no scientific principle, law or discovery. It comes from disciplines *outside* of science.

South Dakota's proposed "heartbeat bill" defined the fetal heartbeat as "cardiac activity or the steady and repetitive rhythmic contraction of the fetal heart…" So defined, the "heartbeat" is first detectable anywhere in a time period from the 34th through the 49th gestational days (*J Cardiovas Dev Disease*; 2022; 9:187-212). The definition refers to the rhythmic contraction of myocytes organized in a tube that conducts a unidirectional flow of plasma and red cells through the vascular tree of the developing embryo. This tube is not then the four-chambered structure we commonly envision. Viewing the heartbeat as a manifestation of *life* is a common and general understanding for many; we all have our own *reactions* to hearing the familiar "lub-dub" or even to thinking about it. It is these *reactions* that we often use to relate life to the heartbeat, *but that is not what science reveals*. Our lawmakers, though, proclaim *as evidentiary science*, this definitional significance for the fetal heartbeat, even though science never offered such a definition.

LGBTQ Health. Current South Dakota laws negatively impact the health of LGBTQ patients through the exclusion of services or drugs related to gender transformation in the state health benefit program and banning school districts from adopting LGBTQ non-discrimination and/or anti-bullying policies. Additional laws impacting LGBTQ patient health are continuously being introduced (see HB 1080 in the 2023 session of the legislature).

Six years ago, State Sen. David Omdahl said of LGBTQ individuals "I'm sorry if you're so twisted you don't even know who you are," and State Rep. Steve Haugaard said the legislature's perpetuation of "confusion in the lives of anyone is a disservice to them" by not doing anything to avert it. At the same time, Florence Thompson, representing South Dakota Parents Involved in Education, claimed without evidence, "The sexual orientation transgender agenda. It's in all of the schools now." In 2023 Norman Woods, director of the Family Heritage Alliance Action, said in a speech in Pierre that there is "a dangerous lie that is whispering in the ears of our children and whispers things like 'you're broken…'" Woods, supporting SB 1080, perpetuates, along with government officials, a notion that gender dysphoria is not real; that gender identity different from one's sex at birth is unnatural ("twisted"); and that health care, and specifically gender-related health care, for LGBTQ individuals is a strictly moral choice and not in the best interests of children.

Studies from 2008 (Drummond et al: *Dev Psych* 44:34 and Wallien et al: *J Am Acad Child Adoles Psych* 47:1413), 2009 (Reed et al: Gender Variance in the UK. The Gender Identity Research and Education Society), 2011 (Steensma et al: *Clin Child Psychol Psych* 16:499) and 2012 (Byne et al: *Arch Sex Behav* 41:759) supported later discredited ideas of gender confusion and desistence. These ideas were further promoted by news articles and popular TV shows (e.g. Dr. Phil). The research, however, conflated patient subsets, had biased samples, had no long-term follow-up and made unfounded assumptions (e.g. lack of response was equated to a return to natal gender). More recent research corrected the errors of this earlier work (e.g. Claahsen-van der Grinten et al: *Euro J Peds* 2021, 180:1349; National Academies of Sciences, Engineering, and Medicine. Understanding the well-being of LGBTQI+ populations. Washington, DC: National Academies Press, 2020; New Eng J Med 2022; 387(21):1919; and New Eng J Med 2023; 388(3):240-50), a science contradicting the basis for what is an objectively non-scientific prejudice.

Mr. Woods and some legislators driven by prejudice rather than a real concern for families do not engage with health professionals on the merits of their views. A conversation at that level would reveal the factual weakness both of their views regarding gender affirmation and of the condition itself. There is, indeed, much yet to learn about how best to treat gender related conditions (*New Eng J Med* 2023; 388(3):275-7; *The Atlantic* www.theatlantic.com/ideas/archive/2023/01/detransition-transgender-nonbinary-gender-affirming-care/672745). Further work to fill the gaps in our knowledge is the province of medicine, not politics. Untrained governmental officials must not dictate to physicians how they can treat their patients based on those officials' prejudices and misunderstandings of human medicine.

Diversity Training and the Medical Curriculum. In 2022 the legislature passed HB 1012 that effectively banned required diversity training in medical school. Its basis was widespread opposition to critical race theory, even though that isn't even mentioned in the law. When a government directly interferes with a course of instruction in our schools based on narrow prejudice rather than broad consensus, it

Extenuating Circumstances

disenfranchises teachers from the very core of their life's work including not only teaching itself, but the content of what is taught. In spite of the bill, our medical school continues to emphasize diversity in its curriculum. This legislative action rejected the collective judgements of educators. We physicians, without any organized attempt at marshalling opposition, simply accepted the decision of those with no medical training whose ideas came from a single Michigan university's restricted historical philosophy.[4]

Even though Wynia from the University of Colorado went so far as to suggest professional civil disobedience[5] in cases where a law requires us to harm our patients, we South Dakota health care professionals have not even taken the step of *actively* pushing back against misrepresentations such as those cited here. We have abdicated our responsibility to our patients, their families, and science for too long. It is time for us as a group, and individuals, to stand up and speak up. We took an oath. Let's honor it.

FOOTNOTES

1. Jay Bhattacharya, Martin Kulldorff, Steven Templeton, Joseph Fraiman, Bret Weinstein, Christine Benn and Tracy Høeg are committee members. All are associated with the Brownsone Institute, founded in 2021 to oppose COVID pandemic restrictions. Most are not practicing physicians; one is an economist and one a biostatistician. Weinstein, a college biology professor, touted ivermectin as a COVID cure.
2. The American College of Pediatricians, founded in 2002, believes there are absolutes that transcend social conventions, that moral considerations (acceptable to the College) must guide medical science and that a mother-father family unit within the context of marriage is optimal for childhood development. It opposes abortion and sex before marriage and supports an absolute right of parents to rear and educate their children. One of their pertinent objectives is to "engender the honest interpretation of scientific pediatric research, without deference to current political persuasions."
3. The American Association of Pro-life Obstetricians and Gynecologists was founded in 1973 as a "special interest group" within the American College of Obstetrics and Gynecology, but became an independent organization in 2013. It "empowers and encourages" its members to practice in the Hippocratic tradition. Members "share the view that human life begins at fertilization," but it does not claim a scientific basis for that view.
4. Hillsdale College representatives have assisted in developing social studies curriculum proposals for South Dakota and provided the chair for the 1776 Commission.
5. Wynia MK. Professional civil disobedience – Medical-Society responsibilities after Dobbs. New Eng J Med, 2022; 24 August 2022.

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Match Day 2023!

Lori Hansen, MD

Congratulations to the Class of 2023 on a successful Match Day. Match Day is the important transition in an aspiring physician's journey from undergraduate to graduate medical education. On the third Friday of every March, thousands of graduating medical students learn where they will be training for residency for the next several years. Nationally, there were 48,156 medical students who entered the 2023 Residency match. In the face of a growing shortage of primary care physicians across the U.S., there was a record number of primary care positions offered in the 2023 Match. Forty-two percent of students at the University of South Dakota Sanford School of Medicine (SSOM) matched into a primary care specialty. SSOM graduates will be training across the U.S. in a wide range of specialties and two students will be training in a military facility. Twenty-three percent of SSOM graduates will be staying in South Dakota for their residency in family medicine, internal medicine, pathology, pediatrics, psychiatry, transitional year or general surgery.

Match Day celebrations were held at the SSOM clinical campuses in Sioux Falls, Rapid City and Yankton, South Dakota. We are proud of the hard work and dedication of our students and look forward to their return to provide compassionate care for patients in our South Dakota communities.

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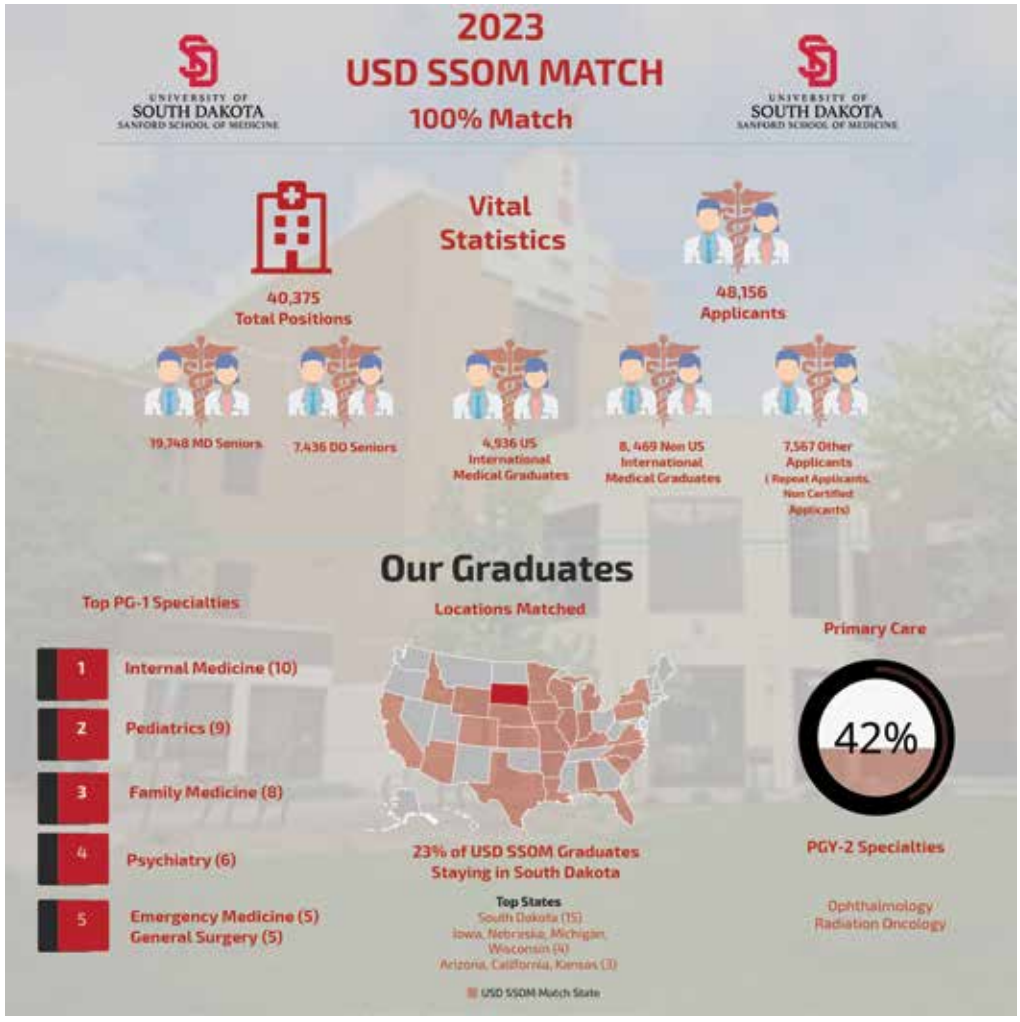


University of South Dakota Sanford School of Medicine 2023 Residency Match Listing

Last Name	First Name	PG1 Inst Name	PG1 Pgm Name	PG1 Location	PG2 Inst Name	PG2 Pgm Name	PG2 Location
Anderson	Ruthellen	U Iowa Hosps and Clinics	Pediatrics	IOWA CITY, IA			
Barwari	Shivon	Larkin Community Hospital	Urology	MIAMI, FL			
Bishop	Brian	University of Kentucky	Surgery-Prelim	LEXINGTON, KY			
Blasius	Lane	University of Nebraska Medical Center	Phys Medicine & Rehab	OMAHA, NE			
Braun	Madisyn	Center for Family Medicine	Family Medicine	SIOUX FALLS, SD			
Chandra	Sanyogita	U Iowa Hosps and Clinics	Internal Medicine	IOWA CITY, IA			
Clem	Alexander	Bassett Medical Center	General Surgery	COOPERSTOWN, NY			
Critzer	Samuel	University of South Dakota	Psychiatry	SIOUX FALLS, SD			
Cucak	Anja	Med Coll Wisconsin Affil Hosps	Anesthesiology	MILWAUKEE, WI			
Dally	Ann	Beaumont Health	Emergency Medicine	ROYAL OAK, MI			
Durant	Ashley	Northwestern McGaw	Emergency Medicine	CHICAGO, IL			
Egge	Justin	Med Coll Wisconsin Affil Hosps	Pediatrics	MILWAUKEE, WI			
Feng	Shelley	University of Kentucky	Surgery-Prelim	LEXINGTON, KY			
Fleming	Zachary	University of South Dakota	Psychiatry	SIOUX FALLS, SD			
Gallivan	Max	U Arkansas COM	Emergency Medicine	LITTLE ROCK, AR			
Gogoi	Maya	U Iowa Hosps and Clinics	Pediatrics	IOWA CITY, IA			
Goldammer	Megan	U Florida COM-Shands Hosp	Pediatrics	GAINESVILLE, FL			
Grassel	Meghan	Center for Family Medicine	Family Medicine	SIOUX FALLS, SD			
Habben	Chase	Mayo Clinic School of Grad Med Educ	Pediatrics	ROCHESTER, MN			
Hansen	Emily	Zucker SOM-Northwell NS	Surgery-Prelim	LONG ISLAND, NY			
Hanten	Brandon	U Kansas SOM	Internal Medicine	KANSAS CITY, KS			
Harris	Mitchell	Baylor Scott & White Medical Center	Psychiatry	TEMPLE, TX			
Hensley	Kjerstin	Beaumont Health	Emergency Medicine	ROYAL OAK, MI			
Hora	Kirby	University of Nebraska Medical Center	Orthopedic Surgery	OMAHA, NE			
Jahnz	Stephanie	Naval Medical Center	Internal Medicine	SAN DIEGO, CA			
Jones	Mason	Creighton University	Internal Medicine	OMAHA, NE			
Keith	Meredith	Center for Family Medicine	Family Medicine	SIOUX FALLS, SD			
Kendall	Ganeva	Kootenai Health	Family Medicine	COEUR D'ALENE, ID			
Koenen	Maria	University of South Dakota	General Surgery	SIOUX FALLS, SD			
Leif	Ethan	Center for Family Medicine	Family Medicine	SIOUX FALLS, SD			
Leigh	Lindsay	U Kansas SOM	Obstetrics-Gynecology	WICHITA, KS			
Loewen	Benjamin	University of Wyoming	Family Medicine	CHEYENNE, WY			
Louwagie	Eli	HCA Healthcare LGH	Internal Medicine	BLACKSBURG, VA			

University of South Dakota Sanford School of Medicine 2023 Residency Match Listing

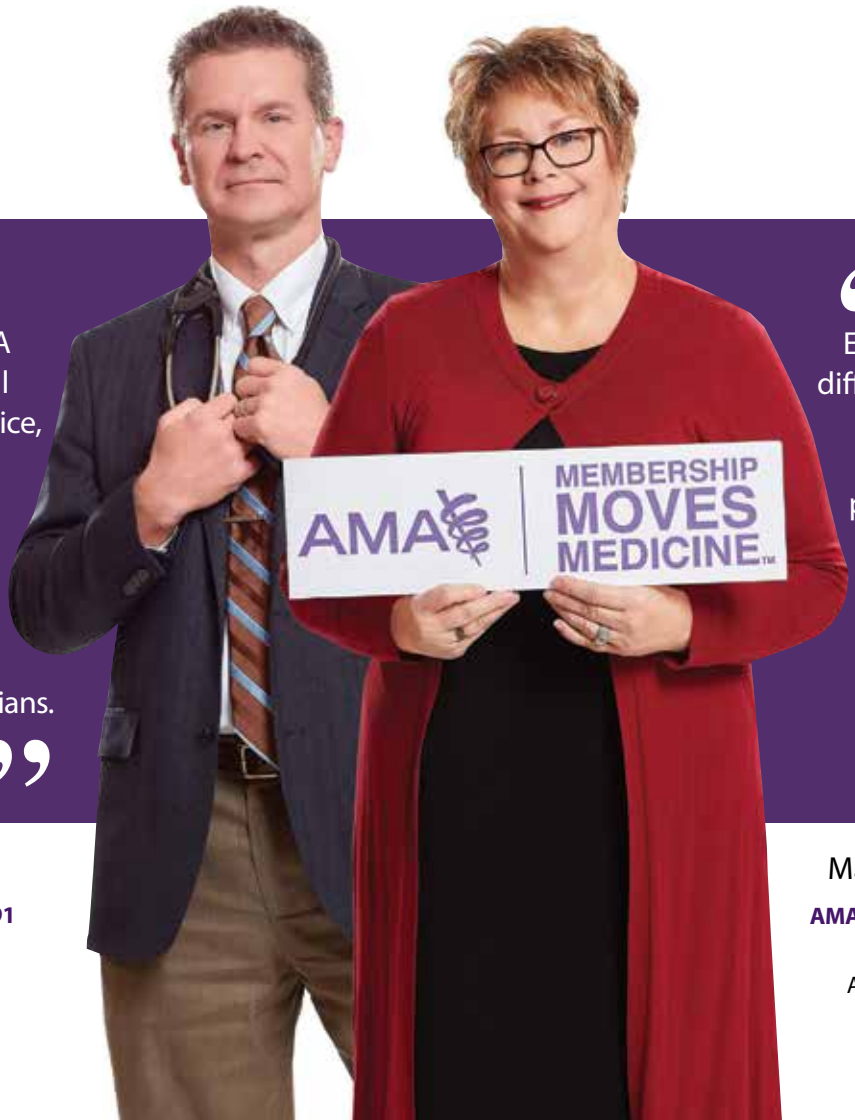
Last Name	First Name	PG1 Inst Name	PG1 Pgm Name	PG1 Location	PG2 Inst Name	PG2 Pgm Name	PG2 Location
Lovrien	Sydney	University of South Dakota	Internal Medicine	SIOUX FALLS, SD			
Lucas	Spencer	Medical University of SC	Vascular Surgery	CHARLESTON, SC			
Mack	Danielle	U Iowa Hosps and Clinics	Pathology	IOWA CITY, IA			
Makela	Tory	Riverside Community Hospital	Surgery-Prelim	RIVERSIDE, CA			
McMahon	Connor	Mayo Clinic School of Grad Med Educ	Psychiatry	ROCHESTER, MN			
Newton	Jazmin	University of South Dakota	Transitional Year	SIOUX FALLS, SD			
Pischke	James	Indiana University SOM	Internal Medicine	VICENNES, IN			
Pollema	Christian	University of Nebraska Medical Center	Psychiatry	OMAHA, NE			
Randall	Halle	University of South Dakota	Pediatrics	SIOUX FALLS, SD			
Richardson	Sophie	Uniformed Services Health Education Consortium	Transitional Year	SAN ANTONIO, TX			
Rohlf	Derek	Louisiana State University	Internal Med-Prelim/Ophthalmology	SHREVEPORT, LA	Louisiana State University	Ophthalmology	SHREVEPORT, LA
Smith	Luke	U Colorado SOM	Neurology	AURORA, CO			
Soyland	Dallas	George Washington University	Neurological Surgery	WASHINGTON, DC			
Stevens	Sawyer	Health Education Services	Family Medicine	FOLEY, AL			
Storm	Emily	University of South Dakota	Pathology	SIOUX FALLS, SD			
Stys	Julia	Ascension St John Hosp	General Surgery	DETROIT, MI			
Thaemert	Jadah	University of South Dakota	Psychiatry	SIOUX FALLS, SD			
Thanel	Paul	Western Michigan Univ Stryker SOM	Emergency Medicine	KALAMAZOO, MI			
Thomsen	Jacob	U Arizona COM	General Surgery	PHOENIX, AZ			
Tobin	Christian	University of South Dakota	General Surgery	SIOUX FALLS, SD			
Torve	Megan	St Louis University SOM	Pediatrics	ST. LOUIS, MO			
Trautman	Ariadne	Med Coll Wisconsin Affil Hosps	Pediatrics	MILWAUKEE, WI			
VanderZee	Brandon	University of Wisconsin	Opthalmic Pathology and Imaging Fellowship	MADISON, WI			
Van Oosbree	Annika	Medical University of SC	Obstetrics-Gynecology	CHARLESTON, SC			
Veldman	Amber	Phoenix Childrens Hospital	Pediatrics	PHOENIX, AZ			
Vlach	Michael	U Kansas SOM	Internal Medicine	KANSAS CITY, KS			
Vollmer	Mykayla	University of South Dakota	Internal Medicine	SIOUX FALLS, SD			
Wellman	Allen	Geisinger Health System	Transitional Year	BLOOMSBURG, PA			
Wilson	Patrick	ECU Health Medical Center	Family Medicine	GREENVILLE, NC			
Wilson	Peter	University of South Dakota	Internal Med-Prelim	SIOUX FALLS, SD	University of Minnesota	Radiation Oncology	MINNEAPOLIS, MN
Wixon	Nicholas	Riverside Community Hospital	Anesthesiology	RIVERSIDE, CA			
Zineldine	Omar	U Arizona COM	Internal Medicine	TUCSON, AZ			

Strengthen the voice of **South Dakota physicians**

Your membership in the South Dakota State Medical Association and the American Medical Association (AMA) ensures you have powerful allies amplifying your voice and advocating for the needs of South Dakota patients and physicians—in your community and across the country.

AMA | MEMBERSHIP MOVES MEDICINE™

SOUTH DAKOTA STATE MEDICAL ASSOCIATION
Values. Ethics. Advocacy.



"The AMA and SDSMA help me "filter out" all the noise in my practice, which allows me to focus on the issues that are most important to me—my patients, the practice of medicine and my fellow physicians."

Robert Allison, MD

AMA MEMBER SINCE: 1991
South Dakota
AMA House of Delegates

"Every locality has a different perspective on what is most important for providing excellent health care. Everyone must be represented when policy is being crafted."

Mary Carpenter, MD

AMA MEMBER SINCE: 1982
South Dakota
AMA House of Delegates

Member News

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

Legal Brief Highlight: Reporting of Impaired Drivers

If a physician believes an impaired driver is a serious imminent threat to a person or the public, the physician should make a report to the Department of Public Safety. Many states have statutes in place that compel physicians to report impaired patients to the requisite governmental agency when they feel that a continuation of their driving privileges presents substantial risk to both themselves and the public at large. South Dakota is not one of those states; however, South Dakota physicians have a general ethical obligation to protect the health and welfare of the public.

HIPAA permits disclosure of confidential health information in certain circumstances. A physician may disclose confidential health information to avert a serious threat to health or safety. A physician who qualifies as a covered entity under HIPAA may use or disclose protected health information if the covered entity, in good faith, believes the use or disclosure is necessary to prevent or lessen a serious and imminent threat to the health or safety of a person or the public. The permissive disclosure described in HIPAA is subject to the limitation that such disclosure must be consistent with the applicable law and standards of ethical conduct for your jurisdiction.

The American Medical Association (AMA) has issued an ethics opinion concerning a physician's duties in regard to patients whom they believe to be impaired drivers. The AMA recommends that the physician undertake a "tactful but candid discussion with the patient and family about the risks of driving" before independently reporting. The AMA recommends this conversation address additional treatment or therapy, or encourage the patient and family to decide on a restricted driving schedule, if possible. Reporting is only recommended in situations where clear evidence of substantial driving impairment implies a strong threat to patient and public safety, and where the physician's advice to discontinue driving is ignored. The AMA also recommends that physicians disclose and explain to their patients their responsibility to report.

More information is available in the SDSMA legal brief *Reporting Impaired Drivers* at sdsma.org. The SDSMA provides legal briefs for member physicians on a variety of rules and regulations that apply to the medical profession.

for the 2023 SDSMA Annual Leadership Conference on June 2!

The 2023 SDSMA Annual Leadership Conference is Friday, June 2 at the Hilton Garden Inn Downtown Sioux Falls. Registration, details and the full schedule will be posted soon at sdsma.org.

Events and Activities

- University of South Dakota Sanford School of Medicine Update
- Update from the American Medical Association
- Panel discussion
- Membership open forum
- SDSMA PAC lunch
- Medical student poster session
- Policy Council meeting
- Membership mixer
- Awards banquet & scholarship recognition

Presentations Will Focus on Substance Use

Opioids and substance use is the focus of this year's conference. Presentations will address the facts of opioid use, what can be done to reverse this crisis, safe prescribing, substance use disorders, and more.

Opioid prescribing continues downward while overdose and death related to illicitly manufactured fentanyl, methamphetamine, and cocaine increase -- further amplified by underlying social needs. Reversing the use and impact of illicit drugs requires physician leadership, an emphasis on overdose prevention/treatment, and coordinating efforts and best practices.

Medical Student Poster Session

Be sure to join us for the Medical Student Poster Session! This opportunity helps students improve their CVs and their competitiveness for medical residencies.

Submit a Policy Issue & Participate in the Open Forum

Do you have a policy issue you'd like to bring to the SDSMA's attention? Make plans to address the SDSMA Policy Council. Policy issues submission forms are available at sdsma.org. Submission forms must be returned to the SDSMA office by May 5. The open forum will be held at the Hilton Garden Inn Downtown Sioux Falls during the Annual Leadership Conference.

South Dakota State Medical Association

Overview

South Dakota's 2023 Legislative Session opened January 10 and continued through March 9, with the 38th legislative day held on March 27. Of the 480 pieces of legislation brought forward, 61 had the potential to impact health care delivery in South Dakota.

During the nine-week session, the SDSMA worked on a wide range of issues to protect the practice of medicine and to enhance the delivery of medical care. Some highlights of the important issues the SDSMA was involved in include:

Promoting the art and science of medicine

The South Dakota State Legislature began its 2023 session with Governor Kristi Noem's State of the State address in which she spoke on several issues of interest to the SDSMA including pregnancy & postpartum care and vaccine mandates.

HB 1235 would have established a personal exemption from any COVID-19 vaccination mandate, requirement, obligation, or demand on the basis that receiving a COVID-19 vaccination violates the person's conscience. Health care facilities would have been exempt from the law if compliance would result in violation of regulations issued by CMS or CDC. The bill failed in the House of Representatives 30-39 after passing the Health and Human Services Committee. The SDSMA opposed the bill.

SB 1 updates the process for adding new debilitating medical conditions for medical cannabis from an administrative rules process within the Department of Health to a legislative process. The bill also places into statute eight specific diagnoses that can be used to certify patient eligibility for medical cannabis. The SDSMA opposed the inclusion of glaucoma and post-traumatic stress disorder as qualifying conditions, due to the lack of evidence for cannabis as an effective treatment. An amendment removing glaucoma from the list was adopted by the House Health and Human Services Committee, and the bill passed both the Senate and House of Representatives by narrow margins.

SB 122 attempted to create opioid prescribing guidelines and require additional physical examinations and administrative documentation for physicians. The proposal was deemed restrictive and out of line with AMA and CDC guidelines. The Senate Health and Human Services Committee defeated the bill on a 6-0 vote with the SDSMA testifying in opposition.

SB 125 would have restricted any school, early childhood program, state agency, elected official, or state employee from adding new immunization requirements to state statute. Most of the proponent testimony during the Senate Health and Human Services Committee focused on the COVID-19 vaccine. The SDSMA opposed the bill, which failed in committee on a 7-0 vote.

SB 130 attempted to amend statute to include an exception for required school entrance vaccinations "because of a sincerely held religious or philosophical belief." The bill was defeated by the Senate Health and Human Services Committee with a 6-0 vote and was opposed by the SDSMA.

Protecting and improving public health

HB 1041 provides an exception to the definition of drug paraphernalia by exempting fentanyl test strips. The SDSMA testified in support during committee hearings and the bill received near unanimous, bipartisan support of the legislature for passage.

HB 1131 sought to prohibit state legislation or state agency rules from creating extreme risk protection orders, also known as red flag laws, and prohibit the enforcement of any federal law or rule pertaining to the same. The SDSMA opposed the legislation and testified against it in the House Judiciary Committee. The bill was defeated by a vote of 9-3.

HB 1162 allows licensed health professionals to dispense or distribute an opioid antagonist to an employer. Practically, the bill allows drug treatment centers to have on hand critical life-saving medications that can be used for someone experiencing an opioid overdose. Similar to HB 1041, another bill aimed at life-saving efforts related to drug use, support of the legislation was unanimous in its passage and was supported by the SDSMA.

HB 1216 was an effort to create and revise criteria for patient visits at certain medical facilities, including adding a physician evaluation component. Opponents, including the SDSMA, were concerned with new requirements for physician evaluation of patients for whom they are not currently serving as the primary practitioner, as well as facility visitation policies being set without regard to the impact of public health at large. The bill was defeated 9-2 by the House Health and Human Services Committee.

Ensuring access to and delivery of quality medical care

HB 1044 was tabled by the Joint Appropriations Committee. The bill would have appropriated $20 million to fund scholarships promoting behavioral health careers. The SDSMA supported this legislation, as well as other initiatives to expand patient access to quality mental health care.

SB 25 authorizes the disbursement of funds to physicians who have met criteria for recruitment assistance. The bill received unanimous, bipartisan support and passed both chambers overwhelmingly. The SDSMA supports this annual legislation.

SB 87 attempted to expand optometric scope of practice to permit injections around the eye, removal of lid lesions, and some laser surgeries. This scope expansion would have allowed optometrists to perform procedures without any experience on live patients and do so after completing a curriculum of just 32 hours. The bill was defeated 7-6 by the House Health and Human Services Committee after passing the Senate. The SDSMA opposed the bill and testified against it.

SB 149 would have created a Medicaid expansion fund to track the disbursements by the state related to the expansion of Medicaid. The SDSMA supported Medicaid expansion during the 2022 general election and supported this bill. The bill passed each chamber in slightly different form and a conference committee was unable to reach consensus. A separate fund isn't necessary for the distribution of funds and instead will be part of the Department of Social Services' budget.

SB 175 would have allowed independent practice of physician assistants. As proposed the bill would have granted independent practice authority upon the completion of 2,080 supervised practice hours. The bill allowed those practice hours to be completed under the supervision of another physician assistant, which isn't allowed in any state. The SDSMA strongly opposed the legislation, and it failed in the Senate by a vote of 14-20.

Advocating on other legislative priorities

HB 1192 and SB 106 both sought to expand postpartum Medicaid coverage to one year, a change supported by the SDSMA. These bills were eventually tabled because the Department of Social Services expanded its ongoing budget to adopt the policy and cover the cost.

HB 1169 proposed to amend statute to state that before an abortion is justified as necessary to preserve the life of a mother, the attending physician, exercising reasonable medical judgment, must determine that by continuing the pregnancy the woman is at serious risk of death or of a substantial and irreversible physical impairment of one or more major bodily functions. The bill was tabled at the request of the sponsor. The SDSMA supported the bill.

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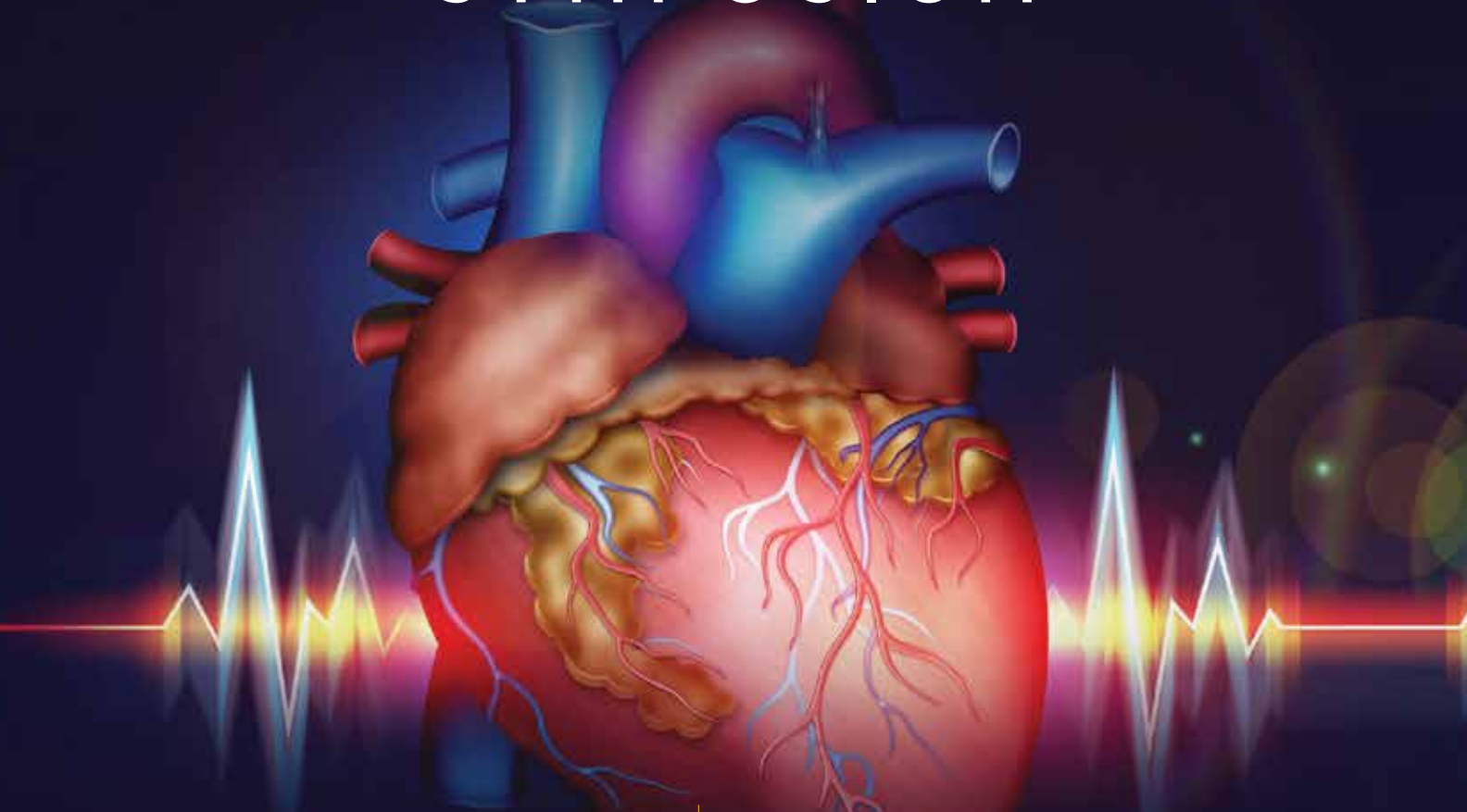

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