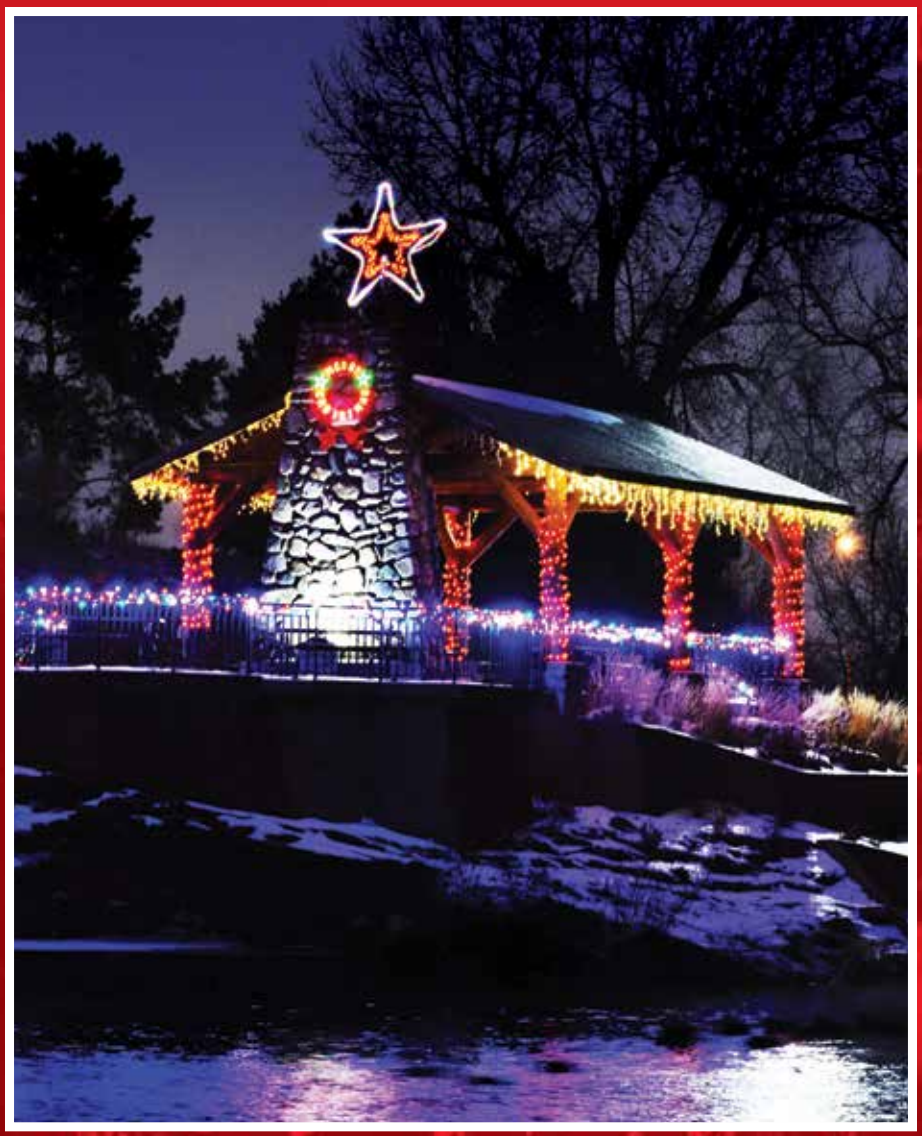
December 2025
Volume 78
Number 12



South Dakota medicine

THE JOURNAL OF THE SOUTH DAKOTA STATE MEDICAL ASSOCIATION

CAREERS WITH PURPOSE

BRENDA, RN, CASE MANAGER
A trusted liaison with a lot of heart, Brenda finds a way to connect every person to their right level of care – often when they need it most.

GROW THE GOOD

It's not just about finding a job – it's about working for an organization that truly cares about people.

Your work matters here.

Good Samaritan™
SANFORD HEALTH

FIND A CAREER WITH PURPOSE AT GROWTHEGOOD.COM

891-196-446 10/25

Contents

December 2025
Volume 78 | Number 12

Editorial

531 The Ethics of Organ Donation: New Challenges and Enduring Issues – Jane Clare Joyner, JD, MSN, RN; Ann Freeman Cook, PhD; Jerome W. Freeman, MD, FACP

President's Comments

534 The Move – Keith Hansen, MD; Tim Ridgway, MD, MACP, FASGE

The Journal

536 Historical Series: Discovery of DNA – Discovery of DNA

538 Atypical Presentation of Temporal Arteritis in a Female Patient: A Case Report – Lucas D. Goetz, MS II; Bailey Thooft, MD; Peter Reynen, MD

543 A Case Report: Incidental Subdural Catheter Placement Complicated by Horner's Syndrome Following Epidural – Meagan Kray, MS IV; Samuel Jensen, MD

546 Understanding Kratom: Pharmacology, Risks, and Relevance to South Dakota Providers – Kianna Thelen, MS IV; Carly Cooper, MS IV; Michelle Schimelpfenig, DO

548 Searching for a Solution: A Review of Deceased Donor Solid-Organ Procurement Protocols and Examination of Innovative Ideas to Address Organ Shortage – Benjamin Limburg, MS III; Sujit Vijay Sakpal, MD, FACS, FICS

552 Epinephrine is Safe in Digital Anesthesia: Debunking the Myth of Finger Necrosis and Correcting Persistent Misinformation in Medical Education – Ben Van Bockern, MS III; Emily Wilde, MS III; Lacey Person, MS II; Cole D. Tessendorf, MD; Robert E. Van Demark Jr., MD

557 Impact of Direct Current Shock on Mortality and Outcomes in Patients with Pulmonary Embolism with Pre-Existing Atrial Fibrillation, Coronary Artery Disease, and Heart Failure: A National Inpatient Sample Analysis (2019–2021) – Hammad Chaudhry, MD; Logan Johnke, MD; Dawood Shehzad, MD; Dawlat Khan, MD; Mustafa Shehzad, MD; Mamoon Ahmed, MD

564 A Case of Idiopathic Retroperitoneal Fibrosis and Inflammatory Abdominal Aortic Aneurysm in a 73-year-old Patient – Michael Roberts, MS IV; Tate Blankespoor, MS IV; Muhammed Hasan, MD; Logan Johnke, MD; Mamoon Ahmed, MD

Primers in Medicine

567 Adult Celiac Disease: Recognition and Diagnosis for the Primary Care Provider – Kennedy Forest, MD; Rafia Irfan Waheed, MD; Joseph Schowalter, DO

Special Features

570 SDSMA PAC Contributors 2025

572 Member News

576 SDBMOE Board News – Margaret B. Hansen, PA, MPAS

Cover photo by K. Remboldt, kerdallremboldt@yahoo.com

South Dakota medicine

The Journal of the South Dakota State Medical Association

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
Keith Hansen, MD

Chief Executive Officer
Tammy Hatting

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

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

South Dakota Medicine
(ISSN 0038-3317)
Published 12 times per year with one special issue by the South Dakota State Medical Association.

The SDSMA thanks the University of South Dakota Sanford School of Medicine for its financial contributions toward the production of South Dakota Medicine.

Subscription price: $50 per year domestic
$65 per year foreign, $8.95 for single copy

Periodicals postage paid at Sioux Falls, South Dakota and additional mailing offices.

Postmaster: Send address changes to
South Dakota Medicine
2600 W. 49th Street, Suite 100
Sioux Falls, SD 57105

SDSMA Home Page: www.sdsma.org

AMA Home Page: www.ama-assn.org

Printer:
Walsworth
P.O. Box 370
Fulton, Missouri 65251-0370

FOSTER GROUP

Your Financial Life, Truly Cared For®

I love what I do, but I'm busy day and night. I need time to secure my family's financial future.

MY DAYS ARE SPENT SEEING PATIENTS. HOW CAN I FOCUS ON SAVING FOR RETIREMENT AND PROTECTING MY LOVED ONES?

SEE HOW DANIEL'S WEALTH STORY UNFOLDS

Get a **complimentary financial confidence report** to learn how we can help you too!

fostergrp.com/daniel

The Team at Foster Group is dedicated to providing comprehensive financial life advice, helping clients turn their vision of success into reality.

These hypothetical case studies, based upon a combination of client scenarios, are provided for illustrative purposes only and do not represent an actual client or an actual client's experience, but rather are meant to provide an example of Foster Group's process and methodology. An individual's experience may vary based on his or her individual circumstances. There can be no assurance that Foster Group will be able to achieve similar results in comparable situations. Further, not all clients will necessarily receive the same advice or level of service as those outlined, as Foster Group provides customized services based upon the nature of each client's needs. No portion of this article is to be interpreted as a testimonial or endorsement of Foster Group's investment advisory services and it is not known whether the hypothetical clients referenced approve of Foster Group or its services.

Different types of investments involve varying degrees of risk, and there can be no assurance that the future performance of any specific investment or investment strategy (including those undertaken or recommended by Foster Group), will be profitable or equal any historical performance level. Not all services will be appropriate or necessary for all clients, and the potential value and benefit of Foster Group's services will vary based upon the client's individual investment, financial, and tax circumstances. The effectiveness and potential success of a financial, tax, and investment plan depends on a variety of factors, including but not limited to the manner and timing of implementation, coordination with the client and the client's other engaged professionals, and market conditions. Past performance does not guarantee future results.

PLEASE SEE IMPORTANT DISCLOSURE INFORMATION at www.fostergrp.com/disclosures. A copy of our written disclosure brochure as set forth on Part 2A of Form ADV is available at www.fostergrp.com.

The Ethics of Organ Donation: New Challenges and Enduring Issues

Jane Clare Joyner, JD, MSN, RN; Ann Freeman Cook, PhD; and Jerome W. Freeman, MD, FACP

Organ transplantation has tremendous potential to save or extend the lives of countless individuals, but the process for obtaining organs for transplantation is an ethically and legally complex matter. This editorial explores some of those issues, both historically and with regard to new and emerging procedures, protocols and practices.

In 1968, the Uniform Anatomical Gift Act (UAGA) was drafted in an effort to harmonize differences in state laws and address a range of ethical issues associated with organ donation and transplantation, including questions concerning the right to authorize donation, the mechanisms for designating status as an organ donor, and potential conflicts between donor rights and the rights of organ recipients. The UAGA is based on the legal framework of gift law and has been described as grounded in the ethical principles of altruism, autonomy and trust. The Act, which was most recently updated in 2006, recognized a donated organ as a voluntary gift based on the principle of consent by the donor or next-of-kin, and it included, among other things, procedures to simplify and standardize the donation process. Within three years of its drafting, all 50 states and the District of Columbia enacted a version of the UAGA.[1]

Relatedly, the growth of organ donation inevitably raised issues concerning the criteria for declaring death in potential donors. These issues were addressed in 1980 through the Uniform Declaration of Death Act (UDDA), which has been adopted, either in whole or in part, by a majority of states. Section 1 of the UDDA states:

> An individual who has sustained either (1) irreversible cessation of circulatory and respiratory functions, or (2) irreversible cessation of all functions of the entire brain, including the brain stem, is dead. A determination of death must be made in accordance with accepted medical standards.[2]

Viewpoints on the UDDA vary and there have been calls for an update of the standard, but recent efforts to revisit this standard were unsuccessful and have been paused.[3] Current state laws (including those based on the UDDA) remain in place to establish death prior to donation after circulatory death (DCD) or donation after brain death (DBD) (a term also described as brain death/death by neurologic criteria, or BD/DNC).[4]

The process for determining death is central to this discussion because organ donation and transplantation have historically been based on the "dead donor rule" (DDR), which was originally conceived as an ethical requirement that a patient will not be killed for their organs or by the act of organ donation.[5] Like the discussion surrounding UDDA, recent literature highlights the ongoing debate on the DRR within the medical community and among other stakeholders concerning both the use and the interpretation of this standard.[6-10]

Despite efforts to standardize aspects of the donation process and ongoing work to encourage organ donation, recent statistics from the Health Resources and Services Administration[11] indicate that over 100,000 individuals are currently on the national transplant waiting list. A recent report on organ donation statistics also notes that while the use of organs from donation has continued to grow, the overall growth in the number of transplants has not kept pace with additions to the waitlist.[12]

In recent years, efforts have been proposed or undertaken to increase both the volume of donated organs and the quality of the retrieved organs, all of which inevitably generate discussion of associated ethics issues. And these ethics issues may be experienced by individuals who have very different values, beliefs, socio-economic status, and cultural influences, thus creating difficult terrain for physicians and families to navigate. For example, the current iteration of the UAGA, as enacted through state laws, is consistent with "opt-in" practices, where either the donor or next-of-kin explicitly grant permission for the donation. One suggested approach to improve the overall number of donated organs has been described as a "presumed consent" or "opt-out" system, where a willingness to donate would be assumed

unless an individual indicates that they do not want to be a donor. A number of ethical concerns have been suggested with a presumed consent system, including a potential loss of autonomy, a loss of public trust or public backlash resulting in decreased donation rates.[13,14]

Recent literature has also discussed efforts to increase the use of DCD as a means to address the ongoing shortage of organs. One example is the use of normothermic regional perfusion (NRP) to improve organ viability and outcomes for organ recipients by restoring a donor's circulation following a declaration of death. Numerous authors acknowledge ongoing ethical discussion about the procedure but argue for efforts to facilitate its use to increase the availability of organs and improve outcomes.[15,16] Other authors, while recognizing the potential benefits of NRP, elaborate further on the potential ethical concerns, discussing whether the resumption of circulation violates the DDR or nullifies the declaration of death; whether cerebral reperfusion might result in pain or suffering; whether specific authorization or consent is appropriate and necessary for NRP; whether the procedure might violate a patient's religious belief; and whether an incorrect use of NRP (and any accompanying publicity) could erode public trust in donation.[17-19]

An article by Rosenbaum[9] discusses some of the ethical issues surrounding a scenario when a patient's desire to make an "imminent death donation" (IDD) or a "life donation prior to planned withdrawal" (LD-PWW) is at odds with the DDR and concerns about accelerating the death of the patient. The author describes this as a "catch-22," and writes:

> A practice such as LD-PWW could theoretically yield more organs. But if LD-PWW is perceived as unethical (surgeons as "vultures" stealing organs from those not quite dead), then, regardless of safeguards, potential donors may be lost.

The organ procurement system relies on DCD and DBD protocols to protect the interests of both organ donors and potential recipients. The topic was recently in the news when the *New York Times* (NYT) published an investigative article highlighting concerns with the organ procurement process and describing a series of troubling incidents where procurement may have been premature.[20] These challenging issues are currently being considered by Congress. Earlier this month, the Oversight Subcommittee of the House Ways and Means Committee conducted a hearing addressing organ procurement organizations' (OPO) accountability.[21] The article referenced recent studies from the *New England Journal of Medicine*[22] and the *Journal of Neurotrama*[23] addressing potential awareness in patients thought to be unresponsive, as well as the accuracy of predictions of recovery within the first 72 hours following a traumatic brain injury. The incidents described in the NYT article raise the horrifying specter of donors undergoing a procurement procedure before the donors have died. The incidents also have implications for the impact of such a scenario on both the family and the medical personnel involved in the care of the patient and the organ retrieval process. Beyond these significant issues, stories such as these may ultimately undermine public trust in the organ donation process and result in an overall decrease in the number of individuals willing to be organ donors. This would have significant implications for individuals currently awaiting organ transplants. The NYT piece highlights the centrality of ethical issues surrounding organ donation and procurement and the need for clear and transparent protocols and policies that are meticulously followed by hospitals.

Aside from ethics issues that come with new procedures, protocols and technology like presumed consent/opt-out, NRP and LD-PWW, some old ethics concerns and challenges endure. An article published over 20 years ago by one of the authors of this editorial articulated some of these concerns, particularly numerous issues pertaining to adequate consent.[24]

The following is not an exhaustive discussion of new and old issues, but offers our perspective on some pressing ethical issues associated with organ donation:

- The right to autonomy and bodily integrity should remain central to a decision to donate organs, particularly if there is a change from the current "opt-in" system to a presumed consent or "out-out" system.
- Regardless of the mechanism of proposed donation to be used, ethical principles suggest that the patient/perspective donor and/or the next-of-kin need a clear understanding of the patient's prognosis and the impact of proposed options for organ donation that may involve interventions *prior to death* (such as ante-mortem insertion of vascular catheters, system heparinization, or potential consequences of LD-PWW) or following death (such as post-mortem procedures like NRP).
- Disclosure of the different protocols for DCD and DBD donation should take place. In cases of DCD, the prospective donor and next-of-kin should be apprised of how DCD takes place, and understand that depending on

the timing of the patient's death, organs may not be viable for donation. Likewise, in cases of DBD, the next-of-kin should be apprised if the donor will remain on a ventilator or mechanical support following a declaration of death and prior to retrieval of organs.

- The prospective donor and family should be aware that interventions and treatment that take place *prior* to the donor's death may present a financial cost to the family.
- In addition to disclosing protocols for determining death and retrieving organs, numerous other ethics issues accompany the challenges of consent, any one of which may influence a donor/family decision. There has been no agreement about the extent to which prospective donors should be informed about the business aspects of the organ procurement system. Both for-profit and non-profit entities are involved with tissue procurement, including corneas, bones and skin. For example, OPOs are non-profit entities that contract with the federal government to coordinate deceased donor organs from specific donor service areas. In addition to assessing donors, obtaining consent, coordinating surgery, and preserving and transporting organs for transplantation, over 80% of OPOs conduct tissue procurement, and these activities reportedly account for 93 percent of OPO profits.[25]
- At the facility level, protocols based on full consideration of ethical implications should be in place to guide the implementation of new organ retrieval methods, including NRP, LD-PWW or other emerging techniques or technology concerning organ donation.
- At a societal level, the lack of access to health care because of financial constraints may result in unequal access to transplantation. For example, donors are regularly recruited from all socio-economic groups, but individuals without financial resources may be unable to access the transplant system if they are unable to demonstrate the ability to cover expenses associated with transplantation.

The decision to donate organs is an altruistic gift made to benefit another human being, and public trust in the system is essential to the ongoing growth and viability of the organ donation system. Scrupulous attention to ethical issues surrounding both current organ donation procedures and proposed changes to protocols, procedures and technology is essential to maintain the integrity and growth of the system.

REFERENCES

1. Sadler BL, Sadler AM Jr. Organ transplantation and the uniform anatomical gift act: a fifty-year perspective. Hastings Cent Rep. 2018 Mar;48(2):14-8.
2. Uniform Law Commission (the National Conference of Commissioners on Uniform State Laws), Chicago, IL. Determination of Death Act. Retrieved from www.uniformlaws.org/viewdocument/final-act-49?CommunityKey=155faf5d-03c2-4027-99ba-ee4c99019d6c.
3. Truog RD, Magnus DC. The unsuccessful effort to revise the Uniform Determination of Death Act. JAMA. 2023 Dec 26;330(24):2335-6.
4. Greer DM, Lewis A, Kirschen MP. The neurologist's imperative in brain death. JAMA Neurol. 2025 Mar 24.
5. Robertson JA. The dead donor rule. Hastings Cent Rep. 1999 Nov-Dec;29(6):6-14.
6. Bernat JL. Clarifying the DDR and DCD. Am J Bioeth. 2023 Feb;23(2):1-3.
7. Nielsen Busch EJ, Mjaaland MT. Does controlled donation after circulatory death violate the dead donor rule? Am J Bioeth. 2023 Feb;23(2):4-11.
8. Rodríguez-Arias D. The dead donor rule as policy indoctrination. Hastings Cent Rep. 2018 Nov;48 Suppl 4:S39-42.
9. Rosenbaum L. Altruism in extremis - the evolving ethics of organ donation. N Engl J Med. 2020 Feb 6;382(6):493-6.
10. Schweikart SJ. Reexamining the flawed legal basis of the "dead donor rule" as a foundation for organ donation policy. AMA J Ethics. 2020 Dec 1;22(12):E1019-24.
11. Health Resources and Services Administration, US Department of Health and Human Services, Rockville, MD. Organ donation statistics. Retrieved from www.organdonor.gov/learn/organ-donation-statistics.
12. Organ Procurement and Transplantation Network (OPTN) and Scientific Registry of Transplant Recipients (SRTR). OPTN/SRTR 2023 Annual Data Report. U.S. Department of Health and Human Services, Health Resources and Services Administration. Retrieved from https://srtr.transplant.hrsa.gov/annualdatareports.
13. DeRoos LJ, Marrero WJ, Tapper EB, Sonnenday CJ, Lavieri MS, Hutton DW, Parikh ND. Estimated association between organ availability and presumed consent in solid organ transplant. JAMA Netw Open. 2019 Oct 2;2(10):e1912431.
14. Glazier A, Mone T. Success of Opt-In Organ Donation Policy in the United States. JAMA. 2019 Aug 27;322(8):719-20.
15. Truog RD, Doernberg SN. In defense of normothermic regional perfusion. Hastings Cent Rep. 2024 Jul;54(4):24-31.
16. Miñambres E, Royo-Villanova M, Domínguez-Gil B. Normothermic regional perfusion provides a great opportunity to maximize organ procurement in donation after the circulatory determination of death. Crit Care Med. 2022 Nov 1;50(11):1649-53.
17. Gardiner D, McGee A. Normothermic regional perfusion for organ donation in the US-the dangers of unregulated adoption. JAMA Netw Open. 2024 Oct 1;7(10):e2440017.
18. Kirschen MP, Lewis A, Rubin MA, Varelas PN, Greer DM. Beyond the final heartbeat: neurological perspectives on normothermic regional perfusion for organ donation after circulatory death. Ann Neurol. 2024 Jun;95(6):1035-9.
19. Peled H, Mathews S, Rhodes D, Bernat JL. Normothermic regional perfusion requires careful ethical analysis before adoption into donation after circulatory determination of death. Crit Care Med. 2022 Nov 1;50(11):1644-8.
20. Rosenthal, BM, Tate, J. A push for more organ transplants is putting donors at risk. The New York Times, 2025 Jul 20; Sect. A. Retrieved from www.nytimes.com/2025/07/20/us/organ-transplants-donors-alive.html.
21. Oversight Subcommittee Hearing on Lives at Stake: Holding Tax-Exempt Organ Procurement Organizations Accountable: Hearing Before the Subcommittee on Oversight, House Ways & Means Committee, 119th Cong. 1st Sess. (Dec. 2, 2025). Retrieved from https://waysandmeans.house.gov/event/oversight-subcommittee-hearing-on-lives-at-stake-holding-tax-exempt-organ-procurement-organizations-accountable/?utm_medium=email&utm_source=newsletter&utm_campaign=wpi_healthbriefand
22. Bodien YG, Allanson J, Cardone P, Bonhomme A, Carmona J, Chatelle C, Chennu S, et al. Cognitive motor dissociation in disorders of consciousness. N Engl J Med. 2024 Aug 15;391(7):598-608.
23. Sanders WR, Barber JK, Temkin NR, Foreman B, Giacino JT, Williamson T, Edlow BL, Manley GT, Bodien YG. Recovery potential in patients who died after withdrawal of life-sustaining treatment: A TRACK-TBI Propensity Score Analysis. J Neurotrauma. 2024 Oct;41(19-20):2336-48.
24. Cook AF, Hoas H, Grayson C. Asking for organs: different needs and different values. J Clin Ethics. 2003 Spring-Summer;14(1-2):37-48.
25. Held PJ, Bragg-Gresham JL, Peters TG, McCormick F, Chertow G, Vaughan WP, Roberts JP. Cost structures of US organ procurement organizations. Transplantation. 2021 Dec 1;105(12):2612-9.

About the Authors:

Jane Clare Joyner, JD, MSN, RN, Department of Neurosciences, University of South Dakota Sanford School of Medicine.

Ann Freeman Cook, PhD, Department of Neurosciences, Ethics Programming, University of South Dakota Sanford School of Medicine.

Jerome W. Freeman MD, FACP, Department of Neurosciences, University of South Dakota Sanford School of Medicine.

The Move

Keith Hansen, MD; and Tim Ridgway, MD, MACP, FASGE

"The Mission of the University of South Dakota Sanford School of Medicine is to provide the opportunity for South Dakota residents to receive a quality, broad-based medical education with an emphasis on family medicine. The curriculum is to be established to encourage graduates to serve people living in the medically underserved areas of South Dakota and is to require excellence in the biomedical sciences and in all clinical disciplines. The University of South Dakota Sanford School of Medicine is to provide to its students and to the people of South Dakota excellence in education, research and service."

Since its inception in 1907, the University of South Dakota Sanford School of Medicine (USD SSOM) has served the people of South Dakota as its only medical school allowing opportunity for South Dakota residents to get an exemplary medical education with an emphasis on family medicine and primary care. This focus increases the probability that after training physicians will practice in rural/frontier areas of the state of South Dakota. Recently it was announced that the 18-month preclinical curriculum and division of Biomedical and Translational Sciences will move to Sioux Falls. What will this change for medical students at the USD SSOM? The professors in the division of Biomedical and Translational Sciences are responsible for educating Pillar 1 (used to be the first and second year) medical students on the biomedical sciences, giving them a strong foundation to be successful and build upon in the clinical clerkships. They also instill in our students the importance for lifelong learning and research to expand our understanding of physiology and pathophysiology. The move to Sioux Falls will enhance students' experience by allowing more convenient access to clinical faculty. Having clinical faculty work in tandem with biomedical sciences faculty will potentially enhance foundational knowledge and retention by utilizing clinical scenarios to emphasize biomedical concepts. Currently a clinical faculty member must cancel an entire half day of clinic or procedures to participate in these sessions in Vermillion. The move will allow clinical faculty more opportunities to be involved in these clinical teaching sessions for the Pillar 1 students without having to cancel a half day of clinic. Another advantage will be easier access to the Parry Center for Clinical Skills and Simulation, which is in the lower level of the Health Science Center in Sioux Falls. Students currently must travel to the Parry Center at least one day per week for simulation exercises. Biomedical and translational research will be enhanced by allowing clinical and biomedical scientists to interact more closely, developing protocols and securing funding for translational research. This collaborative environment can also enhance the ability to recruit scientists to the USD SSOM.

The move to Sioux Falls will not impact the Pillar 2 and Pillar 3 rotations and the focus will remain on training family medicine and primary care physicians for rural South Dakota. The Pillar 2 rotations will remain at the Rapid City, Yankton, Sioux Falls and the FARM sites. Currently the FARM sites include Chamberlain, Milbank, Mobridge, Parkston, Pierre, Spearfish and Vermillion. The FARM program is an innovative Pillar 2 experience where the clinical rotations are in rural sites where the student works as an integral, supervised member of the healthcare team. Alumni of this unique program have preferentially matched into primary care residencies with a desire to serve in rural communities. All students will also be exposed to rural practice in the sophomore family medicine preceptorship at the beginning of Pillar 2, and the required family medicine rural rotation in Pillar 3. These experiences allow the student to better understand the challenges and rewards of practicing medicine in a rural healthcare setting. Pillar 3 will remain the same and allows students more flexibility with time for electives to explore the various medical specialties in which they have an interest.

The move of the preclinical curriculum and division of Biomedical and Translational Sciences to Sioux Falls will not change the mission of the USD SSOM. However, the move will enhance our ability to recruit biomedical scientists, improve collaboration between biomedical and clinical scientists, and allow more clinicians to be involved in Pillar 1 instructional activities with the students. This move will enhance the education of our medical students with a continued goal to ease the healthcare shortage for all of South Dakota.

About the Author:

Keith Hansen, MD, President, South Dakota State Medical Association; Department of Obstetrics and Gynecology, University of South Dakota Sanford School of Medicine.

Tim Ridgway, MD, MACP, FASGE, Dean, University of South Dakota Sanford School of Medicine; Vice President for Health Affairs, University of South Dakota.

South Dakota Physician Well-Being Program

A confidential resource

NO insurance billing.
NO medical record or reporting.

Supporting well-being and healing
for physicians and medical students coping with:

- Career fatigue
- Grief and loss
- Professional and personal life changes
- Financial stressors
- Anxiety and depression
- Addiction
- And more...

Our goal is advocacy, education, and direct support to enrich the lives of physicians and medical students and help them live their best professional and personal lives.

Scan to Learn More

Learn more and get confidential support at:
sdpwbp.org • 605-275-4711

HISTORICAL SERIES

Discovery of DNA

H. Bruce Vogt, MD

If asked the question, "who discovered DNA," many students and physicians, for that matter, would perhaps, not surprisingly, answer "Watson and Crick." That would be incorrect. It was Friedrich Miescher who discovered DNA and many, if not most, would be surprised this was in 1869. Miescher was a Swiss chemist, biologist, and physician. He discovered what he named "nuclein," what we now call nucleic acids, from the nuclei of white blood cells leading to the identification of deoxyribonucleic acid (DNA). The significance of this discovery was not initially apparent. Even at this early date, however, Miescher hypothesized that "nuclein" might be involved in inheritance.[1] Quite the hypothesis in the mid-19th century! Upon reflection, one recalls that what James Watson and Francis Crick discovered was the "structure" of the DNA molecule. This was in 1953, nearly a century after DNA's discovery. And, although Watson and Crick deserve the most credit for the discovery, other scientists contributed significantly, most notably, Rosalind Franklin.[2]

Franklin was a British physical chemist and crystallography expert. She identified an A form (crystalline) and a B form (paracrystalline) form of DNA and discovered that the A form could be converted to the B form simply by increasing the relative humidity in the specimen chamber used in her experiments. The most crucial finding, however, was what was revealed by an x-ray diffraction photo taken by her graduate student Raymond Gosling under her direction.[3] Franklin and Maurice Wilkins,[4] another key figure in the study of DNA, had been studying x-ray diffraction images of the molecule. The image (photo 51),[3] showed the structure of DNA to be helical. Unbeknown to Franklin, Wilkins showed the photo to Watson,[2] who immediately recognized the structure as helical, in fact, a double helix. It is frequently described as a double stranded structure resembling a twisted ladder. It remains controversial as to whether Franklin recognized the structure as helical. There is evidence on both sides of the argument.

In addition to Franklin and Wilkins, Watson and Crick relied on evidence from many other researchers. Between 1885 and 1901, the German biochemist Albrecht Kossel identified the four nitrogen bases that are components of DNA (adenine, thymine, guanine and cytosine), and a 5th base uracil, which replaces thymine in the RNA molecule. He received the Nobel Prize in Physiology or Medicine in 1910 for this discovery. It was American-born chemist Phoebus Levene, however, who determined the basic structure of nucleic acids (which he termed nucleotides) and that they were linked as the molecule's backbone in the order sugar, phosphate, and nitrogen base. This included identifying both the sugars ribose and deoxyribose. Despite these significant achievements, Levene incorrectly proposed that DNA was made up of equal amounts of the four bases in the molecule (tetranucleotide hypothesis).[5] However, it was Erwin Chagraff's[6] research, which determined that there were equal amounts of Adenine and Thymine in a DNA molecule and equal amounts of Guanine and Cytosine, but not equal amounts of all four bases. Franklin's photo 51[3] provided the evidence that, as hypothesized by Levene,[5] sugar and phosphate make up the backbone of the DNA double helix and that phosphate was on the outside of the molecule with the bases facing inward (and forming hydrogen bonds). These findings were crucial to Watson and Crick's model of the DNA molecule. Their paper, as well as, one by Franklin and Goslin, and one by Wilkins were published simultaneously in the journal *Nature* in 1953.

Watson, Crick, and Wilkins won the Nobel Prize in Physiology or Medicine in 1962 for their identification of the structure of DNA. Unfortunately, in the opinion of many, Franklin was not included. This is due to the fact that she died at the youthful age of 37 in 1958 and the Nobel Prize is not awarded posthumously. However, the story might be different if, despite her untimely death, she had been recognized in the publication of the papers by any of the Nobel recipients. Her lack of acknowledgement has been suggested as a prime example of sexism in the scientific community at the time.

REFERENCES

1. Lamm Ehud, Harman Oren, Veigl Sophie Juliane. Before Watson and Crick in 1953 came Friedrich Miescher in 1869. Genetics. 2020;215(2):291-6.
2. Watson JD, Crick FH. Molecular structure of nucleic acids; a structure for deoxyribosenucleic acid. Nature. 1953;171:737-8.
3. Franklin RE, Gosling. Nature. 1953;171:740-41.
4. Wilkins MHF, Stokes AR, Wilson HR. Nature. 1953:171:738-40.
5. Levene PA. Nucleic acids. American Chemical Society. Monograph series 56. 1931.
6. Krege N, Simon RD, Hill RL. J Biochem. 2005;280(24):172-74.

About the Authors:

H. Bruce Vogt, MD, Department of Family Medicine, University of South Dakota Sanford School of Medicine.

Newborn Hearing Screening in South Dakota

SOUTH DAKOTA DEPARTMENT OF HEALTH
DOH.SD.GOV

In 2024 newborn hearing screening was added to the South Dakota newborn screening panel! Thank you for your continued role in supporting early hearing detection as part of comprehensive pediatric care.

EARLY HEARING DETECTION AND INTERVENTION (EHDI) 1–3–6 GUIDELINES

All babies should be screened at birth:

- Re-screened by 1 month
- Diagnosed by 3 months
- Enrolled in early intervention by 6 months

SUPPORT FOR MEDICAL HOMES

Free Hearing Screening Resources for Providers & Families
Educational materials can be found at: https://doh.sd.gov/programs/newborn-screening/hearing/

Find Diagnostic Services Fast
Not sure where to refer? With EHDI-PALS you can search for pediatric audiology services in the area. https://www.ehdi-pals.org/

Making a Referral to Early Intervention
For Infants and young children identified as deaf or hard of hearing, a referral can be made by contacting the service coordinator who serves the county where the family resides. Details: https://doe.sd.gov/birthto3/referral.aspx

Family-to-Family Support
If a baby or child in your practice is diagnosed with hearing loss, refer families to South Dakota Hands & Voices: https://sdhandsandvoices.org/

Coming Summer of 2026: The South Dakota Department of Health will be launching a new information system, designed to help medical homes to monitor all babies as they move through the hearing diagnostic process. As a reminder, newborn screening bloodspot results are currently available in the Iowa Open Elis portal. Providers can set up an access account for Open Elis at: https://shl.uiowa.edu/complete-web-portal-application-form

WHY THE 1-3-6 GUIDELINES MATTER

- Only pediatric audiologists have the expertise and equipment needed for a comprehensive diagnostic evaluation.
- Early identification and intervention lead to optimal outcomes.
- Linguistic input is vital to communication milestones and language development early in life.

Stay current with the latest from Joint Committee on Infant Hearing and AAP: https://www.jcih.org/ and https://www.aap.org/en/patient-care/early-hearing-detection-and-intervention/

This project is supported by the Health Resources and Services Administration (HRSA) of the U.S. Department of Health and Human Services (HHS) under Grant Number B0454576 for Maternal Child Health (MCH) services. Its contents are solely the responsibility of the authors and do not necessarily represent the official views of the Maternal Child Health Bureau/HRSA.

DID YOU KNOW?

Children who pass their newborn hearing screening can develop hearing loss at any point after birth. Those with risk factors or parent/provider concerns should be referred to a pediatric audiologist.

Babies have a higher risk of hearing loss if they experience any of the following:

- A NICU stay longer than five days
- Congenital infections, including Cytomegalovirus, herpes, rubella, toxoplasmosis, syphilis
- Head trauma
- Syndromes such as Usher syndrome
- Chemotherapy
- Low birth weight
- Craniofacial anomalies
- Ototoxic medications
- Family history of hearing loss

Scan for more info. on newborn hearing screening

Atypical Presentation of Temporal Arteritis in a Female Patient: A Case Report

Lucas D. Goetz, MS II; Bailey Thooft, MD; and Peter Reynen, MD

Abstract

Temporal arteritis, or giant cell arteritis, is an idiopathic, autoimmune vasculitis that primarily affects medium- and large-sized arteries. It typically presents with headache, jaw claudication, fever, or visual disturbances. We report an atypical case of temporal arteritis in a 68-year-old female who initially presented with left paraspinal neck pain, malaise, and mild headaches. Over the following weeks, her symptoms progressed to include worsening headache, fever, night sweats, and fatigue. A multidisciplinary team approach led to timely diagnosis and treatment initiation, preventing severe complications. This case illustrates an uncommon presentation of temporal arteritis initially mistaken for musculoskeletal pain and highlights the value of early recognition to prevent irreversible complications such as vision loss.

Introduction

Temporal arteritis, also known as giant cell arteritis, classically presents with headache, temporal tenderness, and jaw claudication. As the disease progresses, systemic constitutional symptoms such as fever, myalgia, night sweats, fatigue, and weight loss are frequently observed.[1] This case describes a 68-year-old female who initially presented with neck pain, malaise, and headache following recent travel—an atypical presentation which ultimately led to the diagnosis of temporal arteritis.

Case Report

Current Presentation

A 68-year-old female presented to the clinic with a 10-day history of left paraspinal neck pain and headaches. Her symptoms began while traveling out of state earlier in the year. She sought chiropractic care during her trip but experienced no relief. Conservative measures including heat, ice, and acetaminophen were minimally effective. She denied any recent trauma, prior history of similar symptoms, known sick contacts, or identifiable triggers.

Over the subsequent months, her neck pain and headaches gradually worsened. She developed additional systemic symptoms including intermittent fevers, night sweats, chills, and profound fatigue. She also reported an unintentional weight loss of approximately 13 pounds over six weeks. As the illness progressed, she began to experience blurry vision, imbalance, and altered taste sensation.

The patient's medical history included well-controlled type 2 diabetes mellitus, essential hypertension, mixed hyperlipidemia, ulcerative colitis, and cervicalgia attributed to multilevel cervical spondylosis.

Upon presentation to the clinic following her return from out-of-state travel, the initial physical examination revealed tenderness over the C5–C6 spinous processes. The patient's neck pain was most pronounced in the left paraspinal musculature near the lower cervical spine, accompanied by bitemporal discomfort. There was no limitation in range of motion, cervical lymphadenopathy, or evidence of torticollis. Palpable myofascial knots were noted in the trapezius muscles at the C5–C6 level. Vital signs were within normal limits.

Laboratory testing was not performed at the initial visit, as the patient's neck pain and headache were initially attributed to a musculoskeletal etiology. At a follow-up appointment one week later, laboratory evaluation—including a complete blood count (CBC), comprehensive metabolic panel (CMP), and C-reactive protein (CRP)—was obtained. Abnormal results are summarized in Table 1. Based on the physical exam findings and recent travel history, the patient was initially referred to infectious disease. However, after an extensive infectious workup yielded negative results, a referral was made to rheumatology for further evaluation.

Additional testing included *Borrelia burgdorferi* PCR, SS-A and SS-B antibodies, and an acute hepatitis panel—all of which were within normal limits. Rosuvastatin was

Table 1. Initial abnormal lab values. CBC and CMP were otherwise normal.

	Initial abnormal lab values	Normal range
WBC	11.4 K/uL (high)	4.5-11.0 K/uL
Platelet count	736 K/uL (high)	140-450 K/uL
Neutrophil % (auto)	77.2% (high)	40.0-75.0%
Absolute neutrophils (auto)	8.80 (high)	1.80-7.70 K/uL
CRP	27.1 (high)	<0.5 mg/dL
ESR	37 mm/Hr (high)	0-30 mm/Hr

WBC = white blood cell, CRP = c-reactive protein, ESR = erythrocyte sedimentation rate

discontinued to rule out statin-associated muscle symptoms as a contributing factor. Due to her persistent neck pain, a lumbar puncture was performed. Cerebrospinal fluid (CSF) analysis included oligoclonal band profiling, cryptococcal antigen, paraneoplastic autoantibody panel, angiotensin-converting enzyme (ACE), lactate dehydrogenase (LDH), VDRL with reflex titer, and West Nile virus IgG/IgM. The only abnormal finding was a positive West Nile IgG; all other results were negative or within normal limits.

Brain MRI was unremarkable, with no acute findings. Cervical spine MRI revealed multilevel cervical spondylosis with central stenosis at the C5–C6 level, along with varying degrees of foraminal stenosis throughout the cervical spine. Additionally, cervical spine x-rays demonstrated multilevel degenerative changes, most prominent at the C5–C6 and C6–C7 levels.

Given the patient's age, new-onset headache, and markedly elevated inflammatory markers, a temporal artery biopsy was pursued to evaluate for temporal arteritis, despite the absence of scalp tenderness or decreased temporal artery pulse. Histopathologic examination revealed a medium-sized artery with lymphohistiocytic inflammation, arterial wall thickening, and focal elastic fiber fragmentation on special staining—findings consistent with temporal arteritis.

Treatment and Follow-Up

Following confirmation of temporal arteritis on temporal artery biopsy, the patient was started on high-dose corticosteroid therapy with prednisone 80 mg daily. Pantoprazole 40 mg daily was prescribed for gastrointestinal prophylaxis, and trimethoprim-sulfamethoxazole (160 mg/800 mg) was initiated three times per week for *Pneumocystis jirovecii* pneumonia (PJP) prophylaxis. Subcutaneous tocilizumab (162 mg weekly) was also added to enhance disease control and facilitate a more rapid steroid taper. The patient was counseled on potential corticosteroid side effects and advised to engage in daily weight-bearing exercise, maintain an active lifestyle, and supplement with calcium and vitamin D to support bone health.

After 10 months of steroid therapy, the patient reported a gradual improvement in symptoms and expressed satisfaction with her outcome. Aside from occasional tension headaches, she denied experiencing scalp tenderness, neck pain, jaw claudication, or fever. She remained on weekly tocilizumab and continued rheumatology follow-up every three months for laboratory monitoring. In addition, she maintained regular visits with her primary care provider for ongoing management of her other chronic conditions.

Discussion

Temporal arteritis is a systemic inflammatory vasculitis primarily affecting medium and large arteries, with an incidence of 20 per 100,000 individuals over the age of 50. It is rare in patients under 50, with a mean age of onset around 75. The exact etiology remains unknown, but known risk factors include advanced age, Scandinavian ancestry, and smoking, with women being particularly at higher risk. The female-to-male ratio for temporal arteritis is approximately 2:1.[2]

A major concern in temporal arteritis is ischemic optic neuropathy, which can lead to irreversible vision loss in the affected eye, potentially involving both eyes. While the temporal artery is the most commonly affected, other arteries originating from the arch of the aorta can also be involved. Temporal arteritis is frequently associated with polymyalgia rheumatica and is the most common form of vasculitis in adults in Western countries.[2]

C-reactive protein (CRP) is a more sensitive marker of inflammation in temporal arteritis than erythrocyte sedimentation rate (ESR), and elevations in both increase the specificity of the diagnosis. Although classic findings such as temporal artery tenderness or diminished pulsation heighten clinical suspicion, their absence does not exclude the disease. In this case, the patient lacked these hallmark signs, but the presence of systemic symptoms and significantly elevated inflammatory markers warranted a temporal artery biopsy. Histopathologic evaluation revealed lymphohistiocytic inflammation, arterial wall thickening,

and disruption of elastic fibers—findings characteristic of temporal arteritis. A positive biopsy is typically defined by transmural inflammation, fragmentation of the internal elastic lamina, and mononuclear infiltrates composed of lymphocytes, histiocytes, and occasionally multinucleated giant cells.

Due to the lack of specific serological or laboratory markers for temporal arteritis, a temporal artery biopsy remains the gold standard for diagnosis, with a sensitivity of 90–95%. Ideally, a biopsy should be performed at the earliest suspicion of temporal arteritis. However, given the risk of irreversible vision loss, corticosteroid treatment should be initiated promptly, and the biopsy should be done within two weeks of starting treatment. In cases where headache is present, the biopsy should be conducted on the symptomatic side. Due to the presence of skip lesions, biopsies should aim to remove longer segments (4 to 6 cm). Although a negative biopsy does not definitively rule out temporal arteritis, especially in the presence of skip lesions, clinical suspicion should remain high.[1,2]

The American College of Rheumatology (ACR) has established diagnostic criteria for temporal arteritis, requiring at least three of the following five criteria[2,3] for a diagnosis (though these were primarily developed for research purposes and are not intended for sole clinical use):

- Age of onset ≥50 years
- New headache
- Abnormalities of the temporal artery (e.g., decreased pulse or tenderness)
- ESR ≥50 mm/h
- Abnormal artery biopsy, which may show predominance of mononuclear cell infiltrates, granulomatous inflammation, vasculitis, or multinucleated giant cells

In this case, the patient initially presented with atypical symptoms, beginning with paraspinal neck pain and malaise. Over time, her condition progressed to include headaches, fever, night sweats, chills, unintentional weight loss, blurry vision, imbalance, and altered taste sensation. This patient met several of the risk factors for temporal arteritis, including her age, gender, and Scandinavian descent. For patients who meet these risk factors and present with new-onset headache and constitutional symptoms, temporal arteritis should remain high on the differential diagnosis.[2] Table 2 outlines additional symptoms that are commonly and less commonly associated with temporal arteritis.

The mainstay of treatment for temporal arteritis is prompt initiation of high-dose corticosteroids to prevent ischemic complications such as irreversible vision loss. Standard regimens include prednisone 40-80 mg daily for uncomplicated cases, or intravenous methylprednisolone

Table 2. Common symptoms at presentation and how common the symptomology is.[1]

Symptom at presentation	Percentage	Note
Headache	≥75%	• May be absent in patients with extracranial involvement • Often insidious in onset and gradually worsens as time goes on • Does not respond well to over-the-counter medications • May be accompanied by scalp tenderness while combing or brushing hair
Polymyalgia rheumatica	40-60%	• 15-20% of patients with polymyalgia rheumatica also have temporal arteritis
Temporal artery symptomology	≤50%	• May present as enlargement, tenderness, nodular swelling, or loss of pulse in the temporal artery • Nodular swelling of the temporal artery carries a high positive likelihood ratio for positive biopsy
Negative temporal artery biopsy	≤50%	• Due to skip lesions, a biopsy may be negative
Fever	40%	• Typically, a low-grade fever • 15% of fevers of unknown origin in patients over 65 are due to temporal arteritis
Jaw claudication	≥30%	• Especially when chewing or talking • Carries a high positive likelihood ratio for positive biopsy
Neurological symptoms	≤30%	• May present as transient ischemia, strokes, mononeuropathies, or peripheral neuropathies • Commonly involves C5 nerve root and causes loss of shoulder abduction
Vision loss	≤15%	• Due to ophthalmic artery or posterior ciliary artery involvement • Often painless and sudden onset • Diplopia may occur before vision loss • May be unilateral or bilateral
Extracranial involvement	10-15%	• Commonly involves thoracic or abdominal aorta (branches commonly involved include subclavian, axillary, carotid, and brachial arteries) • May occur without cranial involvement • Symptoms may include bruits, lack of pulses or asymmetric pulses in upper extremities, asymmetric blood pressure readings in upper extremities, claudication of upper extremities, or Raynaud's phenomenon
Pharyngeal ischemia	≤10%	• May present as a dry or productive cough, hoarseness, or sore throat

when ocular symptoms are present.[2] In our patient, gastrointestinal prophylaxis with pantoprazole and PJP prophylaxis with trimethoprim-sulfamethoxazole were added due to the expected duration and dose of corticosteroid therapy. Tocilizumab, an IL-6 receptor antagonist, was initiated early to support disease control and minimize cumulative steroid exposure. According to current guidelines and expert recommendations, tocilizumab is an effective adjunct in newly diagnosed or relapsing temporal arteritis and is particularly useful in patients at risk for steroid-related complications.[2]

Conclusion

Temporal arteritis is an inflammatory vasculitis that typically affects adults over 50 and carries a risk of irreversible vision loss if not promptly treated. Diagnosis is supported by temporal artery biopsy, and high-dose corticosteroids remain the cornerstone of treatment. This case underscores the importance of considering temporal arteritis even when initial symptoms are atypical, such as paraspinal neck pain or fatigue. Maintaining a high index of suspicion, especially in older female patients with new-onset headaches and systemic symptoms, can lead to earlier diagnosis and prevent serious complications.

REFERENCES

1. Bilton EJ, Mollan SP. Giant cell arteritis: Reviewing the advancing diagnostics and management. Eye (Lond). 2023 Aug;37(12):2365–73.
2. Ameer MA, Granville LJ. Giant cell arteritis (temporal arteritis). StatPearls. Treasure Island (FL): StatPearls Publishing; 2024 May 2 Retrieved from www.ncbi.nlm.nih.gov/books/NBK459376/.
3. Hunder GG, Bloch DA, Michel BA, et al. The American College of Rheumatology 1990 criteria for the classification of giant cell arteritis. Arthritis Rheum. 1990 Aug;33(8):1122–8.

About the Authors:

Lucas D. Goetz, MS II, University of South Dakota Sanford School of Medicine.

Bailey Thooft, MD, University of South Dakota Sanford School of Medicine.

Peter Reynen, MD, University of South Dakota Sanford School of Medicine.

A Case Report: Incidental Subdural Catheter Placement Complicated by Horner's Syndrome Following Epidural Anesthesia

Meagan Kray, MS IV; and Samuel Jensen, MD

Abstract

Background: Epidural anesthesia is commonly used for labor analgesia, but complications such as subdural catheter placement, although rare, can occur. This case report describes a patient who experienced neurological symptoms, including Horner's syndrome, likely caused by incidental subdural catheter placement after an epidural procedure.

Case Presentation: A 32-year-old female, G3P1011, at 37 weeks gestation, presented for scheduled induction of labor due to gestational hypertension. The combined spinal-epidural (CSE) procedure was performed at the L3-L4 intervertebral space without complications initially. However, approximately 1 hour later, the patient developed left-sided facial drooping, left arm weakness, numbness around the mouth, and hypotension. The epidural was initially stopped and repositioned due to suspected subdural catheter placement and later restarted at a reduced infusion rate of 1 mL/hr. Over several hours, the patient's symptoms improved, and she progressed to full cervical dilation. She delivered a healthy baby vaginally without further complications. The patient's neurological symptoms fully resolved by 6 hours post-procedure.

Conclusion: This case highlights the rare complication of subdural catheter placement during epidural anesthesia. The patient's symptoms, including an atypical sensory block and mild motor involvement, were consistent with a subdural block and improved with supportive care, including cessation of the epidural infusion, phenylephrine, and fluid resuscitation. Although subdural catheter placement is rare, it should be considered in the differential diagnosis for patients with unexpected neurological symptoms after epidural anesthesia. Early recognition and management are crucial for preventing complications and improving patient outcomes. In this case, prompt intervention led to complete resolution of symptoms.

Introduction

Epidural anesthesia is a well-established technique for providing labor analgesia. However, rare complications such as subdural catheter placement are important to recognize early. The subdural space, located between the arachnoid and dura mater, is a potential space that can be inadvertently entered during epidural procedures. Unlike the epidural space which terminates at the foramen magnum, the subdural space communicates with the intracranial space. Therefore, cephalad spread of an anesthetic agent into the subdural space can result in significant hemodynamic complications. This case report discusses the clinical presentation, management, and outcomes of a potential subdural catheter placement in a pregnant patient, with a focus on the occurrence of Horner's syndrome and hemodynamic instability.

Case Presentation

A 32-year-old female (ASA 2), G3P1011, at 37 weeks gestation, presented for a scheduled induction of labor due to gestational hypertension. Her pregnancy was further complicated by a low-lying posterior placenta, but she had no significant comorbidities. Upon admission, the patient was hemodynamically stable, with a BP of 122/88 mmHg, pulse 94, and an O_2 saturation of 98% on room air.

After admission, a combined-spinal epidural (CSE) procedure was initiated for labor analgesia without sedation. The patient was positioned in the sitting position, prepped with chlorhexidine gluconate, and was continuously monitored. The epidural was placed at the L3-L4 intervertebral space using anatomical landmarks. A 17-gauge, 9 cm Tuohy needle was inserted 5 cm deep, followed by the placement of a multi-

orifice, 19-gauge epidural catheter, which was threaded 10 cm from the skin level. The catheter was secured, and an initial test dose of 3 mL of lidocaine 1.5% with epinephrine was administered. Correct epidural placement was confirmed by a negative test result, indicated by the absence of signs of intravascular or intrathecal injection—such as immediate tachycardia, hypotension, rapid onset of lower limb motor block, or an unexpectedly high sensory block. Following the negative test dose, a continuous epidural infusion of 14 mL/hr of ropivacaine was started. For the spinal portion of the CSE, clear cerebrospinal fluid (CSF) was observed to ensure proper placement using a 27-gauge pencil-tip needle. One mL of 0.25% bupivacaine and 15 mcg of fentanyl was administered. The patient tolerated the procedure well with no immediate complications. Post-procedure vital signs were BP 127/79 mmHg, pulse 77 bpm, and SpO_2 100% on room air.

Approximately 1 hour after the epidural placement, the patient developed unexpected neurological symptoms, including left-sided facial drooping, left arm weakness, and numbness around the mouth. Her blood pressure initially dropped to 82/42 mmHg, which was managed with phenylephrine. Despite treatment, her symptoms persisted, and the epidural was temporarily stopped. At this time, subdural catheter placement was suspected and the catheter was pulled back approximately 1 cm to reposition in epidural space. Approximately 20 minutes later, the epidural was restarted at a reduced infusion rate of 1 mL/hr. Despite this, the patient continued to experience intermittent hypotension, which was managed with repeated doses of phenylephrine. Throughout the course of neuraxial analgesia, fetal heart tracings remained category I, with a brief deceleration that resolved with maternal repositioning. Her neurological symptoms gradually improved over the next several hours, and by 4 hours after the epidural placement, her blood pressure stabilized, and the symptoms of left arm weakness and facial drooping began to resolve. Sterile vaginal exam (SVE) later revealed continued progress to 10 cm dilation, and the patient delivered a healthy 3270g baby vaginally with an APGAR score of 8 and 9. The placenta was delivered without complications. By 6 hours post-procedure, the patient's facial drooping and left arm weakness had completely resolved, and she had no residual neurological deficits. The epidural catheter was discontinued following delivery.

Discussion

Subdural catheter placement during epidural anesthesia is an exceedingly rare complication with an incidence of 0.82% in lumbar epidural injections.[2] The subdural space lies between the dura mater and the arachnoid, and its unique anatomical features allow for a more cephalad spread of anesthetic agents than the epidural space.[3] This can lead to serious complications, including high sympathetic blockade, neurological deficits, and hemodynamic instability. Horner's syndrome, a hallmark of sympathetic disruption, is an uncommon clinical presentation associated with this condition. It presents with the classic triad of ptosis, miosis, and anhidrosis, often accompanied by other signs like enophthalmos or conjunctival injection. This syndrome occurs because the sympathetic fibers to the eye, originating from the cervical sympathetic chain, are blocked by anesthetic agents that inadvertently enter the subdural space.[3]

The rarity of subdural catheter placement and the associated neurological complications emphasize the need for vigilance during the placement and monitoring of epidural catheters. In obstetric patients, anatomical and physiological changes, such as increased intra-abdominal pressure and venodilation, can predispose to a higher spread of anesthetic agents, increasing the risk of reaching the subdural space.[3] This phenomenon can be exacerbated by variations in epidural anatomy, such as the presence of fibrous septae, scoliosis, or prior epidural attempts, which can create channels that facilitate cephalad spread.[2]

The unique nature of this case lies in its relatively mild and transient neurological presentation compared to other reports in the literature, where symptoms are often more severe and associated with significant hemodynamic instability.[4,5] In contrast to more dramatic presentations of subdural block, this patient's symptoms—left-sided facial drooping, arm weakness, and hypotension—resolved gradually over several hours, and the patient ultimately delivered a healthy infant without any residual neurological deficits. This underscores the importance of early recognition and management of such complications, which can often resolve without long-term effects if promptly addressed.[2]

In our case, Horner's syndrome appeared approximately an hour after epidural catheter placement, with subsequent management involving temporary cessation of the epidural infusion and symptomatic treatment with phenylephrine. Interestingly, the patient did not develop the more severe manifestations often seen in subdural block, such as respiratory depression or profound hypotension. This suggests that in cases where symptoms are mild and transient, conservative management and careful monitoring may be

appropriate.

Unlike typical reports of subdural catheter placement, where there is a more extensive sensory blockade, this patient maintained a sensory level around T4. The fact that the neurological symptoms were limited to Horner's syndrome and resolved spontaneously without the need for extensive intervention suggests that a subdural catheter placement, while a possible cause, might not always lead to the catastrophic outcomes often anticipated. In patients who experience prolonged hypotension or atypical neurologic symptoms, discontinuation of the epidural should be considered if symptoms do not resolve or if maternal or fetal compromise occurs. In this case, the patient's hypotension and symptoms improved with supportive care, allowing for cautious continuation of the epidural at a reduced rate. This case emphasizes that even when Horner's syndrome is identified, it may not necessitate the immediate cessation of neuraxial anesthesia in all instances, particularly when the symptoms are isolated, mild, and transient.

Conclusion

This case highlights the importance of recognizing subdural catheter placement in the differential diagnosis for patients with unusual neurological symptoms following an epidural procedure. Prompt diagnosis, supportive care, and cautious management are crucial for positive outcomes. Awareness of this rare complication can help clinicians provide better care and reduce the risk of further complications.

REFERENCES

1. Bansal S, Turtle MJ. Inadvertent subdural spread complicating cervical epidural steroid injection with local anaesthetic agent. Anaesth Intensive Care. 2003;31:570–2.
2. Lubenow T, Keh-Wong E, Kristof K, Ivankovich O, Ivankovich AD. Inadvertent subdural injection: A complication of an epidural block. Anesth Analg. 1988;67:175-9.
3. Agarwal D, Mohta M, Tyagi A, Sethi AK. Subdural block and the anaesthetist. Anaesth Intensive Care. 2010;38(1):20-6.
4. Bergquist A, Buerger C, Hatfield A, Hofkamp M. A 24-year-old multiparous woman with apparent subdural epidural catheter: Diagnosis and management of an uncommon obstetric anesthesia complication. Proc (Bayl Univ Med Cent). 2023;36(6):764-6.
5. Smith JA, Barker ME, Jones SL. Hemodynamic instability and Horner's syndrome following a labor lumbar neuraxial block: a warning sign. Anesth Analg. 2017;125(5):1562-4.

About the Authors:

Meagan Kray, MS IV, University of South Dakota Sanford School of Medicine.

Samuel Jensen, MD, Sanford USD Medical Center, Sioux Falls, South Dakota.

Don't Just Survive —
Really **Live**

MORE GOOD YEARS

Quitting can the difference between quality time with your family and missing out to catch your breath. Enjoy your years really living. Kicking a tobacco addiction isn't easy, but it'll be worth it!

With South Dakota QuitLine, choose from 3 FREE ways to put out your last cigarette, let the vape pen die and close that tin for good. Leave tobacco in the past, and live more **good** years.

1-866-SD-QUITS

SOUTH DAKOTA QuitLine

Paid for by the South Dakota Department of Health

Call the QuitLine today! | 1-866-737-8487 | SDQuitLine.com

Understanding Kratom: Pharmacology, Risks, and Relevance to South Dakota Providers

Kianna Thelen, MS IV; Carly Cooper, MS IV; and Michelle Schimelpfenig, DO

Abstract

Introduction: Kratom (Mitragyna speciosa), a Southeast Asian botanical, has gained increasing popularity in the U.S. for its stimulant and opioid-like effects. As its use grows, providers face challenges in understanding its pharmacology, clinical risks, and regional impact.

Methods: This narrative review synthesizes existing literature on kratom's pharmacological profile, clinical implications, regulatory status, and poison control trends, with a particular emphasis on South Dakota.

Results: Kratom contains mitragynine and 7-hydroxymitragynine, alkaloids with activity at opioid receptors. Its effects vary by dose, ranging from stimulation to sedation. Though often used for pain and opioid withdrawal, kratom carries risks such as dependency, hepatotoxicity, seizures, and death—particularly when combined with other substances. South Dakota-specific poison control data reveal a significant uptick in kratom-related calls, aligning with national increases.

Conclusion: Kratom remains legal and accessible in South Dakota, contributing to rising clinical encounters. Providers should screen for use, understand its dose-response profile, and be prepared to address associated complications.

Introduction

Kratom (Mitragyna speciosa) is a tree native to Southeast Asia long used for its stimulant and analgesic properties. Recently, kratom has gained popularity in the U.S., including South Dakota, as an herbal supplement marketed for pain relief, mood enhancement, and opioid withdrawal. Despite anecdotal reports of therapeutic benefit, kratom's pharmacological profile and health risks raise concern among clinicians and public health professionals. This review provides an overview of kratom's pharmacology, adverse effects, and legal landscape, with emphasis on its growing relevance for South Dakota providers.

Pharmacology and Mechanism of Action

Kratom's psychoactive effects are mediated by alkaloids—primarily mitragynine and 7-hydroxymitragynine. These compounds act as partial agonists at the mu-opioid receptor and interact with serotonergic and adrenergic systems. At low doses (1–5 g), kratom acts as a stimulant, increasing alertness and energy. At higher doses (5–15+ g), sedative and opioid-like effects emerge, including analgesia and euphoria. Though kratom may not cause respiratory depression to the same extent as traditional opioids, overdose risks escalate when combined with other central nervous system depressants.

Patterns of Use in the U.S. and South Dakota

Kratom is available in multiple forms—powders, teas, capsules, and extracts—through online vendors and local head shops. National surveys estimate over 1.7 million Americans use kratom annually. In rural South Dakota, where access to comprehensive pain management and substance use treatment may be limited, kratom is often used as an unregulated alternative to traditional therapies.

Poison control data provide insights into adverse outcomes. Nationally, kratom-related calls to poison centers increased from 13 in 2011 to over 680 in 2017. According to the 2023 National Poison Data System report, kratom was among the top unregulated substances of concern, with serious medical outcomes reported in over half of cases.

In South Dakota, poison exposures are managed by the Sanford Poison Center in Sioux Falls, in collaboration with the Minnesota Regional Poison Control Center. While detailed kratom-specific statistics are not publicly available, toxicologists and poison center staff report a marked increase in kratom-related calls over the past three years. Providers have noted cases involving gastrointestinal distress, seizures, and hepatic dysfunction—often in the context of polypharmacy.

Unofficial reports suggest a 3- to 4-fold increase in kratom-related poison control inquiries between 2021 and 2024, mirroring national trends.

Adverse Effects and Toxicity

Common short-term effects include nausea, vomiting, constipation, and agitation. More severe complications involve hepatotoxicity, seizures, hallucinations, and withdrawal syndromes resembling those of opioids. Fatalities are rare but documented, particularly when kratom is used alongside other psychoactive agents. Importantly, kratom is not detected in standard urine drug screens, which may delay diagnosis and treatment.

Regulatory Landscape

Kratom is not scheduled at the federal level. Although the DEA proposed a ban in 2016, it was reversed following public backlash. The FDA continues to warn against its use due to safety concerns but has not enforced regulation. In South Dakota, kratom remains legal statewide, though some municipalities have debated restrictions.

Clinical Implications for South Dakota Providers

- Given kratom's availability and the rising number of poison control calls, South Dakota providers must be equipped to:
- Screen for kratom use, especially in patients with chronic pain or opioid use history
- Recognize kratom-related adverse effects, particularly in poly-substance users
- Educate patients about unregulated products and potential harms
- Refer patients to substance use treatment as appropriate

Perioperative Risk and Opioid Dependence

Chronic kratom use may alter opioid receptor sensitivity, leading to tolerance and complicating perioperative pain management. Patients may require high opioid doses or non-opioid adjuncts to achieve adequate analgesia. Kratom withdrawal—marked by agitation, nausea, and hypertension—can occur postoperatively, especially if use is abruptly stopped.

Because kratom is not detected on standard drug screens, preoperative screening is essential to help prevent perioperative events and lead to better control of patients' pain. Its use may also signal underlying opioid use disorder, especially in patients self-medicating for pain. South Dakota providers involved in surgical care should consider kratom use when assessing perioperative risk and coordinate with anesthesia and addiction specialists as needed.

Naloxone for Kratom Overdose

Naloxone (Narcan) may offer benefit in select cases of kratom overdose, particularly when opioid-like symptoms such as respiratory depression or altered consciousness are present. However, its effectiveness is inconsistent due to kratom's mixed pharmacologic profile—while mitragynine and 7-hydroxymitragynine act on mu-opioid receptors, they also modulate adrenergic and serotonergic systems. In one case series of 10 kratom overdose incidents, naloxone was administered in 3 cases; 2 patients showed prompt improvement, while 1 did not respond. These mixed results underscore the need for clinical judgment and further research when considering naloxone for kratom toxicity. Providers should consider naloxone administration if opioid toxidrome is suspected but remain vigilant for additional toxicologic mechanisms.

Conclusion

Kratom use is increasing in South Dakota and poses a multifaceted challenge to healthcare providers. Though it is marketed as a natural remedy, kratom has significant pharmacological activity and risk for harm. Providers should remain vigilant, inquire about use during history taking, and collaborate with poison control and addiction specialists when complications arise. Ongoing surveillance and public health education are essential to mitigate the risks associated with this emerging substance.

REFERENCES

1. Prozialeck WC, Jivan JK, Andurkar SV. Pharmacology of kratom: An emerging botanical agent with stimulant, analgesic, and opioid-like effects. Journal of the American Osteopathic Association, 2012;112(12):792–9.
2. Henningfield JE, Fant RV, Wang DW. The abuse potential of kratom according to the 8 factors of the controlled substances act: implications for regulation and research. Psychopharmacology. 2018;235:573–89.
3. Anwar M, Law R, Schier J. Notes from the field: Kratom (Mitragyna speciosa) exposures reported to poison centers — United States, 2010–2015. MMWR Morb Mortal Wkly Rep. 2016;65(29):748–9.
4. Smith KE, Dunn KE, Rogers JM, Garcia-Romeu A, Strickland JC, Epstein DH. Assessment of kratom use disorder and withdrawal among an online convenience sample of us adults. J Addict Med. 2022;16(6):666–70. doi: 10.1097/ADM.0000000000000986.
5. Galbis-Reig D. A Case report of kratom addiction and withdrawal. WMJ. 2016;115(1):49–52; quiz 53.
6. Peran D, Asan H, Willers J, Dinca D. Case series: Mitragyna speciosa (kratom) poisoning. Toxicology. 2023;488:153493.
7. South Dakota Poison Control Center. Annual Report on Substance-Related Calls. Internal communication. 2024.
8. U.S. Food and Drug Administration. FDA and Kratom. 2022. Retrieved from www.fda.gov/news-events/public-health-focus/fda-and-kratom.

About the Authors:

Kianna Thelen MS IV, University of South Dakota Sanford School of Medicine.

Carly Cooper MS IV, University of South Dakota Sanford School of Medicine.

Michelle Schimelpfenig, DO, University of South Dakota Sanford School of Medicine; Sanford Children's Sioux Falls, South Dakota.

Searching for a Solution: A Review of Deceased Donor Solid-Organ Procurement Protocols and Examination of Innovative Ideas to Address Organ Shortage

Benjamin Limburg, MS III; and Sujit Vijay Sakpal, MD, FACS, FICS

Abstract

As solid-organ transplant capabilities improve, transplants are becoming a life-saving solution for an increasing number of patients. However, the number of organs procured from donors following brain death (DBD) falls far short of the number of waiting recipients. Organs procured from donors following circulatory death (DCD) are often comparable to DBD organs and attempt to address the shortage of viable allografts. Strikingly, consistent protocols governing DCD organ procurement do not exist, with one potential reason being an imperfect understanding of the underlying science and philosophy. Along with improvements to the DCD process, consideration of standardizing the window for withdrawal of life support therapy and pre-mortem nephrectomy present potential avenues to enhance allograft yield. Deserving recipients ultimately stand to benefit from further discussion of DCD procurement within the scientific community and beyond.

Introduction

At present, over 100,000 people are waiting for a life-saving organ in the U.S., with over 90,000 of those awaiting a kidney.[1] Despite significant advances in the technical aspects of transplant operations, anti-rejection immunosuppression, and methods of prolonging organ health outside the body, demand for organs greatly exceeds supply. Of all solid-organ transplants (SOTs) in the U.S. each year, 92% come from brain-dead donors.[1] However, of roughly 2 million deaths yearly in the U.S., only around 12,000 are declared brain-dead. Even if every one of the 12,000 brain-dead patients consented to organ donation and operations were carried out with perfect yields, the number of organs procured via donation following brain death (DBD) would remain overwhelmingly short of the need.

Donation after brain death

The current biological understanding of death is not absolute, especially in transplantation when a body must be alive enough to offer usable organs, yet dead enough to no longer be a patient in the traditional sense of the word. Essentially, a body is alive when it can deliver oxygen and nutrients to its cells to sustain cellular processes and metabolism.[2] Basic life support techniques such as cardiopulmonary resuscitation, and advances like mechanical ventilation and extracorporeal membrane oxygenation, provide ways to prolong patients' lives, indefinitely in certain cases, and can blur the lines between natural death and artificial life.[2–4]

As life support capabilities became increasingly proficient, situations arose in which patients were determined to be irreversibly comatose but were maintained with medically assisted breathing, circulation, and nourishment. For many, life in such a state was neither meaningful nor feasible, yet physicians did not possess legal or widespread ethical backing to discontinue life support. To address this undesirable gray area, brain death was a proposed solution. When a patient loses all neurologic function, they are considered "brain dead." At present, brain death is formally classified as irreversible loss of function of every brain region including the brainstem.[5] Interestingly, the modern legal understanding of brain death was formulated relatively recently as part of the 1981 Uniform Determination of Death Act (UDDA) and remains imperfect. Although brain death is in many cases conclusive, UDDA guidance does not perfectly encompass all means by which a patient can die, and subjective interpretations are occasionally made by those in charge of their care. Thus, brain death criteria are not perfect guidance for death pronouncement.

What is donation following circulatory death?

The first deceased organ donors were actually patients who had died by "cardiac death."[6] Put simply, circulatory death

occurs when the heart stops beating and cannot be restarted. With the advent of DBD criteria, donation following circulatory death (DCD) was phased out in favor of the more objective, reproducible brain death criteria. Shifting preferences to declaration of brain death occurred partially because of decreased liability on the part of physicians and the healthcare team, along with superior function of DBD over DCD allografts at the time. What made DCD organs inferior to those from brain dead patients was primarily the damage caused by prolonged periods of ischemia.[7] Obviously, transplantable organs must be healthy enough to resume function within the recipient. Without modern preservation techniques, which allow organs to remain viable for much longer periods of time outside the body, procuring organs from patients whose hearts were not circulating blood often proved to be too challenging.[7,8] For DCD donors, the stoppage of a beating heart immediately begins the warm ischemia time (WIT), and earlier transplants were utilizing heavily compromised organs from such donors. However, advances in cooling organs within the donor allowed DCD organs to become a feasible option again.

The Maastricht classification system encompasses five possible categories by which someone can be declared dead via circulatory death: dead on arrival, unsuccessful cardiopulmonary resuscitation, awaiting cardiac arrest following withdrawal of care, cardiac arrest after brain death, and in-hospital cardiac arrest.[9] Cardiac arrest following withdrawal of care is the most controllable, given the ability to wait to withdraw care until the patient is prepared for procurement in the preoperative area or operating room, and is thus the main avenue for DCD because it's most amenable to limiting WIT. DCD is currently endorsed by the World Health Organization and recognized as legitimate by most major health institutions worldwide.[9] Importantly, current technical improvements are prolonging the time organs can be outside the body, making procurement operations more efficient and allowing for quicker transport time and grafting into the recipient.

The challenge for physicians caring for potential organ donors is first and foremost their duty to care for the donor while they remain a living patient. Focus must also be on finding an ideal approach to navigate the dying process so that the organs remain viable for transplant. Evidently, the main barrier to longevity of DCD organs is the damage caused by WIT and the best remedy is to limit how long the body is without adequate circulation. However, because of the need to have a dead patient first, the heart must completely stop beating so that death can be declared. Herein lies the potential superiority of DBD organs compared to DCD: DBD donors essentially remain on life support throughout the entire procurement process, including with medically assisted circulation, as opposed to the DCD procedure which involves an inevitable period without circulation.

Enhancing DCD

A) Shortening and standardizing the window for withdrawal of life support therapy

How long a deceased donor must be without a heartbeat to assure absence of autoresuscitation has yet to be comprehensively studied, but reports thus far approximate a maximum of 60 seconds.[10] A recent 10-year data review did discover two cases in which resumption of spontaneous circulation (ROSC) occurred between 2 to 3 minutes after the absence of a heartbeat.[11] However, even if a limited subset of data suggest the possibility of ROSC after multiple minutes of no heartbeat, consideration must be devoted to whether the waiting period should be longer than every documented instance of ROSC. Also, does anecdotal evidence outweigh the extraordinary demand for transplantable organs which could be spared from ischemic damage? That question begs consideration in the context of standardizing the waiting period even if shortening the window after withdrawal of life support therapy isn't being directly examined.

At the least, if not shortened, standardization of the waiting period after heartbeat cessation for DCD should be strongly considered. Depending on the cultural and ethical views of a particular region, it is natural for healthcare practices to exhibit some variance. However, given the interconnectedness of SOT and the high standards for graft function in recipients, adoption of consistent criteria should be emphasized. Standard practice for the procurement procedure involves the procurement team waiting in a separate operating room until the potential donor's heart stops beating. They then move into position to begin procurement once a predetermined stand-off period without a heartbeat has passed. Inherent policy differences arise during this step. At one hospital in Sioux Falls, a five-minute designated waiting period is the current practice. However, this is not standard at hospitals across the country. For example, Choubey et al. found that of 58 OPOs surveyed, 12 waited less than five minutes, 41 waited exactly five minutes, and five did not share specific hospital policies.[12] Furthermore, the ASTS recommends a two-minute stand-off period but acknowledges variation due to a lack of conclusive data.[13]

Logistically, such protocols would not be difficult to implement. The United Network for Organ Sharing (UNOS) is under contract by the US federal government to facilitate SOTs between donors and recipients, separated by thousands of miles in some cases. However, the organ allocation process does not account for possible differences in organ quality because of different DCD procurement policies. One specifically stated statute of the UNOS mission is to "bring together the organ transplant community to develop policies that make the best use of the limited supply of organs."[14] If the goal of an Organ Procurement and Transplantation Network (OPTN) is to not only provide the maximum number of transplantable organs, but also to make *the best use* of them, DCD donation policies should be consistent within the network of the OPTN. Exact parameters specifying the aforementioned stand-off window, and thus "permanent" lack of circulatory function, would serve to standardize the DCD procurement process.

B) Refined advanced directives

Of the four core principles of biomedical ethics, patient autonomy is of significant importance in this discussion. Currently, the choice to become an organ donor is ambiguous and provides little guidance on specifics such as becoming a DCD organ donor; it is possible that with education one would choose to be a DBD donor and not a DCD donor, or vice versa, along with further detailed specifications for one's own medical care during the deceased donation process. Undoubtedly, a patient's wishes must guide their medical and end-of-life care. In that light, clearer advanced directives could partly address issues with DCD donation. For example, could a patient express the desire to only have a waiting period of one minute after cessation of heartbeat, with the expressed wish being enough to justify DCD procurement of that patient's organs one minute after heartbeat cessation? Furthermore, reinforcement for lucid advanced directives benefits physicians and institutions. The ethical physician seeks to do well by their patients, and physicians left in the unclear position of declaring death using variable criteria could lose motivation to procure organs from DCD donors simply because they don't want to break rules. Normalizing detailed communication between patients, or caretakers, and physicians on wishes about being an organ donor would encourage positive progress for transplant medicine.

Pre-mortem nephrectomy

Dr. Paul Morrissey suggested that with proper informed consent to DCD donation, a single kidney could be procured before the patient is even declared dead.[15] Although not currently utilized, pre-mortem single nephrectomy prior to the death of the donor may be ethically defensible and is an interesting topic to explore. For such a procedure to be acceptable within the current framework of SOT, must it abide by the DDR? The DDR is the de facto first principle for any organ procurement policy, yet different interpretations prevail. For example, the sole difference between pre-mortem nephrectomy and currently practiced living kidney donations is really in how soon the living donor is expected to die.

Depending on one's understanding of the DDR, a different conclusion could be reached regarding pre-mortem nephrectomy. If it's interpreted that organ donors must *be dead* before organ procurement, pre-mortem nephrectomy would be unethical. However, if the DDR is framed such that procurement does not *cause* the death of the patient, pre-mortem nephrectomy would be permissible. The former statement is already widely disobeyed through the well-accepted processes of living donor kidney and liver procurements. Thus, the conception of the DDR as an ethical norm prohibiting causation of death by organ procurement is the practiced standard. Within this framework, pre-mortem nephrectomy ostensibly aligns with the DDR.

Conclusion

In the U.S. and worldwide, hopeful recipients of a SOT vastly outnumber available donors. DBD organs are a significant source of donated organs, but innovation is acutely necessary to work to lessen the deficit. Through improved organ preservation and operative techniques, DCD organs are becoming increasingly functional with quality approaching that of DBD organs. With the increase in DCD organs come new ethical questions which can only be addressed through discourse amongst the many disciplines related to organ transplantation. Many important questions lack clear, objective answers, but with further discussion on how to approach organ donation, the medical community can better seek achievement of the overarching goal of service to patients in need of organ transplantation.

REFERENCES

1. HRSA. Organ donation statistics. www.organdonor.gov/learn/organ-donation-statistics. 2024.
2. Shemie SD, Gardiner D. Circulatory arrest, brain arrest and death determination. Front. Cardiovasc. Med. 2018 Mar 13;5:15.

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:

Benjamin Limburg, MS III, University of South Dakota: Sanford School of Medicine.

Sujit Vijay Sakpal, MD, FACS, FICS, Avera Medical Group Transplant and Liver Surgery, Avera McKennan Hospital and University Health Center, Sioux Falls, South Dakota; Department of Surgery, University of South Dakota Sanford School of Medicine; Department of Internal Medicine, University of South Dakota Sanford School of Medicine.

University of South Dakota Sanford School of Medicine

Seeks Chair of Emergency Medicine

Sioux Falls, South Dakota

The University of South Dakota (USD) Sanford School of Medicine (SSOM), located in Sioux Falls, announces a nationwide search for an accomplished academic emergency medicine physician leader to serve as the Inaugural Chair, Department of Emergency Medicine. This position reports directly to the Dean of Sanford School of Medicine and Vice President for Health Affairs at the University of South Dakota, Tim Ridgway, M.D., MACP, FASGE.

Goals of the new Department will include expanding community-based faculty numbers and engagement across the state of South Dakota and strengthening educational opportunities for students interested in pursuing a career in emergency medicine. The establishment of a new department will enhance students' competitiveness for emergency medicine residency programs nationally and will expand the pool of residency-trained emergency medicine physician graduates with ties to the state who may be recruited to practices throughout South Dakota.

The Chair of the Department of Emergency Medicine for the University of South Dakota Sanford School of Medicine will be a respected board-certified academic emergency medicine physician with leadership experience in a medical school or similar environment. This individual will have demonstrated abilities as a leader, educator, clinician, and scholar. The position is based in Sioux Falls, but the chair is expected to work effectively and collegially with colleagues throughout the state, advancing the mission of the department in both rural and urban areas. SSOM is a community-based medical school with a tradition of excellence and is highly ranked in family and rural medicine.

To view the full Leadership Profile, which includes additional information about the University of South Dakota Sanford School of Medicine, or to apply, visit

bit.ly/ssom-emchair

Applications will be considered until January 7, 2026.

Please direct all nominations and expressions of interest to USD-ChairEM@agbsearch.com

UNIVERSITY OF SOUTH DAKOTA
SANFORD SCHOOL OF MEDICINE

Together in teaching, innovation and compassion

Epinephrine is Safe in Digital Anesthesia: Debunking the Myth of Finger Necrosis and Correcting Persistent Misinformation in Medical Education

Ben Van Bockern, MS III; Emily Wilde, MS III; Lacey Person, MS II; Cole D. Tessendorf, MD; and Robert E. Van Demark Jr., MD

Abstract

The application of epinephrine in digit procedures has historically been a subject of debate in medical education. Trainees are often advised to avoid its use in fingers, toes, and other terminal areas due to concerns about tissue ischemia and necrosis. However, numerous studies demonstrate that, when used at appropriate concentrations, epinephrine can be safely administered in these regions, challenging long standing misconceptions. The belief that epinephrine causes digital necrosis stems from outdated reports from the early to mid-20th century, many of which involved procaine or cocaine—anesthetics with inherent vasoconstrictive properties that may have compounded the effects of epinephrine. Additionally, procaine alone has been shown to impair tissue viability, likely contributing to observed necrosis. Some case reports of digital necrosis did not involve epinephrine at all, suggesting alternative causes. Despite this, cautious use of epinephrine in digital anesthesia has been incorporated into contemporary medical education. Many reputable textbooks, journals, and online learning resources continue to advise caution against the use of epinephrine in digits, even in light of extensive research suggesting otherwise. When used at an appropriate concentration (1:100,000) alongside an appropriate reversal agent such as phentolamine, epinephrine in the digits has been demonstrated to be safe, with very few reports of necrosis. The use of epinephrine in hand procedures aligns with current best practices and should not be viewed as controversial. Bridging the gap between evidence and education is essential for training clinicians who are confident, competent, and guided by up-to-date, evidence-based practices.

Introduction

During a busy call night, an orthopedic resident prepares to inject local anesthetic into a patient's finger. As the resident draws up lidocaine with epinephrine, a medical student asks, "I thought we weren't supposed to use epinephrine in fingers?" The resident hesitates. "That's what I always heard too, and some older literature warns against it, but I've seen it used during hand procedures." The attending physician reassures them both: "It's safe. The evidence has been clear for years."

Moments like this highlight a persistent problem in medical education: outdated dogma conflicting with modern evidence. Despite strong data supporting epinephrine's safety in digital blocks, trainees still encounter cautionary messages in textbooks and review resources—leading to hesitation and perpetuation of outdated beliefs.

For much of the 20th century, epinephrine was believed to be contraindicated in areas with end-artery blood supply—such as the fingers, toes, ears, nose, and penis—due to concerns over ischemia and necrosis. This belief stemmed from anecdotal reports using outdated anesthetics at excessive concentrations. However, the hesitation to use epinephrine in end-artery anesthetization remains deeply embedded in medical culture. The purpose of this review is to trace the historical roots of this misconception and summarize current evidence supporting the safety of epinephrine in the hand.

Pharmacology of Epinephrine and Its Role in Local Anesthesia

Epinephrine regulates cardiovascular and metabolic functions via dose-dependent adrenergic activation.[1] At higher doses epinephrine causes beta-1 and alpha-1 activation leading to increased heart rate, contractility, and vasoconstriction - useful for reducing surgical bleeding.[1,2]

When combined with local anesthetics such as lidocaine, epinephrine serves multiple functions. It prolongs anesthesia

by constricting blood vessels, slowing systemic absorption of the anesthetic, enhancing efficacy and reducing toxicity.[2,3] Epinephrine also improves hemostasis, enhances visualization, and intensifies anesthesia, increasing comfort during delicate hand procedures.[3]

Despite these benefits, hesitancy to use epinephrine in the fingers and hands persists due to misconceptions about its safety and potential to cause ischemia and necrosis.[4] However, extensive modern research has debunked this myth, demonstrating that epinephrine is safe for use in local anesthesia when administered at appropriate concentrations.[4]

Origins of the Myth: Misinterpretation of Early Studies

The longstanding belief that epinephrine causes digital necrosis stems from anecdotal reports and outdated teachings dating back to the early-to-mid 20th century. During this time, several isolated case reports linked epinephrine use in the fingers to ischemic complications, including necrosis and even amputation.[3]

Early reports were largely based on cases involving procaine or cocaine, local anesthetics with intrinsic vasoconstrictive properties. When these agents were combined with epinephrine (often in much higher concentrations than those used today), the risk of compromised perfusion was amplified.[3] In an exhaustive literature review, Thomson et al. examined all reported cases of finger infarction associated with local anesthetic use from 1880 to 2005. Out of 48 documented cases, only 21 involved epinephrine.[5] Interestingly, this means that more than half of the infarctions occurred without epinephrine at all, suggesting the involvement of confounding factors.[5] Additionally, variables contributing to necrosis in these studies consisted of overdistention of the tissue, poor surgical technique, using too much epinephrine, post- and pre-operative management, as well as the type of local anesthetic used.[3,5] Many early cases also involved procaine, an older anesthetic with a highly acidic pH that can harm tissue viability. Despite poor methodology and outdated drugs, these cases heavily influenced medical culture.

Misconceptions in Medication Training

Despite decades of evidence disproving the myth of epinephrine-induced digital necrosis, the misconception persists in clinical practice and education. Trainees encounter conflicting messages; some resources warn against its use in digits, while others highlight its safety, leading to confusion and hesitation despite clear supporting data.[5]

Many major textbooks still caution against using epinephrine in end-artery areas. Textbooks such as *Bunnell's Surgery of the Hand* (5th Ed.), *Frederick Christopher's Minor Surgery* (3rd–6th Ed.), *Schwartz's Principles of Surgery*, *Campbell's Operative Orthopaedics* (4th-8th Ed.), all editions of *Green's Operative Hand Surgery* and most notably the 54th edition of the *Physicians' Desk Reference* published in 2000.[3] The *Physicians' Desk Reference* echoed this, stating in 1973, "The use of any vasoconstrictor drug is not recommended in surgery involving the digits, nose, ears, or penis," and later advising in 1980 (through its 54th edition), that "Solutions containing a vasoconstrictor should be used cautiously and in carefully circumscribed quantities in areas of the body supplied by end arteries or having otherwise compromised blood supply."[3]

This outdated advice persists even in widely used orthopedic teaching references and educational platforms. The *Handbook of Fractures* (Egol, 5th edition) states that "epinephrine should not be used for a digital block." It gives the mnemonic advising against its use in "fingers, nose, penis, and toes."[6] Similarly, Academic Life in Emergency Medicine (ALiEMU), an e-learning platform for emergency medicine, includes content that advises providers to avoid the use of epinephrine in digital blocks due to "historical concerns."[7] These examples reflect the ongoing gap between current best evidence and commonly used educational resources, contributing to the confusion and hesitation among trainees and providers.

Modern Evidence Supporting the Safety of Epinephrine

In a 2004 prospective study of 3,110 consecutive cases of epinephrine use in the fingers and hand, there was no evidence of infarction, necrosis, or any tissue loss. Bupivacaine was used in 391 of the cases, while lidocaine was used in the rest. Doses of epinephrine were 1:100,000 or less. In addition to this combination, physicians were prepared to use the rescue agent, phentolamine, should it be required.[8]

Nodwell and Lalonde (2003) demonstrated that phentolamine effectively reverses epinephrine-induced vasoconstriction. In a randomized, blinded study, 22 subjects received lidocaine (1.8 mL, 2%) with epinephrine (1:100,000) in three sites on each finger. After one hour, one hand was injected with phentolamine and the other with saline. Fingers returned to normal in 85 minutes with phentolamine vs. 320 minutes with saline. The study confirmed phentolamine's reliability and also showed that blood flow and capillary refill were maintained throughout the study.[9]

A widely used and universally accepted use of epinephrine in

the hand and digits is during Wide Awake Local Anesthesia No Tourniquet (WALANT) procedures. This surgical technique relies on the use of lidocaine and epinephrine to perform common surgeries of the hand; the benefits of which include improved patient safety, greater access to surgical care, and enhanced intraoperative diagnosis and assessment.[10] Epinephrine is essential to WALANT, eliminating the need for a tourniquet. However, some surgeons unfamiliar with WALANT remain concerned about field visualization and the risk of finger necrosis. A survey of American Society for Surgery of the Hand (ASSH) members found that, of 869 respondents, 79% had performed at least one WALANT procedure. Of the 266 who hadn't, only 16% preferred tourniquets and 2% avoided WALANT due to epinephrine concerns.[11] The growing acceptance of WALANT further challenges outdated fears about epinephrine in the hand.

Contraindications

Despite the use of epinephrine at appropriate concentrations (1:100,000) in combination with lidocaine in the hand has been shown repeatedly to be safe, there have been few cases of necrosis that have prompted contraindications for its use.[4,12–18] Zhang et al. (2016) reported a case of digital necrosis in a 63-year-old smoker following trigger finger release. Lidocaine with epinephrine was injected into the flexor sheaths and subcutaneous tissue of three fingers. The patient presented the next day with bulla and fingertip ischemia; phentolamine was not administered due to concern that necrosis had already occurred, and two fingertips were subsequently amputated.[18]

Delayed ischemia is another adverse effect of epinephrine in combination with lidocaine in the hand. In a 2021 case by Van Demark et al., a 71-year-old man with significant vascular history underwent repair of a small finger flexor tendon and radial digital nerve. Six hours after discharge, he presented with a cold, discolored finger. He returned to the hospital where phentolamine was administered approximately 12 hours post-repair and by the following day, capillary refill was fully restored.[4] Although rare, additional contraindications such as Raynaud's or cardiovascular disease warrant caution. With proper technique and phentolamine on hand, epinephrine remains safe—even in high-risk patients.

Summary

The belief that epinephrine is unsafe in fingers has been thoroughly disproven. When used at appropriate concentrations (1:100,000) in combination with lidocaine, epinephrine has been shown to be both safe and effective. Its use in WALANT procedures has revolutionized hand surgery by improving visualization, eliminating tourniquet-related risks, and expanding access to care.

Nevertheless, this outdated concern continues to appear in textbooks, board review materials, and educational platforms, causing confusion among students, residents, and even experienced clinicians. These misconceptions drive hesitation and slow adoption of evidence-based care. Epinephrine should no longer be controversial—it is the standard of care. Bridging the gap between literature and clinical practice is essential to training confident, competent, and evidence-driven medical providers.

REFERENCES

1. Butterworth JF, Strichartz GR. Molecular mechanisms of local anesthesia: a review. Anesthesiology. 1990;72(4):711-34.
2. Lalonde D, Bell M, Benoit P, Sparkes G, Denkler K, Chang P. A multicenter prospective study of 3,110 consecutive cases of elective epinephrine use in the fingers and hand: the Dalhousie Project clinical phase. J Hand Surg. 2005;30(5):1061-7.
3. Denkler K. A comprehensive review of epinephrine in the finger: to do or not to do. Plast Reconstr Surg. 2001;108(1):114-24.
4. Van Demark RE Jr, Hewitt C, Smith VJ. Phentolamine rescue for digital ischemia following lidocaine with epinephrine local anesthesia: case presentation, review of the literature and treatment options. SD Med. 2021;74(11):532-6.
5. Thomson CJ, Lalonde DH, Denkler KA, Feicht AJ. A critical look at the evidence for and against elective epinephrine use in the finger. Plast Reconstr Surg. 2007;119(1):260-6.
6. Egol KA, Koval KJ, Zuckerman JD. Handbook of Fractures. 5th ed. Philadelphia: Lippincott Williams & Wilkins; 2015.
7. Hill B. ALiEMU CAPSULES Module 5: Procedural sedation & analgesia in the ED. ALiEM. February 10, 2016. Retrieved from www.aliem.com/aliemu-capsules-procedural-sedation-analgesia-ed/.
8. Lalonde D, Martin A. Epinephrine in local anesthesia in finger and hand surgery: the case for wide-awake anesthesia. J Am Acad Orthop Surg. 2013;21(8):443-7.
9. Nodwell T, Lalonde D. How long does it take phentolamine to reverse adrenaline-induced vasoconstriction in the finger and hand? A prospective, randomized, blinded study: the Dalhousie Project experimental phase. Can J Plast Surg. 2003;11(4):187–90.
10. Fish MJ, Bamberger HB. Wide-awake local anesthesia no tourniquet (WALANT) hand surgery. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2025. Retrieved from www.ncbi.nlm.nih.gov/books/NBK570646/.
11. Grandizio LC, Graham J, Klena JC. Current trends in WALANT surgery: A survey of American Society for Surgery of the Hand members. J Hand Surg Glob Online. 2020;2(4):186-90.
12. Zhu AF, Hood BR, Morris MS, Ozer K. Delayed-onset digital ischemia after local anesthetic with epinephrine injection requiring phentolamine reversal. J Hand Surg. 2017;42(6):479.e1-4.
13. Ravindran V, Rajendran S. Digital gangrene in a patient with primary Raynaud's phenomenon. J R Coll Physicians Edinb. 2012;42(1):24-6.
14. McNamara CT, Greyson M. Digital ischemia after lidocaine with epinephrine injection in a patient with primary Raynaud's phenomena. Case Rep Plast Surg Hand Surg. 2022;9(1):193-6.
15. Hutting KH, van Rappard JRM, Prins A, Knepper AB, Mouës-Vink CM. [Digital necrosis after local anaesthesia with epinephrine]. Ned Tijdschr Geneeskd. 2015;159:A9477.
16. Ruiter T, Harter T, Miladore N, Neafus A, Kasdan M. Finger amputation after injection with lidocaine and epinephrine. Eplasty. 2014;14:ic43.
17. Sama CB. Post-traumatic digital gangrene associated with epinephrine use in primary Raynaud's phenomenon: lesson for the future. Ethiop J Health Sci. 2016;26(4):401.
18. Zhang JX, Gray J, Lalonde DH, Carr N. Digital necrosis after lidocaine and epinephrine injection in the flexor tendon sheath without phentolamine rescue. J Hand Surg. 2017;42(2):e119-23.

About the Authors:

Ben Van Bockern, MS III, University of South Dakota Sanford School of Medicine

Emily Wilde, MS III, University of South Dakota Sanford School of Medicine

Lacey Person, MS II, University of South Dakota Sanford School of Medicine

Cole D. Tessendorf, MD, University of South Dakota Sanford School of Medicine

Robert E. Van Demark Jr., MD, Sanford Orthopedics and Sports Medicine, Sioux Falls, South Dakota; Department of Surgery, University of South Dakota Sanford School of Medicine.

PHYSICIANS
VEIN CLINICS
Nationally Recognized Vein Experts

WE CHECK ALL THE BOXES

- ☑ Office procedures keep **COSTS DOWN** for patients.
- ☑ Back to work **SAME DAY**.
- ☑ **NO** sedation procedures.
- ☑ In-office. **NO** hospital stay needed.
- ☑ We are an extension of **YOUR** practice.
- ☑ Accepts most insurances.
- ☑ Better results for **YOUR** patients.
- ☑ Certified by the American Board of Venous and Lymphatic Medicine.

HEALTHY LEGS.
Healthy Life.

North Dakota • South Dakota • Iowa
Minnesota • Illinois • Pennsylvania

888.PVC.VEIN
MidwestVeins.com

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

South Dakota Medicine

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

Impact of Direct Current Shock on Mortality and Outcomes in Patients with Pulmonary Embolism with Pre-Existing Atrial Fibrillation, Coronary Artery Disease, and Heart Failure: A National Inpatient Sample Analysis (2019–2021)

Hammad Chaudhry, MD; Logan Johnke, MD; Dawood Shehzad, MD; Dawlat Khan, MD; Mustafa Shehzad, MD; and Mamoon Ahmed, MD

Abstract

Background: Pulmonary embolism (PE) remains a major cause of cardiovascular mortality, particularly in patients with underlying atrial fibrillation (AF), coronary artery disease (CAD), and heart failure (HF). The use of direct current (DC) shock in this complex population is not well studied and may pose risks.

Objective: To evaluate the association between DC shock and in-hospital outcomes—including mortality, length of stay (LOS), and total hospital charges (TOTCHG)—in PE patients with pre-existing AF, CAD, and HF, using the National Inpatient Sample (NIS).

Methods: A retrospective analysis was conducted using 2019–2021 NIS data. Adult patients hospitalized with PE and diagnoses of AF, CAD, and HF were included. Survey-weighted logistic and linear regressions were used to assess the association between DC shock and outcomes, adjusting for demographics, comorbidities, and hospital characteristics.

Results: Among 1,152 unweighted (5.76 million weighted) patients, 14.7% received DC shock. DC shock was associated with a nearly fourfold increase in mortality (aOR = 3.90, 95% CI: 1.64–9.27, p = 0.002). DC shock recipients were younger and had lower comorbidity scores. Mortality disparities were observed among racial/ethnic minorities and older adults, with significant regional variation in DC shock use. Higher comorbidity scores were linked to increased hospital charges, but DC shock did not significantly impact LOS.

Conclusion: DC shock in PE patients with AF, CAD, and HF is associated with increased in-hospital mortality and reflects broader disparities in care by age, race, and geography. These findings highlight the need for risk-stratified treatment app

Introduction

Pulmonary embolism (PE) remains a life-threatening condition, accounting for approximately 100,000 annual deaths in the United States.[1] Its clinical complexity escalates in patients with pre-existing atrial fibrillation (AF), coronary artery disease (CAD), and heart failure (HF), who face compounded risks of thromboembolism, arrhythmia, and hemodynamic collapse.[2] AF, for instance, increases the likelihood of right atrial thrombus formation, while CAD and HF exacerbate myocardial vulnerability to ischemic stress during PE.[3-4] These overlapping pathologies create a "perfect storm" of cardiovascular instability, complicating therapeutic decisions.[5]

Direct current (DC) shock, though a cornerstone of emergent cardiac care, remains controversial in this population. While it is widely used to terminate life-threatening arrhythmias like ventricular fibrillation, its role in PE patients with multisystem cardiovascular disease is poorly defined.[6] Studies suggest that aggressive interventions like DC shock may paradoxically worsen outcomes in frail or comorbid patients, yet evidence remains fragmented.[7-9] For example, Kucher et al.[4] reported that hemodynamic instability in PE often stems from right ventricular failure, which may not respond to rhythm-focused interventions like DC shock.[5]

Compounding these clinical challenges are systemic inequities. Older adults, who constitute nearly 60% of PE-related hospitalizations, frequently face therapeutic nihilism due to ageism or unsubstantiated assumptions about frailty.[10] Similarly, racial disparities persist as demonstrated by patients identifying as a racial or ethnic minority experiencing up to 7 times higher mortality in PE compared to White patients, a gap attributed to delayed diagnosis, inequitable access to anticoagulation, and socioeconomic barriers.[11-12] Regional variations in care—such as the availability of pulmonary embolism response teams (PERTs)—further fragment outcomes.[13] Using the National Inpatient Sample (NIS), this study evaluates DC shock's association with mortality, length of stay (LOS), and costs in PE patients with AF, CAD, and HF, while probing demographic and systemic determinants of care.

Methods

Data Source and Study Population

Data obtained from the 2019–2021 NIS was analyzed for this study. The NIS is a publicly available inpatient healthcare database in the United States and is part of the Healthcare Cost and Utilization Project (HCUP), sponsored by the Agency for Healthcare Research and Quality (AHRQ). It contains detailed data on hospital inpatient stays from U.S. community hospitals, including information on diagnoses, procedures, demographics, length of stay, costs, and discharge outcomes. Since 2012, the NIS has used a 20% stratified sample of all inpatient discharges, with the discharge, not the hospital, as the sampling unit. Stratification is based on hospital characteristics including but not limited to region, teaching status, bed size, ect. Hospitals are not individually selected at random; instead, all eligible community hospitals that participate in the HCUP State Inpatient Databases are included in the sampling frame. From this broad pool, a stratified random sample of discharges is drawn to ensure national representativeness. Each sampled discharge is weighted to produce national estimates, making the NIS a valid and reliable resource for large-scale epidemiologic and health services research.[14]

Selection in this retrospective study included adults (≥18 years) with PE (*ICD-10-CM*: I26.xx) and pre-existing AF (I48.x), CAD (I25.1x), and HF (I50.x). Exclusion criteria included pregnancy, traumatic PE (*ICD-10-CM*: S26-S29), and discharges with missing mortality or cost data. The final cohort represented 1,152 unweighted patients (weighted N = 5,760,000).

Variables and Statistical Analysis

- **Exposure:** DC shock (*ICD-10-PCS*: 5A2204Z).
- **Outcomes:** In-hospital mortality, length-of-stay (LOS), and total hospital charges (TOTCHG).
- **Covariates:** Age, gender, race, ZIP income quartile, Charlson Comorbidity Index (CCI), hospital region, insurance status, and hospital size.

Survey-weighted logistic regression (mortality and DC shock) and linear regression (LOS, TOTCHG) were performed using Stata MP-Parallel Edition 18.0. Subpopulation analyses accounted for clustering, stratification, and discharge-level sampling weights.[15] Model fit was assessed using design-adjusted F-statistics and R^2 values. Statistical significance was set at $p < 0.05$.

Results

Baseline Characteristics

The study cohort consisted of 1,152 patients with pulmonary embolism (PE) and pre-existing AF, CAD, and heart failure. The mean age was 74.6 years (±12.3), with 39.1% identifying as female and 21.8% as African American (Table 1). Due to small sample sizes, individuals identifying as Asian, Pacific Islander, Native American, or other racial/ethnic groups were combined into a single category ("Other") to preserve statistical power and protect patient confidentiality. We acknowledge the heterogeneity within this group and recognize this as a limitation of our analysis.

The comorbidity burden was high, with a mean Charlson Comorbidity Index (CCI) score of 5.8 (±2.1). When stratified, 7.81% had a CCI of 1, 16.41% had a CCI of 2, 24.31% had a CCI of 3, and 51.48% had a CCI ≥4.

A total of 14.7% of patients received direct current (DC) shock during hospitalization. Patients who received DC shock were younger (mean age = 69.4 years vs. 75.8 years, $p < 0.001$) and had a lower CCI score compared to those who did not undergo DC shock.

Regarding insurance status, privately insured patients were significantly younger (mean age = 63.2 years vs. 76.8 years for Medicare patients, $p < 0.001$). The majority of patients were insured under Medicare (80.97%), followed by private insurance (10.63%), Medicaid (6.43%), and self-pay/uninsured (1.97%).

Primary and Secondary Outcomes

DC shock was associated with a 3.90-fold increased odds of mortality (95% CI: 1.64–9.27, $p = 0.002$) after adjustment

Table 1. Patient characteristics.

Total participants	1,152
Age (by decade)	
Age 40-49	50 (4.3%)
Age 50-59	112 (9.7%)
Age 60-69	256 (22.2%)
Age 70-79	392 (34.0%)
Age 80+	342 (29.7%)
Mean age	74.57 (± 12.3)
Sex	
Male	702 (60.9%)
Female	450 (39.1%)
Race	
White	818 (71.0%)
Black	251 (21.8%)
Hispanic	45 (3.9%)
Other	38 (3.3%)
Region	
Northeast	192 (16.7%)
Midwest	296 (25.7%)
South	472 (40.9%)
West	192 (16.7%)
Insurance status	
Medicare	933 (80.97%)
Medicaid	74 (6.43%)
Private	122 (10.63%)
Self-pay or uninsured	23 (1.97%)
DC shock utilization	
Received shock	82 (7.1%)
No Shock	1,070 (92.9%)
Charlson Comorbidity Index Score (CCI)	
1	90 (7.81%)
2	189 (16.41%)
3	280 (24.31%)
≥4	593 (51.48 %)
Income by quartile	
Quartile 1 (Lowest)	347 (30.14%)
Quartile 2	298 (25.89%)
Quartile 3	300 (26.06%)
Quartile 4 (Highest)	207 (17.91%)
Hospital size	
Small	246 (21.35%)
Medium	364 (31.60%)
Large	542 (47.05%)

(Table 2). Older age independently predicted mortality (aOR = 1.022 per year, $p = 0.048$) but inversely correlated with DC shock use (aOR = 0.954, $p = 0.003$), suggesting that younger patients were more likely to receive DC shock.

Racial disparities in mortality were observed, with a composite group of Native American, Asian/Pacific Islander, and other races which were evaluated together experiencing a disproportionately higher mortality (aOR = 7.23, 95% CI: 1.48 – 35.39) while the group categorized as White demonstrated statistically significant lower mortality rates (aOR = 0.28, 95% CI: 0.08 – 0.92). Additionally, hospital Region 4 (West) demonstrated significantly decreased DC shock use (aOR = 2.01×10^{-7}, $p < 0.001$).

Higher CCI scores (≥5) increased TOTCHG by 27,814 ($p = 0.035$–0.050). Privately insured patients were significantly younger ($\beta = -17.45$ years, $p < 0.001$) but showed no mortality difference ($p = 0.312$). Outcomes including hospital size and patient income quartile did not demonstrate statistically significant differences in DC shock use or mortality.

Discussion

DC Shock: A Double-Edged Sword

The near fourfold mortality risk associated with DC shock in this cohort aligns with prior studies suggesting that aggressive interventions may destabilize critically ill PE patients.[16] For instance, Chatterjee et al. (2013) reported increased stroke risk post-DC shock in patients with renal insufficiency, underscoring the hazards of procedural interventions in comorbid populations.[17] There are several proposed mechanisms that may explain the increase in mortality. First, DC shock in PE patients with AF/CAD/HF may exacerbate right ventricular strain by abruptly increasing myocardial oxygen demand, particularly in those with pre-existing ischemic cardiomyopathy.[18] It is well established that electric interventions used in treating arrhythmias have profound impacts on the myocardium, not only structurally, but also on a cellular level. These are hypothesized to be secondary to either high heat associated with DC current or cellular membrane breakdown due to electroporation injury. This injury disrupts ion and molecule homeostasis within cardiac myocardium leading to myocardial edema. This injury has been shown in animal models to impair both right and left ventricular systolic function. Additionally, left ventricular diastolic dysfunction following electrical shock has been observed.[19] Further human studies have also shown that cardiac hemodynamics following electrical shock can reduce cardiac index such that the duration and extent of

Table 2. Adjusted odds ratio for use of DC shock and mortality.

Characteristics		DC shock use (aOR)	95% confidence interval	Mortality (aOR)	95% confidence interval
Age		0.969	0.944 - 0.995	1.022	1.0 - 1.045
Sex					
	Male	0.8	0.41 - 1.56	0.75	0.47 - 1.19
	Female	1.25	0.64-2.45	1.34	0.84 - 2.15
Race					
	White	2.03	0.87 - 4.74	0.28	0.08 - 0.92
	Black	0.36	0.09 - 1.65	1.72	0.92 - 3.23
	Hispanic	0.67	0.09 - 5.06	1.32	0.39 - 4.45
	Other	7.23	1.48 - 35.39	7.76	1.94 - 31.07
Region					
	Midwest	1.08	0.3 - 3.88		
	Northeast	0.67	0.22 - 2.03		
	South	0.79	0.24 - 2.57		
	West	2.01E-07	4.84e-08 - 8.30e-07		
Insurance status					
	Medicare	0.39	0.11 - 1.29	1.15	0.30 - 3.41
	Medicaid	2.16	0.73 - 6.40	1.31	0.54 - 3.14
	Private Insurance	1.95	0.78 - 4.88	0.63	0.25 - 1.60
	Self-pay or uninsured	3.68	0.81 - 16.66	0.68	0.09 - 5.17
Charlson Comorbidity Index Score (CCI)		0.562	0.344 – 0.916		
	1	0.3	0.1 - 0.86	0.75	0.23 - 2.49
	2	0.14	0.04 - 0.53	1.21	0.42 - 3.50
	3	0.29	0.09 - 0.98	1.29	0.42 - 3.98
	≥4	0.26	0.03 - 2.17	1.21	0.28 - 5.33
Income quartile					
	Quartile 1 (Lowest)	1.18	0.43 - 3.22	1.45	0.75 - 2.80
	Quartile 2	1.01	0.34 - 3.0	0.76	0.41 - 1.42
	Quartile 3	0.74	0.22 - 2.50	0.58	0.30 - 1.13
	Quartile 4	1.03	0.25 - 4.30	0.73	0.36 - 1.47
Hospital size					
	Small	1.12	0.44 - 2.87	0.38	0.10 - 1.42
	Medium	0.38	0.10 - 1.42	1.34	0.44 - 4.08
	Large	1.11	0.39 - 3.14	1.11	0.39 - 3.14

cardiac dysfunction is proportional to the amount of energy delivered.[20]

On a cellular level, defibrillation has been demonstrated to induce several changes. Examination of myocardial tissue following electrical shocks has shown damage to mitochondrial membranes and disturbances to oxidative metabolism.[21] In addition, myocardial tissue samples show alterations in protein and DNA structure within myocardial cells and changes such as metabolic remodeling, increased oxidative stress, and increases in inflammatory markers.[22]

Alternatively, the observed association may reflect confounding by indication, as DC shock is preferentially administered to hemodynamically unstable patients with higher baseline mortality risk. However, simply dismissing the associations without further investigation would be premature. More granular and prospective data can further

Table 3. Adjusted associations between DC shock and clinical outcomes.

Outcome	Adjusted OR/β (95% CI)	p-value
In-hospital mortality	3.90 (1.64–9.27)	0.002
LOS (days)	0.83 (-0.65–2.31)	0.271
TOTCHG (USD)	8,610 (-6,210–23,429)	0.255
Age (per year)	1.022 (1.000–1.045)	0.048

help tease out the true risk versus benefit of pursuing DC shock in this patient population and how clinical practice may be influenced.

Age and Therapeutic Nihilism

Older adults' reduced likelihood of receiving DC shock raises ethical concerns. While age-related comorbidities like frailty or diastolic dysfunction may justify cautious intervention, systematic undertreatment risks perpetuating inequities.[23] Dharmarajan et al. (2018) found that older adults with acute cardiopulmonary conditions are often excluded from guideline-directed therapies, even when clinically eligible.[23] This "risk-aversion paradox"—where fear of harm leads to underuse of potentially beneficial interventions—warrants scrutiny. While clinicians may hesitate to use DC shock in older adults due to concerns about procedural risks, frailty, or reduced physiological reserve, age alone should not be the primary determinant of care decisions. Frailty scores and functional status assessments may be more appropriate tools for guiding therapy than chronological age alone. Additionally, shared decision-making tools, as advocated by the American Geriatrics Society, could help reconcile patient preferences with evidence-based care, ensuring that treatment plans align with both clinical appropriateness and individual goals.[24]

Race and Structural Inequities

The elevated mortality demonstrated in the composite group consisting of Asian, Pacific Islander, Native American, and others mirrors broader disparities in cardiovascular care. Structural and socioeconomic barriers, such as reduced healthcare access, lower insurance coverage rates, geographic disparities in hospital resources, and underrepresentation in clinical trials, likely limit optimal treatment for minority groups.[26] The concentration of lower-resourced hospitals in predominantly minority communities may further contribute to discrepancies in care, as these institutions often have fewer specialized providers and reduced access to high-cost interventions. Underrepresentation in clinical trials compounds these issues by limiting race-specific data on treatment efficacy and safety, further perpetuating inequitable care. Expanding community-based PE education programs in underserved areas could help mitigate delays in care, while broader interventions—such as increasing access to telemedicine and mobile health units, promoting minority participation in research, and implementing provider education programs to reduce implicit bias—may help bridge the gap. Future studies should investigate whether standardizing treatment protocols or expanding access to advanced therapies could improve PE outcomes and reduce racial disparities. Churchwell et al. (2020) identified racism as a root cause of these disparities, emphasizing the need for systemic reforms.[5]

Regional Variations: A Call for Standardization

Based on the results, western U.S. (Region 4) demonstrated a significantly lower utilization of DC shock compared to other regions, while other regions did not show significantly different rates in usage. This would suggest potential differences in regional practice patterns for managing PE patients with pre-existing AF, CAD, or HF and may reflect variation in hospital protocols, provider preferences, or access to specialized cardiac care. Differences in clinical decision-making could stem from institutional policies that favor pharmacologic management over procedural interventions, regional variations in provider training, or disparities in hospital resources and capabilities.

Although data from mortality rates among the regions was not available, the lower use of DC shock in the West raises important questions regarding treatment selection criteria and patient outcomes. It is possible that hospitals in this region apply stricter thresholds for performing DC shock, reserving it for select cases where its benefits are more clearly established. Alternatively, regional differences in patient severity or contraindications to DC shock could also influence these trends.

Future research should further explore the factors driving this regional variation by integrating hospital-level characteristics, patient severity indices, and provider-level decision-making patterns. Standardized national guidelines—such as the 2019 AHA/ACC/HRS AF management update—could help promote more consistent approaches to rhythm management in PE patients with underlying cardiac conditions. [3] Additionally, incorporating evidence-based risk stratification

tools within healthcare systems may aid in optimizing the selection of patients most likely to benefit from DC shock, reducing variability in its application across different regions.

Costs and Comorbidity Burden

The nonsignificant LOS prolongation with DC shock (p = 0.271) contrasts with studies linking invasive procedures to extended hospitalization. [12] However, the robust association between CCI scores and TOTCHG (p = 0.035–0.050) highlights the economic burden of multimorbidity reinforcing the idea that underlying chronic illnesses—rather than procedural interventions alone—drive healthcare costs. Patients with high comorbidity burdens may require more intensive monitoring, greater use of supportive therapies (e.g., inotropes, oxygen therapy), and increased post-discharge care, all of which contribute to rising hospitalization expenses. Optimizing comorbid conditions (e.g., HF exacerbations) through multidisciplinary care teams may mitigate costs while improving outcomes.[13]

Strengths, Limitations, and Future Directions

This study leverages the NIS's nationally representative design to address gaps in PE-comorbidity literature. Use of the NIS allows for broad generalizability across a wide array of hospital settings which may strength the external validity of the findings demonstrated. Unlike prior studies that have focused on a single variable or aspect of PE treatment, this study evaluates the impact several different comorbid conditions on the outcomes this select patient group. However, retrospective analyses cannot establish causality, and unmeasured confounders (e.g., PE severity, troponin levels) may bias results. [18] As noted above, a composite group of individuals identifying as Asian, Pacific Islander, Native American, or other racial/ethnic groups combined into a single category ("Other") may have introduced a degree of heterogenicity into the results. Future studies would benefit from identifying sample sizes within each of these racial groups better assess each separately.

There may be a degree of confounding simply by indication alone as it can be reasonably hypothesized that patients receiving a DC shock are likely sicker and at greater risk of mortality and complications. Additionally, ICD-10 coding inaccuracies and the NIS's lack of longitudinal follow-up limit granularity. Future prospective studies should integrate echocardiographic data and patient-reported outcomes to refine risk stratification.

Conclusion

DC shock is associated with increased mortality in PE patients with AF, CAD, and HF, with pronounced disparities across age, race, and region. These findings advocate for risk-stratified treatment algorithms, equity-focused quality initiatives, and cost-effective comorbidity management. As the U.S. healthcare system grapples with an aging, increasingly comorbid population, addressing these inequities is not just a clinical imperative—it is a moral one.

REFERENCES

1. Goldhaber SZ, Bounameaux H. Pulmonary embolism and deep vein thrombosis. Lancet. 2012;379(9828):1835-46.
2. Piazza G, Campia U, Hurwitz S, et al. Registry of patients with pulmonary embolism and atrial fibrillation. Am J Cardiol. 2018;122(12):2075-81.
3. January CT, Wann LS, Calkins H, et al. 2019 AHA/ACC/HRS Focused Update of the 2014 Guideline for Management of Patients With Atrial Fibrillation. J Am Coll Cardiol. 2019;74(1):104-32.
4. Kucher N, Rossi E, De Rosa M, Goldhaber SZ. Massive pulmonary embolism. Circulation. 2006;113(4):577-82.
5. Churchwell K, Elkind MSV, Benjamin IJ, et al. Call to action: structural racism as a fundamental driver of health disparities. Circulation. 2020;142(24):e454-68 .
6. HCUP National Inpatient Sample (NIS). Agency for Healthcare Research and Quality. 2021.
7. Lumley T. Analysis of complex survey samples. J Stat Softw. 2004;9(1):1-19.
8. Chatterjee S, Sardar P, Biondi-Zoccai G, Kumbhani DJ. Moderate to severe renal insufficiency and the risk of stroke with direct-current cardioversion for atrial fibrillation. Am Heart J. 2013;166(2):324-9.
9. Dharmarajan K, Strait KM, Tinetti ME, et al. Treatment for multiple acute cardiopulmonary conditions in older adults hospitalized with pneumonia. J Am Geriatr Soc. 2018;66(3):447-53.
10. Carnethon MR, Pu J, Howard G, et al. Cardiovascular health in African Americans: A scientific statement from the American Heart Association. Circulation. 2017;136(21):e393-423 .
11. Rodriguez F, Foody JM, Lopez L, et al. Young Hispanic women experience higher in-hospital mortality following acute myocardial infarction. J Am Heart Assoc. 2015;4(12):e002089 .
12. Manemann SM, Chamberlain AM, Roger VL, et al. Perceived discrimination and cardiovascular health disparities: data from the Jackson Heart Study. Ethn Dis. 2020;30(1):89-96.
13. Kabrhel C, Rosovsky R, Channick R, et al. A multidisciplinary pulmonary embolism response team: initial 30-month experience with a novel approach to delivery of care to patients with submassive and massive PE. Chest. 2016;150(2):384-93.
14. Agency for Healthcare Research and Quality. HCUP National Inpatient Sample (NIS). 2021.
15. Lumley T. Complex Surveys: A Guide to Analysis Using R. Wiley; 2010.
16. Kucher N, Goldhaber SZ. Management of massive pulmonary embolism. Circulation. 2005;112(2):e28-32 .
17. Chatterjee S, Sardar P, Mukherjee D, Lichstein E. Direct current cardioversion for atrial fibrillation in patients with left atrial thrombus. Am J Cardiol. 2013;112(6):905-9.
18. Konstantinides SV, Meyer G, Becattini C, et al. 2019 ESC Guidelines for the diagnosis and management of acute pulmonary embolism. Eur Heart J. 2020;41(4):543-603.
19. Fischer K, Becker P, Mongeon FP, Yu J, Nadeshalingam G, Friedrich MG, Guensch DP. Feature tracking strain analysis detects the onset of regional diastolic dysfunction in territories with acute myocardial injury induced by transthoracic electrical interventions. Sci Rep. 2022 Nov 14;12(1):19532.
20. Tokano T, Bach D, Chang J, Davis J, Souza JJ, Zivin A, Knight BP, Goyal R, Man KC, Morady F, Strickberger SA. Effect of ventricular shock strength on cardiac hemodynamics. J Cardiovasc Electrophysiol. 1998 Aug;9(8):791-7.
21. Schirmer U, Hemmer W, Lindner KH, Anhäupl T, Wieser T. Ultrastructural alterations in the right and left ventricular myocardium following multiple low energy endocardial countershocks in anesthetized dogs. Pacing Clin Electrophysiol. 1997 Jan;20(1 Pt 1):79-87.

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:

Hammad Chaudhry, MD, Department of Internal Medicine, University of South Dakota Sanford School of Medicine.

Logan Johnke, MD, Internal Medicine Residency Program, University of South Dakota Sanford School of Medicine.

Dawood Shehzad, MD, Internal Medicine Residency Program, University of South Dakota Sanford School of Medicine.

Dawlat Khan, MD, Department of Internal Medicine, University of South Dakota Sanford School of Medicine.

Mustafa Shehzad, MD, Internal Medicine Residency Program, Hackensack University Medical Center.

Mamoon Ahmed, MD, Department of Internal Medicine University of South Dakota Sanford School of Medicine.

Let's Be Clear:

Overdose prevention is healthcare.

Opioid use disorder is a medical condition — and it deserves the same care as any other diagnosis.

Medications like methadone, buprenorphine and naltrexone are proven to be safe and effective. They help patients find stability, prevent returns to use and save lives.

But still, stigma prevents people from accessing them. It's our job to change that.

Find resources, training and materials on MOUD at **LetsBeClearSD.com/care**

SOUTH DAKOTA,
Let's Be Clear
PREVENTION. TREATMENT. RECOVERY.

This publication was supported by the Centers for Disease Control and Prevention of the US Department of Health and Human Services as part of a financial assistance award totaling $1,865,943 with 100 percent funded by CDC/HHS and by award number TI087773; SAMHSA. The views expressed in written materials or publications and by speakers and moderators do not necessarily reflect the official policies of the CDC/HHS; nor does mention of trade names, commercial practices, or organizations imply endorsement by the U.S Government.

A Case of Idiopathic Retroperitoneal Fibrosis and Inflammatory Abdominal Aortic Aneurysm in a 73-year-old Patient

Michael Roberts, MS IV; Tate Blankespoor, MS IV; Muhammed Hasan, MD; Logan Johnke, MD; and Mamoon Ahmed, MD

Abstract

Retroperitoneal fibrosis (RPF) involves an inflammatory fibrous retroperitoneal development that surrounds the abdominal aorta. Oftentimes, extension into the iliac arteries and ureters is seen, which can cause hydronephrosis and lead to renal failure. There are a variety of causes for RPF including an idiopathic presentation, complication of infection such as syphilis or tuberculosis, or a complication of autoimmune disease. A case of idiopathic retroperitoneal fibrosis in a 73-year-old male will be examined. The patient presented with bilateral flank pain, hematuria, and low urine output that had lasted for about 4 days. Initial laboratory workup had shown increased levels of BUN at 49 mg/dL, CRP at 61.6 mg/L, and creatinine at 6.28 mg/dL. A 5.1 cm abdominal aortic aneurysm extending into the bilateral common iliac arteries was seen on CTA of the abdomen and pelvis. Surrounding the aneurysm was a soft tissue mass that had extension into the ureters bilaterally and caused hydronephrosis. Rheumatological and infectious workup of the soft tissue mass was negative. Vascular surgery performed endovascular aneurysm repair and urology performed bilateral cystoscopy and placed ureteral stents. High dose steroids were prescribed for a month. On follow up, CTA imaging showed a reduction of the mass as well as return of inflammatory markers to baseline.

Introduction

Retroperitoneal fibrosis (RPF) is a rare fibro-inflammatory disease with variable etiologies, primarily affecting the abdominal aorta. It can extend to the iliac arteries and bilateral ureters, leading to ureteral obstruction and, in severe cases, renal failure. Symptoms often include flank pain, hematuria, and low urine output, fever, nausea, and fatigue.[1] Other symptoms secondary to compression such as varicocele, hydrocele or deep vein thrombosis can be seen.[1] The estimated incidence of RPF ranges from 0.1 to 1.3 cases per 100,000 people annually, with a prevalence of 1.4 cases per 100,000.[3] Etiologies of RPF include an idiopathic presentation or a sequel to autoimmune conditions such as IgG4-related disease or infections such as tuberculosis. Out of these, the most common cause is an idiopathic presentation. Here, we present the case of a 73-year-old man who developed acute renal failure secondary to postrenal obstruction caused by idiopathic retroperitoneal fibrosis (IRPF).

Case Presentation

A 73-year-old man with a history of cerebellar degeneration with ataxia, restless leg syndrome, and tobacco use disorder presented to the emergency department with bilateral flank pain, hematuria, and low urine output for the past four days. His initial vital signs revealed elevated blood pressure. Physical examination was unremarkable, except for chronic ataxia and a global tremor. Laboratory results showed a creatinine level of 6.28 mg/dL (baseline 0.7-0.9 mg/dL), BUN 49 mg/dL (baseline 7-25 mg/dL), and CRP 61.6 mg/L (baseline ≤ 5.0 mg/L). A CTA of the abdomen and pelvis revealed a 5.1 cm abdominal aortic aneurysm extending into the bilateral common iliac arteries. The aneurysm was surrounded by a soft tissue mass extending to the bilateral ureters, causing hydronephrosis as seen in Figure 1 and 2.

Urinalysis showed a large amount of blood (3+), 100 mg/dL of protein, and >30 RBCs/hpf, while leukocyte esterase and bacteria were negative. The patient's acute renal failure was suspected to be secondary to postrenal obstruction. Urology was consulted and performed a bilateral cystoscopy with ureteral stent placement. Vascular surgery performed an endovascular aneurysm repair for the inflammatory infrarenal abdominal aortic aneurysm.

The patient underwent an extensive workup for rheumatological and infectious causes of the soft tissue

mass, including ANA, ANCA, SPEP, IgG4, and tests for histoplasmosis, hepatitis, syphilis, and TB, all of which were negative. A biopsy of the mass was advised against due to its close proximity to the aneurysm and the associated risk of rupture. The patient was eventually diagnosed with idiopathic retroperitoneal fibrosis (IRPF) and started on high-dose steroids with plans for a prolonged taper. One month after initiating steroids, repeat abdominal imaging

Figure 1. CTA abdomen pelvis showing infrarenal abdominal aortic aneurysm with a soft tissue draping over the aorta extending to bilateral common iliac arteries.

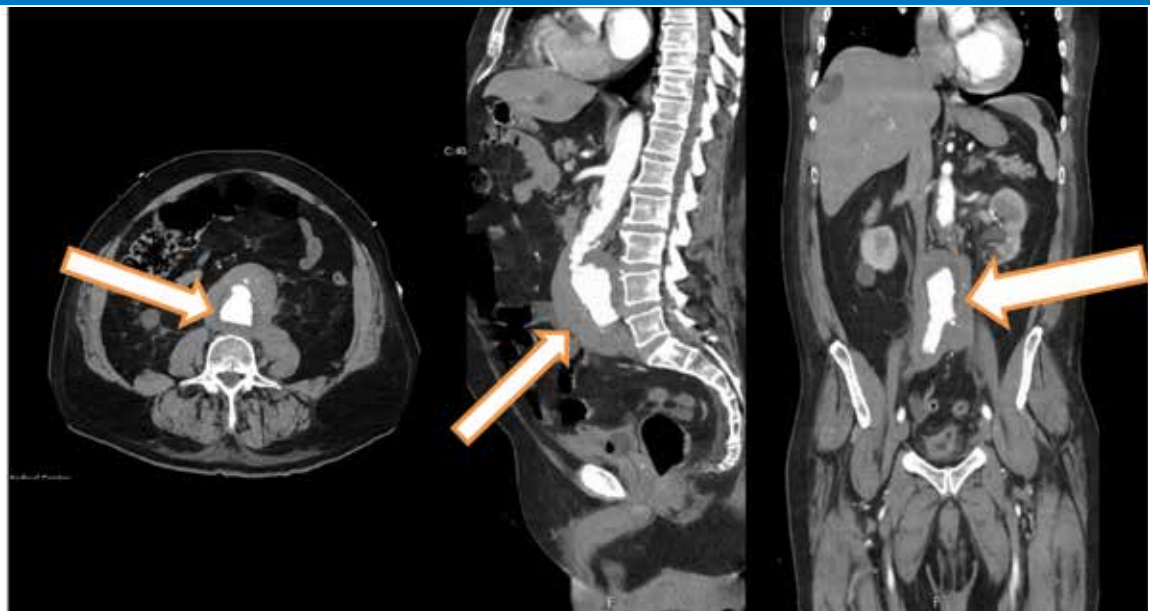
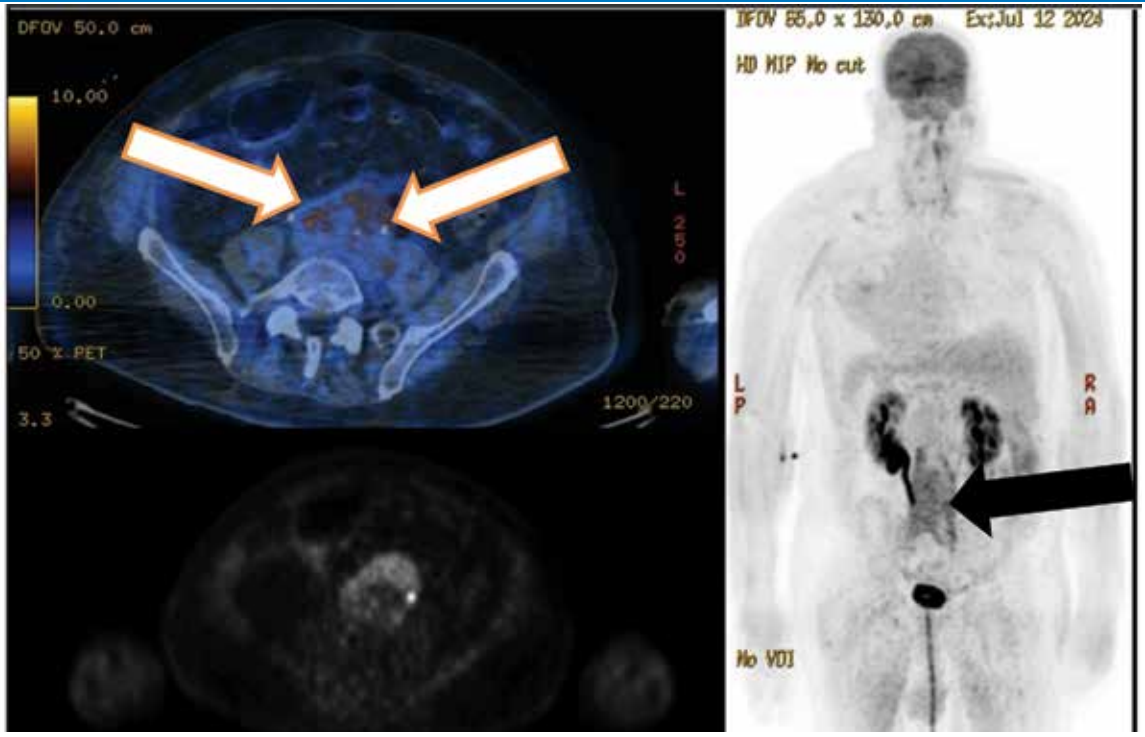


Figure 2. PET CT showing hypermetabolic soft tissue encasing the abdominal aorta and common iliac arteries.

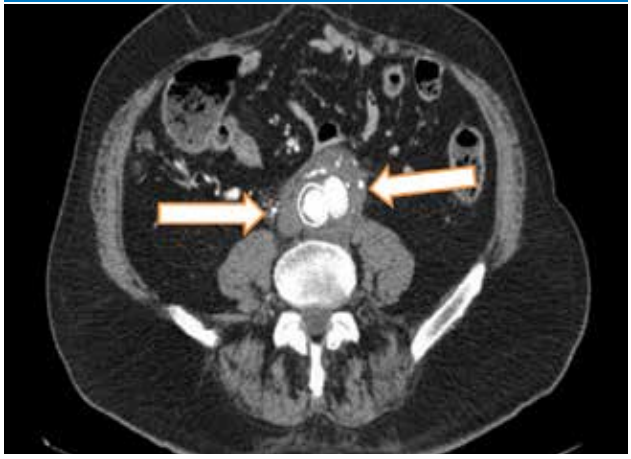


via CTA showed a reduction in the mass size as seen in Figure 3, and inflammatory markers, including CRP, had returned to normal levels.

Discussion

Retroperitoneal fibrosis (RPF) often presents as a fibrotic development in the retroperitoneum that can cause acute renal failure and ureteral obstruction. This was seen in this patient who presented with bilateral flank pain, hematuria, and low urine output and had laboratory results showing elevated creatinine, BUN, and CRP levels. The incidence of renal failure in RPF cases are present in about 42-95%

Figure 3. Repeat CTA abdomen pelvis showing aortoiliac graft in place and improved appearance of soft tissue thickening around the aorta.



of cases.[1] Other presentations can include sequelae of compression such as edema of lower extremities, varicocele, hydrocele, and deep vein thrombosis.[1] Systemic symptoms include fever, nausea, and fatigue.[1] The most common etiology is idiopathic, accounting for over 75% of cases.[1] Other causes include infections such as syphilis and tuberculosis, as well as autoimmune conditions like IgG4-related disease and vasculitis. The disease typically presents between the ages of 55 and 60 and is more common in males, with a male-to-female ratio of 2:1.[2] Pediatric cases are thought to be rare.[1] Diagnosis is usually made with the help of imaging such as ultrasonography, abdominal CT scan or MRI.[1] However, idiopathic retroperitoneal fibrosis (IRPF) is a diagnosis of exclusion to rule out underlying malignancies and infections.[1] Thus, the use of cancer screening is beneficial to aiding a diagnosis.[1] Although this patient had a soft tissue mass that surrounded his aortic aneurysm, a biopsy to rule out malignancy was not advised due to the risk of causing a rupture of the aneurysm. Biopsy may be used when imaging studies are equivocal or the patient is found to be refractory to primary therapy.[1] Although a biopsy was not performed for the presented patient, a diagnosis of RPF was supported by the imaging studies performed, negative infectious workup, and a positive response to high-dose steroid therapy.

The primary treatment for IRPF involves high-dose steroids with a prolonged taper, although there are no established guidelines for management. Some studies suggest the addition of immunosuppressive agents such as azathioprine, cyclophosphamide, or rituximab to the steroid regimen.[2] In this patient, bilateral cystoscopy with ureteral stent placement was used to help relieve the postrenal obstruction. Furthermore, the abdominal aortic aneurysm was repaired with endovascular surgery.

Conclusion

Through this case, a presentation of idiopathic retroperitoneal fibrosis was examined in a 73-year-old male. Recognition of the symptoms such as renal failure and ureteral obstruction in the context of a retroperitoneal fibrotic mass that primarily affects the abdominal aortic aneurysm is important. In the case of this patient, extension of the abdominal aortic aneurysm into the bilateral iliac arteries was seen. He also had bilateral flank pain, hematuria, and low urine output that were consistent with ureteral obstruction. Although most occurrences of IRPF are idiopathic, it can also be caused by autoimmune conditions, syphilis, and tuberculosis. Diagnosis is done with the help of ultrasonography, abdominal CT scan or MRI or tissue biopsy. The most common treatment regimen includes high dose steroids. However, the addition of immunosuppressive agents has been thought to be helpful.[2]

REFERENCES

1. Urban ML, Palmisano A, Nicastro M, Corradi D, Buzio C, Vaglio A. Idiopathic and secondary forms of retroperitoneal fibrosis: a diagnostic approach. La Revue de Médecine Interne. 2015 Jan;36(1). Retrieved from www.sciencedirect.com/science/article/pii/S0248866314006638.
2. Vaglio A, Maritati F. Idiopathic retroperitoneal fibrosis. Journal of the American Society of Nephrology 2016 Jul [cited 2025 Apr 12];27(7). Retrieved from https://pubmed.ncbi.nlm.nih.gov/26860343/.
3. Van Bommel EFH, Jansen I, Hendriksz TR, Aarnoudse ALHJ. Idiopathic retroperitoneal fibrosis: prospective evaluation of incidence and clinicoradiologic presentation. Medicine [Internet]. 2009 Jul [cited 2025 Apr 13];88(4). Retrieved from https://pubmed.ncbi.nlm.nih.gov/19593223/.

About the Authors:

Michael Roberts, MS IV, University of South Dakota Sanford School of Medicine.

Tate Blankespoor, MS IV, University of South Dakota Sanford School of Medicine.

Muhammed Hasan, MD, Internal Medicine Residency Program, University of South Dakota Sanford School of Medicine.

Logan Johnke, MD, Internal Medicine Residency Program, University of South Dakota Sanford School of Medicine.

Mamoon Ahmed, MD, Department of Internal Medicine, University of South Dakota Sanford School of Medicine.

Adult Celiac Disease: Recognition and Diagnosis for the Primary Care Provider

Kennedy Forest, MD; Rafia Irfan Waheed, MD; and Joseph Schowalter, DO

Abstract

Celiac disease is an immune-mediated enteropathy in response to dietary gluten causing malabsorption and a multitude of systemic changes. Although referral to a gastroenterologist is recommended, a primary care physician plays a crucial role in the recognition, diagnosis, and management of this condition. Systemic manifestations outside of the traditionally recognized gastrointestinal signs should prompt a primary care physician to begin the initial work up of this common gastrointestinal disease.

Introduction

Celiac disease (CD) is an autoimmune disorder that is initiated by an immune-mediated reaction to gluten, a protein present in common foods consumed around the world including wheat, barley, and rye. This immune reaction creates an inflammatory response that affects the mucosal lining of the small intestine resulting in nutrient malabsorption. CD is a multisystem disorder that can manifest in a variety of symptoms, making the diagnosis challenging even for experienced clinicians. Delayed diagnosis can lead to the development of complications such as anemia, osteoporosis, and even lymphoma.[1] This review aims to highlight the diverse presentation, diagnosis, and management of CD in the primary care setting.

Pathophysiology

The pathophysiology of CD is complex as various factors play a role including genetic, environmental, and immunological components. Most patients diagnosed with CD have either one or both human leukocyte antigens (HLA) HLA-DQ2 and HLA-DQ8. Although the penetrance of celiac disease is low, these genotypes are an important component of disease development. The most influential environmental allergen that leads to CD symptom development in a genetically susceptible individual is gluten. Other factors that further increase susceptibility include enteric virus infections, gut microbiota imbalance, surgery, pregnancy, and trauma.[2]

Gluten ingestion and breakdown lead to the release of gliadin peptides that are not completely digested in those with CD, triggering an immune response. Tissue transglutaminase (TTG) deamidates gluten peptides, which enhances their binding affinity to HLA-DQ2 or HLA-DQ8 molecules found on antigen-presenting cells. This results in the activation of CD4+ T cells in the lamina propria and the release of pro-inflammatory cytokines leading to tissue damage. Furthermore, B-cells also produce various autoantibodies, including anti-TTG, anti-gliadin, and anti-endomysial antibodies. This persistent intestinal inflammation leads to mucosal changes including villous atrophy, crypt hyperplasia, and intraepithelial lymphocytosis. These alterations to the small intestine result in impaired nutrient absorption.[2]

Clinical Presentation

CD can present with a wide range of gastrointestinal symptoms including diarrhea, abdominal pain, bloating, nausea, and vomiting. Many patients present with nonspecific symptoms including generalized fatigue, weight loss, anxiety, depression, and peripheral neuropathy. Many laboratory abnormalities are often seen in CD patients including iron deficiency anemia, vitamin D deficiency, and unexplained elevation in aminotransferase levels.[1] Conditions listed in Table 1 should cause a physician to consider CD if other clinical symptoms correlate with the disease.

Diagnostic Testing

Patients with the above-mentioned signs, symptoms, and laboratory abnormalities should be tested for CD. Additionally, the American College of Gastroenterology (ACG) recommends testing symptomatic patients with first-degree relatives with CD and even considering testing asymptomatic first-degree relatives.[1] ACG also recommends testing for CD in patients with diarrhea-predominant irritable bowel syndrome (IBS).[3]

Table 1. List of disorders which are common in patients who suffer from CD (>2 times the prevalence of general population).

Common primary care diagnoses to consider testing for CD[1]
Chronic iron deficiency anemia
Premature osteoporosis
Unexplained weight loss
Abnormal elevated liver enzymes
Dermatitis herpetiformis
Peripheral neuropathy
Oral aphthous ulcers
Thyroid disease
Irritable bowel syndrome

Initial testing for CD includes immunoglobin A (IgA) anti-tissue transglutaminase (TTG) along with total IgA levels. It is essential to test total IgA levels, as patients with IgA deficiency need additional testing including immunoglobulin G (IgG) TTG or deamidate gliadin peptide (DGP) to further investigate the diagnoses.[4] To ensure accurate diagnosis, patients need to continue gluten in their diet while the diagnostic tests are performed, as excluding gluten from the diet before testing can result in false negative results.[1] If a patient has stopped gluten consumption, gluten should be reintroduced into the diet and serological testing should be performed in three months.[4] Genetic testing for HLA-DQ2 and HLA-DQ8 can also be beneficial in these cases. If neither allele is present, CD can be essentially excluded, however, even if one allele is present, it requires a gluten challenge followed by serological testing.[1] Primary care providers should refer patients with elevated TTG-IgA, TTG-IgG, or DGP-IgG to a gastroenterologist for further evaluation and ongoing management. A gastroenterologist can perform upper endoscopy to collect multiple biopsies of the duodenum as a confirmatory test and to evaluate the extent of mucosal changes.[1]

Management

CD is a lifelong condition without a definitive cure. Although there are currently clinical trials being conducted surrounding newer therapeutic modalities, the primary management recommendation at this time is the complete avoidance of dietary gluten.[1] In addition to referral to a gastroenterologist for specialized long-term care, another important initial step is referral to a registered dietician experienced in managing gluten intolerance. A dietician can perform a comprehensive assessment of an individual patient's ability and willingness to adhere to a gluten free diet and can offer education on how to achieve this lifestyle.[5] Patients with newly diagnosed CD should be evaluated for micronutrient deficiencies including iron, folic acid, and vitamins D and B12.[1]

Although rare, a potential life-threatening manifestation of CD is celiac crisis in which a patient experiences an acute onset of severe diarrhea leading to dehydration, metabolic disturbances, and potentially renal and neurological dysfunction. This condition requires hospitalization to provide fluid resuscitation and correct electrolyte imbalances. Parenteral nutrition can be necessary due to the severe malabsorption in patients. The role of corticosteroid use in this scenario continues to be studied as currently published case studies have shown mixed outcomes.[6]

Monitoring

The unfortunate reality in the management of CD is that complying with an entirely gluten-free diet can be extremely challenging. Patients should be regularly monitored for adherence to diet and development or persistence of symptoms. Monitoring compliance with diet can be evaluated by both clinical symptoms and rechecking of IgA TTG or IgA (or IgG) DGP antibodies. It is recommended that patients are scheduled for a follow-up visit for three to six months following the initial diagnosis and initiation of appropriate dietary changes. In that time, the expected response includes progressive relief of symptoms, a decrease of antibody titers, and improvement of nutritional deficiencies. Follow-up of these parameters should be conducted at the one-year mark to evaluate for continued improvement or relapse. Most commonly TTG-IgA is monitored closely and will return to a normal value in the setting of strict gluten avoidance.

In cases where there is a relapse of symptoms or overall lack of improvement despite strict adherence to a gluten-free diet, upper endoscopy to obtain intestinal biopsies is strongly recommended if not yet performed. Commonly, the persistence of symptoms or abnormal laboratory testing is a result of non-adherence to the diet, and close follow up for diet assessment with the dietician is recommended.[1] CD unresponsive to treatment requires further evaluation by a gastroenterologist to investigate refractory CD or disorders associated with CD including microscopic colitis, pancreatic exocrine dysfunction, and enteropathy-associated lymphoma.[1]

Conclusion

CD is a chronic immune-mediated condition that can be a life-altering diagnosis for affected patients. There are common primary care conditions that may point to a diagnosis other than the typical gastrointestinal manifestations. Although

the dietary adjustments needed to achieve a gluten-free diet can be challenging, with the support and guidance of a dietitian and physicians familiar with the diagnosis of CD, patients can live a normal life with little to no lasting effects.

Takeaways

- Primary care doctors should consider testing for CD in patients with conditions seen in Table 1
- CD testing must be performed prior to the patient eliminating gluten from their diet
- It is essential to test total IgA levels when testing TTG-IgA, as patients with IgA deficiency could have a false negative result
- Patients with newly diagnosed CD should be evaluated for micronutrient deficiencies including iron, folic acid, and vitamins D and B12
- Referral to a gastroenterologist and registered dietician with experience in CD management can be helpful for long term monitoring and success of treatment

REFERENCES

1. Rubio-Tapia A, Hill ID, Semrad C, Kelly CP, Greer KB, Limketkai BN, et al. American college of gastroenterology guidelines update: Diagnosis and management of celiac disease. Am J Gastroenterol. 2023;118(1):59-76.
2. Voisine J, Abadie V. Interplay between gluten, HLA, innate and adaptive immunity orchestrates the development of coeliac disease. Front Immunol. 2021;12:674313.
3. Lacy BE, Pimentel M, Brenner DM, Chey WD, Keefer LA, Long MD, et al. ACG clinical guideline: Management of irritable bowel syndrome. Am J Gastroenterol. 2021;116(1):17-44.
4. Husby S, Murray JA, Katzka DA. AGA clinical practice update on diagnosis and monitoring of celiac disease-changing utility of serology and histologic measures: Expert review. Gastroenterology. 2019;156(4):885-9.
5. McDermid JM, Almond MA, Roberts KM, Germer EM, Geller MG, Taylor TA, Sinley RC, Handu D. Celiac disease: An academy of nutrition and dietetics evidence-based nutrition practice guideline. J Acad Nutr Diet. 2023;123(12):1793-807.
6. Soni P, Gogia P, Kundavaram R. Celiac crisis: A life-threatening complication of celiac disease. Middle East J Dig Dis. 2024;16(4):206-10.

About the Authors:

Kennedy Forest, MD, Internal Medicine Residency Program, University of South Dakota Sanford School of Medicine.

Rafia Irfan Waheed, MD, Internal Medicine Residency Program, University of South Dakota Sanford School of Medicine.

Joseph Schowalter, DO, Avera McKennan Hospital and University Health Center, Sioux Falls, South Dakota.

SDSMA PAC Membership 2025

South Dakota State Medical Association Political Action Committee

Chair's Club $1,000+

Ty Hanson, DO

Tim Ridgway, MD

Senate Club $500-$999

Tony Berg, MD

Mary Carpenter, MD

Michael Eide, MD

Mandi Greenway Bietz, MD

Matt Herber, MD

Dawn Hill, MD

Laura Hoefert, MD

Amy Kelley, MD

Terry Larsen, DO

Lucio Margallo II, MD

Stephan Miller, MD

Joy Mueller, MD

Alexander Schabauer, MD

Erica Schipper, MD

Barbara Smith

Thavam Thambi-Pillai, MD

House Club $350-$499

Scott Boyens, MD

Nielsen Burns, MD

Thomas Dean, MD

Brooke Eide, MD

Daniel Heinemann, MD

Anthony Loewen, MD

House Club $350-$499

Michael McHale, MD

Megan Smith, MD

Grace Wellman

Matthew Witte, MD

Member $200-$349

Jared Daniel, MD

William Fuller, MD

Jenny Guido, MD

Benjamin Aaker, MD

Robert Allison, MD

Mark Bubak, MD

Amy Cook, MD

Rachel Edelen, MD

Andrew Ellsworth, MD

David Elson, MD

Stephen Foley, MD

Michael Gerlach, MD

Steven Giuseffi Jr., MD

Lori Hansen, MD

Karl Heilman III, MD

Donald Knudson, MD

James MacDougall, MD

Sara Marroquin, MD

Sean McGrann, MD

Benjamin Meyerink, MD

Jacob Quail, MD

Matthew Simmons, MD

Taylor Slingsby, MD

Member $200-$349

Sarah Smith, MD

Jennifer Tinguely MD, MPH

Katherine Van Demark, MD

Keith Vollstedt, MD

Victoria Walker, MD

Jason Wickersham, MD

$50-$199

Jerome Bentz, MD

Lawrence Finney, MD

Brian Wilson, DO

Student Member $25+

Holly Goehring

Your SDSMA PAC membership is very important in candidates who understand and embrace our philosophy and vision for the future of healthcare. To contribute to SDSMA PAC, please visit sdsma.org.

This list shows contributions as of November 12, 2025

Content submitted by Children's Nebraska

Children's Nebraska Named Nebraska's First and Only Level I Pediatric Trauma Center

Children's Nebraska, the region's pediatric health care leader, has been verified as a Level I Pediatric Trauma Center by the American College of Surgeons (ACS), making it the first and only pediatric hospital in Nebraska to earn this distinction.

Level I verification is the highest designation for a trauma care center, recognizing hospitals equipped to provide the most advanced and comprehensive treatment for critically injured children.

"This is the achievement of a moonshot for Children's Nebraska and for our entire state. Becoming Nebraska's only Level I Pediatric Trauma Center reflects our unwavering commitment to providing the highest level of care to children and families when every second counts," said Patrick B. Thomas, MD, MMCi, FACS, trauma medical director. "It's a powerful testament to our community's trust and to pediatricians across the region who rely on us to deliver world-class trauma care close to home."

As a verified Level I Pediatric Trauma Center, Children's provides:

- 24/7 access to pediatric trauma specialists, including surgeons, emergency physicians, anesthesiologists, neurosurgeons, orthopedic surgeons and critical care teams.
- Child-focused facilities and equipment, designed exclusively for pediatric patients.
- Comprehensive, coordinated care spanning emergency response, surgery, rehabilitation and recovery support.
- Leadership in education, research and injury prevention, advancing pediatric trauma science and sharing best practices statewide.
- Regional collaboration, serving as a referral and resource center for hospitals throughout the region.

A Defining Moment of Teamwork

This milestone reflects the tireless efforts and collaborative spirit of the entire Children's team. From physicians, surgeons and nurses to therapists, administrative staff and support services, every individual played a crucial role in achieving this goal.

"This designation was earned through the dedication and expertise of our incredible trauma team – surgeons, nurses, respiratory therapists, emergency physicians, intensivists, interpreter services, rehabilitation specialists, social workers, child life and countless others who work tirelessly behind the scenes to ensure that every child receives coordinated, compassionate and lifesaving care," said Dr. Thomas. "Their commitment to excellence truly made this possible."

Children's Nebraska continues to lead the region in advancing pediatric trauma care, ensuring that children and families have access to the most specialized, lifesaving care right here at home.

Scan the QR code or visit ChildrensNebraska.org/Trauma to learn more about our pediatric trauma center.

Children's NEBRASKA

Member News

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

Don't Let This Be Your Last Issue!

Renew your SDSMA membership for 2026 to continue your subscription to *South Dakota Medicine* and have fees waived for submitted manuscripts. Other member benefits include:

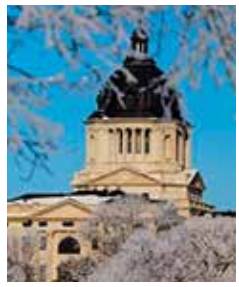
- Be represented in Pierre and Washington, D.C., with the SDSMA's strong pro-medicine advocacy agenda.
- Keep your membership in your District Medical Society to maintain your local involvement.
- Keep your members-only access to sdsma.org to read South Dakota Medicine and access other secure content.
- Have your name included in the SDSMA Member Directory at sdsma.org.

Visit sdsma.org to renew today! See a full member benefits listing at sdsma.org. Questions? Email membership@sdsma.org.

Follow us on Social Media!

Stay up to date with the latest from the South Dakota State Medical Association. We are on Facebook, Twitter, and Instagram! Follow and connect with us.

Advocacy Opportunities for Physicians and Medical Students in Pierre – 2026 Legislative Session



USD SSOM Medical Student and SDSMA Day at the Legislature

February 2-3

- February 2 – Social hour/reception at AmericInn, Fort Pierre – first-year USD SSOM medical students, SDSMA members, and legislators
- February 3 – Day at the Legislature – State Capitol Building, lobbies during floor session.

Please mark your calendar and plan to attend. More details will be provided soon.

Immunization Advocacy Day – South Dakota Families for Vaccines

February 12

- 10:30-1:00 – Speak with legislators about the importance of pro-vaccine policies and serve lunch
- 1:00-3:00 – Attend House and Senate sessions
- Sign up at www.sdfamiliesforvaccines.org/2026dayofadvocacy

SDSMA Doctor of the Day Program

The SDSMA is looking for volunteers for the association's Doctor of the Day Program at the State Capitol during the 2026 legislative session. This volunteer opportunity involves one day at the State Capitol in Pierre by providing basic medical assistance to elected representatives and staff as needed. The legislative session begins in January. Sign up to be a Doctor of the Day at sdsma.org.

Doctors of the Day have the unique opportunity to interact with legislators and get a first-hand look at the legislative process and how it affects the practice of medicine. Your presence at the Capitol shows legislators not only your expertise, but also your concern for the health of South Dakotans. It is critical that volunteer physicians are serving each day of session. Every year we receive requests from PAs and NPs who wish to participate in the program. Questions? Contact Justin Ohleen at 605.336.1965 or johleen@sdsma.org.

Copic Tips: Caring for Patients Unable to See Mental Health Specialists in a Timely Manner

There is a nationwide shortage of mental health providers and often long delays from the time of referral to the time of available consultation. As a consequence, many PCPs and other providers are trying to help these patients that they would typically expect a psychiatrist to be managing and monitoring with medications. Simultaneously, the providers do not want to inadvertently expose themselves to allegations of negligence in the event of adverse outcomes.

Fortunately, best practices for these scenarios are relatively straightforward. As with all patient care conversations, shared decision making should be appropriately documented. This serves to reduce risk of allegations that a provider is "practicing outside of their usual scope" or "should have referred to a specialist." Of course, no actions can guarantee against lawsuit risk, but the following ideas are helpful and important considerations:

Care notes should be completed contemporaneously to the patient visit. Delayed completion of notes creates a time window for an adverse outcome to occur prior to documentation. If a note does not already contain information about the delayed availability of a specialist and the clinical judgment that was exercised in making the care plan, then adding this "after the fact" when a bad outcome has occurred may lack credibility to outside reviewers.

Documentation should clearly state that your judgment was that a referral was indicated and that you made the referral. The means of communicating this to the patient should be included. If you or your staff call the specialist, then that should also be documented contemporaneously.

If an AI tool or a scribe is used for documentation of the encounter, you should read the note to ensure that it accurately reflects your awareness of the importance of the diagnosis and efforts at referral. Additionally, documentation of patient management should include any medications, testing, and follow-up plans.

Planning regular follow ups with the patient while waiting for the consultation and documenting this is helpful. This should include the follow-up schedule and the patient's understanding.

Suicide is an impulsive act and even an experienced psychiatrist has difficulty in predicting if/or when a patient will become suicidal. In a clinical scenario such as this one, it is important to perform and document appropriate suicidal ideation (SI) screening and to contract for safety with the patient where they agree that if SI occurs, they will immediately call appropriate resources, most likely you or 911.

The Substance Abuse and Mental Health Services Administration (SAMHSA) offers a printable safety plan template that includes references to the 988 system and links to helpful information for family members. Get it here: www.samhsa.gov/sites/default/files/988-safety-plan.pdf.

Fortunately, although there is some risk in caring for patients with mental health issues prior to their establishing a relationship with a psychiatrist, you are not held to an impossible standard of instantaneous consultations. To protect yourself and your patients, best practices are to clearly and contemporaneously document the situation, why you are implementing a particular plan, and maintain a regular check in and reassessment of patients waiting for a specialist consultation.

This information was provided by Copic, the endorsed carrier of the SDSMA.

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

South Dakota Medicine

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

Member News

For Your Benefit:

Membership Services from the SDSMA

SDSMA Membership Services works hard to ensure that you have the programs and services you want and need, as well as marketing the association to potential new members. We want to hear from you if you have questions, concerns or ideas on how we can serve you better, or if you know of a potential new member. It's your association and we'll work with you to make it the best it can be.

If you'd like more information about Membership Services give us a call at 605.336.1965, visit www.sdsma.org, or email membership@sdsma.org. We never take your membership in SDSMA or your input and activism for granted.

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

SDSMA Delegates Attend American Medical Association Meeting

SDSMA Delegates attended the American Medical Association Interim Meeting in National Harbor, Maryland, November 14-18. Read more about the meeting in an upcoming issue of *South Dakota Medicine*. Pictured are Mary Carpenter, MD; Robert Allison, MD; and Robert Summerer, DO.

SDSMA Board and Policy Council Meet in Ft. Pierre

The SDSMA Board of Directors and Policy Council met November 6 and 7 in Ft. Pierre, both with full agendas, and a special recognition for Dr. Mary Carpenter as she retires from serving on the SDSMA board for more than 30 years. Thank you to the physicians who volunteer their time to serve on the board and council.

Let Us Be Your Physician Advocates

SOUTH DAKOTA STATE MEDICAL ASSOCIATION

Values. Ethics. Advocacy.

ADVOCACY

Participate in opportunities to have your voice heard. The SDSMA advocates at the state and federal levels on key health care issues impacting patients and physicians.

PROFESSIONAL GROWTH

Grow leadership skills through the SDSMA Health Leadership Institute, committee involvement, collaborative efforts, and events.

EDUCATION

Access education programs on current topics and publication opportunities in the peer-reviewed medical journal *South Dakota Medicine.*

WELL-BEING

Navigate the stressors of today's ever-changing healthcare landscape and prioritize wellness with the South Dakota Physician Well-Being Program.

sdsma.org

SCAN TO LEARN MORE!

605.336.1965 • membership@sdsma.org

SOUTH DAKOTA
BOARD OF MEDICAL & OSTEOPATHIC EXAMINERS

101 N. Main Ave., Suite 301 Sioux Falls, SD 57104
Phone 605-367-7781 Fax 605-367-7786
www.sdbmoe.gov sdbmoe@state.sd.us

Shutterstock

The medical team representing the Board's regulated professions

South Dakota Board of Medical and Osteopathic Examiners

- **Protects the health and welfare of the state's citizens by ensuring that qualified medical health care professionals are licensed to practice in South Dakota**
- **Establishes regulations by proposing legislation and adopting administrative rules**
- **Provides Safe Haven, supports, and promotes confidential monitoring of its healthcare providers**
- **Licenses and regulates 14,900 licensees**

President **Vice President**


Jennifer L. Tegethoff, MD


Christopher Dietrich, MD


Brittany Azure Bearstail, MD


Scott Blanchard, DO


Maurice A. D. Chessmore, MD


Cartney Gilkerson, GC


Richard Hainje


Natalie Holt, MD


Julie Kalahar, OTR/L


Kathryn Kassin, PA-C


Gary Langerock, NRP


Tryg Odney, ATC


Clay Pavlis, MD


Heather Spies, MD


Lisa Stark, RD/LN


Rachel Sunne, MD


Marissa Trosen, RRT

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

What keeps you up at night shouldn't wait until morning.

If an unexpected outcome occurs, you need someone to talk to now. With Copic, you get a 24/7 hotline staffed by experienced physicians. Day or night, they can offer guidance and help you consider options to navigate the way forward. We're here for the humans of healthcare.

Copic is proud to be the endorsed carrier of the South Dakota State Medical Association. SDSMA members may be eligible for a 10% premium discount.

copic™

copic.com | 800.421.1834

Help Shape the Future of Medicine in South Dakota

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

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

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

Send Your Contributions Today To:
South Dakota State Medical Association Foundation
2600 W 49th St Ste 100, Sioux Falls, SD 57105
Or contribute online at www.sdsma.org

SOUTH DAKOTA
State Medical Association Foundation
Shaping the Future of Medicine in South Dakota