March 2022
Volume 75
Number 3

South Dakota medicine

THE JOURNAL OF THE SOUTH DAKOTA STATE MEDICAL ASSOCIATION

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Office of Publication
2600 W. 49th Street, Suite 100
Sioux Falls, SD 57105
605.336.1965
Fax 605.274.3274
www.sdsma.org

Editor
Keith Hansen, MD

SDSMA President
Kara Dahl, MD

Chief Executive Officer
Barbara A. Smith

SDSMA Vice President
Mark East, MS

Staff Editor
Elizabeth Reiss, MS
–ereiss@sdsma.org, 605.336.1965

Advertising Representative
Elizabeth Reiss, MS
–ereiss@sdsma.org, 605.336.1965

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GIFTING WITH A PURPOSE: PRACTICAL STRATEGIES TO POSITIVELY IMPACT YOUR NEXT GENERATION

CALEB BROWN, CFP®, *Lead Advisor*

Investing early and often is one of the keys to financial independence. When people are in their teens and early twenties, there generally is little room to save and invest and it might not seem like a priority. Skip ahead a generation or so, and people likely will be making more money. They will also be spending a lot more on family, home, education, etc. And if they haven't made it a practice of saving money, it still might feel like there is little left over to invest.

The problem with starting in the second half of life is not only that it's hard to make space for something new in the budget, but also that a person misses out on the power of compounding. Let me illustrate with a calculation.

Scenario 1

- At age 20, invest $15,000 at 6%.
- At age 50, the value of the $15k investment would be approximately $90,000.
- At age 65, the value of the $15k investment would be approximately $220,000.

Scenario 2

- No investing before age 50.
- At age 50, invest $9,000 every year until age 65 at 6%.
- At age 65, the value of this $9k investment every year will be $220,000.

In scenario 1 only $15,000 was invested, but in Scenario 2 you invested over $135,000! Clearly, in Scenario 1, you can see time doing the heavy lifting.

So, maybe you're in a position to gift money every year to children or grandchildren. This is a common practice for some of our clients. For 2022, an individual can gift $16,000 per person. (A married couple could give $32k to one person in a year.) This would not show up as income and would not require filing of a gift tax return. Imagine if that $16,000 was used to help your children or grandchildren save more money early and often. Here are some ideas to consider:

- Set up a Roth IRA, and fund the maximum for them.
- If they maximize the 401k plan at their job, help replenish their cash flow.
- Pay $16k of their mortgage so they could save that money.
- Similar to the above, go to their county's assessor site and pay their property taxes.
- Set up an UTMA account. This is an account designed for minors, and it's a great way to teach investing habits.

These are all examples of gifts, but the idea is gifting with a purpose. The purpose is to help them invest early and often to increase the probability of their financial independence. Because saving money early and often can be difficult, consider how you might help those closest to you to do more of it. Foster Group takes the time to truly know our clients—not just their financial goals, but what's in their hearts. If you'd like to put together a plan to do this, please give us a call!

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Physician Burnout

Kara Dahl, MD
President, South Dakota State Medical Association

It comes as no surprise that the number of physicians who report feeling burned out is increasing across the U.S. Even prior to this pandemic, many physicians were feeling increased distress at work; however, this was magnified greatly over the past two years as we continue to face new and evolving challenges.

Physician burnout is often defined as work-related emotional exhaustion, loss of enthusiasm for one's work, depersonalization, reduced personal accomplishment and a sense of professional dissatisfaction and failure. Some physicians use the term moral injury as burnout may imply that the problem is with the individual physician. Moral injury is repeated insults to a person's morality and core values. Some physicians who report having symptoms of burnout may actually be suffering from moral injury. For simplicity in this article, I use the term burnout; however, I recognize that this is a complicated systemic problem and many physicians do experience moral injury as well. According to Medscape, which recently surveyed more than 13,000 physicians, the number of physicians who are feeling burned out across all specialties is 47 percent which is up from 42 percent last year. Emergency physicians rated the most burned out with 60 percent, followed by critical care at 56 percent, OB/GYN 53 percent, and infectious disease and family medicine both at 51 percent. These statistics are extremely alarming. Physician burnout is a significant public health crisis and we, as leaders within healthcare, need to be at the forefront of finding and implementing solutions. To do this, one must address the numerous causes of physician burnout which are often complex and intricately embedded within our current healthcare system in the U.S.

Many factors contribute to physician burnout. Physicians tend to be emotionally intelligent people with high resiliency who have good coping skills developed and honed throughout the many stressful years in college, medical school and residency. It is harmful and simply untrue to imply that a physician suffering from burnout/moral injury is emotionally weak or incapable of handling stress when in fact it is usually the opposite. It is also important to note that physicians suffering from burnout are often excellent physicians, however studies demonstrate that when physicians report feeling burned out that it can negatively impact patient care and safety. One study revealed physicians who are burned out are significantly more likely to make a serious medical error. Additionally, physicians who are burned out also have higher rates of depression, substance abuse, and suicide. It is imperative that we help find ways to improve our current healthcare system, so that physicians can provide the best possible care to themselves and their patients.

As mentioned above, burnout is multifactorial. Physicians commonly site lack of autonomy and too many bureaucratic tasks as key contributors to burnout. They are often trying to manage unrealistic expectations and are faced with increasing performance measurements, increasing and excessive workloads, decreased pay, constant time pressures, inefficient work processes including electronic medical records and dealing with the many hurdles that insurance companies have in place such as prior authorizations and denials of coverage. As well as worrying about poor patient outcomes, bad online reviews, and potential for lawsuits leading to defensive documentation.

Last month I wrote about increasing emotional intelligence and techniques for stress management which is a great start when facing symptoms of burnout, however, there needs to be a more large-scale, dynamic change in healthcare because physician burnout at its core is a systemic problem that continues to grow. The AMA has engaged healthcare CEOs to examine and address burnout within health systems. Some examples of areas that administrators are focusing on are regular physician well-being check-ins using measurable data, addressing burdensome clerical tasks, using team-based approaches to eliminate certain tasks that could be done by others in the healthcare team, and evaluating physician turnover and early retirement. There are several other ways physician wellbeing is being evaluated and addressed based on local needs. I hope that we as physicians continue to be leaders in healthcare, advocating for the betterment of our patients and colleagues at both a local and national level. The SDSMA is developing a program separate from the licensing board and physician's employer for those who may be struggling or looking for assistance. It will offer comprehensive and confidential resources to physicians that are also protected from discovery. Look for the program's launch soon.

In conclusion, if you are feeling the effects from burnout or are concerned that you are becoming burned out from the increased stress and/or moral injury of practicing medicine in a strained and often dysfunctional healthcare system, you are not alone. Please reach out to a trusted friend, partner, mentor, or coach for support as you find ways to optimize your situation. Physicians need to prioritize taking care of themselves, our health and wellness is essential for the well-being of our patients, especially as we care for others in these difficult and trying times.

Enough?

April K. Willman, MD, FAAP

Dry cracked hands
Washing all the time
It's never enough

Close my eyes
Pray for loved ones
Don't let the invisible killer win

Selfishly hoping day after day after day
And knowing inside
It's never enough

From heroes to zeros
It's not our fault
Just trying to help

Wave after wave
Keep working, keep helping
It's never enough

Washing and praying
And hoping and caring
Is it enough?

About the Author:
April K. Willman, MD, Clinical Associate Professor, Pediatrics, University of South Dakota Sanford School of Medicine; Yankton Medical Clinic, Yankton, South Dakota.

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ST-Segment Elevation Myocardial Infarction Due to an Ectatic Infarct-Related Coronary Artery: Case Series and Review of the Literature

Maheedhar Gedela, MD; Sharoon Samuel, MD; Christopher M. Van Hove, MD; Tomasz Stys, MD; and Adam Stys, MD

Abstract

Coronary artery ectasia is an infrequent finding seen in a localized or diffuse fashion in patients undergoing coronary angiogram. This angiographic entity is attributed to coronary artery atherosclerosis. The ectatic coronary artery segment may be a culprit and perpetuate the thrombus formation in patients with acute myocardial infarction due to the altered normal laminar flow and deranged platelet and endothelial activation. Besides, it may lead to slow flow/no-reflow during the percutaneous coronary intervention and constitutes a significant management challenge. In this article, we report three patients with ST-segment elevation myocardial infarction from the culprit ectatic infarct-related artery and discuss the various management strategies.

Introduction

Coronary artery ectasia (CAE) is uncommon, predominantly seen in patients with coronary artery atherosclerosis. CAE is described as an epicardial coronary artery with a luminal diameter ≥1.5-fold larger than an adjacent, normal coronary artery segment. More than 50 percent of cases of CAE are caused by atherosclerosis.[1] Due to the low incidence of CAE, there is limited information on it contributing to acute myocardial infarction (AMI) as well as what its clinical outcomes are.[2] Moreover, management of CAE-related AMI is typically derived from small observational studies and case reports. CAE poses a significant management dilemma, though, due to the associated high thrombus burden and alteration in platelet activity and the coagulation system. Here, we describe three cases of ST-segment elevation myocardial infarction (STEMI) due to an infarct-related artery with coronary ectasia and review the available literature on this topic.

Case 1

A 68-year-old male with no significant past medical history presented to an outlying facility with symptoms of on-and-off chest pain for two days and worsening of the chest pain with diaphoresis one hour before arrival. An EKG was performed, and it revealed a 1 mm ST-segment elevation in leads AVR, V1, and V2 and de Winter T-waves in leads V4 and V5, with reciprocal ST-segment depressions in leads II, III, and AVF (Figure 1). The patient was diagnosed with anteroseptal STEMI and received tenecteplase (TNKase) before being transferred to our institution for a facilitated percutaneous coronary intervention (PCI). The patient's emergent coronary angiogram revealed an acute, thrombotically occluded proximal left anterior descending (LAD) artery with TIMI 2 flow (Figure 2). The patient then underwent a Penumbra CAT RX Indigo mechanical aspiration thrombectomy and subsequent Medtronic Resolute Onyx 4.5x18 mm stent placement in the infarct-related proximal LAD artery through right radial access. The final angiogram was excellent, showing TIMI 3 flow (Figure 3). However, due to the no-reflow phenomenon during the procedure, tirofiban infusion was continued for 18 hours. The patient was discharged two days later with a regimen of aspirin, clopidogrel, and atorvastatin. In addition, he was started on losartan, metoprolol succinate, and spironolactone due to the heart failure with reduced ejection fraction (HErEF) of 30-35 percent. A week later, the patient received placement of a BIOTRONIK Orsiro 2.5x13 mm stent in the noninfarct-related proximal left circumflex (LCX) artery into the obtuse marginal 1 artery.

Case 2

An 82-year-old female with a significant medical history of

Figure 1. Electrocardiogram (EKG) showing 1 mm ST-segment elevation in leads AVR, V1, and V2 (red arrows) and de Winter T-waves in leads V4 and V5 (asterisk), with reciprocal ST-segment depressions in leads II, III, and AVF (black arrows).

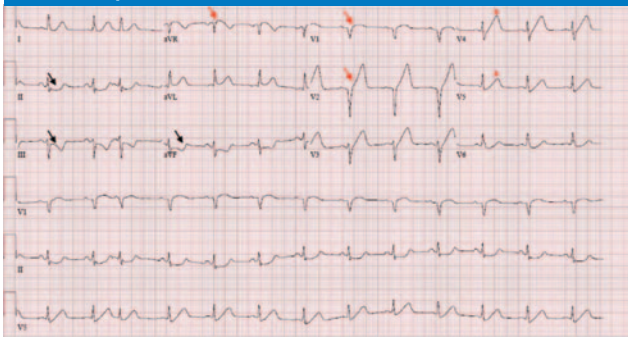


Figure 2. Coronary angiogram cranial (CRA) view showing thombotic occlusion of the proximal left anterior descending (LAD) artery. Arrow represents ectatic segment with thrombus.

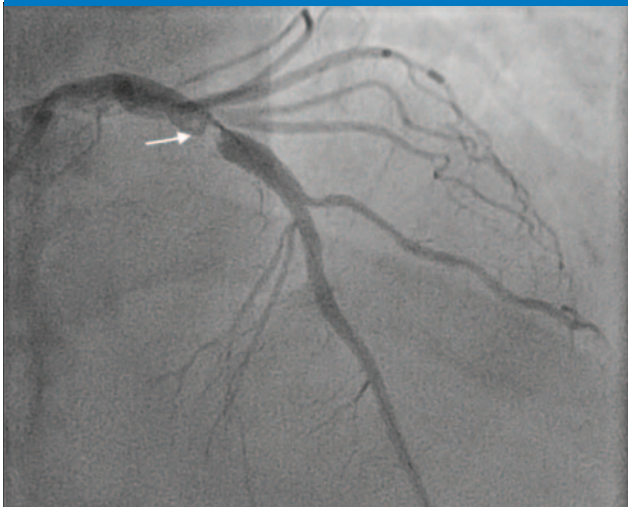
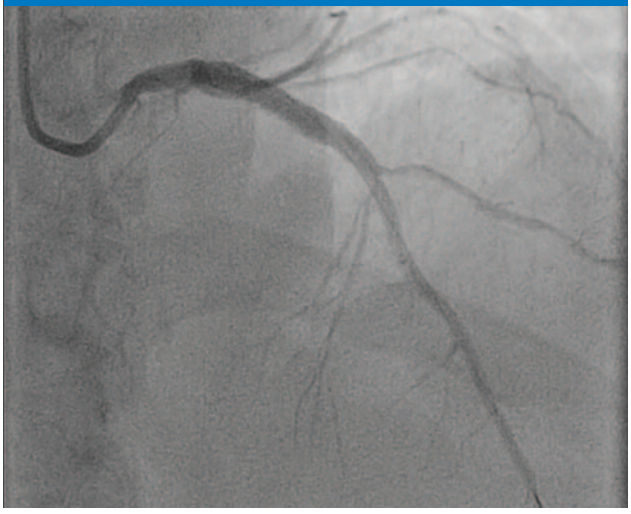


Figure 3. Coronary angiogram CRA view showing final good angiographic result in the ectatic infarct related LAD artery.



hypertension and stage III chronic kidney disease presented to an outlying facility with substernal chest pressure for two hours with radiation to the mid-back. Her EKG showed a new left bundle branch block (Figure 4). She was diagnosed with STEMI and transferred to our facility for the primary PCI. TNKase was not administered due to her advanced age, frailty, low body mass index, and renal dysfunction. Her emergent coronary angiogram revealed acute occlusion of the proximal through distal LAD artery with a heavy thrombus burden and TIMI 1 flow (Figure 5). The patient underwent a Penumbra CAT RX Indigo mechanical aspiration thrombectomy and placement of an Abbott XIENCE 4.0x33 mm stent in the infarct-related proximal and mid segment of the LAD artery through right radial access, with restoration of TIMI 3 flow (Figure 6). She got a tirofiban infusion for 18 hours due to the large thrombus burden and slow-flow phenomenon. The patient was started on aspirin and ticagrelor and discharged three days later. She

Figure 4. EKG showing new onset left bundle branch block.

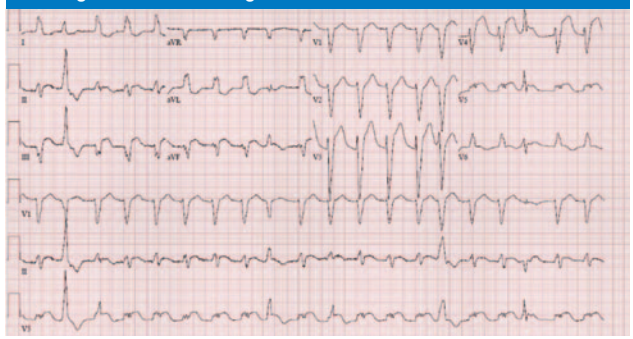


Figure 5. Coronary angiogram right anterior oblique (RAO) CRA view showing acute occlusion of the LAD with a large thrombus burden. Arrow represents significant filling defect with haziness in the ectatic segment.

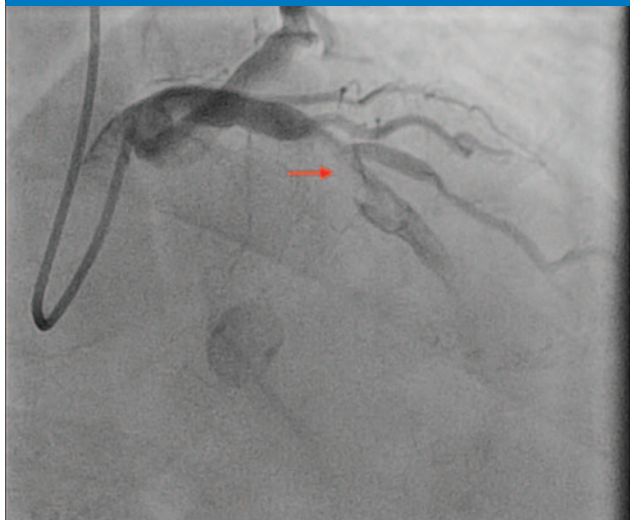


Figure 6. Coronary angiogram RAO CRA view showing good angiographic result in the LAD artery. Arrow represents ectatic segment after the stent in the LAD.

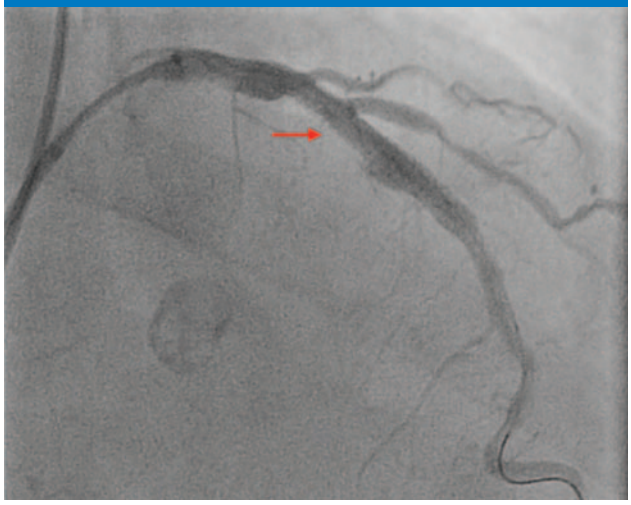


Figure 7. EKG showing ST-segment elevations in leads II, III, AVF, V5, and V6 (red arrows), with ST-segment depressions in leads V1, V2, I, and AVL, and a complete heart block.

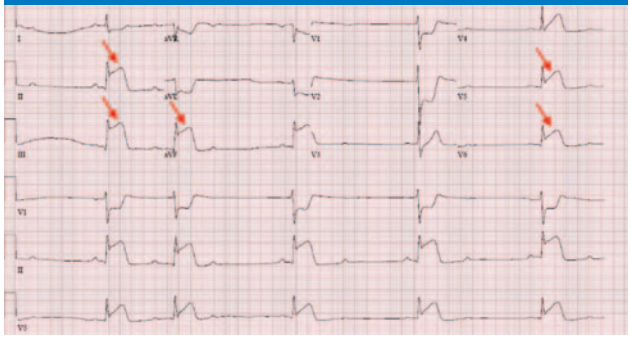
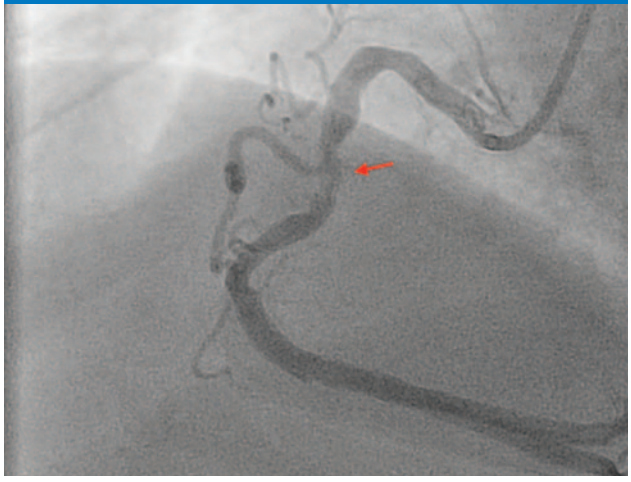


Figure 8. Coronary angiogram left anterior oblique (LAO) CRA view showing acute occlusion of proximal and mid segment of right coronary artery (RCA). Arrow represents thrombotically occluded ectatic segment.



had been started on metoprolol succinate due to the HErEF with an ejection fraction of 15-20 percent but not on angiotensin-converting enzyme inhibitors or an aldosterone antagonist due to the acute kidney injury.

Case 3

A 53-year-old male with a history of hypertension, hyperlipidemia, tobacco use, and obesity presented to an outside facility following a blackout episode and severe substernal chest pain while hunting. He was noted as having ST-segment elevations in leads II, III, AVF, V5, and V6, with ST-segment depressions in leads V1, V2, I, and AVL, and a complete heart block (CHB) on the EKG (Figure 7). He received TNKase for the inferior STEMI and was transferred to our facility for the facilitated PCI. The ST segments improved and the CHB resolved following the TNKase infusion. An emergency coronary angiogram performed through the right femoral access showed thrombotic occlusion of the proximal and mid segment of the right coronary artery (RCA), with TIMI 2 flow (Figure 8). A temporary transfemoral venous pacemaker was inserted through the right femoral vein for the transient CHB. The patient underwent a Penumbra CAT RX Indigo mechanical aspiration thrombectomy and then placement of an Abbott XIENCE 4.0x23 mm stent and a Medtronic Resolute Onyx 4.5x15 mm stent in the proximal and mid RCA. However, he required several boluses of adenosine and nicardipine because of the no-reflow phenomenon. Despite this, the infarct-related artery (IRA) showed improvement to TIMI 1-2 flow only (Figure 9). Thus, we placed an intra-aortic balloon pump and initiated tirofiban infusion for 18 hours. Though the final coronary flow was slow, his ST-segment elevations were resolved completely, and the patient was chest pain-free after the PCI was performed. He had been started on aspirin and clopidogrel and was discharged three days later.

Discussion

CAE is defined as the dilatation of a coronary arterial segment to a diameter of at least one and half times that of the contiguous, normal coronary artery diameter.[3] The prevalence of an IRA with CAE leading to AMI is 2.6-4.9 percent.[2,4] However, CAE has been observed in 0.1-0.44 percent of patients who underwent coronary angiography for suspected coronary artery disease (CAD).[5] CAE is concurrently associated with CAD, and atherosclerosis is also implicated as a major contributor to CAE. Other causes of CAE include congenital, various acquired pathological conditions including connective tissue disorders such as

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*Truth Initiative Survey, January 2021

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Marfan syndrome and Ehlers-Danlos syndrome, autoimmune conditions such as systemic lupus erythematosus and rheumatoid arthritis, vasculitides such as Kawasaki disease and polyarteritis nodosa, chest trauma, iatrogenic complications stemming from coronary angioplasty, the usage of cocaine and amphetamines, and sickle cell disease.[6,7]

Historically, various studies have shown an association with the male gender, hypertension, dyslipidemia, smoking, and stroke. In contrast, diabetes mellitus (DM) is less prevalent in patients with CAE.[1,8-10] The RCA is most commonly involved as an ectatic IRA (EIRA) causing STEMI, followed by the LAD artery and the LCX artery.[11] The culprit lesion is usually located on the proximal or mid segment of an EIRA, and it tends to have a larger mean luminal diameter compared to the culprit lesion segment of a non-EIRA.[1] Due to the thrombogenicity of the ectatic segments, the rates of prior and current AMI are higher in patients with CAE.[12] The thrombogenicity of the ectatic regions, though, is suspected to be secondary to stasis, low shear stress, and spasm in the coronary arteries.[13] The ectasia alone may cause AMI even in the absence of concurrent severely stenosed coronary arteries.[10,14]

There are four distinct types of CAE according to Makris et al.: type 1, with diffuse ectasia in two or three arteries; type 2, with diffuse ectasia in one artery and localized ectasia in another artery; type 3, with diffuse ectasia in one artery; and type 4, with discrete ectasia in one artery.[15] In a case-control study by Schram et al., CAE was observed as a strong and independent predictor of the no-reflow phenomenon in patients with STEMI who underwent primary PCI after adjusting for multiple variables including age, gender, DM, hypertension, smoking, previous myocardial infarction, previous coronary artery bypass graft surgery (CABG), thrombus grade, TIMI flow of 0-1 pre-PCI, and RCA-related ectasia.[3] The no-reflow phenomenon occurs significantly more often in patients with EIRAs, especially when the vessel diameter is greater than 4.0 mm in diameter, and is due to the high thrombus burden in the ectatic portion of the coronary artery, contributing to the potential distal microembolization of the coronary vasculature.[2,11] Although a patient with AMI from an EIRA has a large thrombus burden in the ectatic area and an associated slow coronary flow state, the measured myocardial infarct size may not be significantly different when compared to that of a patient with AMI due to a non-EIRA.[12]

The exact pathophysiology of CAE is not clearly understood. There is an upregulation of platelet reactivity and the coagulation system as indicated by Liang et al., in a study that showed that patients with CAE and AMI had higher mean platelet volumes and fibrinogen levels.[12] Previous histopathological studies revealed a decrease in fiber content in the media and obliteration of the internal and external laminae, leading to attenuation of the vessel wall, with a thickness of less than 1 mm in the regions of CAE.[15] Karabulut et al. studied the interaction of preinfarction angina and CAE in patients with AMI who were treated with primary PCI. They observed that CAE is more prevalent in patients with preinfarction angina, and 61.5 percent of the ectatic arteries were deemed to be infarct-related.[8]

There is no clear consensus when it comes to the management of STEMI caused by an IRA with CAE due to the lack of randomized controlled trials. Most evidence is derived from retrospective case-control studies and anecdotal case reports of patients with CAE. Theron et al. reported on the successful reperfusion of an IRA with an ectatic RCA with intracoronary streptokinase administration in patients with acute inferior myocardial infarction.[17] Similarly, intracoronary administration of low-dose TNKase in a severely ectatic RCA was shown to reduce a large thrombus burden during primary PCI.[18] Avoidance of nitroglycerin has been suggested due to its worsening of coronary flow in patients with CAE with existing sluggish flow, as it may lead to further thrombus formation.[5] Also, despite the higher periprocedural and postprocedural use of glycoprotein IIb/IIIa inhibitors and periprocedural anticoagulation for EIRAs, no significant increase in major bleeding has been reported.[1]

A recent study showed that patients with CAE and AMI who were on antiplatelet agents preadmission had lower rates of AMI.[12] Early administration of systemic-loading doses of aspirin and clopidogrel and intracoronary-loading doses of tirofiban has been reported to augment the distal flow of the CAE-IRA in patients with AMI undergoing primary PCI.[8] This was supported by a small study in which there was overall increased fibrinolytic activity and serum D-dimer levels in patients with CAE compared to angiographically normal coronary arteries.[19] There have been case reports showing that the addition of oral anticoagulation (OAC) prevents further recurrences of AMI due to ectatic IRAs.[20,21] A recent large observational study showed that, with warfarin, the targeted therapeutic range (TTR) is maintained ≥60 percent in patients with CAE and AMI, the occurrence of nonfatal MI and cardiac death is lower when compared to a TTR below 60 percent or in the absence of OAC.[22]

Although there was a higher utilization of excimer laser coronary angioplasty (ELCA) in STEMI patients with an EIRA (20.5 percent; n=8/39) compared to a non-EIRA (6 percent; n=42/705) in one study, the authors did not look into PCI procedural outcomes when ELCA was used.[16] Thus, the utility of ELCA in STEMI patients with an EIRA who tend to have a large thrombus burden remains unknown. Other treatment options include lone aspiration thrombectomy, pulse-spray thrombolysis, and a "pharmaco-mechanic" strategy including sequential intracoronary thrombolysis, manual aspiration thrombectomy, and mesh-covered stent implantation.[14,23,24] Stent malapposition may occur due to an irregularity in the coronary artery contour, and intracoronary imaging can be considered.[25]

Fujii et al. conducted a retrospective study to compare the immediate PCI outcomes and angiographic or clinical outcomes at 30 days and 360 days in STEMI patients with EIRAs (*n*=39/744) versus non-EIRAs (*n*=705/744).[16] The final TIMI grade 3 flow and angiographic success was 53.8 percent in the ectatic group and over 90 percent in the nonectatic group. In contrast, the all-cause mortality was significantly better in the ectatic group compared to the nonectatic group at 30 days and 360 days, surprisingly. Despite the initially poor TIMI 3 flow rates following the initial PCI for patients with ectasia, nearly 85 percent with TIMI ≤2 flow showed restoration of TIMI 3 flow at one year following a coronary angiogram. Conversely, only 25 percent of the nonectatic group showed improvement irrespective of the final blush grade.[16] Erden et al. noted a significantly lower ST-segment resolution and collateral vascular development in EIRA patients with AMI compared to non-EIRA patients with AMI.[2]

Bogana Shanmugam et al. observed that patients with EIRA STEMI may not achieve a final TIMI flow grade of 3 (or even 1 or 2), with low angiographic and procedural success compared to non-EIRA STEMI patients.[1] Despite this, the in-hospital composite clinical cardiovascular events, including mortality, recurrent MI, unstable angina, and the need for CABG, did not differ significantly between those groups. However, upon the long-term mean follow-up of 36.6±14.1 months, patients in the EIRA group had a higher composite clinical event rate compared to the non-EIRA group, with no difference in all-cause mortality. Interestingly, patients with EIRA who were treated with stenting had angiographic and procedural success and a low rate of in-hospital cardiovascular events. At the end of long-term follow-up, there were no significant differences in all-cause mortality or composite clinical cardiovascular events between the two groups.[1]

Boles et al. reported that in spite of increased amounts of inflammatory markers in patients with CAE and AMI who required urgent primary PCI, their major adverse cardiac events and/or mortality upon two-year follow-up did not vary when compared to those with AMI due to coronary artery atherosclerosis.[9] However, Doi et al. found an increase in cardiac death and nonfatal MI in patients with CAE and MI upon a 49-month follow-up, and these observations were noted again in a multivariate Cox proportional hazards model and propensity score-matched analyses.[22] One study reported a 2 percent mortality rate in patients with STEMI and acute coronary syndrome and attributed this low number to undergoing revascularization appropriately.[10] Despite the less favorable and acute in-hospital and procedural outcomes, the overall long-term prognosis for patients with CAE and AMI appears to be, in general, encouraging and not significantly different from non-CAE-mediated AMI, despite the higher incidence of the no-reflow phenomenon, distal embolization, and microvascular dysfunction.

We did not observe DM as a comorbidity of the patients described in this paper which is in line with the reported literature in patients with EIRA causing STEMI. We felt the need to use glycoprotein IIb/IIIa inhibitors in our cases due to the high thrombus burden and slow/no reflow phenomenon periprocedural period. We used thrombectomy, especially Penumbra CAT RX Indigo mechanical aspiration thrombectomy in all our patients due to the large thrombus burden. Despite the TIMI flow of 1-2 in our case no.3, the post procedural hospitalization course was uneventful.

Conclusion

CAE is rare and associated with a high thrombus burden and the no-reflow phenomenon in STEMI due to EIRAs. The long-term prognosis seems to be favorable in general, regardless of poor, acute postprocedural and in-hospital outcomes. Sometimes, a single strategy may not work, and the institution of combined treatment modalities is required to treat the high thrombus burden associated with EIRA-related AMI.

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About the Authors:

Maheedhar Gedela, MD, Sanford Cardiovascular Institute, Sanford Heart Hospital, Sioux Falls, South Dakota; University of South Dakota Sanford School of Medicine, Sioux Falls, South Dakota; Mount Sinai Hospital and Icahn School of Medicine at Mount Sinai, New York.

Sharoon Samuel, MD, Sanford Cardiovascular Institute, Sanford Heart Hospital, Sioux Falls, South Dakota; University of South Dakota Sanford School of Medicine, Sioux Falls, South Dakota.

Christopher M. Van Hove, MD, Sanford Cardiovascular Institute, Sanford Heart Hospital, Sioux Falls, South Dakota; University of South Dakota Sanford School of Medicine, Sioux Falls, South Dakota.

Tomasz Stys, MD, Sanford Cardiovascular Institute, Sanford Heart Hospital, Sioux Falls, South Dakota; University of South Dakota Sanford School of Medicine, Sioux Falls, South Dakota.

Adam Stys, MD, Sanford Cardiovascular Institute, Sanford Heart Hospital, Sioux Falls, South Dakota; University of South Dakota Sanford School of Medicine, Sioux Falls, South Dakota.

Use of Nitrous Oxide for Minimal Sedation in Pediatric Outpatients: A Survey Analysis of Patient Experience and Parent/Guardian Satisfaction

Mir Ali, MD; Kayla Munger, MD; Ali Haines, MD; Laura Rezac, MD; and Emily Yoon, MD

Abstract

Introduction: The use of minimal and moderate sedation in the pediatric population allows for the successful completion of many procedures in both hospital and outpatient settings without the risks involved with general anesthesia. Sanford Children's outpatient sedation clinic had been using oral midazolam for minimal sedation but began using inhaled nitrous oxide in January 2019. The current study examines patient experience and parent/guardian satisfaction with use of inhaled nitrous oxide for minimal sedation.

Design/Methods: A survey was designed to evaluate parent/guardian satisfaction with nitrous oxide for pediatric sedation in various outpatient procedures. Parents'/ guardians' understanding of the sedation and procedural logistics were surveyed as well as their satisfaction with the child's comfort, recovery time, and overall satisfaction were assessed.

Results: Thirty-seven surveys completed by parents/guardians of patients ages 1-16 years were collected. Average age of the patient was 6 years old, with 22 female and 15 male patients. Outpatient procedures for which the nitrous oxide sedation was used included 30 botulinum toxin injections, 5 VCUG, 1 vaccine administration, and 1 IV placement. Mean survey results were 9.6 (95 percent CI, 9.3-9.9) for satisfaction of recovery time, 8.5 (95 percent CI, 7.7-9.3) for control of discomfort, and 9.1 (95 percent CI, 8.5-9.7) for overall satisfaction.

Conclusions: When evaluating nitrous oxide as an agent for minimal sedation in pediatric procedures, parents/guardians were most satisfied with the recovery time and least satisfied with its ability to control discomfort. Overall, we concluded that nitrous oxide is a moderately good agent for pediatric patients receiving minimal sedation.

Introduction

Sedation has long been used in the pediatric population to help perform both invasive and noninvasive procedures more comfortably. Providing sedation can help ease the associated trauma and allows for pediatric patients to remain still for the procedure's duration. The American Academy of Pediatrics goals of sedation include safety, anxiolysis, analgesia, amnesia, and the ability to enable completion of the procedure successfully.[1] In addition, the sedation would ideally have a rapid onset of action and quick recovery upon its discontinuation.[2] Both minimal and moderate sedation allow for depressed cognitive functioning and consciousness during the procedure while the patient is still able to protect his/her own cardiac and respiratory function.[2] The use of minimal and moderate sedation allows for the successful completion of many procedures in both hospital and outpatient settings and avoids the risks involved with general anesthesia.

Nitrous oxide (chemical structure is N_2O) is commonly referred to as "laughing gas" and is a colorless, odorless gas at room temperature. While the use of nitrous oxide as an adjunctive induction agent for anesthesia in both dentistry and the operating room has been well established, it is only recently that the use of nitrous oxide has become a popular, effective, and valuable method of sedation for the pediatric patient in emergency rooms and outpatient settings.[2] Nitrous oxide is an inhaled agent that induces analgesia, amnesia, and anxiolysis by acting on opioid receptors and the GABA inhibitory pathway.[2,3] Benefits of using nitrous oxide include its simple and noninvasive delivery method,

its rapid onset of action and quick recovery after discontinuation, its safety profile, and its effectiveness at providing minimal and moderate sedation.[2,3] Most patients experience no side effects, but the most frequently reported adverse effects include headache, nausea, and vomiting.[2]

Sanford Children's outpatient clinic began using inhaled nitrous oxide in January 2019 for minimal sedation involving short procedures such as voiding cystourethrogram (VCUG), botulinum toxin injections, and other various simple procedures. The goal of this study was to evaluate parent or guardian satisfaction with use of nitrous oxide gas for minimal sedation at Sanford Children's outpatient clinic by administration of surveys. We hypothesized that the use of nitrous oxide would demonstrate high parent/guardian satisfaction overall. Results from this study are intended to establish a baseline satisfaction score with the use of nitrous oxide gas for pediatric sedation.

This study is the second arm of the previously published study by Pickthorn et al. in 2019 entitled "Use of midazolam for minimal sedation in pediatric outpatients: a survey analysis of patient experience and parent-guardian satisfaction." [4] The methods, participants, survey design, procedure, and statistical analysis are nearly identical in the two separate studies. Eventually, the results from this nitrous oxide study can be used for comparison to the results of Pickthorn et al. study on the use of midazolam for pediatric sedation, as well as other possible sedative alternatives. The results of such comparison studies will help guide future research work to improve overall pediatric medical practice regarding analgesic agents, should it be indicated.

Methods

This research was approved by both the University of South Dakota (USD) Institutional Review Boards (IRB) and Sanford Hospital, Sioux Falls, South Dakota. Since the study did not meet the FDA definition of clinical investigation for a human subject, Sanford accepted the work as non-human research according to the Protocol Template for Human Research Determination (HRP-503c). The work was approved as a Survey Research Application by the USD IRB. The Plan Do Study Act model for healthcare quality improvement was followed.

Inclusion criteria utilized for our study was modeled along the lines of a previous study with midazolam sedation in the pediatric population. Two essential elements were required to participate in the study. First, participants were required to be parents/guardians of a pediatric patient (ages 0-17) undergoing minimal sedation via nitrous oxide. Secondly, the pediatric patient must have successfully completed the simple outpatient procedure using nitrous oxide as the sedation. The four procedures participants underwent included voiding cystourethrograms (VCUG), botulinum toxin injection, vaccine administration, and intravenous catheter placement. All pediatric patients were seen in the outpatient setting at Sanford Children's Hospital, Sioux Falls, South Dakota.

Excluded from the study were patients with a contraindication to the sedation method or those who had previously experienced an adverse reaction to nitrous oxide sedation. There were three surveys that were not included due to sedation method substitution from nitrous oxide to midazolam during the procedure. These patients either did not tolerate the delivery route of nitrous oxide and/or the nitrous oxide did not provide adequate sedation. Because the current study focused on satisfaction with nitrous oxide, these surveys did not qualify for inclusion in the data analysis. Surveys of participants were still considered complete if all numerical response questions were answered but one or more of the surveillance or background questions were left unanswered. Surveys were also considered complete if most of the numerical response questions were complete despite an omission in questions 1-7.

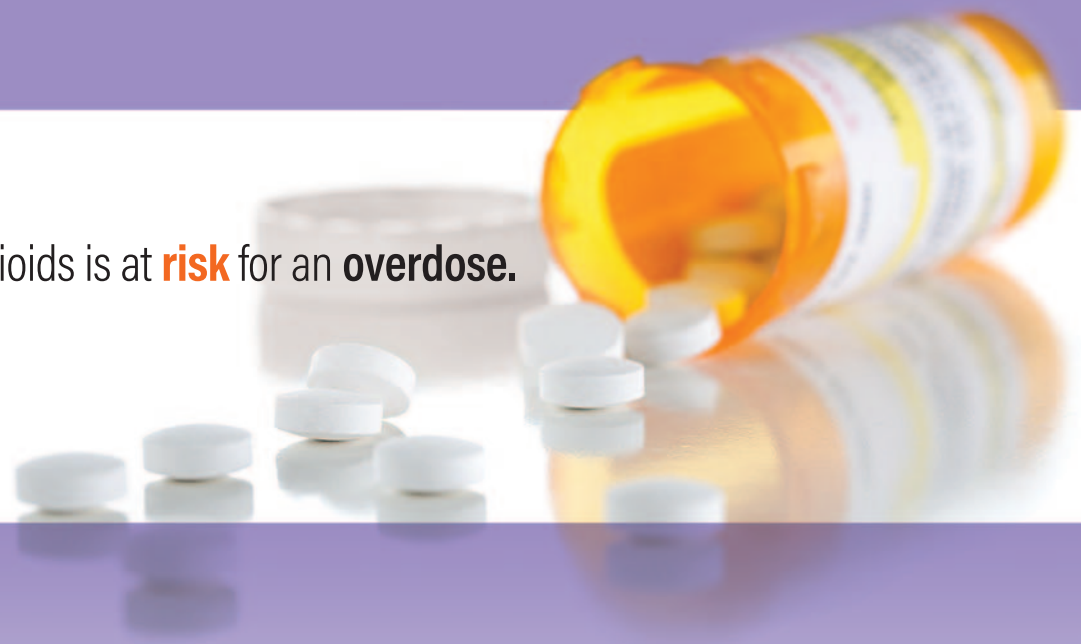
Survey Design

The survey design in this study was identical to the survey formulated in the first arm of the study done by Pickthorn et al., which assessed the use of oral midazolam sedation. The 10-question survey included questions 1-7 assessing the background of the patient and the procedure logistics, while questions 8-10 assessed satisfaction using a numerical scale of 1-10 (1 being "not satisfied" to 10 being "extremely satisfied." There was an optional comments section concluding the survey. The survey did not have patient or parent/guardian identifying information and the surveys were not linked to the patient's electronic medical record. The utilized survey is included in Appendix A at sdsma.org.

Procedure

Initial contact with participants was via written communication in which an envelope was provided to parents/guardians of eligible participants. Included in the envelope was a 'consent to participate in research' letter explaining the prospective observational study and its aims. Furthermore, it reiterated that participation is strictly

Anyone taking opioids is at **risk** for an **overdose.**



Help your patients recognize the symptoms of an opioid **overdose.**

Most opioid overdoses in South Dakota are *accidental*. If your patients have opioids in their homes, *remind them* of what to look for if an overdose is suspected.

What to watch for:

- Small, constricted "pinpoint pupils"
- Falling asleep or loss of consciousness
- Slow, shallow breathing
- Pale, blue, or cold skin or fingernails
- No response when they rub the middle of the chest with knuckles
- Vomiting, choking, or gurgling sounds

What can be done:

Suggest to patients that they keep naloxone on hand. **It can save a life.** Encourage them to keep their loved ones safe by taking these easy, preventative steps:

- Get naloxone for FREE from any pharmacy
- Order a FREE medication lock box for safe storage
- Order a FREE Dispose Rx packet to safely get rid of unused or expired medication or find a take-back location

Refer your patients and their families to the

Resource Hotline
1-800-920-4343

It's FREE, confidential, and available 24/7

For more about the State Unintentional Drug Overdose Reporting System (SUDORS) data, visit **AvoidOpioidSD.com**

Avoid Opioid
PRESCRIPTION ADDICTION

Brought to you by the South Dakota Department of Social Services. Funding made possible through a Substance Abuse and Mental Health Services Administration (SAMHSA) grant. Nothing in this PSA constitutes a SAMHSA endorsement of content, services, or policies.

voluntary with no direct benefit or compensation to be had from participation. Also included in the envelope was the satisfaction survey to be filled out after the procedure in the patient's outpatient hospital room. The envelope with the completed survey inside was then placed in a secured drop-box. Data was manually entered into a Microsoft Excel file for analysis and double-checked by another researcher for accuracy. Any responses on the survey that were not completed were left blank in the analysis file. Quality analysis was conducted via a two-check confirmation system by research team members.

Results

Data was collected over a 10-month period from May 2019 to February 2020 at the Sanford Children's Hospital outpatient center. Participant response rate was 100 percent. There was no patient-identifying information on any of the documentation. Microsoft Excel (Version 1806; 2016) was used to enter and analyze the data from the post-procedure surveys. Primary outcome was measured by the subjective parent/guardian satisfaction score reported on the surveys following the procedure (rated on a scale of 1-not satisfied to 10-extremely satisfied).

Over the collection period of the study, a total of 37 parents/guardians of patients participated in the study using the exclusion criteria detailed above. All participants consented to be enrolled into the study and their responses were collected and analyzed. Thirty surveys were from botulinum toxin injections, five were VCUG procedures, one was for vaccine administration, and one was an intravenous catheter placement. All survey responses confirmed that the sedation administration was described to them beforehand and that the child received sedation. Of these 37 participants, 34 of them had received sedation before in a similar setting. Patient demographics consisted of 15 males and 22 females. The age range was 1 year through 16 years old with an average age of 6 years. Survey results are shown as the mean, standard deviation, and 95 percent confidence interval in Table 1. On a scale of 1-10, the mean answer for question 8, "How satisfied are you with your child's recovery time after sedation?" was 9.5 (95 percent CI, 9.2-9.8). The mean answer for question 9, "On a scale of 1-10, how well do you feel that the sedation controlled your child's discomfort?" was 8.5 (95 percent CI, 7.7-9.3). The mean answer for question 10, "On a scale of 1-10, what is your overall satisfaction with the sedation used for this procedure?" was 9.1 (95 percent CI, 8.5-9.7). Questions 1-7 were employed in order to survey parents'/guardians' understanding of the sedation and procedural logistics. For example, these included "Was your child given sedation?", "Were there any adverse reactions to the sedation?", and "Did your child's procedure start on time?" The parents/guardians were also educated on the most common adverse effects prior to administration of nitrous oxide. There were two adverse events subjectively reported by the parents/guardians as "nausea, little vomit" and "groggy."

Table 1. Summary of numeral data from satisfaction survey.

Question	Mean	Standard deviation	95% confidence interval
Satisfaction with recovery time (question 8)	9.5	0.9	±0.30
Control of discomfort (question 9)	8.5	2.4	±0.81
Overall satisfaction (question 10)	9.1	1.7	±0.60

Discussion

The intent of this prospective observational study was to establish a baseline satisfaction score for nitrous oxide sedation used in pediatric outpatient procedures as determined by a parent/guardian survey at the Sanford Children's Hospital. By examining the satisfaction of nitrous oxide use in the pediatric population, we establish a data set pertaining to satisfaction scores that can be utilized in future studies for comparison with other sedation methods.

Patients included in this study were successfully sedated with nitrous oxide in order to complete his or her respective procedure. We found that generally, parents/guardians were satisfied with nitrous oxide sedation overall (9.1 with 95 percent CI, 8.5-9.7). Especially rated highly on the survey was satisfaction with recovery time of nitrous oxide (9.5 with 95 percent CI, 9.2-9.8). This finding is in line with nitrous oxide's rapid onset and short duration of effect,[2,3] which are qualities that make it attractive for use in simple outpatient procedures. In comparison to the other satisfaction scores, a lower satisfaction score was found with the control of discomfort (8.5 with 95 percent CI, 7.7-9.3). In addition, the adverse effects reported throughout the study were minimal and mild, supporting nitrous oxide's strong safety profile. Overall, we feel that the study demonstrated that nitrous oxide yielded good parent/guardian satisfaction.

Limitations

The biggest limitation of this study was the small sample size, which makes it difficult to generalize the results to a larger population. The small sample size can be

attributed to the limited time period for data collection. It is expected that a larger number of responses could be collected with a longer survey capture window.

Future Work

In the future, the satisfaction scores for use of nitrous oxide obtained in this study could be compared to those of oral midazolam obtained in Pickthorn et al.'s 2019 study, using a retrospective study design. Further, a prospective, blinded, randomized, control study could be performed to directly compare satisfaction with nitrous oxide to satisfaction with midazolam. It may also be interesting to compare patients of the same age and gender who underwent the same outpatient procedure to minimize variables and better evaluate any differences in satisfaction between oral midazolam and inhaled nitrous oxide. A study focused on comparing the duration of recovery with midazolam versus nitrous oxide might also be of special interest from a public health standpoint; faster recovery can lead to a shorter total duration of procedure, which might in turn improve efficiency and ultimately decrease healthcare costs in this setting.

Conclusion

When evaluating nitrous oxide as an agent for minimal sedation for the pediatric population undergoing outpatient procedures, parents/guardians were most satisfied with the duration of recovery from undergoing sedation. A lesser satisfaction was noted regarding its ability to control the patient's discomfort as perceived by the patient's parent/guardian. Overall, we concluded that nitrous oxide is a moderately good agent for pediatric patients receiving minimal sedation in the outpatient setting.

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About the Authors:

Mir Ali, MD, Assistant Professor, University of South Dakota Sanford School of Medicine, Sioux Falls, South Dakota; Pediatric Critical Care Intensivist, Sanford Children's Specialty Clinic, Sioux Falls, South Dakota; Sanford Hospital, Sioux Falls, South Dakota.

Kayla Munger, MD, University of South Dakota Sanford School of Medicine, Sioux Falls, South Dakota.

Ali Haines, MD, University of South Dakota Sanford School of Medicine, Sioux Falls, South Dakota.

Laura Rezac, MD, University of South Dakota Sanford School of Medicine, Sioux Falls, South Dakota.

Emily Yoon, MD, University of South Dakota Sanford School of Medicine, Sioux Falls, South Dakota.

Susac Syndrome Presenting as Recurrent Strokes

Abdelmohaymin Abdalla, MD; Mansi Oberoi, MD; Mohamed Abdallah, MD; and Tony Oliver, MD

Abstract

Susac syndrome (SS) is a rare clinical entity that affects primarily young women and might result in significant morbidity. The triad that leads to suspecting the disease has classically been involvement of the brain, retina and inner ear. The likely pathology of the disease is thought to be immune mediated endotheliopathy; given its clinical and, possibly, pathological remission with immunosuppressive therapy. Here we describe an uncommon recurrent stroke in a young female that unfolds to Susac syndrome at the end. We also reviewed the literature behind diagnosis and treatment. Delayed diagnosis is associated with worse morbidity and mortality, and the most important predictor of long-term prognosis in the reported cases is the time to diagnosis. Therapies tried (with variable success) include corticosteroids, IVIG, plasmapheresis, cyclophosphamide, mycophenolate mofetil, and rituximab. The prognosis of SS is difficult to predict given the absence of strong clinical or radiographic features to suggest better/worse prognosis at the time of initial diagnosis. Brain MRIs and hearing/vision impairment have never normalized in previously studied cohort of patients.

Background

Susac syndrome (SS) is a rare disease that affects the brain, retina, and the inner ear. It is thought to be autoimmune in nature, but the exact etiology is not known.[1] Dr. John Susac originally described this clinical syndrome in 1975, when he saw two patients with suspected granulomatous angiitis (now known as primary CNS vasculitis). Many years later, he made the distinction between SS and multiple sclerosis (MS) based on the fact that the former almost always affect the corpus collosum.[2] The same clinical triad was also known as retinocochleocerebral vasculopathy, RED-M (retinopathy, encephalopathy, deafness associated microangiopathy), and SICRET (Small infarcts of cochlear, retinal and encephalic issues).[3-5] In 2003, Dr. Susac suggested a diagnostic pathway that involves central supratentorial demyelination along any two of the three organs commonly affected by the disease; namely encephalopathy, hearing loss and branch retinal artery occlusion (BRAO).[6,7] This myriad of clinical and neurological manifestations is treatable with immunosuppression. However, late diagnosis and subsequent late initiation of treatment are reported to be fatal.[8]

No diagnostic or treatment algorithm has been established for SS, but in most cases, escalation of immunosuppressive therapy is required early in the disease course. Immunosuppression with mycophenolate mofetil, rituximab; followed by addition of cyclophosphamide in refractory cases is most commonly used.[9] Here we present a clinical mystery of recurrent strokes in a middle-age female that later evolved into a presentation of SS. We also present a literature review about diagnostic imaging and treatment options/success.

Case Presentation

A 56-year-old Caucasian woman presented to the emergency department (ED) with dizziness that started three weeks prior to presentation. She described bilateral sensation of spinning around her head, which was exacerbated with head movement, which was more profound on the right side. She also complained of headaches and minimal hearing impairment. The patient denied head and neck trauma, vision changes, swallowing difficulty, facial droop, motor weakness, sensory disturbances, or bowel or bladder dysfunction.

Her past medical history was significant for seropositive rheumatoid arthritis, which was well controlled with hydroxychloroquine and methotrexate. The patient is a former smoker of seven pack years who quit 17 years ago but does not drink alcohol or use illicit drugs.

On physical examination, the patient was alert and oriented, and did not appear to be in acute distress. Pupils were equal and reactive to light and the extraocular eye movements were intact. The patient had horizontal nystagmus bilaterally, more on the right. Ear examination revealed no drainage

from ears and no fluids was seen behind the tympanic membranes. Facial sensation and movement, motor and sensory examination of upper and lower extremities was normal. Heart, chest, abdomen, and skin exam was unremarkable.

Laboratory tests obtained in the ED, including complete blood count, complete metabolic panel and coagulation profile were within normal values. Magnetic Resonance Imaging (MRI) brain without the use of intravenous contrast revealed subacute infarcts involving the central/left aspect of the splenium of the corpus callosum, left parietal white matter, left basal ganglia, and anterior right cingulate sulcus (Figure 1). Computerized tomography (CT) angiography of the head and neck arteries was unremarkable, and the patient was admitted to the stroke unit and was started on aspirin and clopidogrel for secondary stroke prevention. Transthoracic echocardiogram, cardiac rhythm on telemetry did not reveal any abnormalities. Therefore, an implantable loop recorder (ILR) was placed at time of hospital discharge to look for intermittent cardiac arrhythmia.

The patient was later re-admitted to the hospital four days later with dizziness, headache, and confusion. She had mild weakness on the left side of her body with reduced power (+3). Repeat MRI showed new strokes in the left corona radiata with expansion of the previous infarct into the corpus callosum. ILR was interrogated but no cardiac arrhythmia was detected. The patient was discharged afterwards to inpatient rehabilitation. A few days later, the patient developed increased confusion/disorientation, anxiety, and speech difficulties. She also reported worsening hearing loss on the left ear, fullness, and some tinnitus. Repeat MRI of the brain revealed multiple new areas of infarction in bilateral cerebral hemispheres (Figure 2). Cerebral angiogram was done to evaluate vasculitis but was normal. MRI brain with and without intravenous contrast revealed enhancing lesions and areas of leptomeningeal enhancement. Lumbar puncture (LP) and cerebrospinal fluid (CSF) analysis showed elevated protein at 252 (normal range: 50-80 mg/dL), but other CSF studies: bacterial cultures, cytology, meningitis/encephalitis polymerase chain reaction (PCR) viral panel, angiotensin converting enzyme level, myelin basic protein, multiple sclerosis (MS) panel, N-methyl D-aspartate (NMDA) were normal.

During the following two weeks, the patient's mental status waxed and waned from awake and alert but slightly confused, to minimally responsive at times. Electroencephalogram (EEG) was negative for seizure. Repeat MRI brain showed new white matter lesions in the

Figure 1. MRI brain without contrast revealed subacute infarcts involving the central/left aspect of the splenium of the corpus callosum (yellow arrow) and left basal ganglia (blue arrow).

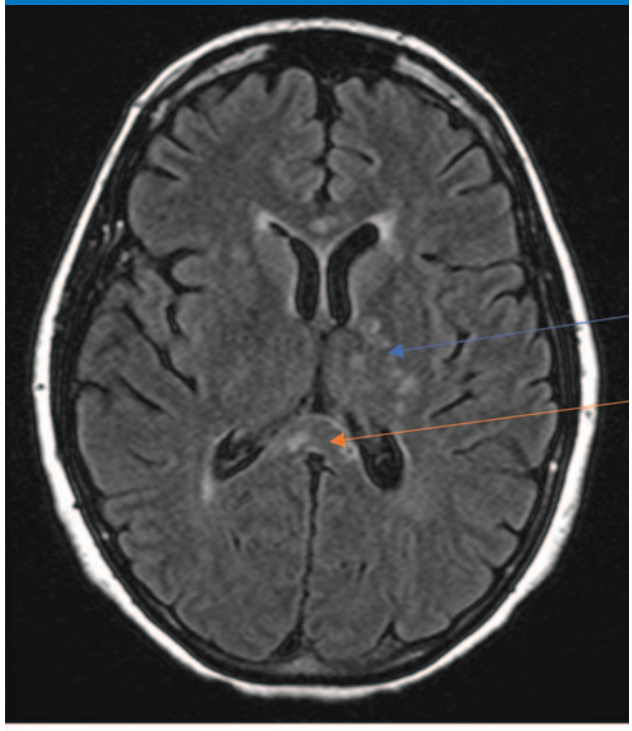
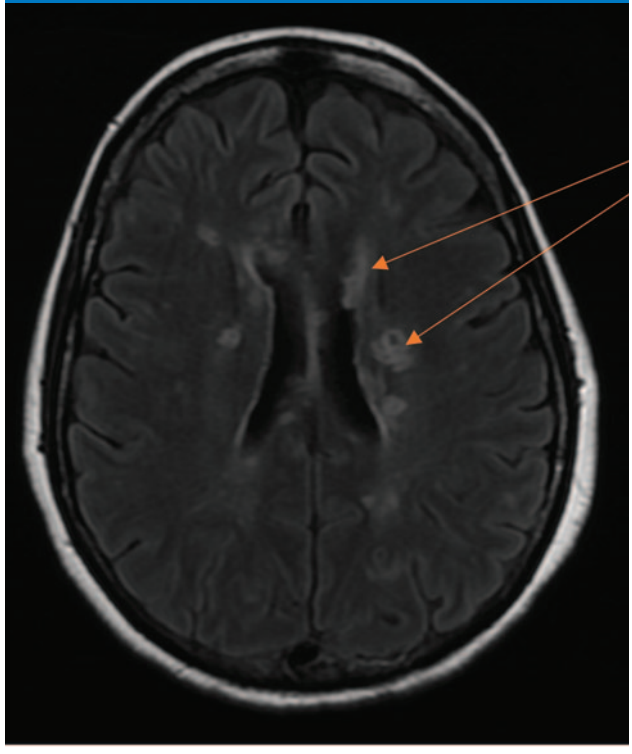


Figure 2. Repeat MRI brain revealed multiple new areas of infarction in bilateral cerebral hemispheres (arrows).



left temporal lobe. LP was then repeated, and it showed high protein at 277. Other CSF studies were again unremarkable. Due to the continued cognitive impairment/encephalopathy and unrevealing workup, brain biopsy for leptomeningeal enhancements was obtained, and it only showed fragments of benign brain parenchyma and meninges with focal hemorrhage with no definitive etiology/diagnosis. The patient was started on a full week course intravenous methyl-prednisone (1mg/kg) and intravenous immunoglobulin (IVIG). Her mentation started to improve on this regimen, but she continued to develop episodes of new focal weakness. Due to non-resolving hearing deficit when she is alert and awake; audiological evaluation was done, which showed bilateral asymmetric sensorineural hearing loss, left ear worse than right. The patient did not complain about vision changes initially but later had blurred vision in her left eye. Due to her recurrent strokes and recurring admission to the hospital, full ophthalmology assessment could not be completed. However, because of the high suspicion for SS, sedated fluorescein angiogram was obtained, and it showed multifocal arterial occlusion of left eye. After revising the European Susac Consortium diagnostic criteria (Table 1); a definitive diagnosis of Susac syndrome was made based on the combination of multiple stroke-like episodes/confusion, retinal artery branch occlusion and hearing loss.[10] Treatment with cyclophosphamide and prednisone was initiated, but unfortunately, the patient sustained a significant impairment from recurrent strokes. The patient, however, did not develop any new neurological symptoms for at least nine months since starting cyclophosphamide.

Discussion

SS is one of the rarest neurological disorders that occurs more commonly in young females. Between 1979 and 1994, only four cases were described in the literature and all of them were females.[2,5] The disease is usually diagnosed at a very late stage, not only due to rarity but also secondary to the wide variety of clinical mimickers, including MS (the most commonly suspected diagnosis), Meniere's disease, lupus encephalopathy, aseptic meningitis, sarcoidosis (and other granulomatous diseases), Lyme disease and complicated migraine.[2,3,11] Similar to our patient's story, the clinical course of most reported cases follows an acute fluctuating course with recurrence of symptoms ranging from weeks to years (up to 23 years) after the onset of the disease.[12] Headaches and confusion (encephalopathy) are the most common symptoms reported in the literature, which are thought to be due to autoimmune leptomeningeal involvement.[13] Other common symptoms include hearing loss, vertigo, ataxia, muscle pains and even urinary dysfunction.[1,14] Before committing to such rare diagnosis, most connective tissue diseases, infections, demyelinating disorders and cerebrovascular diseases should be excluded using appropriate testing.[5]

Rennebohm et al. proposed an immune mediated endotheliopathy as the likely etiology of this condition given its clinical and, possibly, pathological remission with immuno-

Table 1. Proposed diagnostic criteria for diagnosing SS.

I. Definitive diagnosis of Susac syndrome: Each criterion (1, 2, and 3) with the sub-criteria (i, ii) must be fulfilled. **1. Brain involvement** i. Symptoms and clinical findings: new cognitive disorder and/or behavioral changes and/or new focal neurologic symptoms and/or new headaches. ii. Images: typical findings in cranial MRI - rnultifocal hyper-intensity, small round lesions, at least one of them in the corpus callosum ("snowball") in T2-weighted sequences (or FLAIR). **2. Retinal involvement.** i. No clinical findings and symptoms are required. ii. Ophthalmic examination: OARR or HPR in fluorescein angiography or characteristic signs of ischemia of the retinal branch in fundoscopy or TCO-DE. **3. Vestibule-cochlear involvement.** i. Symptoms and clinical findings: new tinnitus and/or hearing loss and/or peripheral vertigo. ii. Examination of the function of the inner ear: The hearing loss must be supported by an audiogram; vestibular vertigo must be supported by specific diagnoses.
II. Probable diagnosis of Susac syndrome: Incomplete triad, only two of the three previous criteria are met, 1-3.
III. Possible diagnosis of Susac syndrome: In all other patients who show some clinical and/or paraclinical findings from the previous triad but do not meet I or II, Susac syndrome should be included in differential diagnoses, but should not be considered as the most likely diagnosis.

HPA – hyperfluoroscence of the arterial wall; OARR – arterial occlusion of the retinal branch; TCO-DE – spectral domain of optical coherence tomography

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suppressive therapy. Interesting enough, he linked SS to dermatomyositis based on how they look alike in terms of natural history, immunopathogenesis and treatment.[15] There is no description of a familial pattern of SS.[1,13] Histopathology of lesions was described mostly as pre-capillary arteriolar inflammation with associated hypoxic damage to nearby neurons secondary to edema of the vessel wall and impaired diffusion of oxygen[1,13]

After a thorough review of the medical history and a detailed clinical examination (followed by a large panel of negative autoimmune and infectious workup), SS diagnosis should be confirmed using MRI of the brain along with detailed eye exam including fluorescein angiography to measure retinal blood flow. Non-specific markers of inflammation and CSF protein elevation (along pleocytosis) has been reported in most of the patients.[3,16] Contrast-enhancing white matter lesions documented on brain MRI involves many regions including deep grey matter (basal ganglia/thalamus), posterior fossa (70 percent) and leptomeningeal enhancement (33 percent). However, corpus collosum involvement is universal.[6] This latter finding served as the basis of distinguishing SS from MS. Retinal findings of SS can be detected on ophthalmoscopy, and fluorescein angiography is often needed to document BRAO.[4] Another interesting feature of SS is the lack of correlation between BRAO and reported vision deficits.[17] The same applies to sensorineural hearing loss which is thought to be secondary to an associated encephalopathy.[3]

Brain biopsy in many SS case reports was done mainly secondary to confounding clinical presentation or on postmortem examination. In the first reported case of SS, the author suggested using callosal lesions on MRI as a cornerstone for diagnosis (plus the other two criteria of SS triad) in order to avoid brain biopsy.[6] Reversible infarcts, necrotizing vasculitis, and amyloid angiopathy are among the most documented pathology findings.[3] To diagnose SS without a brain biopsy; corpus collosum white matter changes should prompt audiometry and testing for BRAO in encephalopathic patients.

Various immunosuppressive therapies had been reported with variable success. No randomized or crossover clinical trials have been done to date given the clinical rarity and the possibility of spontaneous remission. The most important predictor of long-term prognosis in the reported cases is the time to diagnosis; with delayed diagnosis resulting in severe disability and even mortality. Therapies used include corticosteroids, IVIG, plasmapheresis, cyclophosphamide, mycophenolate mofetil, and rituximab.[3,5,8,9,14]

The prognosis of SS is difficult to predict given the absence of strong clinical or radiographic features to suggest better/worse prognosis at the time of initial diagnosis. A six years follow up study of nine patients using hearing assessment, eye exams (including fluorescein angiography), and brain MRI showed clinical remission of encephalopathy with the use of steroids, and all patients were able to return to work by one year. However, brain MRI never normalized in these patients, and hearing and vision loss never normalized or even improved. There was no correlation between clinical course of SS and brain MRI, eye exam or hearing assessment findings on diagnosis.[18] Our patient did not have a follow up brain MRI or an eye exam; but was followed clinically and with routine care offered to those receiving immunosuppressive therapy.

Conclusion

SS can mimic recurrent acute strokes in younger patients (especially females). It is a rare clinical entity; however, early recognition and initiation of immunosuppressive therapy can slow disease progression and may reduce morbidity and mortality. The clinical, radiographic, treatment regimens, and prognosis continue to unfold as more cases are added to the literature.

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About the Authors:

Abdelmohaymin Abdalla, MD, Department of Internal Medicine, University of South Dakota Sanford School of Medicine, Sioux Falls, South Dakota.

Mansi Oberoi, MD, Department of Internal Medicine, University of South Dakota Sanford School of Medicine, Sioux Falls, South Dakota.

Mohamed Abdallah, MD, Department of Internal Medicine, University of South Dakota Sanford School of Medicine, Sioux Falls, South Dakota.

Tony Oliver, MD, Department of Internal Medicine, University of South Dakota Sanford School of Medicine, Sioux Falls, South Dakota.

BRINGING INNOVATION TO ORTHOPEDIC CARE AT SANFORD HEALTH

Harvey Oliver, MD, is an orthopedic surgeon and Sanford Health's new medical director of shoulder arthroplasty. He provides individualized, high-quality care for shoulders, knees and joint restoration, while always looking for innovative opportunities.

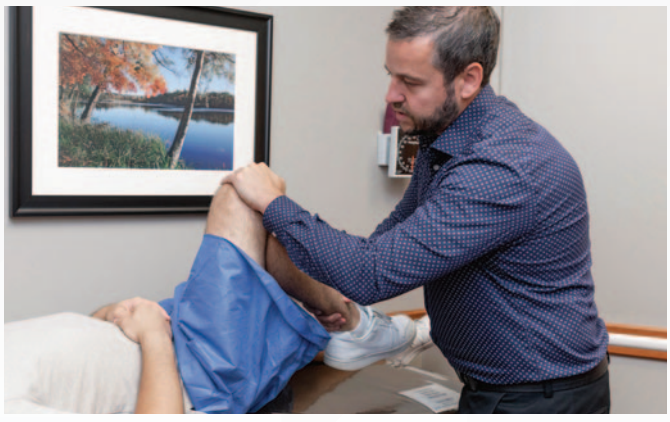
"We can take care of any shoulder issue. Whether it's a young person with an injury or an older patient with arthritis," said Dr. Oliver.

Typically, Dr. Oliver sees older patients dealing with years of shoulder pain who have exhausted all of their non-surgical treatment options. Dr. Oliver offers shoulder replacement procedures that aim to reduce pain and restore function.

"The happiest patients we see are shoulder replacement patients. They go from dealing with pain every day to having better function. It makes a huge difference in their quality of life," he said.

To help support better outcomes for his patients, Dr. Oliver offers patient-specific surgical planning. Many joint replacement programs provide generic implants, but Sanford Health uses imaging to create unique 3D computer models of each patient's shoulder. These images are then used when planning the procedure, choosing an implant and creating guides to use during the surgery.

In tougher cases, like if a patient previously fractured their shoulder, Dr. Oliver can use 3D printing to create a physical model of their unique shoulder joint to hold and study before operating.



Dr. Oliver also explores new ways to learn more about shoulder rehabilitation and further develop current treatments.

He now leads a rotator cuff tear clinical study that is trying to use a patient's own cells to treat a partial tear without surgery. Patients in the study have their adipose-derived stem cells harvested and injected into their injured shoulder.

> ***"We're always looking for the next best option instead of staying complacent and doing the same things we've always done,"***

said Dr. Oliver. In addition to developing their research offerings, Sanford Health's orthopedic team is looking to build on current services and specialties to better care for patients close to home.

"We're always expanding our services and becoming more specialized. We can take care of the simplest to the most complex issues, so patients don't have to travel to get specialized care," he said. "They can get it here in South Dakota."

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Non-Resolving Perihepatic Abscess Following Spilled Gallstones Requiring Surgical Management

Muhammad Waleed, MD; Muhammad Hassaan Arif Maan, MD; Muhammad Soban Arif Maan, MD; and Muhammad Arsalan Arshad, MD

Abstract

Introduction: Laparoscopic cholecystectomy (LC) is the treatment of choice for symptomatic cholelithiasis and is among the most frequently done procedures in United States. Spillage of gallstones occurs in up to 30 percent of these procedures and is associated with rare but important complications including abscess formation.

Case Description: We present a case of 44-year-old man with a peri-hepatic abscess developed three years after a laparoscopic cholecystectomy. Multiple percutaneous drainages and antibiotic courses had failed to provide a definitive resolution. CT scan showed signs of a developing abscess but no stones. A diagnostic laparoscopy was performed, and multiple retained stones were visualized. It was converted to open laparotomy and the abscess was drained along with resection of portions of liver and diaphragm. The patient remained vitally stable with no fever spikes following the procedure.

Discussion: Spillage of gallstones should be seriously considered in all patients presenting with peri-hepatic abscess with a history of previous LC, even if the imaging studies do not provide evidence of stones. Percutaneous drainage and antibiotics may provide temporary relief, but a surgical intervention is often the definitive management.

Introduction

Laparoscopic cholecystectomy (LC) is the treatment of choice for symptomatic cholelithiasis and is among the most done procedures in U.S. It is commonly associated with two main complications: bile duct injury and retention of gallstones in the peritoneal cavity. Whereas the bile duct injury has received significant attention, the issues related to the retained gallstones are often overlooked. Spillage of gallstones occurs in up to 30 percent of these procedures and is associated with rare but important complications including abscess formation.[1] These complications often have an unusual and remote presentation as they can take up to 20 years to manifest thus making a challenging diagnosis. These complications can result in significant morbidity and can have a devastating impact on the quality of life. In this manuscript, we highlight a case of spilled gallstones leading to recurrent intra-abdominal abscess.

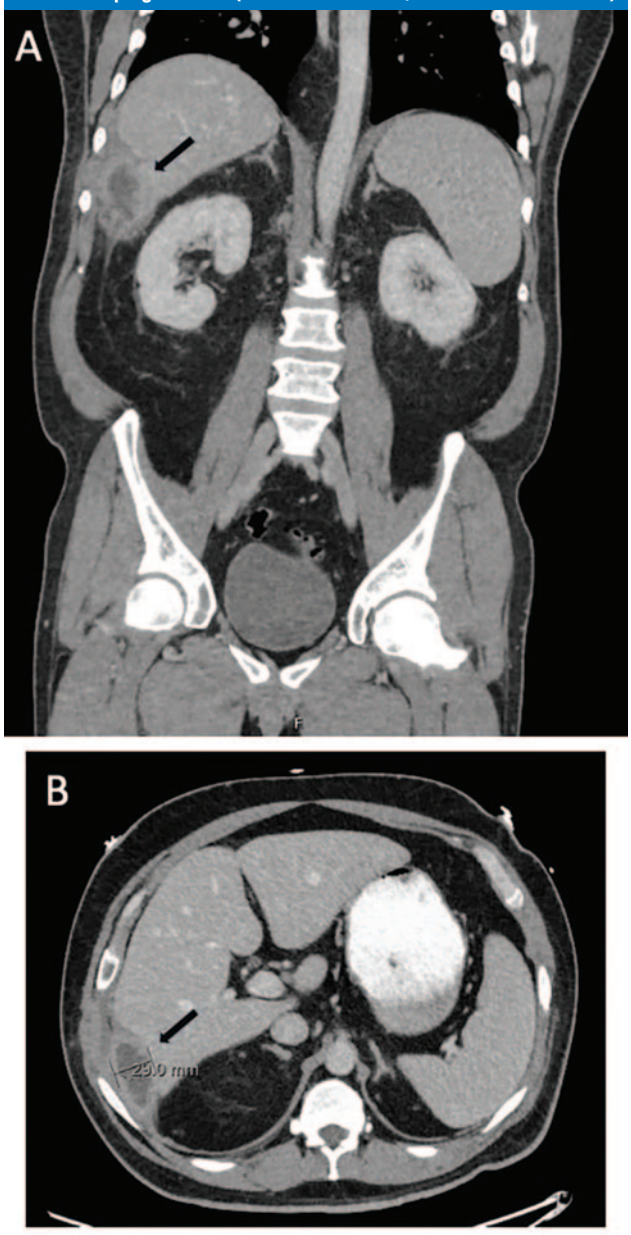
Case Description

A 44-year-old man, known case of diabetes mellitus type-2, GERD, hyperlipidemia and hepatitis C, presented to the emergency department with worsening abdominal pain for two weeks. Pain was felt most in right upper and lower quadrants. It had progressed from intermittent to a constant pain and was associated with a burning sensation, but no radiation. The pain worsened with movement and relieved with rest and ibuprofen. The patient was also tachycardic and had a low-grade fever.

The past surgical history was remarkable for a LC three years back. The past medical history was significant for a right peri-hepatic fluid collection (which was thought to be an abscess), first identified four months back, due to which the patient had made multiple hospital visits. On initial visit, peri-hepatic fluid was not enough for drain placement, but CT guided aspirate grew E. coli and patient was discharged on Levaquin with improvement. He presented again with similar complaint at which interventional radiology guided drain was placed and 2 cc of fluid was drained. Drain was subsequently removed and based on sensitivities of E.coli, patient was discharged on cefalexin. He remained on cefalexin until he presented again.

The patient presented to us again with similar complaint and was started on piperacillin/tazobactam prophylactically and laboratory investigations were ordered. There was no significant leukocytosis or transaminitis and the blood cultures came back negative. CT abdomen showed irregular, ring-enhancing complex fluid collection in the right perihepatic space with increasing size which was suspicious for a developing abscess (Figure 1 A and B). A CT-guided pigtail drainage was placed and 15 ml of purulent fluid was removed. Gram stain and culture grew E.coli and piperacillin/tazobactam was continued throughout hospital stay.

Figure 1. CT abdomen showing irregular, ring-enhancing complex fluid collection (arrow in 1A and 1B) in right perihepatic space suspicious for a developing abscess (1A: coronal section; 1B: transverse section).



The patient's high HbA1c level at 10.4 was thought as one of the causes of the non-resolution of the abscess. Moreover, there were concerns of spilled gallstone from the previous cholecystectomy which could act as a possible nidus for the infection. General surgery consulted, and the patient underwent diagnostic laparoscopy which was converted to open laparotomy with drainage of perihepatic abscess, resection of the segment VI of liver and a small piece of diaphragm. Multiple retained stones were removed from the abscess cavity.

After the procedure, the patient remained vitally stable with no fever spikes. Piperacillin/tazobactam were continued, and pain was optimally managed. Toward the end of hospital stay, the patient was deemed stable for discharge on oral antibiotics. Based on the culture sensitivities of drained fluid, ID recommended continuing oral Bactrim DS (sulfamethoxazole and trimethoprim) until follow up as outpatient. Repeat CT scan abdomen/pelvis showed progressive involution of peri-hepatic abscess and patient was taken off of antibiotics. On follow up with primary care provider, he continued to do well off of antibiotics.

Discussion

Spillage of gallstones is reported in 6-30 percent of LCs with stone retention in the peritoneal cavity in about 2.4 percent of the cases.[1] When LC is performed under the setting of acute cholecystitis, the edematous and delicate gall bladder wall can be torn easily.[2] The stones spilled can lead to complications in 0.08-2.3 percent of the patients.[3] In their literature review, Zehetner et al. and Nooghabi et al. identified multiple different complications associated with spilled stones including peritonitis, cellulitis, septicemia, fistula and abscess formation (Table 1).[2-4] Among these, abscess formation is the most common, accounting for 60 percent of the total complications.[5] Gallstones left in the peritoneum shelter viable enteric and non-enteric bacteria and thus can act as a nidus for infection.[3] These stones can cause inflammation which can lead to abscess, granulomatous reaction and can erode into abdominal organs.[6] Recurrent intra-abdominal abscesses have diverse causes, but usually result from an extension of infection or inflammation. Some common causes include, perforation of hollow viscus, diverticulitis, crohn's disease, pancreatitis, pelvic inflammatory disease, appendicitis as well as any condition that can lead to peritonitis.[7] However, in light of recent cholecystectomy, spilled gall stones should remain high on list of differentials.

The complications often have unusual presentations resulting in delayed diagnoses. With median time of about five months, the complications are reported to present up to 20 years after the original procedure. The diagnosis is often not made at the initial presentation and is only established after recurrence of the abscess.[8]

Imaging studies such as ultrasound, CT and MRI are generally helpful in establishing a diagnosis.[5] Typically, pure cholesterol stones and those with low calcium content are difficult to visualize on CT.[3] The imaging done in our patient did not show any evidence of retained stones which made the diagnosis difficult. However, similar findings of rim-enhancing intra-abdominal collections on CT scan in absence of gallstones has been reported in literature.[9]

Apart from antibiotics, various treatment modalities for a recurrent abscess due to spilled stones include imaging-guided percutaneous drainage, endoscopic stone removal

Table 1. Possible complications of spilled gallstones.[2,4]

Abdominal related complication
▪ Sub hepatic abscess
▪ Retro hepatic abscess
▪ Sub phrenic
▪ Intra peritoneal
▪ Right flank
▪ Paracolic and retroperitoneal
Intestinal complications
▪ Ileus
▪ Volvulus
Biliary tract complications
▪ Icterus
▪ Liver abscess
Thoracic complication
▪ Empyema and pleural effusion
▪ Pneumonia
Reproductive complications
• Chronic pelvic pain
• Infertility
Fistulas
▪ Umbilical or trocar site
▪ Bilio-cutaneous
▪ Skin
▪ Colo-cutaneous
▪ Colo-vesical
▪ Urinary tract
Miscellaneous
▪ Stone in hernia sac
▪ Wound infection
▪ Acute appendicitis
▪ Gluteal abscess
▪ Peritonitis
▪ Recurrent *Staphylococcal bacteremia*
▪ Vesical granuloma
▪ Dyspareunia
▪ Middle colic artery thrombosis

via percutaneous drainage tract and surgical drainage. The best treatment approach can vary depending on the site and size of the stones as well as patient's health.[10] Surgical approach is preferred by some authors as they believe it ensures a complete evacuation leading to less recurrence.[10,11] Percutaneous drainage is a less invasive measure which can be considered prior to surgical approach, especially in patients with superficial abscess or those who would not tolerate a surgery well.[10] However, as in our experience, percutaneous drainage and antibiotics may not provide long-term benefit until the main source of infection; the stone, is removed via a surgical intervention.

In an ideal scenario, the integrity of gallbladder should remain intact during removal; however, the inflammation leads to weakening of wall and distension in the organ making rupture likely.[3] The maneuvers associated with gallbladder perforation during the procedure include retracting gallbladder to visualize critical view while dissecting cystic duct and artery, separating it from the liver bed and extracting it via the incision in abdominal wall.[12] Careful dissection and pulling the gallbladder via a retrieval bag through port site incision as well as aspirating fluid from a tense gall bladder can decrease the risks of a perforation. Whereas spillage of stones does not necessitate conversion to an open procedure, attempt to retrieve the stones and thorough irrigation and suction of the abdominal cavity should be performed.[5]

In conclusion, despite the low incidence, complications from gall stone spillage can cause significant morbidity, especially in cases where multiple conservative treatment attempts have failed. Spillage of gallstones should be seriously considered in all patients presenting with peri-hepatic abscess with a history of previous LC, even if the imaging studies do not provide evidence of stones. Percutaneous drainage and antibiotics may provide temporary relief, but a surgical intervention is often the definitive management.

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About the Authors:

Muhammad Waleed, MD, Resident, Department of Internal Medicine, University of South Dakota Sanford School of Medicine, Sioux Falls, South Dakota.

Muhammad Hassaan Arif Maan, MD, Medical College, Aga Khan University, Pakistan.

Muhammad Soban Arif Maan, MD, Medical College, Aga Khan University, Pakistan.

Muhammad Arsalan Arshad, MD, Resident, Department of Internal Medicine, University of South Dakota Sanford School of Medicine, Sioux Falls, South Dakota.

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Delayed Presentation of a Flexor Digitorum Profundus Avulsion Injury: A Case Report

Nathan Whitsell, MD; and Robert Van Demark Jr., MD

Abstract

Avulsion of the flexor digitorum profundus (FDP) tendon is a relatively common injury in athletes. Also known as "jersey finger," it can also occur in nonathletes, and is often not initially diagnosed. Early diagnosis and repair are essential to regaining optimum return of function. We report a case of a 37-year-old woman who was seen six weeks following an undiagnosed FDP avulsion injury. Due to finger stiffness and the delayed diagnosis, she was treated with occupational therapy to maximize finger range of motion. The anatomy, classification, diagnosis, and treatment options for FDP avulsion injuries are discussed. The goal of this paper is to increase awareness for this injury, resulting in early diagnosis and prompt treatment.

Introduction

Hand injuries are common and comprise approximately 10 percent of all emergency department visits and up to 20 percent of all injuries treated.[1] Tendon injuries of the hand can include both extensor or flexor tendons and must be evaluated when treating injuries to the hand. Most tendon injuries have an obvious clinical presentation and are readily apparent, such as a mallet finger deformity caused by an extensor tendon avulsion. This is not always the case when dealing with an FDP avulsion injury. FDP tendon avulsion injuries are less common and are usually not diagnosed promptly.[2] A delay in diagnosis of this injury can result in suboptimal outcomes that can adversely impact hand function.[2]

Avulsion of the flexor digitorum profundus (FDP) tendon insertion occurs when the flexed distal interphalangeal joint (DIP) is suddenly hyperextended. This injury is commonly referred to as "jersey finger" or "rugby finger." This name is derived from the common injury mechanism seen in American football and rugby, when a tackler flexes their fingers to grab an opponent's jersey and the opponent pulls away leading to forced hyperextension of the flexed finger.[2-4] Early diagnosis and timely repair are key to successful treatment. We present a patient who was seen six weeks after an undiagnosed FDP avulsion injury. Due to the late presentation, surgery was not indicated, and she was treated with occupational therapy.

Case Report

A 37-year-old right-handed woman injured her right ring finger when her finger was caught between a bicycle handlebar and metal railing. Two days after the injury, she presented to an orthopedic urgent care clinic. She complained of a sharp throbbing pain over the proximal interphalangeal (PIP) joint. Physical examination demonstrated edema over the PIP joint with decreased range of motion secondary to pain and swelling. Initial radiographs were read as normal and she was diagnosed with a volar plate injury. She was discharged with an Alumifoam splint and told to progress

Figure 1. Lateral radiographs five weeks after the injury demonstrating a large bony fragment at the level of the PIP joint (blue arrow) which was avulsed from the distal phalanx (red arrow).

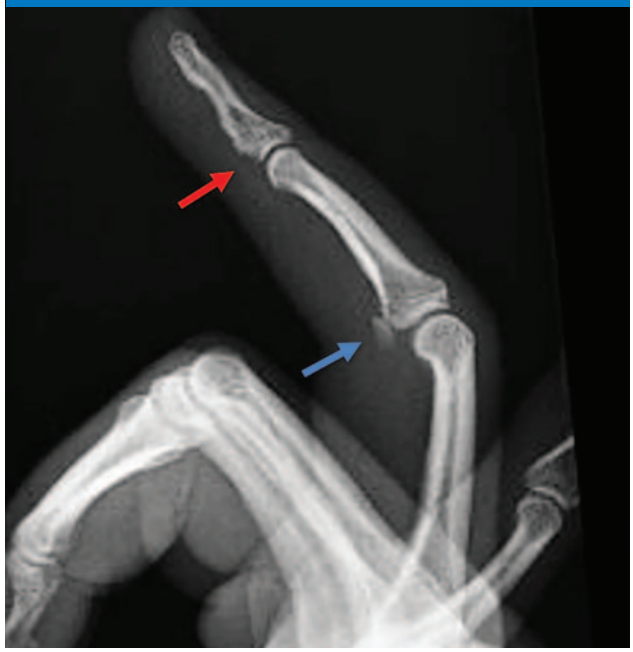
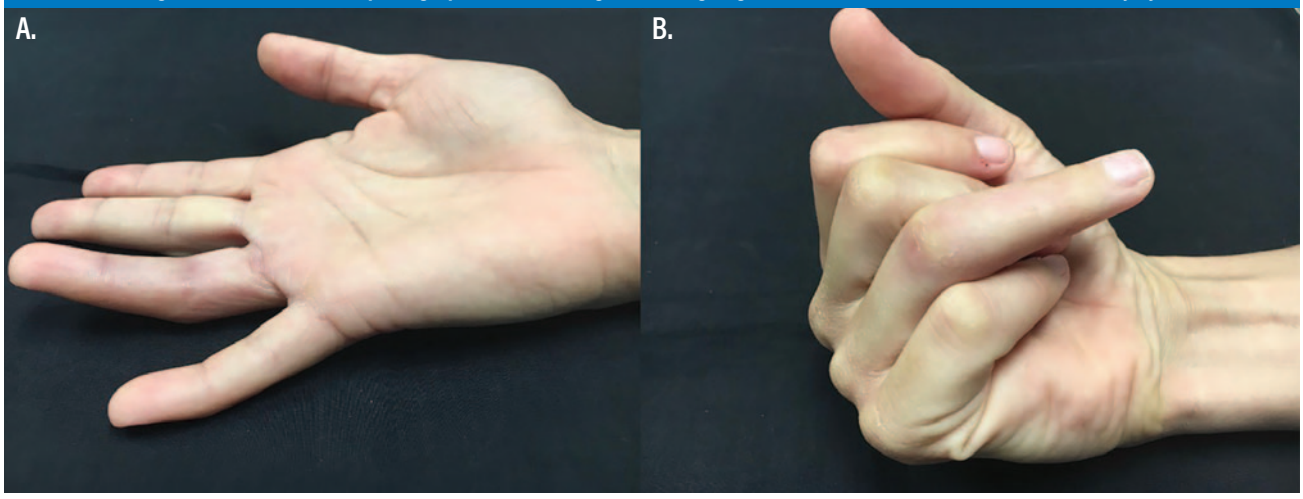


Figure 2A and 2B. Clinical photograph demonstrating lack of ring finger flexion consistent with a FDP avulsion injury.



to buddy taping over the next week. Five weeks after the initial visit, she saw her primary care provider for persistent pain, edema, and stiffness. A repeat true lateral radiograph of the finger revealed a bony avulsion injury from the distal phalanx with the fracture fragment located at the volar aspect of the PIP joint (Figure 1).

She was referred to our clinic for further evaluation. Physical examination revealed moderate edema of the right ring finger without ecchymosis or erythema. Palpation along the flexor tendon sheath was painful with no tenderness noted in the palm over the annular (A1) pulley. The PIP joint was stiff, and a flexion contracture was noted. Range of the motion of the PIP joint was -25/60 degrees. There was no active flexion of the ring finger DIP joint (Figure 2A and 2B). Ultrasound findings of the ring finger were consistent with a Type II FDP avulsion injury. Due to the delay in diagnosis and amount of PIP joint stiffness, we felt that she was not a candidate for surgical repair of the tendon avulsion. She started an occupational therapy program to improve finger motion. Four weeks later, PIP joint motion had improved but she still lacked FDP function.

Discussion

Traumatic avulsion of the FDP tendon (i.e., jersey finger) is not uncommon, and occurs in athletes and nonathletes.[5] In 1933, McMaster showed experimentally that a rupture rarely occurs in the substance of a normal tendon, and that a normal tendon usually ruptures at its bony insertion.[6] Forced hyperextension of the DIP joint during contraction of the profundus tendon can avulse the tendon from its distal phalanx insertion, resulting in proximal retraction to a variable degree. The extension force may be powerful enough to fracture part of the distal phalanx along with the tendinous insertion.[7] The ring finger is involved in more than 75 percent of cases, but the injury has been reported to occur in all digits.[3,8]

The flexor digitorum profundus tendons travel through the carpal tunnel and course along the volar side of the palm and phalanx before passing distally through the Chiasm of Camper and inserting at the base of the distal phalanx. The vincula supply blood vessels and nerves to the FDP tendon.[5,9] The long (vincula longa) and short (vincula breve) vinculum nourish the phalangeal portion of the tendon (Figure 3).[4]

Figure 3. Figure of Camper's Chiasm and the vincula longus and vincula brevis. *Used with permission of Orthobullets.*

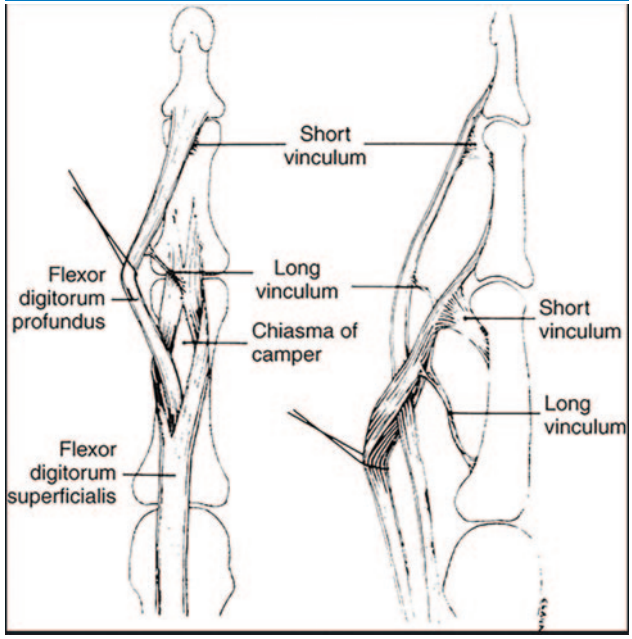
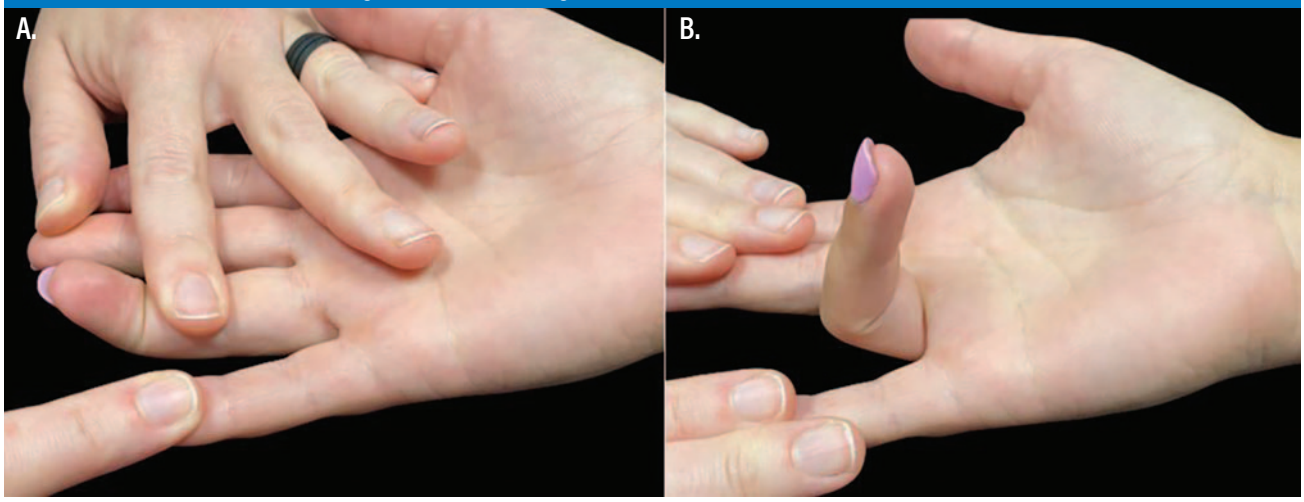


Figure 4A and 4B. Testing for FDP function (4A) and FDS function (4B).



The vinculum brevis profundus (VBP) derived network extends from the bifurcation of the flexor digitorum superficialis (FDS) tendon bifurcation to the DIP joint.[9] The long vincula supply the tendon at the level of the proximal interphalangeal (PIP) joint.[4] With proximal retraction of the tendon in the palm, the vincula vascular supply will be disrupted, leading to impaired tendon vascularity which affects tendon healing.

Leddy and Packer described the classification system for FDP avulsion injuries.[4] Their classification system is based on the level of tendon retraction and presence of a fracture (Table 1). In Type I, the avulsed tendon retracts into the palm resulting in no active flexion at the DIP joint, and a tender mass in the palm.[4] Proximal retraction of the tendon into the palm disrupts both the long and short vinculum leading to loss of the tendon blood supply.[3-5, 8]

Type II FDP avulsion injuries are the most common type, and occur when the tendon retracts proximally to the level of the proximal interphalangeal (PIP) joint.[4] The long vinculum arises at the level of the PIP joint and is preserved while holding the tendon at this level. The VBP is disrupted but preservation of the long vinculum allows blood supply to the tendon.[3-5,8] In Type II injuries, a small volar cortical avulsion can occur and may also prevent the tendon from retracting more proximally.

In Type III, a large bony fragment is avulsed from the distal phalanx and catches on the A4 pulley of the middle phalanx preventing the tendon from retracting beyond the middle phalanx. This is less common than the previously described types.[4] In contrast to Type I and Type II, both vincula are intact and blood supply is preserved.[3,5] Lateral radiographs of the finger show the large bony fragment just proximal to the level of the DIP joint.

Leddy and Packer detailed Types I through III, but their original classification scheme has since been expanded to describe two additional distinct injury patterns. Type IV injuries are unusual and are the result of two distinct injuries. The presumed mechanism occurs when a large bony avulsion fragment of the volar base of the distal phalanx is incarcerated at the A4 pulley, and continued force

Table 1. Classification of FDP avulsion.

Type	Description
I	The tendon retracts to the palm. Vincular system completely disrupted.
II	The tendon retracts to the level of the PIP joint. Long vinculum intact. Short vinculum disrupted.
III	The tendon is attached to a large bony fragment entrapped on A4 pulley. Vincular system remains intact.
IV	The tendon separates from bony fragment and retracts. Vincular system depends on level of tendon retraction.
V	Avulsion of an osseous fragment with concomitant fracture of the distal phalanx.

through the tendon results in a superimposed rupture of the FDP tendon from the bony fragment.[3,5,10] Following avulsion from the bony fragment, the tendon may retract into the finger or palm resulting in disruption of the blood supply to the tendon. Type V FDP avulsion injuries involve bony avulsions coupled with a distal phalanx fracture. Types IV and V are less common than the other three types.

FDP avulsions are common but are often not recognized initially. A delay in diagnosis can have an adverse effect on the treatment and overall function of the hand.[4,8] The inability to flex the DIP joint is often overlooked and may be misdiagnosed as a sprain.[2] Most patients present with ecchymosis, swelling, and pain along the course of the tendon of the affected finger.[7,8] The classic physical finding is loss of active DIP flexion. To accurately diagnose an avulsion injury, the provider must be aware of this injury, obtain a detailed history including mechanism of injury, and test for both FDS and FDP tendon function. The FDP and FDS tendons are tested separately (Figure 4A and 4B) to diagnosis the FDP tendon rupture.[5] It is also important to palpate the flexor tendon of the involved finger. Physical examination often demonstrates swelling and tenderness at the DIP joint and along the entire tendon sheath.[2] A point of maximal tenderness may give an indication to the level of the avulsed tendon stump. Localizing the extent of tendon retraction is a critical part of the preoperative assessment. Three to four weeks after an FDP injury, patients may also present with flexion contracture of the proximal interphalangeal joint.[2] Prompt referral to a hand surgeon should be made immediately following diagnosis.

Lateral radiographs of the involved digit are important to assess for bony avulsion fragments and phalanx fractures.[2,3,5] In the acute setting, additional imaging is generally not necessary because surgical repair is indicated regardless of the level of tendon retraction. However, in chronic injury, ultrasound or MRI may be used for distal tendon stump localization.[5] Tendon stump location determines tendon blood supply and dictates treatment options for chronic injuries.[3]

The prognosis and treatment of FDP avulsions are time dependent and multifactorial: they include the extent of proximal tendon retraction, the status of the vincula, and the size of the bony fragment.[5,8] FDP avulsion injuries diagnosed acutely (up to 10 days post-injury) should be surgically repaired immediately.[2,3,7] Early repair results in the best outcome while delays in surgical correction may lead to permanent loss of function.[7] The level of tendon retraction and tendon blood supply will ultimately dictate the timeline for primary surgical repair.

Type 1 FDP avulsions retract to the level of the palm and vincula blood supply is fully disrupted. Optimal treatment is early reinsertion of the tendon to the distal phalanx. Leddy and Packer reported that the tendon became necrotic and retracted if not reinserted within 7-10 days of injury.[4,10] Type II FDP avulsions retract to the level of the PIP joint leaving the long vinculum intact maintaining some blood supply. Type II is best treated by early insertion of the tendon into the distal phalanx but may be repaired weeks or even months later in certain cases.[3-5,10] Type III avulsion injuries have preserved blood supply and limited tendon retraction due to a large bony fragment caught in the A4 pulley. Treatment of Type III requires open reduction and internal fixation of the osseous fragment to the distal phalanx.[3,8] Preservation of the vincula system allows for late primary repair due to the relatively healthy tendon.[5] In Types IV and V, the osseous avulsion fragment is openly reduced followed by reinsertion of the tendon, as described for Type I and II injuries.[8] However, other surgical methods for Type IV and V injuries have been described with varying results.[3]

Failure to recognize and adequately repair FDP avulsion injuries may limit treatment options. In chronic Type II and Type III injuries, delayed tendon repair may be considered, but chronic Type I injuries cannot be repaired.[3] For chronic lesions in which the patient is relatively asymptomatic, no treatment is recommended.[3-5,7,8,10] Patients who experience loss of PIP range of motion may benefit from an aggressive therapy regimen.[3] Chronic cases that present with symptomatic instability of the DIP joint may be treated with arthrodesis or tenodesis.[3-5,8] Select patients with a need for manual dexterity or young highly motivated patients who will comply with therapy may be candidates for tendon grafting. Results of tendon grafting vary, and the patient must be aware that a tendon graft does not usually allow complete recovery of their primary range of motion.[5,7,8]

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About the Authors:
Nathan Whitsell, MD, University of South Dakota Sanford School of Medicine, Sioux Falls, South Dakota.
Robert Van Demark Jr., MD, Sanford Orthopedics and Sports Medicine, Sioux Falls, South Dakota.

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Improving Rates of Vitamin D Supplementation in Somali Immigrant Infants: A Prospective Interventional Pilot Trial

Jeffrey S. Cook, MD; Allison Kimball, MD; Linda Zittleman, MSPH; and Mark K. Huntington, MD, PhD

Abstract

Purpose: Rates of vitamin D deficiency and nutritional rickets have been rising over the past several decades, particularly in high-risk infants. This pilot study assessed the impact of providing free vitamin D supplements, a culturally-appropriate educational brochure, and a brief counseling session about the importance of both vitamin D supplementation and breastfeeding to the parents of Somali infants at routine office visits from newborn through 6 months of age at three Federally Qualified Health Centers in Colorado. We also assessed the impact this intervention had on rates of breastfeeding.

Methods: Twenty-five Somali infants aged 24 weeks or less were identified by searching electronic health records and enrolled into a historic control group. The parents were then surveyed by phone regarding breastfeeding and vitamin D supplementation. Subsequently, 37 families with newborn Somali infants were identified and enrolled into the intervention arm of the trial.

Results: The intervention group had a higher rate of vitamin D supplementation compared to the historical control group (67 vs. 48 percent, p=0.011) without significantly impacting breastfeeding rates.

Conclusion: These results suggest a practical way to increase vitamin D status in this high-risk population. Trial not registered as it was a pilot study, not a phase II to IV prospective clinical trial.

Introduction

Vitamin D is essential for healthy bone metabolism and the prevention of nutritional rickets. Current guidelines recommend that infants consume 400 international units (IU) of vitamin D daily.[1-3] Human milk, however, typically contains 20-60 IU or less of vitamin D per liter;[4,5] as a result, breastfed infants often do not receive the recommended daily minimum level of vitamin D. Risk factors for vitamin D deficiency include breastfeeding in the absence of vitamin D supplementation, dark skin pigmentation, lack of sunlight exposure (whether due to clothing, use of sunscreen, winter months, and/or living at higher latitudes), and low maternal vitamin D status.[3,5-11] Immigrant or refugee status has also been identified as a risk factor.[12-15] Over the last several decades, there has been an increase in the rates of nutritional vitamin D deficiency and rickets, primarily in dark-skinned, breastfed infants. There has also been increased awareness of the high prevalence and adverse clinical effects of vitamin D deficiency, which is generally defined as a serum 25-hydroxyvitamin D level less than 20 nanograms per milliliter (50 nanomoles per liter).[1,2,5,9,16]

Supplementing breastfed infants with vitamin D has been shown to improve vitamin D status and prevent rickets. Although several professional societies have updated their guidelines about vitamin D supplementation, these recommendations have not been widely adopted.[17-19] The reasons for this are not completely understood but may include lack of knowledge among healthcare providers and parents about both the importance of vitamin D supplementation and the recommendations from current guidelines.[10,18-21] Some providers may not recommend vitamin D supplementation due to a belief that doing so may negatively impact breastfeeding.[19] Dark-skinned, breastfed, immigrant infants living at northern latitudes represent an extremely high-risk population for vitamin D deficiency and rickets, yet there are few data regarding how to improve rates of vitamin D supplementation and, ultimately, vitamin D status in this population.[12-14]

This study assessed the effects that an intervention comprised of an educational brochure, free supplements, and patient counseling on vitamin D supplementation had

upon the rates of vitamin D supplementation and breastfeeding in infants born to Somali immigrants.

Methods

Design

This is a prospective interventional pilot trial using a historical control group. Randomization was not performed due to ethical concerns about creating a control group which might not receive currently recommended vitamin D treatment.

Population

This study was conducted at three Federally Qualified Health Centers in northern and central Colorado—one in a rural community, one in a mid-sized agricultural city, and the third in Denver. A historical control group to establish pre-intervention baseline rates consisted of 25 infants. The electronic health records of the three sites were searched to identify all Somali families with infants aged 24 weeks or less. All the parents identified were invited to participate in the study. Staff medical interpreters at the study sites were oriented to the study. They then surveyed participating parents by phone about rates of breastfeeding and vitamin D supplementation utilizing a written script and recorded answers onto a customized response form.

Subsequently, 37 families with newborn Somali infants were identified and enrolled in the intervention arm of the trial.

Intervention

The intervention consisted of three components: (1) a culturally-appropriate educational brochure, (2) free vitamin D supplement drops (brand name *D-Vi-Sol*™ [Enfamil], Mead-Johnson, Chicago, Illinois), and (3) a brief counseling session regarding the benefits of both breastfeeding and vitamin D supplementation. The intervention was conducted once at either the several-day-old or the two-week well child visit.

The educational brochure was created by two health care providers who practiced at one of the study sites. It emphasized both the benefits of breastfeeding and vitamin D supplementation, the steps for vitamin D administration, and instructions for obtaining free supplement refills. English and Somali versions of the brochure were created, with the Somali language brochure back-translated into English to verify content accuracy. Instructions for administering vitamin D included pictograms, as some data suggest that a significant number of Somali immigrants are illiterate in their native language.[12,22-24] A copy of the brochure is available in English and Somali at sdsma.org.

At the time of enrollment and all subsequent well child visits, participating parents were surveyed about breastfeeding and vitamin D supplementation. The survey was verbally administered by medical assistants or healthcare providers utilizing standard translation protocols available at each clinical site. The survey was similar to that used for the control arm, with the addition of a question assessing the need for supplement refills.

Study data were collected and managed using REDCap (Research Electronic Data Capture, Vanderbilt University, Nashville) electronic data capture tools. Descriptive analysis was conducted to compare characteristics in the historical and intervention groups. Quantitative comparison of the rates of vitamin D supplementation between the historical and intervention groups employed Yates' chi square, with significance level set at $p < 0.016$ using Bonferroni's correction to account for experiment-wise error rate.

Ethics

This study was reviewed and approved by the Colorado Multiple Institutional Review Board (COMIRB) at the University of Colorado Denver. Verbal informed consent was obtained prior to patient enrollment.

Results

There were 25 infants enrolled in the historical control group with ages ranging from 4 weeks to 6 months and 37 infants in the intervention group, with ages ranging from 2 to 3 days to 6 months. (Table 1).

Table 1. Age of infant at the time of survey.

Age	Control N = 25 (%)	Intervention N = 37 (%)
0-4 weeks	1 (4%)	18 (49%)
5-12 weeks	5 (20%)	6 (16%)
13+ weeks	7 (28%)	2 (5%)
Age missing	12 (48%)	11 (30%)

In the historical control group, 100 percent of infants had initiated breastfeeding and 80 percent of those were still breastfeeding at the time they were surveyed. In the intervention group, 97 percent of infants initiated breastfeeding and 92 percent were continuing to breastfeed at the time they were surveyed (Table 2).

Approximately half of the infants in the control group had

Table 2. Rates of breastfeeding and of vitamin D supplementation.[a]

Characteristic	Control group N (%)	Intervention group N (%)
Initiated breastfeeding	25 (100%)	36 (97%)
Currently breastfeeding *(of those who initiated breastfeeding)*	20 (80%)	33 (92%)
Breast and formula feeding	20 (80%)	26 (72%)
Exclusive breastfeeding	0 (0%)	10 (28%)
Initiated vitamin D supplementation *(of those who initiated breastfeeding)*	12 (48%)	24 (67%)*
Currently supplementing with vitamin D *(of those who initiated breastfeeding)*	9 (36%)	16 (44%)**

[a]The rates of both initiation and continuation of vitamin D supplementation differed significantly between the historical control and the intervention groups (*$p = 0.011$ and **$p < 0.0001$, respectively)

ever been supplemented with vitamin D. Thirty-six percent of infants in the control were still receiving vitamin D at the time they were surveyed. In the intervention group, almost two-thirds of infants had initiated vitamin D supplementation; 43 percent of the infants were still supplementing with vitamin D at the time they were surveyed (Table 2).

Discussion

Providing Somali infants with free vitamin D supplements along with a culturally-appropriate educational brochure and parental counseling was associated with a significant improvement in the rates of initiation of vitamin D supplementation. Higher rates of ongoing vitamin D supplementation were also noted. Contrary to earlier reports,[19] vitamin D supplementation did not adversely affect breastfeeding rates in this population; rather, a trend toward improvement was noted. Additionally, more women in the intervention group reported exclusive breastfeeding. The educational component of this intervention emphasized the benefits of breastfeeding; however, further study of the relation between vitamin D supplementation and breastfeeding rates will be needed to clarify this apparent discrepancy.

While similar results have been demonstrated previously,[25] to our knowledge this represents the first time this clinical question has been investigated in North America. In addition to demonstrating the efficacy of the intervention, baseline rates of initiating breastfeeding in the Somali immigrant population and the relative frequency of e xclusive versus mixed (i.e., formula supplemented) breastfeeding were also established. These data have not been previously reported for this population.

Human milk contains insufficient levels of vitamin D for many at-risk infants, including breastfed infants of Somali immigrants in the U.S. Vitamin D supplementation improves vitamin D status and can prevent nutritional rickets. Providing families with vitamin D at the time of their office visit removed the barrier of filling a prescription while the educational brochure reinforced the importance of both breastfeeding and vitamin D supplementation.

A strength of this study was its multi-site, community-based design, both of which improve generalizability. The patient-education brochure was developed by healthcare providers at one of the clinics where Somali immigrant families receive care. The methods that improved vitamin D supplementation in this high-risk population could be easily replicated in other clinics.

A significant limitation of this study was missing age data of the infants and the relatively young age of intervention group compared to control; either may contribute to differences between the two comparison groups. Additional limitations include the non-randomized study design, the small study size, and the short trial duration. Longer term follow-up could help assess the durability of the intervention. Patient enrollment rates were not collected, which would have strengthened the study design. Finally, the relative contributions of the brochure, vitamin D supplements and counselling to the outcomes could not be quantified.

Conclusion

This study of infants born to Somali immigrants demonstrates that an intervention comprised of an educational brochure, brief counselling and free samples of vitamin D supplement was associated with improved rates of vitamin D supplementation and increased breastfeeding rates through the first six months of life. This type of low-cost, locally derived, culturally appropriate intervention can improve vitamin D supplementation and breastfeeding in a vulnerable population. Few other data regarding how to improve vitamin D status in this population exist.

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Conflict of interest statement – One author was employed by the High Plains Research Network, Aurora, Colorado, during the time of active investigation. High Plains Research Network is a practice-based research network associated with the University of Colorado School of Medicine. The authors have no relevant financial or non-financial interests to disclose.

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About the Authors:

Jeffrey S. Cook, MD, Faculty Physician, North Colorado Family Medicine Residency Program, Greeley, Colorado; Associate Clinical Professor, University of Colorado School of Medicine.

Allison Kimball, MD, Staff Physician, Sunrise Community Health, Evans, Colorado.

Linda Zittleman, MSPH, Co-Director, High Plains Research Network, University of Colorado Denver; Anschutz Medical Campus, Aurora, Colorado.

Mark K. Huntington, MD, PhD, Director, The Center for Family Medicine, Sioux Falls, South Dakota; Professor, University of South Dakota Sanford School of Medicine, Sioux Falls, South Dakota.

A Young Adult with White Matter Changes and Recurrent Stroke Symptoms

John Fanta, MD; Eric Roach, MD; Anthony Breemo, MD; and Andrew Ridder, MD

Abstract

White matter changes on MRI can be a diagnostic puzzle as a large group of inflammatory, autoimmune, infectious, and neoplastic conditions can present in this way. An otherwise healthy 36-year-old male presented with his second episode of unilateral weakness, the first episode occurring five years previously. He did not have sensory or cerebellar symptoms with the current or previous episode. He reported that his grandfather, father, two of his aunts, and an uncle had multiple sclerosis (MS), dying in their 40s-50s from their disease. The MRI during his first hospitalization revealed acute ischemia as well as diffuse white matter hyperintensities. The current MRI revealed new ischemic changes as well as progression of the white matter hyperintensities with notable temporal lobe involvement. While small vessel disease and multiple sclerosis can present similarly, the history of stroke, lesion distribution, and family history suggested an alternative diagnosis. Due to high clinical suspicion, genetic testing was performed for CADASIL (cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy) and confirmed the diagnosis. This case report describes the approach to the adult with white matter changes and describes the typical presentation and findings of CADASIL, the most common heritable cause of stroke and vascular dementia in adults.

Introduction

Progressive white matter changes on MRI can be caused by a heterogenous group of disorders that range from the relatively common disorder of small vessel ischemic disease and multiple sclerosis to a variety of other acquired or rare genetic conditions. In the adult patient with confluent and symmetric white matter changes it is first important to assess for acquired conditions, including those caused by infection, malignancy, or drugs.[1] If these causes are ruled out, the provider may then be left to consider inherited causes of leukodystrophies. The history and MRI findings offer important clues in developing a differential diagnosis. In obtaining the history, care should be taken to uncover all associated symptoms as well as a comprehensive family history. Additionally, although non-specific, the pattern of white matter changes on MRI can be helpful in developing a differential diagnosis. Through this process and with appropriate clinical suspicion, specific gene testing can then be obtained to aid in confirming the suspected diagnosis.

Case

An otherwise healthy 36-year-old male presented to his local emergency department after developing right-sided facial droop and dysphasia for one day. Review of systems was otherwise unremarkable. Examination showed facial droop but no other focal neurological deficits and no sensory or cerebellar findings. He was admitted to the hospital for further workup and management of a presumed ischemic stroke. Interestingly, family history was reported by the patient to be positive for multiple sclerosis (MS) in his grandfather, father, two aunts and an uncle who died in their 40s and 50s from their disease. Admission brain MRI showed periventricular acute ischemia in the corona radiata as well as progressive white matter changes in the temporal lobes from a previous scan (Figure 1). Medical history was remarkable for a hospitalization five years previously for left-sided facial weakness as well as left-sided upper and lower extremity weakness. At that time, he did not have any sensory deficits or cerebellar symptoms and an extensive workup was performed including CSF analysis, echocardiogram, and assessment for infectious and autoimmune etiologies (Table 1 & 2). These studies were largely unrevealing. He was discharged on aspirin given the ischemic nature of the disease although a clear diagnosis was not established during this previous hospitalization and he was lost to further neurologic follow-up. Given this thourough workup from his previous admission, a limited workup was performed during his current admission, with several tests

Figure 1C-F. T2 FLAIR from 2015 (C & E) and 2020 (D & F) showing progression of extensive, diffuse cerebral white matter hyperintensities with classical involvement of the anterior temporal lobes (circled). Small punctate lesion of the midbrain (red arrow) which is also common in CADASIL.

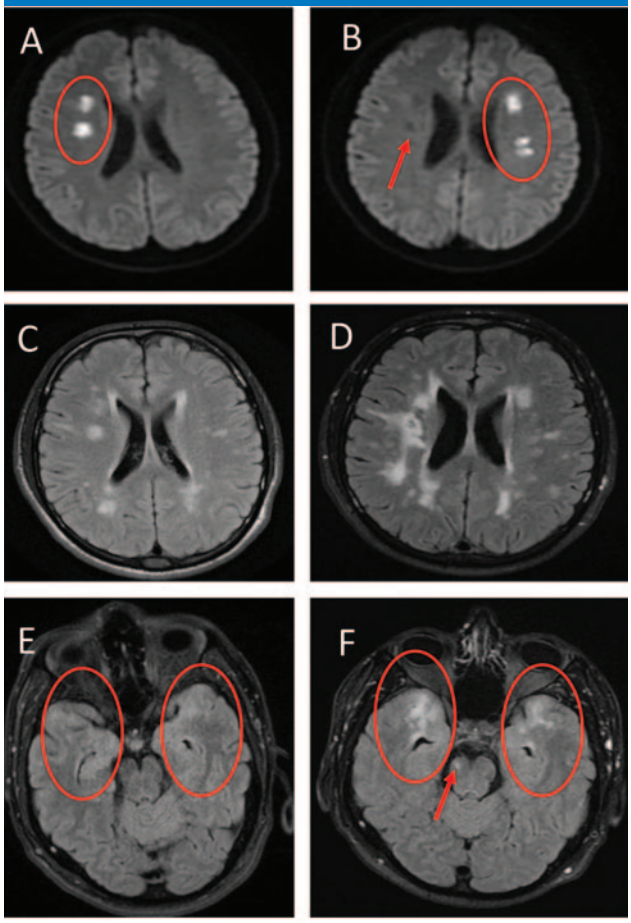


Table 1. Select blood tests.

	2015 admission	Current admission
Anti-myeloperoxidase	WNL	WNL
SS-A antibody	124 (N<100)	WNL
Scl-70 scleroderma Ab	WNL	WNL
Paraneoplastic antibodies	Negative	Not tested
CRP	<0.5	<0.5
HIV 1 & 2 antigen & antibody	Non-reactive	Non-reactive
RPR	Not tested	Non-reactive
Full hepatitis panel	Not tested	Negative

Table 2. Selected CSF results (2015 admission).

		Normal range
CSF WBC	1	N<5
CSF RBC	2	N=0
CSF total protein	33 mg/dL	N=15-45
CSF glucose	58 mg/dL (N=40-70)	N=40-70
CSF oligoclonal bands	Negative	Negative
CSF West Nile IgM Ab	0.0	Negative
Neuromyelitis optica IgG	Negative	Negative
JC virus PCR	Not detected	Not detected
Lyme disease PCR	Not detected	Not detected

being repeated and being negative. To complete the work-up from his previous admission, a six-vessel cerebral angiography was performed and was normal.

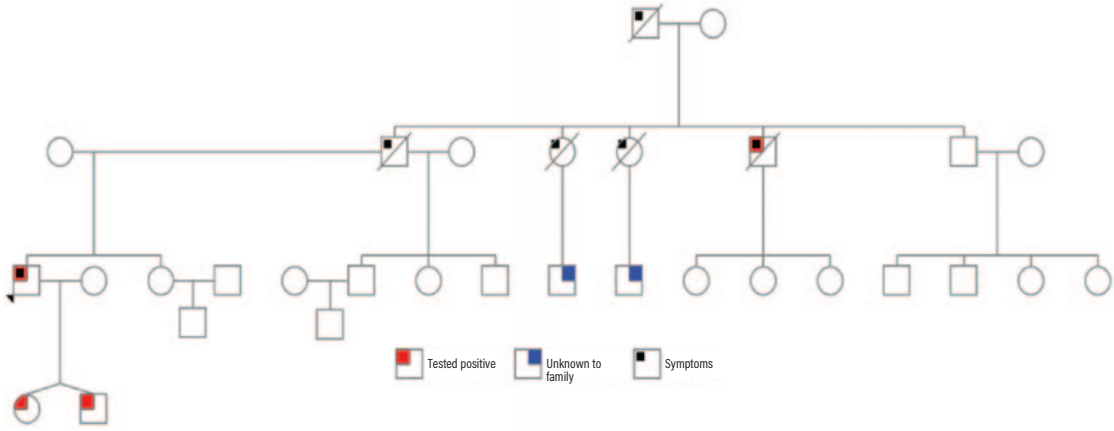
Despite the patients reported family history of MS, this diagnosis seemed unlikely given the ischemic changes on his imaging, the negative oligoclonal bands in the CSF on his previous admission and his extensive family history of neurological disease.

With this negative workup for inflammatory, autoimmune, and infectious causes, the diagnosis of Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts and Leukoencephalopathy (CADASIL) was suspected based on the patient's recurrent strokes, family history and location of MRI white matter changes. Outpatient gene testing was performed which later revealed a NOTCH3 c.457 cystine to thymine pathogenic heterozygous missense mutation confirming the diagnosis. The patient was informed of the autosomal dominant nature of this disease and was encouraged to reach out to family members about the need for genetic testing. A three generation pedigree shows the extent of involvement within the patient's family (Figure 2).

Discussion

This case reveals important points in the evaluation of the adult with white matter changes. The differential diagnosis is broad and includes various metabolic, genetic, and acquired causes.[1] Figure 3 shows an algorithm for the workup of leukodystrophy in the adult patient.[1] The most common diagnosis to consider is small vessel disease. However, this patient's age, the sub-acute/chronic progression, absence of significant vascular risk factors, and lack of brainstem and basal ganglia involvement made this cause

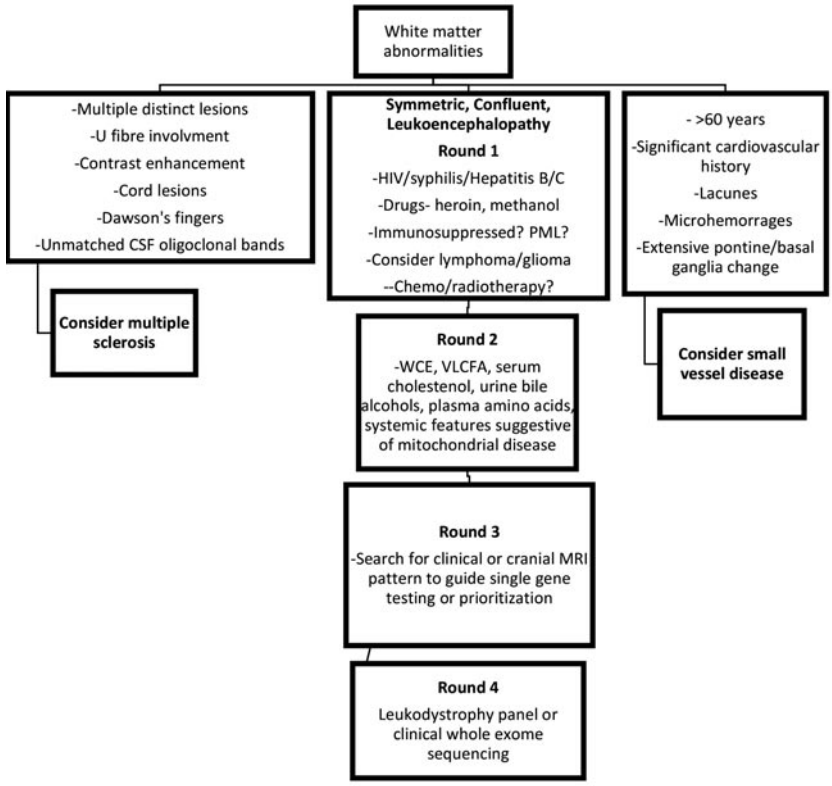
Figure 2. Three-generation pedigree revealing the extend of familial involvement. Many relatives are still in the process of being tested, with many family members' health history unknown to the proband.



unlikely in this patient. A second important etiology to consider is MS because it is treatable and just as common (2.0/100,000) as CADASIL (10-40/million).[1,2] To review, in MS, lesions must be separated in space and time per the McDonald criteria (Please see reference for a complete review of the diagnosis of MS).[3] Lesions are demyelinating rather than ischemic and rarely show restricted diffusion even if imaged acutely and the lesions are active. Although lesions can occur anywhere in the CNS, they are classically seen along the corpus callosum and the periventricular, juxta-cortical, and infratentorial areas.[4] In this patient the corpus callosum was spared and workup in 2015 was negative for CSF-specific oligoclonal bands which are usually found in cases of MS.[5] Additionally, while there is some degree of heritability with MS, such an extensive family history is very atypical.[4]

The next step involves ruling out acquired disorders such as hepatitis, neurosyphilis, HIV, cancer, immunosuppression, or drug-induced causes. If these causes are not suspected, the provider can assess for more rare genetic syndromes such as lysosomal storage disorders. Specific testing includes

Figure 3. Schema demonstrating workup and evaluation of white matter abnormalities.



PML – progressive multifocal leukoencephalopathy; WCE – white cell enzymes; VLCFA – very long chain fatty acids

evaluation for serum long chain fatty acids (X-linked adrenoleukodystrophy) and specific white cell enzymes (Krabbe disease, metachromatic leukodystrophy) as well as serum cholesterol/urinary bile alcohols (Cerebrotendinous xanthomatosis) and plasma amino acids (methylenetetrahydrofolate reductase deficiency and homocystinuria).[1] In this case these tests were not performed due to high suspicion of CADASIL based on the clinical picture. Yet another class of disorders that a provider must consider is mitochondrial disease. These disorders are a heterogenous group that typically have systemic symptoms, frequently involve the eyes and muscles, and commonly are associated with leukoencephalopathy.[6] Mitochondrial disorders are confirmed through molecular analysis based on clinical suspicion.[6]

CADASIL fits into a group of vascular leukoencephalopathies caused by specific genetic mutations. Other disorders in this group include CARASIL (cerebral autosomal recessive arteriopathy with subcortical infarcts and leukoencephalopathy) and CARASAL (cathepsin A-related arteriopathy with strokes and leukoencephalopathy). HTRA1 gene testing is used to identify CARASIL while CTSA gene testing is used to identify CARASAL.[1] CARASIL and CARASAL do share some similar clinical characteristics to CADASIL but they are far less common.[7,8]

CADASIL is an autosomal dominant condition with involvement of small cerebral arteries frequently causing ischemic strokes along with migraines and behavioral changes.[9] It results from cysteine-altering mutations of the epidermal growth factor-like repeat (EGFr) domain of the NOTCH3 transmembrane receptor family located on chromosome 19q12.[10-12] These mutations lead to receptor fragments accumulating in the adventitia and media layers of cerebral arteries, causing vascular smooth muscle cell degeneration and arterial wall thickening.[10, 11] These changes inevitably lead to ischemic strokes in patients in their 20s-70s. Stroke is the most common finding in CADASIL occurring in 60-85 percent of patients. Strokes are almost invariably subcortical and frequently present as lacunar syndromes. Other neurological symptoms that frequently occur are migraine and dementia. Migraine with aura is typically the first symptom that patients develop, starting around the age of 30. As the disease progresses, mood disturbances, apathy and cognitive deficits become prominent and patients typically become bedridden by age 65. Unfortunately, there is no effective treatment. Disease management primarily focuses on symptomatic treatment and aggressive secondary prevention of strokes, including anti-platelet and statin therapy, along with management of cardiovascular risk factors such as diabetes and hypertension.[9]

Findings on brain MRI can offer important clues in making the diagnosis. Changes can be noted 10-15 years before disease onset, evolving from periventricular punctiform and nodular lesions to diffuse, symmetric abnormalities involving the external capsule and temporal lobes. Anterior temporal lobe involvement is the most specific finding, presenting in approximately 67 percent of patients.[9] Lacunar infarcts in the pons, thalamus, and lentiform nuclei also develop as the disease progresses.[9] Observing these findings on MRI should increase clinical suspicion for CADASIL.

CADASIL should be considered in the differential diagnosis of a young adult presenting with recurrent stroke symptoms with a strong family history of a degenerative, inflammatory, or ischemic neurological disease, especially in the absence of other vascular risk factors. Challenges to making the diagnosis include heterogenous symptoms, relatively non-specific MRI findings, and limited resources for genetic testing. Although this case demonstrates a typical presentation of CADASIL, the diagnosis was delayed by the patient reporting a family history of multiple sclerosis. This highlights the need to know the unique MRI findings of these two disorders to avoid ineffective treatments and provide an accurate prognosis for patients.

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About the Authors:

John Fanta, MD, PGY1, Indiana University; University of South Dakota Sanford School of Medicine, Sioux Falls, South Dakota.

Eric Roach, MD, PGY1, University of South Dakota Sanford School of Medicine, Sioux Falls, South Dakota.

Anthony Breemo, MD, Assistant Professor of Medicine, Avera McKennan Hospital and University Health Center, Sioux Falls, South Dakota.

Andrew Ridder, MD, Assistant Professor of Medicine, Avera McKennan Hospital and University Health Center, Sioux Falls, South Dakota.

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Understanding and Overcoming COVID-19 Vaccine Hesitancy in South Dakota

Brittney A. Meyer, PharmD; Filip Viskupič, PhD; and David L. Wiltse, PhD

The COVID-19 pandemic has had a major impact on the lives of South Dakotans. As of Feb. 22, 2022, COVID-19 has caused the loss of 2,467 lives since March of 2020 and has accumulated a total case count of 235,116.[1] As this number only accounts for reported cases, the actual affected population is likely much higher. South Dakota has also had a higher fatality rate of the disease than 29 other states, despite experiencing the first COVID-19 wave months after the hardest hit coastal states.[2] Unfortunately, January 2022 saw record numbers of infections, and community spread currently remains high or substantial for 59 of the 66 counties in South Dakota.[1]

After nearly two years, work to manage the COVID-19 pandemic continues. Scientists agree that vaccinating as many people as possible is the best way to control the spread of the virus, but practitioners and public health officials in South Dakota have experienced challenges in encouraging people to get vaccinated. South Dakota had a relatively swift rollout when the vaccines first became available to the public and for a time the state stayed ahead of the national vaccination rate. However, since then, vaccinations have slowed despite the explosive growth in cases brought by the Delta and Omicron variants. Some groups of South Dakotans are vaccinating at lower rates than the rest of the population, including Republicans and evangelical Christians.[3] Given the emergence of these new highly infectious variants, increasing vaccination rates continues to be important. However, the lingering resistance to vaccination and the presence of conspiracy theories about the virus and about vaccines make accomplishing this objective an uphill battle.

We at *The SDSU Poll* research group seek to understand why some South Dakotans are so reluctant to follow COVID-19 mitigation guidelines, particularly to be vaccinated, and to study how to overcome this resistance. The SDSU Poll fielded several surveys across South Dakota during the last year to achieve this objective. While we have uncovered some deep-seated obstacles to vaccination, we have also identified opportunities and areas of focus for progress. Our goal is to share our findings with medical and public health professionals and with the public.

Researchers at the SDSU Poll were interested in determining the impact of different messengers on people's intentions to follow a variety of COVID-19 mitigation practices, pre- and post-vaccination. It is widely agreed that effective health communication is vital for managing the pandemic.[4-7] And while it is understood that the language of a message is important, existing evidence suggests people's dispositions are also significantly impacted by the messenger.[8-10] Since a patient's level of trust in a particular messenger is an important component in determining the effectiveness of a health communication message, messenger impact was also a matter of central interest to us.

In April 2021, over 3,000 South Dakotans over the age of 18 participated in our statewide survey. We found that among those South Dakotans who were not vaccinated at that time, only about 20 percent answered that they were "very likely" or "likely" to get the shot.[3] We designed a "survey experiment," to evaluate whether it is possible to convince the unvaccinated South Dakotans to receive the vaccination.[11] Participants were randomly split into groups and read an identical message on the importance of vaccination, which was attributed to a religious, political, or medical figure from South Dakota. Immediately thereafter, participants were asked about their vaccination intentions. We found that of the three, only the religious messenger treatment group showed a statistically significant difference in increased interest of vaccine uptake compared to the control group ($t=1.97$, $p=0.049$). Interestingly, when asked about their intention to get more information about COVID-19 vaccines, we observed that those who read the statement from the medical messenger were less inclined to seek out more information about the vaccines. Yet, the religious messenger was particularly effective in reaching certain subsets of the sample that have shown greater vaccine hesitancy. The response from evangelicals to the religious messenger was particularly strong ($t=2.85$, $p=.004$), as well as from those under age 65 ($t=2.04$, $p=0.042$). Overall, we found that religious leaders might be the most effective public health messengers and that messaging from

public health institutions and political messengers was not effective.

We were also interested in discovering how to encourage the already vaccinated population to maintain COVID-19 mitigation practices post-vaccination, as these people often let their guard down.[12,13] Vaccinated respondents were subjected to an experiment on their attitudes about continued mitigation practices after vaccination.[14] These respondents were treated similarly to those in the first experiment. Participants were given identical messages, attributed to either a religious, political, or medical figure from South Dakota, encouraging people to continue wearing masks, avoiding large gatherings, avoiding poorly ventilated spaces, and staying 6 feet away from others. The results were strikingly similar to the first experiment: only the messaging from the religious figure seemed to consistently resonate with the vaccinated respondents across the various mitigation practices (mask wearing t=2.28, p=0.023; gatherings t=2.22, p=.027; ventilation t=3.27, p=.001; distancing t=3.17, p=.002). In total, the results of both experiments underscore the strength of religious leaders as public health messengers, emphasizing a need to collaborate with leaders from the religious community to reach hesitant individuals.

As part of the same survey, we investigated the role an individual's trust in doctors plays in the decision to receive a COVID-19 vaccination. A typical citizen does not have detailed information about the virus or the vaccine; therefore, the decision to get vaccinated is to a large extent driven by the trust they have in the government that is approving the vaccine and in the doctors who are often the source of information on the vaccine. We found that South Dakotans who have more trust in physicians are more likely to get vaccinated.[14] Results from statistical analysis showed that respondents most trusting of physicians had an 80 percent probability of getting vaccinated, while the probability of vaccination was 11 percent for those with the lowest level of trust in physicians.

Our research has uncovered significant challenges to increasing adherence to mitigation guidelines, particularly vaccination, as the COVID-19 pandemic continues to affect the lives of South Dakotans. We have also found places where progress might be made. For example, we found that when it comes to communicating important health information, it is not only the message that matters, but also the messenger. *Who* is doing the talking matters. Having an awareness of how patients are processing information for important health decisions may be helpful. Since patients are less likely to seek out additional information after exposure to messages from a public health official, and because many South Dakotans are receptive to messaging from religious leaders, medical providers and public health officials are encouraged to collaborate with religious leaders in their COVID mitigation efforts.

Our research also supports the large body of knowledge that the trust a patient has in their doctor is integral to their decisions, making the presence of trust between patients and doctors crucial. The physician's recommendation to vaccinate remains influential for overcoming vaccine hesitancy. When it comes to making that recommendation, motivational interviewing can be helpful as it is useful in situations in which people have a high ambivalence to change, have a low desire to change or uncertainty about changing, and place a low level of importance on change due to insufficient understanding of the benefits to change or drawbacks of remaining in the current situation.[15] Motivational interviewing can also be a shared resource between both healthcare providers and religious leaders to facilitate behavioral change in the hesitant population.[16] Motivational interviewing takes time to learn and practice, but with more people engaging in this style of communication, transmission will slow with each additional resulting decision to vaccinate.

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Please note: Due to limited space, we are unable to list all references. You may contact South Dakota Medicine at ereiss@sdsma.org for a complete listing.

About the Authors:

Brittney A. Meyer, PharmD, Associate Professor, Pharmacy Practice and Affiliate Researcher at The SDSU Poll, College of Pharmacy and Allied Health Professions, South Dakota State University, Brookings, South Dakota.

Filip Viskupič, PhD, Assistant Professor, Political Science; Research Associate, The SDSU Poll, School of American and Global Studies, South Dakota State University, Brookings, South Dakota.

David Wiltse, PhD, Associate Professor, Political Science; Director; The SDSU Poll, School of American and Global Studies, South Dakota State University, Brookings, South Dakota.

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Member News

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

Call for Nominations — SDSMA Awards

Nominations for SDSMA Awards are open! The SDSMA Awards honor physicians for their contributions to health care, service to patients, dedication to improving medicine in South Dakota, or service to the SDSMA.

Nominations are now being accepted at sdsma.org for the following awards:

- Distinguished Service Award
- Community Service Award
- COPIC Humanitarian Award - *the recipient of this award designates a $10,000 donation from COPIC to be provided to a healthcare related 501(c)(3) organization in South Dakota!*
- Outstanding Young Physician Award
- Richard P. Holm, MD, Media Award
- Young at Heart Award

Are there people that come to mind who deserve to be recognized? Nominate them today! Award recipients are honored at the awards banquet during the SDSMA Annual Leadership Conference scheduled for June 3, 2022.

An individual may work right alongside of you, serve on a committee with you, volunteer in your organization or community, or have made a significant impact in the medical profession. Help your colleagues and supporters get the recognition they deserve!

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2022 SDSMA Annual Leadership Conference is June 3

The 2022 SDSMA Annual Leadership Conference is Friday, June 3 at the Hilton Garden Inn Downtown Sioux Falls.

Events & Activities

- USD SSOM update
- American Medical Association update
- Panel discussion
- Membership open forum
- Policy Council meeting
- SDSMA PAC Lunch
- Membership mixer
- Awards banquet & scholarship recognition

This year's conference theme is social determinants of health. With presentations, discussions, networking opportunities and the banquet, the Annual Leadership Conference is a great time to share ideas and learn from fellow members.

The conference is a benefit of your membership and there is no conference registration fee. Online registration will soon be available at sdsma.org.

Ticket purchase required for some events and meals.

PA Independent Practice Bill Defeated

Senate Bill 134, which would have removed physician supervision of PAs and allowed PAs to practice independently, was defeated in the State Senate last week by a vote of 16 yea 19 nay.

A huge thank you to SDSMA members for raising the voice of physicians to defeat this legislation for the second year in a row. SDSMA members advocated for patients by showing elected representatives the value of team-based medicine and the risks of inadequate training hours for PAs to practice independently.

SDSMA members, along with AMA President Gerald Harmon, MD, testified before the Senate Health and Human Services Committee in strong opposition of the bill. Despite the argument made by the bill's proponents independent practice of PAs would increase access to care in rural areas, evidence shows that scope expansions do not create an influx of PAs in rural areas.

"The answer to our rural health care access issue is not SB 134. It is to continue to focus on team-based care, telemedicine, and training providers who will be more likely to practice in South Dakota," said SDSMA President Kara Dahl, MD, in her testimony.

Dr. Harmon discussed in his testimony the legislation's lack of provisions and guardrails for patients safety. He stated, "The bottom line is simple: enactment of SB 134 would make South Dakota an outlier compared to the rest of the country.

Member News

Legal Brief Highlight: Ending the Physician-Patient Relationship

Once a patient-physician relationship is begun, a physician generally is under both an ethical and legal obligation to provide services as long as the patient needs them. There may be times, however, when a physician is no longer be able to provide care. It may be that the patient is noncompliant, unreasonably demanding, threatening to you and/or your staff or otherwise contributing to a breakdown in the patient-physician relationship. Or, it may be necessary to end the relationship simply due to relocation, retirement or unanticipated termination by a managed care plan and/or employer.

Challenges can arise when the physician desires to terminate the relationship, yet the patient continues to present for treatment. Further, if the relationship is terminated without proper notice, or in a way that harms the patient, civil liability could result. The requirement of assistance and an opportunity to make alternative arrangements applies regardless of the reason the physician desires to terminate the relationship, including but not limited to failure to pay, failure to follow the physician's advice, or general bad behavior on the part of the patient.

A physician must provide notice of his or her intent to terminate the relationship so that the patient has sufficient time to make alternative arrangements. Thirty days' notice of termination is recommended, and it is also appropriate to provide a referral or other resources the patient can use to locate a new physician. The physician must continue to treat the patient while the patient seeks a replacement caregiver.

A provider remains responsible for record maintenance regardless of the termination of the physician-patient relationship. In addition, the patient is entitled to a copy of his or her medical records upon request.

More information is available in the SDSMA legal brief *Termination of the Physician-Patient Relationship* at sdsma.org. Through the SDSMA Center for Physician Resources, the SDSMA has developed more than 50 legal briefs that are available to members. In addition, the Center develops and delivers programs for members in the areas of practice management, leadership and health and wellness.

SDSMA Meets with Congressional Delegation

SDSMA members and staff met with Rep. Dusty Johnson and Sen. Mike Rounds on Feb. 15-16, and will meet with Sen. John Thune March 8. Meeting with the congressional delegation allows an opportunity to weigh in on federal issues of importance to South Dakota physicians and patients.

Topics of discussion include Medicare physician payment reform and its impact on patient access, Medicare telehealth benefits and the need to keep telehealth benefits available to patients post-pandemic, and prior authorization's jeopardization of patient care.

Attending the meetings are Drs. Kara Dahl — President; Mary Carpenter — Delegate to the AMA; Rob Allison —Delegate to the AMA; Robert Summerer — Alternate Delegate to the AMA; and Barb Smith — CEO.

Medicaid Expansion Fails to Pass Senate

A bill proposed in the legislature that would have expanded South Dakota's Medicaid program failed to pass the State Senate last month. Expansion would have covered more than 40,000 South Dakotans.

South Dakota still has another opportunity to expand Medicaid. The question of whether to expand Medicaid will be on the ballot as a constitutional amendment in the Nov. 8, 2022 General Election. Medicaid expansion would provide access to care for those in the coverage gap. In addition, rural healthcare facilities and communities would benefit from the economic impact of expansion when hundreds of millions of dollars will be put into local economies to improve rural access, prevent rural hospital closure, and increase healthcare related jobs.

SDSMA is part of a coalition, South Dakotans Decide Healthcare, which is urging a 'yes' vote on the proposed constitutional amendment, named Amendment D, this fall.

CDC New Guidelines For Opioid Prescriptions

The CDC has proposed new guidelines for prescribing opioids that remove its previous recommended ceilings on doses for chronic pain patients and instead encourage physicians to use their best judgment. The recommendations are the first comprehensive revisions of the agency's opioid prescribing guidelines since 2016.

The American Medical Association (AMA) has urged the CDC to reconsider its guideline on opioid prescriptions, arguing that the guidelines have proved devastating for patients with pain. The AMA stated the new draft guideline provides a path to remove arbitrary prescribing thresholds, restore balance, and support comprehensive, compassionate care.

PATIENT EDUCATION:

The Physician-Patient

Kelly Evans-Hullinger, MD

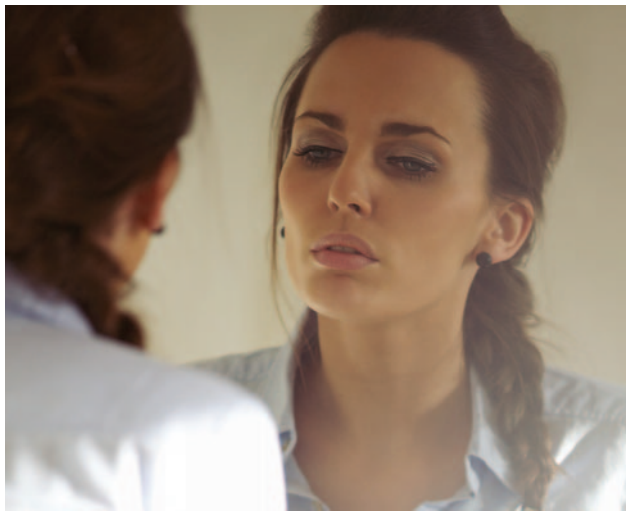
Last spring, I was at home washing my hands, and as I glanced up into the mirror, I noticed something unusual. My bathroom light hit my neck just right as I swallowed and there it was: a prominent lump. I diagnosed myself with a thyroid nodule and wondered how I, a physician, had failed to notice this large protuberance before that moment.

Thyroid nodules are quite common. In some cases, they are noticed by the patient (like me) or are found on exam. In many cases they are found on accident when someone has an imaging test like a CT scan, MRI, or ultrasound, done for some other reason. The vast majority of thyroid nodules are benign, only five percent or less representing thyroid cancer.

Typically, if a thyroid nodule is found, thyroid labs and a formal thyroid ultrasound will be recommended. The size and characteristics of the nodule on the ultrasound helps to guide whether a fine needle aspiration (a type of biopsy) should be performed. Many nodules are fluid filled and small, which we know conveys almost no risk of being cancerous, so those can be watched without biopsy.

In my case the nodule was medium sized, two centimeters in diameter, and had slight irregularity such that it was "mildly suspicious" and did warrant biopsy. As a physician-patient awaiting my procedure, I knew that the data said my nodule was still very low risk of being cancerous, but I still had some anxiety about the worst-case scenario.

My colleague, a surgeon, performed my fine needle aspiration expertly the next month. The procedure was easy,



done in the office with minimal discomfort. She drained out enough fluid that I no longer had a visible neck lump afterward. My results returned benign, a huge relief.

My thyroid nodule story is a typical one and leaves me with the following advice for others. If a nodule is characterized as benign on ultrasound, rest assured, as these guidelines are sound and based in excellent data. If your doctor recommends a biopsy, try not to lose too much sleep; the procedure is very tolerable, and still most nodules are benign.

I had the good fortune of knowing that even if my mass turned out to be cancer, most thyroid cancers have excellent cure rates. However, I am oddly grateful to have had a small taste of the health stress my patients deal with daily. I hope it improves my doctoring.

Kelly Evans-Hullinger, M.D. is part of The Prairie Doc® team of physicians and currently practices internal medicine in Brookings, South Dakota. Follow The Prairie Doc® at www.prairiedoc.org and on Facebook featuring On Call with the Prairie Doc® a medical Q&A show celebrating its twentieth season of truthful, tested, and timely medical information, broadcast on SDPB and streaming live on Facebook most Thursdays at 7 p.m. central.

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