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Contents

June 2024
Volume 77 | Number 6

Editorial

243 Visions of Our Future: AI and Clinical Wisdom – Jerome W. Freeman, MD, FACP

President's Comments

246 Community Health Centers: Caring For Patients From All Walks of Life – Jen Tinguely, MD, MPH

The Journal

248 A Rare Multinuclear Lesion Secondary to Multifactorial Ischemic Stroke: A Case Report on Eight-and-a-Half Syndrome – Brandon Vander Zee, MD; Omar Zineldine, MD; Lien Diep-Plagie, MD, MHS, MPH

252 Variation in Consult and Referral Patterns for Facial Lacerations by Emergency Department Provider Type: A Retrospective Cohort Study – Zachary Edward Lawrence, MD; Sydney Bormann, MD; Li Cao; Hoang Lawrence Nguyen, MD

258 An Unusual Case of Takotsubo Cardiomyopathy with Cardiac Arrest: The Importance of Clinical Context in Implanted Cardioverter Defibrillator Candidacy – Jiannan Huang, MD; Muhammad Hamza Saad Shaukat, MD; Ibrahim Munaf Ahmed, MD; and Scott Pham, MD

262 A Presentation of Disseminated Nocardia Paucivorans Infection: A Case Report – Logan Johnke, MD; Sanyogita Chandra, MD; Anthony Breemo, MD

266 Suspected Pseudocholinesterase Deficiency During Left Thyroid Lobectomy and Isthmusectomy: A Case Report – Jennifer Boleyn, MD; Madison McLaury, MD; Stephanie Wieman, MD

Primers in Medicine

270 Post-Intensive Care Syndrome – An Opportunity For Collaborative Transition Between the Intensive and Primary Settings – Dawood Shehzad, MD; Mitchell Van Kalsbeek, MD; Mustafa Shehzad, MD; Mamoon Ahmed, MD

Images in Medicine

274 Raoultella planticola Urinary Tract Infection Presenting as Hyperbilirubinemia in a 3-Day-Old Infant – David Jensen, MD; Anthony Drazick, MS IV; Rochelle Boote, MD, FAAP; Peter Paul Lim, MD, FAAP

Special Features

281 Take the Survey – Why Did You Pursue a Career in Healthcare? – Fran Rice

282 SDSMA PAC Membership 2024

283 2024 Annual Leadership Conference Diamond Sponsors – Thank You!

285 Member News

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

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It's inspiring, especially working in the financial & investment industry, to see clients who love to make an impact beyond their own bottom line. To many of the clients that we're fortunate to work with, charitable giving strategies are a large part of how we assist them. One of the best strategies for charitable giving is to open and fund a Donor Advised Fund (DAF).

Many have heard of and use DAF's. DAF's could be a great solution for clients who have a strong desire to make a charitable impact through their lifetime while saving some tax along the way. DAFs can be great planning tools for tax-efficient giving.

While I'm not aware of any research to back this up, the clients I work with who have DAF's enjoy them. What are some of the things they've told me they enjoy?

- DAF's help them to be intentional with their giving. The DAF makes it easy to put their goodwill into practice, because they can automatically contribute to it on a monthly, quarterly or yearly basis.
- When they fund their DAF, those charitable dollars need a home. Several of my clients have told me how much they enjoyed researching organizations to give grants to. In fact, I just met with a client last month who shared that they were looking through the website for a specific organization. Through the search, they discovered many similar organizations.
- Having a DAF has led many of my clients to consider what they really care about. It has led to good conversations between spouses and partners about the connection between personal mission and charitable organizations.

Another advantage of a DAF is the ability to name a single donor or multiple successor donors. This is a great way to pass on the value of giving from one generation to the next. Most people are familiar with naming a beneficiary on investment accounts. With a DAF, you can name charitable organizations as beneficiaries just like with any other account, which also may make sense in your estate plan. However, you can also name successor donors. A successor donor would be an individual(s) who would "inherit" the DAF at your passing and would take over the responsibilities to find causes for those dollars to support. It allows your heirs to inherit funds that are specifically earmarked for charitable purposes, so that they can participate in finding causes to support with those funds.

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Visions of Our Future: AI and Clinical Wisdom

Jerome W. Freeman, MD, FACP

Increasingly, I have been pondering the nature of wisdom, especially as artificial intelligence (AI) becomes more prevalent in clinical medicine. The reality of AI is all around us, and its impact on clinical medicine will steadily increase. Of course, AI will influence multiple professions in society as a whole. Recently I was talking to an architect who noted that she had queried an AI site with the instruction to develop a family summer dwelling of 2,000 sq. ft. for a rural site. She was quickly provided with a paragraph proposing design features and a cost estimate.

Among physicians, conversations about the medical potential of AI are increasingly common. Already a few clinicians in our community are using AI technology to generate coherent clinic notes based on the content of the physician/ patient verbal communication with each other. In a future where AI analyzes patient data and generates sophisticated differential diagnoses, very rare conditions like stiff person syndrome or anti-NMDA receptor encephalitis may be readily identified. And instances of mystifying occult malignancy may be promptly resolved.

A 2023 JAMA essay on AI and undergraduate medical education referenced examples of AI doing better than medical students on clinical reasoning tests and often getting the correct diagnosis for unknown clinical pathological cases in the *New England Journal of Medicine*. Our students, worried about their future roles in a world of AI, may believe that the reassuring, personal presence of a medical expert can never be replaced. Data provided by Ayers et al. threaten such complacency by comparing chatGPT's response to patients' healthcare questions with the responses provided by actual physicians. Chatbox responses were rated as being significantly more empathetic.

It is reasonable for seasoned clinicians and medical students to wonder "where in the world are we headed" and "how quickly"? Of course, societal fears exist that AI of the future will eliminate jobs and greatly alter workflow in many professions. Given the expanding acceptance of AI in medicine, it seems vitally important for educators to stay abreast of AI developments and to critically ponder how physicians in the future will optimally interact with AI. Throughout recorded history, physicians and other healers have been admired for having wisdom. As our technologies advance, I think it is very appropriate to ponder the components of wisdom and why it will be important for physicians of the future to couple wisdom with the thoughtful use of AI.

A clear-cut definition and consensus about what wisdom means does not seem to exist. Mostly, we recognize wisdom when we see it. Multiple attributes of clinical wisdom might be postulated. I propose five —covenant, kindness, context, judgment, and well-being.

Covenant can be used in the realm of religious beliefs or secular relationships. In medicine, covenant connotes a promise and understanding that the foremost intention of the physician will be patients' welfare. When confronted with illness or injury, patients are vulnerable and dependent. Necessarily, clinical care operates optimally if patients trust clinicians to always act in their best interest.

A second key aspect of wisdom is **kindness.** Some may question why kind behavior warrants being considered an essential component of wisdom. From the standpoint of utility and effectiveness, kindness can enhance both the perception and reality of wisdom. Patients yearn to be treated kindly, and shower praise and respect upon physicians who are perceived as being kind. Similarly, professional colleagues are often more helpful when they are treated in a kind fashion. In other words, kindness pays dividends and a wise clinician is very aware of this. Students and clinicians who are kind are much more likely to reap the benefits of trust and collaboration. Sanford School of Medicine (SSOM) has recognized the potency of kindness and has made it the focal point of the current strategic plan for the medical school.

As one contemplates diagnostic and treatment quandaries in medicine, there is a growing understanding that the variables of **context**, the social/relational aspects of care, shape decision-making. At SSOM, the four-year ethics curriculum specifically identifies and promotes consideration of such contextual issues as emotional intelligence, disagreement,

and conflict, boundary concerns, and the inevitable uncertainty and ambiguity that accompanies clinical work.

Most of us agree that **judgment** is a crucial aspect of wisdom. Judgment is based in part on attaining sufficient knowledge and experience. An immersion into the complexities of diagnosis and treatment promotes a seasoned approach to the scientific, as well as social/behavioral, aspects of care. Learning from one's mistakes is championed as an important attribute of wisdom. Recognizing and responding to myriad ethical and value elements of clinical medicine is paramount.

To include personal **well-being** as an element of wisdom may at first seem improbable. But a complementary connection between wisdom and well-being has been noted.[3,4] A person who possesses wisdom, necessarily enjoys a professional confidence and finds joy and meaning in work. Unfortunately, many of today's students and practicing physicians worry about "burnout." SSOM colleague, Dr. Ann Cook, has previously been awarded NIH grants to study rural physician practices. Her work revealed that clinicians did not lament the scientific challenges of medicine, but struggled with ethics concerns and contextual controversies that can accompany medical practice. Often these physicians felt poorly prepared to confront such issues, and thus experienced frustration and burnout. Such dismay continues for some of today's physicians, regardless of community size or practice setting. Students take note of faculty unhappiness when they encounter it and share their observations with each other. One way to counter the negative influence of faculty discontent is to ensure that medical education candidly examines medical practice challenges and provides tools to counter inevitable dilemmas. This forthright approach with students will promote coping skills and help sustain personal well-being into the future.

Life would be simpler if wisdom could be succinctly defined and easily acquired. Medical students inevitably worry that the clinical wisdom they admire may prove difficult to attain. The elements of wisdom that I have proposed are best nurtured gradually. During medical school and residency a focus on each element is warranted. Hopefully, trainees attain confidence and competence in the specifics of wisdom, not just superficial familiarity from casually observing skilled mentors.

This essay began with the premise that AI surrounds us and will alter many aspects of clinical care, supplanting some of the traditional functions of the physician. A crucial question, of course, is whether clinical wisdom will help future clinicians successfully adapt to expanding AI capabilities. To pose the issue as a query, one might ask "what value" does clinical wisdom provide?

I firmly believe that attention to the attributes of wisdom will enable future clinicians to effectively utilize, as well as critique, potent AI capabilities. Clinicians now, and in the future, will benefit from a quiet confidence that medicine will always be personal, as well as scientific. Wisdom that is systematically acquired and nurtured can help show us the way forward. Specifics matter here. The five elements of wisdom— covenant, kindness, context, judgment, and well-being— can integrate AI capabilities into clinical medicine, while sustaining relevance and vitality in the physician/patient relationship.

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Community Health Centers: Caring For Patients From All Walks of Life

Jen Tinguely, MD, MPH
President, South Dakota State Medical Association

Greetings! My name is Jen Tinguely and I am honored to serve as your SDSMA president for the next year. I am a family practice physician and public health professional who works for the City of Sioux Falls. My clinical practice is located within Falls Community Health which is a federally qualified community health center in downtown Sioux Falls. I consider our clinic a tiny gem in the community and I am using this platform as an opportunity to share information about the work we do at our health center as well as share information about what community health centers offer across the country.

Community health centers provide primary and preventative care services to patients from all walks of life regardless of their ability to pay using a sliding fee discount scale. Often seen as "safety net providers," community health centers tend to care for patients who do not have health insurance or are underinsured. Of course, patients with insurance (both public and private) are welcome at a community health center. At our clinic, 43% of the 10,776 patients we saw in 2023 were uninsured. This was a very nice improvement from years past in which 50-51% of all our patients were uninsured. This is thanks to Medicaid expansion which took place in July 2023 and the growth of Marketplace options for patients.

Another spinoff as a "safety net provider" is that community health centers are frequently found in rural areas of the country and serve as a local healthcare resource for residents who prefer to receive their care locally versus driving to the "big city" to see their medical provider. In North and South Dakota, community health centers are located in 52 different communities and serve over 136,000 patients each year. There are community health centers located in every corner of South Dakota, including Faith (population 368) in the northwest; Martin (population 938) in the southwest; Aberdeen (population 28,495) in the northeast; and Elk Point (population 2,149) in the southeast. Rural Health Care, Inc. operates multiple community health centers in the central part of the state meaning that anywhere you look, you can probably find a community health center! In 2022, over 30.5 million patients were served by a community health center across the U.S.

Operational funding for community health centers is provided by the federal government through a base grant (Section 330 of the Public Health Service Act). While many issues in Washington D.C. are polarizing and challenging, community health center funding has thankfully had strong bipartisan support for decades. Dating all the way back to Lyndon B. Johnson's War on Poverty, community health centers have been seen as instrumental in providing quality healthcare to local residents. The first two community health centers were started in 1965 in Boston, Massachusetts, and Mound Bayou, Mississippi. The clinic in Boston served a medically underserved part of the inner city and the clinic in Mound Bayou served a very rural and very poor part of the state.

Community health centers carry a proud legacy of caring for the most vulnerable residents in a community and you will find incredibly passionate staff working in community health centers. Many centers, like ours, enjoy having a myriad of services available to patients. There are often dental clinics embedded within the health center and many health centers also provide mental health and chemical dependency services. Our clinic has all of this along with dietary services, pharmacy services (including an on-site pharmacy for patients with no insurance), social work/care coordination and community health workers. Falls Community Health is the contracted agency working with Lutheran Social Services to provide medical care for our newly arrived refugees in the Sioux Falls area. This results in a very vibrant clinic in which you'll hear multiple languages being spoken on any given day! Another unique aspect of our clinic is that our medical providers are comfortable providing medication-assisted treatment for patients who are struggling with substance use disorders. We take a lot of pride in the humanistic way we treat our patients who are working so hard towards sobriety.

Over the next few months, I plan to talk more about the issues that matter to me which include mental health challenges (for both patients and providers), substance use disorders and the stigma that patients face, and social justice in medicine. And, of course, we can anticipate topics related to the legislative session that is just around the corner! It truly is an honor to serve as your SDSMA president and I look forward to meeting you as I visit our medical districts across the state.

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A Rare Multinuclear Lesion Secondary to Multifactorial Ischemic Stroke: A Case Report on Eight-and-a-Half Syndrome

Brandon Vander Zee, MD; Omar Zineldine, MD; and Lien Diep-Plagie, MD, MHS, MPH

Abstract

Internuclear ophthalmoparesis (INO) is a horizontal eye movement disorder that is associated with a lesion at the medial longitudinal fasciculus (MLF). One-and-a-half syndrome occurs when the lesion involves the MLF and the ipsilateral abducens nuclei or the paramedian pontine reticular formation (PPRF) in the dorsomedial tegmentum of the pons. When the lesion is large enough, the fascicles of the facial nerve (CNVII) can also be involved, resulting in an ipsilateral facial nerve palsy. In combination with one-and-a-half syndrome, this condition becomes eight-and-a- half syndrome (EHS). Here, we describe a unique case of EHS in a 72-year-old male with multiple ischemic stroke risk factors who presented with INO, conjugate gaze palsy, ipsilateral facial palsy, and a transient contralateral hemiparesis. Recognizing this pattern of neurologic deficits improves localization of the lesion, prevents misdiagnosis of Bell's Palsy, and expedites proper treatment.

Background

Internuclear ophthalmoparesis (INO) is a horizontal movement disorder of the eyes associated with a lesion involving the medial longitudinal fasciculus (MLF) in the dorsomedial brainstem tegmentum of the pons or the midbrain. One-and-a-half syndrome, first reported by Fisher in 1967, occurs when a lesion results in ipsilateral conjugate horizontal gaze palsy (the "one") and ipsilateral INO (the "half").[1,2] The paramedian pontine reticular formation (PPRF) is responsible for controlling horizontal gaze movements via neurons that stimulate the abducens nerve and oculomotor nuclei. The abducens nucleus contains two distinct sets of neurons; one set innervates the ipsilateral lateral rectus muscle and the other set crosses midline via the MLF to innervate the oculomotor subnucleus that corresponds with simultaneous contralateral eye adduction.[3,4] The proximity of the PPRF and abducens nerve nucleus to the ipsilateral MLF means that a lesion in this area may impair both ipsilateral horizontal gaze and cause an ipsilateral INO, resulting in one-and-a-half syndrome.[5,6]

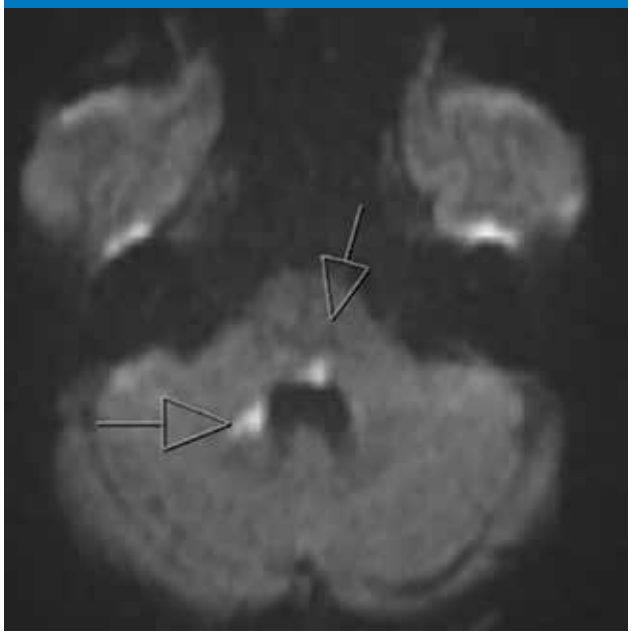
Because of the density of cranial nerve nuclei in the brainstem, it is plausible that a lesion in this area can cause a multitude of different clinical symptoms depending on the precise location of the lesion. When a dorsomedial brainstem lesion also involves the fascicle of the ipsilateral facial nerve (CN VII) at the level of the facial colliculus, this syndrome becomes eight-and-a-half syndrome (EHS), comprising all the features of one-and-a-half syndrome with the addition of an ipsilateral peripheral facial nerve palsy.[7] Here we describe a case of a 72-year-old man who presented with EHS with a transient contralateral hemiparesis.

Case

A 72-year-old male with a past medical history of hyperlipidemia, hypertension, diabetes mellitus, atrial fibrillation, chronic kidney disease and extensive tobacco use presented to the emergency department (ED) with 5 days of intractable nausea, vomiting and generalized weakness. This was accompanied by acute onset dizziness, ataxia and predominantly right lower leg weakness that developed several hours prior. The patient also possessed a left facial droop with inability to close left eye or raise left eyebrow. In addition, he was significantly hypertensive with an initial blood pressure of 217/122 that was appropriately treated. While still in the ED, the patient experienced considerable resolution of his gait instability and weakness, but his left facial paralysis persisted. The rest of his comprehensive neurological exam was unremarkable. He was not a candidate for thrombolysis with tPA, but was administered clopidogrel, aspirin, and IV heparin drip. Laboratory studies (EKG, CMP, CBC, urinalysis) were non-contributory. A Head and Neck CT angiogram obtained showed 50% stenosis of the right carotid bifurcation but was otherwise negative. A

brain MRI revealed small, acute infarcts at the left posterior pontomedullary junction and the right medial cerebellum (Figure 1).

Figure 1. MRI of the brain without contrast reveals small, acute infarcts (arrows) at the left posterior pontomedullary junction and the right medial cerebellum



The next day, the patient complained of worsening double vision and eye movement difficulty. On right gaze, the right eye abducted, but the left eye failed to adduct (Figure 2A). On left sided gaze, the right eye failed to adduct and the left eye failed to abduct (complete left conjugate gaze paralysis) (Figure 2B). Vertical eye movements were intact, but left sided facial weakness was still prominent, with inability to fully close the left eye or wrinkle the left forehead. (Figure 3). The rest of

Figure 2. In 2A, the right eye correctly abducts but the left eye fails to adduct on attempted right horizontal gaze. In 2B, the right eye fails to adduct, and the left eye fails to abduct on attempted left horizontal gaze.

Figure 3. Patient displaying inability to close left eye or raise left eyebrow in addition to loss of left nasolabial fold.

the comprehensive neurological exam was normal, including full strength in both extremities bilaterally. The patient was discharged after stabilization but had minimal resolution of his horizontal gaze and facial nerve palsies. 10 months post discharge, the patient has had some advancement in oculomotor function, with improvements in right eye adduction and left eye abduction. Double vision was still persistent with attempted right gaze but absent on left gaze. The left facial nerve palsy has shown minimal improvement, but his extremity weakness is completely resolved.

Discussion

Here, we describe a unique case of a patient with multiple ischemic stroke risk factors who presented with Internuclear ophthalmoplegia (INO), conjugate gaze palsy, ipsilateral facial palsy, and a transient contralateral hemiparesis, which correlates with a diagnoses of eight-and-a-half syndrome (EHS). INO is a phenomenon in which a person is unable to adduct their eye ipsilateral to an MLF lesion and results from brainstem lesions that involve the MLF.[2] Demyelination, infarction, and space occupying masses are among the most common causes of INO. [2,5,8] In a case series of 410 patients, Kean reported infarction (38%) was the most common cause of INO, followed by demyelination (34%), trauma (4.9%), tentorial herniation (4.9%), infection (4.1%) and tumors (4.1%). Interestingly, atypical causes made up 28% of INO cases.[9]

Conjugate horizontal gaze is the ability of the eyes to work in unison to gaze in one horizontal direction. The paramedian pontine reticular formation (PPRF) is the primary horizontal gaze center and is located in the pons.[3,10] The PPRF sends signals towards the ipsilateral abducens nerve

and contralateral MLF, and therefore, lesions affecting this region result in a horizontal conjugate gaze palsy.[3,6] Lesions that involve both the MLF and ipsilateral PPRF can lead to the neuro-ophthalmologic condition known as one-and-a-half-syndrome. C. Miller Fisher was the first to use the term to describe the combination of horizontal gaze palsy, the "one", and INO, the "half".[1] Rarely, in addition to the MLF and PPRF, the ipsilateral facial nerve fascicle may be involved resulting in an ipsilateral lower motor neuron pattern of facial nerve palsy.[7,8,11] The constellation of conjugated horizontal gaze palsy, ipsilateral internuclear ophthalmoplegia, and ipsilateral lower motor neuron cranial nerve VII palsy was termed "eight-and-a-half syndrome" (EHS) by Eggenberger.[7]

Though EHS is rare, its most common etiology is due to vascular compromise, such as ischemia or infarction at the level of the pons via occlusion of the penetrating arteries. The second most common cause is demyelination, often secondary to multiple sclerosis or neuromyelitis optica spectrum disorders.[12,13] Other reported, but even less common etiologies of eight-and-a-half syndrome include intracerebral hemorrhage,[13] vasculitis,[7] and space occupying lesions such as tumors,[14] tuberculoma,[15] capillary telangiectasia,[16] or cavernoma.[5,6,8] Overall, it is reasonable to suspect a cerebrovascular cause of eight-and-a-half syndrome when the patient is older than 45 and has multiple vasculopathic risk factors such as hypertension, diabetes, tobacco use and hyperlipidemia, as seen in this case. On the contrary, it is prudent to evaluate for a demyelinating, autoimmune process in patients younger than 45 who lack cerebrovascular disease risk factors. However, regardless of underlying risk factors, a thorough workup should be conducted that in most cases includes cerebrovascular imaging and MRI studies, CSF analysis, autoimmune seropositivity's (anti-nuclear antibodies, Anti-Ro, Anti-La, aquaporin-4, & myelin oligodendrocyte glycoprotein antibodies), inflammatory markers (CBC, ESR, CRP) and other tests pertinent to patient history. Additionally, a thrombophilia panel could be considered in cases where patients lack vasculopathic risk factors but have evidence of infarction on imaging studies.

As was seen in our case, it is possible that patients with EHS also present with contralateral hemiparesis. If this hemiparesis is transient, it is very likely the result of edema from the lesion that has extended ventrally to affect the corticospinal tract or the medial lemniscus.[17,18] If the contralateral hemiparesis persists, it is likely the lesion is directly affecting the corticospinal tract and medial lemniscus. In conjunction with the other neurological manifestations of EHS, this syndrome has been termed "nine syndrome".[19] Our patient's hemiparesis resolved quickly while in the ED, but his ophthalmologic and facial symptoms persisted, which aligned with imaging to confirm a strictly EHS presentation and not a nine syndrome.

There is a highly variable degree of patient symptom improvement or resolution following the diagnosis and possible treatment of eight-and-a-half syndrome, especially due to the rare nature of the disease and the lack of available follow up data. Based on past reviews of the literature, it is estimated that around 50% of patients receive partial resolution and 30% of patients undergo complete resolution of oculomotor symptoms.[12] At ten months follow up, our patient has had partial resolution of his oculomotor symptoms. Nonetheless, patients should continue to be monitored for improvement following stroke symptoms in addition to aggressively treating any associated risk factors.

Conclusion

Eight-and-a-half syndrome (EHS) is a rare neurological manifestation of horizontal gaze paresis, ipsilateral INO, and ipsilateral facial nerve palsy. This syndrome is associated with a lesion in the dorsomedial tegmental area of the pons and is most often secondary to cerebrovascular ischemic, demyelinating, or space-occupying lesions at this level. We presented a case of a patient possessing many vascular risk factors with a clinical presentation and MRI findings consistent with eight-and-a-half syndrome. Overall, recognizing the unique pattern of neurologic deficits in EHS improves localization of the lesion, prevents misdiagnosis of Bell's Palsy, and expedites proper treatment.

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Variation in Consult and Referral Patterns for Facial Lacerations by Emergency Department Provider Type: A Retrospective Cohort Study

Zachary Edward Lawrence, MD; Sydney Bormann, MD; Li Cao; and Hoang Lawrence Nguyen, MD

Abstract

Introduction: Facial lacerations are a common reason for emergency department (ED) visits in the U.S. Proper laceration repair is imperative as poor wound management can lead to functional and aesthetic impairment and significantly impact patient quality of life. For the best outcomes and long-term scar reduction, treatment by and follow-up with a plastic surgeon or facial trauma specialist is recommended. The present study examines variations in facial trauma specialist consultation and referral by ED provider type for adult patients at hospitals within a large rural South Dakota health system.

Methods: Records for patients above the age of 18 who received treatment for facial lacerations between January 1, 2017 and January 1, 2022 were retrospectively reviewed across multiple hospitals in South Dakota, spanning a large rural catchment area. Multivariable logistic regression and Fisher's exact test were performed to examine the relationship between ED provider type and the probability of receiving specialty consult and/or referral.

Results: One hundred fifty-four ED visits were included in the analysis. Among these patients, 53 received specialty consult and/or follow-up referral and 101 were treated without consult or referral. ED provider type was significantly associated with the probability of having a specialty consult (OR = 5.11, 95% CI [1.05, 24.96]). When the patients had a certified nurse practitioner (CNP) as their ED provider, they had a significantly higher chance (40%) of receiving specialist consultation.

Conclusion: For patients presenting to the ED with facial lacerations, facial trauma specialist consultation and referral for follow up varies based on provider type. CNPs placed specialist consultations more often than other ED provider types.

Introduction

Facial lacerations are a common reason for emergency department (ED) visits in the U.S. with approximately 2 million patients presenting annually.[1,2] Proper laceration repair is imperative, as poor wound management can lead to functional and aesthetic impairment and significantly impact patient quality of life.[3] For the best outcomes and best long term scar reduction, treatment by and follow-up with a facial trauma specialist (i.e. a plastic surgeon or oromaxillofacial surgeon) is recommended.[4-6]

Patients may prefer laceration repair by a facial trauma specialist; however, the distribution of facial trauma coverage by specialists in the ED is regionally dependent and literature shows that many facial lacerations are repaired by nonspecialists including emergency physicians and advanced practice providers.[2,7] Patterns of patient referral to specialty services vary by physician preference and variation in protocol between institutions.[2] Only 61% of emergency or trauma services at teaching hospitals in the U.S. are reported to have protocols in place for referral of patients with facial injuries and more than 70% of patients undergo repair by a nonspecialist.[5,8]

Advanced practice providers including certified nurse practitioners (CNPs) and physician assistants (PAs) play an integral role on interdisciplinary teams in the emergency department and commonly perform facial laceration repairs.[9] Data assessing facial specialist consult and referral practices for facial laceration repair in the emergency department is limited. This study aims to examine variations in facial trauma specialist consultation and referral between ED

X-ALD and Hearing

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In consultation with the South Dakota Newborn Screening Advisory Committee, the South Dakota Department of Health has added X-ALD (X-linked adrenoleukodystrophy) to South Dakota's newborn screening panel. Additionally, all babies born in South Dakota will be required to have an initial hearing screening before leaving the hospital or by one month of age.

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provider types for adult patients requiring repair of facial lacerations at hospitals within a large rural South Dakota health system.

Methods

Records for patients above the age of 18 who received treatment for facial lacerations between January 1, 2017 and January 1, 2022 were retrospectively reviewed across multiple emergency departments at hospitals within a South Dakota health system, spanning a large rural catchment area. Information from each encounter was entered into a REDCap database. Recorded patient demographics included age, sex, race, insurance status, alcohol and/or illegal substance intoxication at time of presentation, and discharge disposition. Age groups consisted of age less than or equal to 49, age 50-69, and age greater than or equal to 70. Races were categorized as African American/Black, American Indian or Alaskan Native, White, and all other races. Insurance status was grouped as commercial, Medicaid, Medicare, self-pay, worker's compensation, and all other payers. ED provider types were recorded as CNP, PA, or physician. Laceration repair and/or other procedures performed were recorded. Consultation in the ED and follow-up referral to a facial trauma specialist was recorded.

Propensity score matching was performed via R package "MatchIt" to stratify the sample into groups based on age, gender, race, and insurance type. Multivariable logistic regression and Fisher's Exact Test were performed via SPSS v 25 to examine the relationship between ED provider type (physician, CNP, PA) and the probability of having specialty consult, referral for follow up appointment, both a consult and referral, or no consult or referral.

Results

Out of 474 records reviewed, 154 records were randomly selected and included in the analysis (n = 154), and patient demographics are shown in Table 1. ED provider type was a CNP in 15 (9.7%) of cases, PA in 26 (16.9%) of cases, and physician in 113 (73.4%) of cases. Each PA was required to confirm the plan of care with a physician prior to patient discharge, though no CNPs were required to. Laceration repairs were completed for 131 (85.1%) patients. Twenty-five patients (16.2%) received a specialty consult with a facial trauma specialist. Eight patients (5.2%) received a specialty consult without follow-up referral, 17 (11.0%) received a specialty consult and follow-up referral, 28 (18.2%) received no specialty consult but were referred for follow-up, and 101 (65.6%) had no consult or follow-up referral.

Table 1. Patient demographics.

		Mean	Std. deviation
Age		44.38	21.4
		N	Percent
Age group	<= 49	97	63
	50-69	29	18.8
	>= 70	28	18.2
Sex	Female	51	33
	Male	103	67
Race	African American/Black	11	7.1
	White	125	81.2
	American Indian or Alaskan Native	16	10.4
	All other races	2	1.3
Insurance	Commercial	63	40.9
	Workers' comp	7	4.5
	Medicaid	14	9.1
	Medicare	35	22.7
	Self pay	31	20.1
	All other payers	4	2.6
Drug and/or alcohol use	No	124	80.5
	Yes	30	19.5

ED provider type was significantly related to patients receiving consults and follow-up referrals (OR = 5.11, 95% CI [1.05, 24.96]). When patients had a CNP as their ED provider, they had a 40% chance of receiving specialty consult. Patients with a PA as their provider had a 12% chance of receiving a consult and patients with a physician as their provider had a 14% chance. Patients initially seen by a CNP were 2.9 and 3.3 times more likely to receive specialty consult than those seen by a physician or PA, respectively.

ED provider type was also significantly related to a patient receiving both a consult in addition to a follow up referral (p=0.01); patients with CNPs as their provider had more consults only and those seen by physicians had more consult and follow up referrals than the other two provider types. Age, gender, race, and insurance status were not associated with having a specialty consult or follow-up referral.

Discussion

Facial injuries resulting in long-term deformity and scarring are empirically known to cause psychological detriment due to the functional and social importance of the face.[11,12] The face plays a key role in an individual's identity and is central to both self-image and perception by others. Increased scar size is associated with significantly higher ratings of self-

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consciousness and anxiety.[12] Patients may desire laceration repair from a plastic surgeon or facial trauma specialist; however, many emergency departments do not have protocols in place for specialty consultation when treating facial lacerations.[5] There is limited data assessing patient and emergency department-related factors that influence if a patient will receive consultation or referral for follow-up with a plastic surgeon or facial trauma specialist.

To our knowledge, there have been no previous studies assessing the variation in specialty consultation or referral by provider type. The present study determined that ED provider type is significantly associated with patients receiving consultation with a facial trauma specialist. Patients initially seen by CNPs were significantly more likely to receive specialty consultation. Advanced practice providers, including CNPs and PAs play a key role in successful interdisciplinary health care within the ED and much of their procedural skills are developed through on-the-job training.[9] Interestingly, patients initially seen by CNPs were more likely to receive specialty consultation compared to those initially seen by PAs. The exact cause of this is unclear, though procedural training requirements have been shown to vary between CNPs and PAs based on institution and geographic location, making comparison difficult.[9] It is plausible that the number of consults by PAs may have been more influenced by supervising physicians compared to CNPs, as CNPs in South Dakota have full practice authority and are not required to confirm their plan of care with another provider.[10]

In this study, the likelihood of specialty consult or referral when treating patients with facial lacerations was statistically unrelated to age, race, insurance status, or substance intoxication. These results differ from the literature which has demonstrated that uninsured patients receive less care than privately insured patients in the ED, even when they have similar diagnoses.[13] In contrast to our data, a study assessing how insurance type affects pediatric facial laceration management in the ED showed that those with public insurance received fewer subspecialty consultations for facial lacerations.[14]

Research assessing outcomes of facial laceration repair by specific provider type is limited. To our knowledge, this is the first study comparing consultation and referral patterns of different types of ED providers when managing adult patients with facial lacerations. Though we found a difference in consultation and referral frequency based on provider type, further research is needed to elucidate the factors responsible for this difference.

Limitations exist within this study. We were unable to account for the severity of lacerations or other injuries which may impact the likelihood of receiving specialist consultation or referral. Additionally, results of our study may not be generalizable due to the study setting of EDs within a rural South Dakota health system.

In summary, this study concludes that the likelihood of facial trauma specialist consult and referral for patients presenting to the ED with facial lacerations varies based on provider type. Future research is needed to determine the impact this variation has on patient outcomes. Furthermore, the results of this study prompt further exploration of training variation between CNPs, PAs, and physicians when it comes to the management of facial lacerations.

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An Unusual Case of Takotsubo Cardiomyopathy with Cardiac Arrest: The Importance of Clinical Context in Implanted Cardioverter Defibrillator Candidacy

Jiannan Huang, MD; Muhammad Hamza Saad Shaukat, MD; Ibrahim Munaf Ahmed, MD; and Scott Pham, MD

Abstract

Takotsubo syndrome (TTS), also known as stress-induced cardiomyopathy, is characterized by acute heart failure, reversible left ventricular dysfunction, and other complications such as life-threatening arrhythmias. The management of TTS is challenging due to its unpredictable clinical course and the lack of evidence-based treatment recommendations. In this case report, we present a 71-year-old female who developed TTS with ventricular tachycardia (VT) cardiac arrest following septic shock and an exploratory laparotomy for appendicitis. Despite the presence of VT cardiac arrest and a left ventricular ejection fraction of 30-35%, an implanted cardioverter-defibrillator (ICD) was not indicated due to the rapid and satisfactory recovery of the patient's ventricular function. This case highlights the importance of considering the clinical context and the transient nature of TTS in the decision-making process for ICD candidacy.

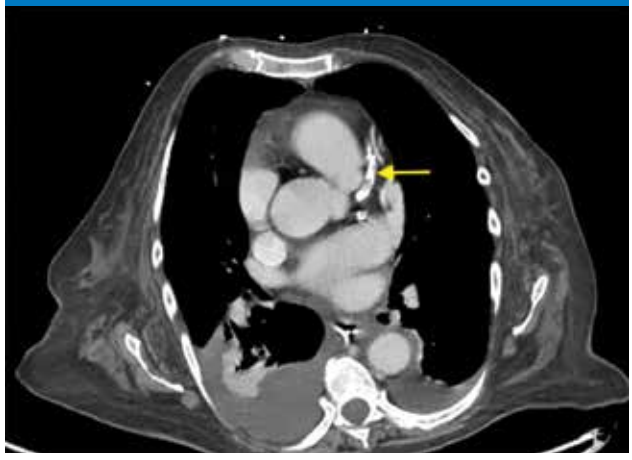
Introduction

Implanted cardioverter defibrillator (ICD) is commonly recommended for primary and secondary prevention of sudden cardiac death (SCD). However, clinical context should be considered while deciding ICD candidacy. We report an unusual scenario when ICD was not indicated in a patient with Takotsubo syndrome (TTS) despite a left ventricular ejection fraction (LVEF) ≤ 35% and ventricular tachycardia (VT) cardiac arrest history.

Case Presentation

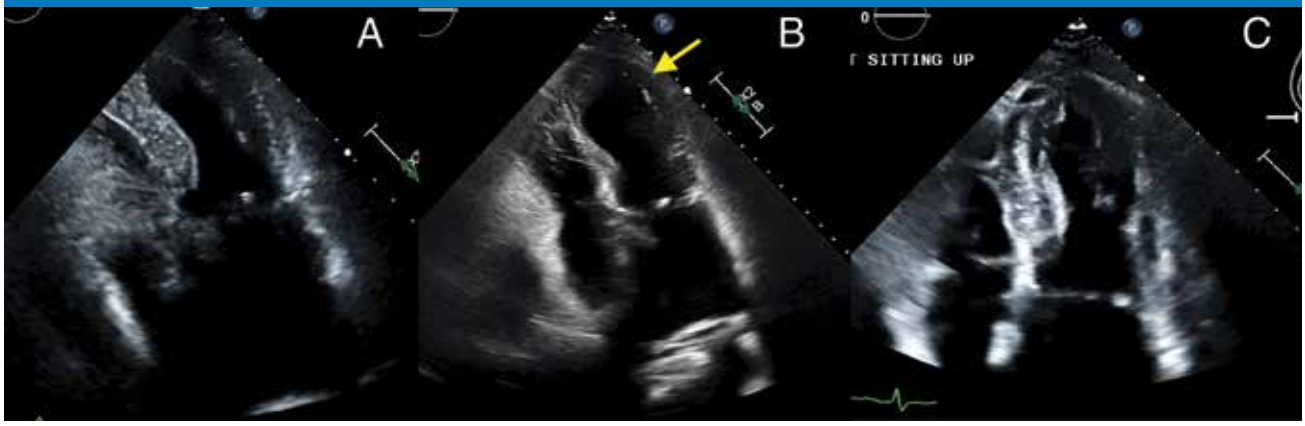
A 71-year-old female presented to our hospital with profound diarrhea and abdominal pain for several days. Her past medical history included hypertension, hyperlipidemia, alcohol use disorder, and psychiatry comorbidities, including panic attack history and bipolar disorder. She had no known cardiac conditions. Upon admission, her initial vital signs were significant for blood pressure of 83/57mmHg, and the physical exam was nonrevealing. Laboratory workup showed leukocytosis, anemia, and acute creatinine elevation. An abdomen and pelvis computed tomography (CT) showed probable appendicitis with features suggesting possible perforation. Notably, incidental findings of coronary artery calcifications were reported (Figure 1). She was admitted to the intensive care unit for management of septic shock and later underwent an exploratory laparotomy with appendicectomy. Her echocardiogram prior to the surgery revealed LVEF of 60 to 65% without valve vegetations or wall motion abnormality (Figure 2A).

Figure 1. Computed tomography scan of the abdomen and pelvis, including the lower part of chest. Heavily calcified left anterior descending artery was demonstrated (arrow).



Three days following the surgery, the patient had a monomorphic ventricular tachycardia cardiac arrest. Return of spontaneous circulation (ROSC) was achieved after 2 minutes of cardiopulmonary resuscitation without electronic defibrillation or epinephrine administration. Emergent laboratory workup revealed potassium of 2.7 meq/L (3.5-

Figure 2. End-systolic four chamber view of transthoracic echocardiograms. (A) Echocardiogram on hospitalization day 2 showed normal left ventricular systolic function. (B) Apical ballooning (arrow) was found on an echocardiogram on hospitalization day 5. The midsection and apex of the left ventricle ballooned out, while the area near the cardiac base contracted normally. This abnormal left ventricular shape during systole resembles a Japanese octopus trap. (C) Echocardiogram on hospitalization day 13 showed recovered left ventricular systolic function with a largely normal contraction contour.

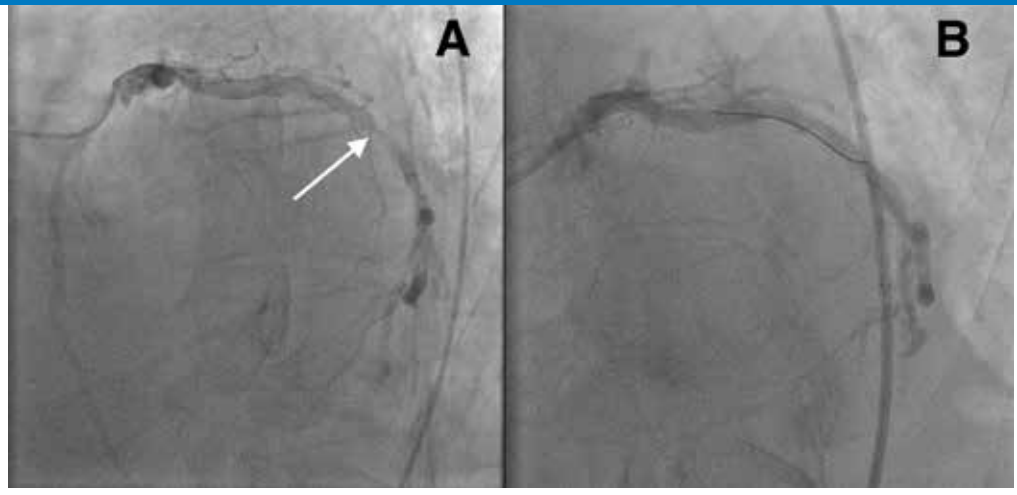


5.1 meq/L) and magnesium of 1.8 mg/dL (1.6-2.6 mg/dL), which were repleted, as well as elevated troponin of 0.664 ng/mL (0-0.013 ng/mL), BNP of 3338 pg/mL (0-100 pg/mL). An electrocardiogram (ECG) showed transient ST-segment elevations and T wave inversions in leads V1-3 and a normal QTc. An acute coronary syndrome (ACS) was suspected. The patient was started on amiodarone drip for ventricular tachycardia and heparin drip for suspected ACS.

After the patient was hemodynamically stabilized, a repeat echocardiogram was performed showing a reduced LVEF of 30 to 35% with apical ballooning (Figure 2B), consistent with stress-induced cardiomyopathy, or TTS. To evaluate potential underlying ischemic cardiac disease, a subsequent diagnostic left heart catheterization was performed, revealing severe branch disease (Figure 3A). A percutaneous coronary intervention was planned as the patient gradually recovered from her underlying infectious disease with appropriate treatment.

Ten days after the VT cardiac arrest, during the same hospitalization, the patient was clinically stable with no evidence of residual infection. Repeat echocardiogram showed significant LVEF recovery to 55-60%, and apical ballooning was no longer observed (Figure 2C). These findings indicated a rapid and satisfactory recovery from TTS. Together with her clinical improvement without further arrhythmia recorded on telemetry, an ICD was not implanted. She underwent a percutaneous coronary intervention (PCI). A drug-eluting stent was successfully deployed to her obtuse

Figure 3. Left heart catheterization films. (A) Coronary angiogram showed stenosis (arrow) at the middle segment of the obtuse marginal branch (OM). (B) Patent stent with improved blood perfusion to the OM.



marginal artery (Figure 3B). The patient was discharged to a skilled-nursing facility after a 37-day hospital stay. At a four-month follow-up, she remained in the facility for continued physical rehabilitation. Her exercise tolerance was limited by osteoarthritis but she denied cardiac symptoms.

Discussion

Takotsubo syndrome, or stress-induced cardiomyopathy, is an acute, nonischemic heart failure syndrome characterized by reversible left ventricular dysfunction. While the exact pathophysiology remains elusive, hypotheses exist involving dysregulation of the central autonomic nervous system, ischemia-mediated stunning due to multivessel epicardial or microvascular spasm, and circulating catecholamine surge.[1] Postmenopause,[2] female sex category, and pre-existing mental illness,[3] as was presented in our patient, predispose an individual to this stress-related pathology. Emotional triggers can proceed to TTS, giving this disease entity other names such as "broken heart syndrome." However, physical stressors such as critical illness, surgery, medications, drug intoxication or withdrawal can also be the cause.[1]

The diagnosis of TTS often requires coronary angiography with left ventriculography to exclude acute myocardial infarction due to the clinical resemblance. Notably, 10-29% of TTS patients have concomitant CAD.[3] Therefore, different diagnostic criteria have acknowledged the presence of CAD not being an exclusion criterion.[3,4] A definite diagnosis requires confirmation of the reversible nature of the condition. However, some clinical features are highly predictive of stress cardiomyopathy (Table 1).[3] The pathognomonic echocardiographic finding is ballooning of the left ventricular wall, resembling the shape of the Japanese Octopus trap.[5] Apical ballooning constitutes 75-80% of all TTS cases.[1]

Table 1. 2018 InterTAK diagnostic criteria of Takotsubo syndrome.[3]

1	Patients show transient[a] left ventricular dysfunction (hypokinesia, akinesia, or dyskinesia) presenting as apical ballooning or midventricular, basal, or focal wall motion abnormalities. Right ventricular involvement can be present. Besides these regional wall motion patterns, transitions between all types can exist. The regional wall motion abnormality usually extends beyond a single epicardial vascular distribution; however, rare cases can exist where the regional wall motion abnormality is present in the subtended myocardial territory of a single coronary artery (focal TTS).[b]
2	An emotional, physical, or combined trigger can precede the takotsubo syndrome event, but this is not obligatory.
3	Neurologic disorders (e.g. subarachnoid hemorrhage, stroke/transient ischemic attack, or seizures) as well as pheochromocytoma may serve as triggers for takotsubo syndrome.
4	New ECG abnormalities are present (ST-segment elevation, ST-segment depression, T-wave inversion, and QTc prolongation); however, rare cases exist without any ECG changes.
5	Levels of cardiac biomarkers (troponin and creatine kinase) are moderately elevated in most cases; significant elevation of brain natriuretic peptide is common.
6	Significant coronary artery disease is not a contradiction in takotsubo syndrome.
7	Patients have no evidence of infectious myocarditis.[b]
8	Postmenopausal women are predominantly affected.

[a]Wall motion abnormalities may remain for a prolonged period of time or documentation of recovery may not be possible. For example, death before evidence of recovery is captured.
[b]Cardiac magnetic resonance imaging is recommended to exclude infectious myocarditis and diagnosis confirmation of takotsubo syndrome.

The management of TTS mainly involves close monitoring, identifying and correcting the underlying triggers, and treating its complications, such as hypotension, cardiogenic shock, arrhythmias, and thromboembolism. There is no prospective randomized clinical trial available up to this day. It is imperative to remember the self-limiting nature of TTS and apply the principle of "do no harm".[6] Cautious decision on permanent device implantation is a good example.

ICD is a widely used implantable device in preventing SCD in systolic heart failure (HF) and ventricular arrhythmias, both of which are common in the acute phase of TTS. Up to 45% of TTS patients have reduced LVEF.[6] Life-threatening arrhythmias including VT, ventricular fibrillation (VF), asystole, and complete atrioventricular block occur in 13.5% of TTS patients.[7] From the HF management perspective, the 2022 AHA/ACC/HRS guidelines recommended ICD therapy for primary SCD prevention in heart failure with reduced ejection fraction (HFrEF) patients with a LVEF≤35% and other selected characteristics (New York Heart Association (NYHA) class II or III symptoms on chronic guideline-directed medical therapy, reasonable expectation of meaningful survival for more than one year).[8] In the approach to survivors of SCD due to VT or VF, ICD is also recommended for secondary SCD prevention in those anticipated to have meaningful survival greater than one year with or without ischemic heart disease.[9] Reduced all-cause mortality was proven for primary and secondary SCD prevention in the abovementioned conditions.

Notwithstanding the common ICD indications in arrhythmia and HF management, the uniqueness of its transient nature in TTS should be taken into consideration. A retrospective study of prospectively collected data conducted by Stiermaier et al. analyzed the permanent device use in 286 consecutive TTS patients.[7] 25 patients in this study suffered from life-threatening ventricular arrhythmias, among whom 3 died during the hospital stay; One received an ICD

after resuscitation due to VF, and all of the remaining 21 patients had no ventricular arrhythmias on follow-up. These data support a conservative approach to TTS-related ventricular arrhythmia management without permanent ICD implantation.

Conclusion

Takotsubo cardiomyopathy is an acute but reversible left heart failure syndrome. In contrast to the early belief that it represents a benign syndrome due to its self-limiting clinical course, severe complications and mortality are not infrequent, including acute HFrEF and ventricular arrhythmias with cardiac arrest. Traditional indications of ICD do not necessarily apply in the acute phase of TTS.

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A Presentation of Disseminated *Nocardia Paucivorans* Infection: A Case Report

Logan Johnke, MD; Sanyogita Chandra, MD; and Anthony Breemo, MD

Abstract

Nocardia paucivorans is a rarely cultured bacteria most commonly found in immunocompromised hosts, and rarely, may result in dissemination across multiple organ systems. Infection and subsequent clinical manifestations are often vague and nonspecific, making timely diagnosis difficult. Due to the infrequency of *N. paucivorans* infection, no consensus treatment has yet been established. We report a case of an immunocompromised patient with disseminated nocardiosis with infective endocarditis and other extrapulmonary involvement.

Introduction

Nocardia is a ubiquitous environmental bacteria found in soil flora worldwide. It is an aerobic, gram-positive bacteria that has been found to cause both localized and systemic infections in humans. It appears as branching, filamentous rods that stain weakly acid-fast. Although over 80 different species of Nocardia have been identified, only half are considered pathogenic.[1] *N. paucivorans* is a recently discovered species of the Nocardia family and shares similar characteristics to other pathogenic Nocardial species.[2] The primary route of transmission is through inhalation of aerosolized bacteria found in soil, although direct inoculation through trauma has been reported.[1]

Pathogenesis is not fully understood due to the complexity of the cellular mechanisms virulent *Nocardia* strains utilize to infect host cells. Macrophages in immunocompetent individuals readily eliminate most *Nocardia* strains; however, the rate of elimination by oxygen-independent or oxygen-dependent mechanisms is considerably slower than the rate of organisms such as *Staphylococcus aureus*.[3] Particularly virulent strains of *Nocardia* have been found to be resistant to oxidative killing mechanisms, thought to be due in part by the high levels of catalase activity within virulent *Nocardia* strains. Host T lymphocytes are important mediators in the defense against *Nocardia* infection. While the role of T cells in nocardial infection is primarily the activation of macrophages and cellular immune response, early studies in mice suggest that T lymphocytes are directly involved in killing certain strains of *Nocardia*, due to the similar structure of *Nocardia* cell wall and T-lymphocyte membranes which allows anti-Thy-1.2 plus complement to degrade the cell wall and kill *Nocardia*.[3] As a result of *Nocardia*'s unique mechanisms to evade the host, immunocompromised individuals with deficiencies in macrophage activity or T-lymphocyte function are at a much greater risk of local and disseminated *Nocardia* infections.[3]

While *Nocardia* is most commonly found in immunocompromised patients, a limited number of cases have been described in immunocompetent individuals.[4] Local soft tissue infections are most associated with immunocompetent individuals while immunocompromised hosts often present with more severe disseminated infection. Among immunocompromised hosts, those with a history of solid organ transplant are most at risk of nocardiosis.[3] According to a study conducted by Velidakis et al, nocardial infective endocarditis (IE) was associated with mortality rates as high as 26.9%.[5] We present a case of a 76-year-old immunocompromised male with a diagnosis of *N. paucivorans* IE with dissemination to the CNS and soft tissue.

Case Report

A 76-year-old Caucasian male with a history of aplastic anemia on chronic immunosuppressive therapy was admitted to the hospital for significant weakness, hypotension, dyspnea and persistent fevers. Infectious disease and pulmonology had evaluated the patient two months prior for persistent dyspnea. Computerized tomography (CT) imaging of the chest revealed a left upper lobe mass, pleural thickening and effusion concerning for malignancy or an infectious process. Subsequent bronchoscopy with endobronchial ultrasound (EBUS) and bronchial washings were negative for malignancy. Infectious workup for fungal serologies,

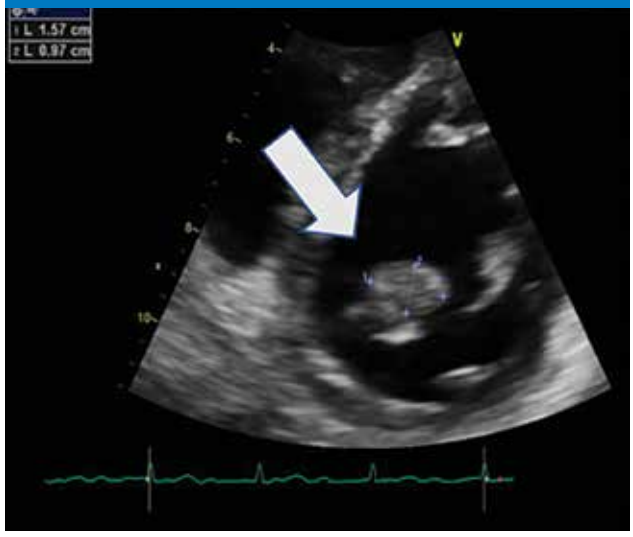
histoplasmosis and blastomycosis urine antigen, Q fever antibody, toxoplasma PCR, and TB quantiferon was completed. The results returned positive for Q fever IgG phase II titer. Bronchoalveolar lavage samples for acid-fast bacilli smear were negative. The remaining infectious disease panel also was negative. The patient was treated for acute Q fever with two weeks of oral doxycycline (100mg twice a day). The patient was later retested and Q fever PCR and phase II IgM titer returned negative making chronic Q fever infection unlikely.

Approximately one month after completing the antibiotics, he reported progressive weakness, anorexia, persistent fevers and chills prompting hospitalization. He presented with septic shock (hypotension and sinus tachycardia) requiring IV fluid boluses and vasopressors. He was afebrile on presentation. Oxygen saturation on pulse oximetry was 94% on room air. Laboratory investigation revealed a hemoglobin of 7.5 g/dL (14.0-17.5 g/dL) slightly below his baseline of 8-9 g/dL, thrombocytopenia of 64 K/uL (140-450 K/uL), C-reactive protein of 8.5 mg/dL (<0.5 mg/dL) and a lactic acid of 1.6 mmol/L (<2 mmol/L). The remainder of his CBC, BMP and urinalysis was unremarkable. Chest X-ray was unremarkable. CT of the chest revealed worsening pleural thickening in the right upper lobe compared to his prior CTs as well as a small pleural effusion and a wedge-shaped hypodensity in the upper spleen concerning for infarct. Head CT revealed small nonspecific areas of parenchymal enhancement predominantly within the cortical regions of the posterior frontal and parietal lobes. Brain magnetic resonance imaging (MRI) revealed several ring enhanced lesions with adjacent restricted diffusion and numerous lesions demonstrating microhemorrhage within the brain and cerebellum concerning for septic emboli (Figure 1). A transthoracic echocardiogram (TTE) revealed a large mobile mitral subvavlular echodensity 1.6 x 1.0 cm suggestive of a vegetation (Figure 2).

Cardiology and cardiothoracic surgery evaluated the patient and was deemed a poor surgical candidate for valvular repair due to his existing comorbidities and clinical status. In light of prior positive *Coxiella brunetti* serologies and negative blood cultures from his prior infectious workup, he was started on doxycycline in addition to vancomycin and cefepime. Additionally, blood cultures were collected and held for 10-day culture. His conditioning improved and he was titrated off vasopressors. He developed a painful lesion on his upper lip, an attempt to aspirate was done, however a lack of free fluid hindered this and no sample was collected

Figure 1. Brain MRI illustrating numerous ring-enhancing lesions enhancement.

Figure 2. Transthoracic echocardiogram illustrating a mobile mitral vegetation measuring approximately 1.6 x 1.0 cm.



or cultured. An induced sputum culture was collected and sent for further analysis.

Unfortunately, the patient developed expressive aphasia on day six of his admission. Acute stroke workup revealed a cerebral embolism with infarction involving left superior cerebellar hemisphere and right corpus callosum without hemorrhage on CT imaging. This finding was concerning for septic embolus from his known mitral valve vegetation.

On day 10 of admission, peripheral blood cultures showcased positive branching and beaded gram-positive rods suspicious for *Nocardia spp*. Confirmatory PCR testing confirmed *N.*

Figure 3. Antibiotic sensitivity found in the *Nocardia* species isolated from AFB culture.

	Nocardia s M.I.C.	RX
Amikacin	<=0.5	S
Ceftriaxone	<=1	S
Ciprofloxacin	<=0.12	S
Imipenem	0.5	S
Linezolid	<=0.5	S
Moxifloxacin	0.06	S
Tobramycin	<=2	S
Trimethoprim/Sulfamethoxazole	0.06/1.2	S

paucivorans. The culture antibiotic sensitivity is shown in Figure 3. Antibiotics were changed to imipenem (500mg IV q6h) and trimethoprim/sulfamethoxazole (TMP-SMX) (5mg/kg trimethoprim component IV q8h). Doxycycline was continued.

On day 11, he developed confusion, worsening aphasia, and facial droop. Head CT revealed a large right frontoparietal hemorrhage. Computed tomography angiography (CTA) showed an aneurysm in middle cerebral artery with active hemorrhage. Surgical intervention was deemed inappropriate by neurosurgery. He was transitioned to comfort cares on hospice and the patient passed away on day 18.

Discussion

Among individuals infected with *N. paucivorans*, a recent analysis found that up to 30% of patients experienced systemic dissemination with 80% of those cases involving dissemination to the brain.[6] Among other *Nocardia* species, the reported rate of cerebral dissemination with systemic *Nocardia* infection is 30-80%, suggesting a predilection for CNS involvement in *N. paucivorans*. However, due to the small sample size of the current data, additional investigation is required.[6]

Diagnosis of *Nocardia* is challenging due to its indolent course. While infections may resemble infective endocarditis (IE), the empiric treatment for the most common offending organisms for IE often consists of a combination of vancomycin, gentamycin, and a cefepime or another anti-pseudomonal agent.[7] Unfortunately these agents generally have either poor CNS penetration or are generally insufficient in the treatment of disseminated *Nocardia*. Symptoms of nocardiosis are highly variable and non-specific, with the most common manifestation being pneumonia with cough, dyspnea, and hemoptysis, as well as systemic signs of fever, weight loss, and night sweats.[1] Extrapulmonary involvement, notably CNS involvement is most commonly seen in the immunocompromised individuals, manifesting with symptoms of space occupying lesions (headache, nausea, vomiting, and other focal deficits). Although *Nocardia* infections are rare, it is important to consider them in the infectious disease differential in an immunocompromised patient. Gram staining and culture with specific medias such as buffered charcoal yeast extract can reveal *Nocardia* in up to two-thirds of infected patients.[8] Unfortunately, blood cultures of patients with suspected *Nocardia* infection may require for up to 2 weeks to yield results, so starting patients at an increased risk of *Nocardia* infection on empiric treatment is reasonable until *Nocardia* can be ruled out.

Due to the scarcity of disseminated *Nocardia* infections, no clinical trials currently guide optimal antibiotic choice. Therefore, antibiotic susceptibility should be assessed by culture and susceptibility to guide treatment. The most frequently reported antibiotic of choice in literature is TMP-SMX dosed at 5-10 mg/kg trimethoprim component divided in 2-4 daily doses.[8] This treatment recommendation aligns with the antibiotic susceptibility found in this patient's *Nocardia* strain. No formal guidelines exist for treatment duration; however, 12-months of antibiotic therapy is the most common recommendation by experts.[8] While immunocompromised status is the most significant risk factor for *Nocardia* infection, primary prophylaxis of nocardiosis in at risk populations is not standard practice. Evidence from case-control studies show that the dose of TMP-SMX for prophylaxis against Pneumocystis pneumonia (160mg/800mg daily) is not sufficient to prevent nocardiosis and it is not known if higher doses of prophylaxis are beneficial. Moreover, the rate of nocardiosis among at-risk populations is <1%, making universal prophylaxis likely unneccesary.[8]

While the overall mortality and morbidity of *N. paucivorans* remains unclear due to a lack of data, the estimated mortality in all species of *Nocardia* remains high regardless of treatment with 1-year mortality rates of 32% and up to 50% mortality in patients with dissemination into the CNS.[1] This may be due in part to a failure to consider this organism in the diagnostic differential and provide appropriate treatment early in the disease course. For diagnosing *Nocardia*, testing by PCR or culture of the suspected source should be performed early in the disease course. While culture remains the gold standard for confirming diagnosis, PCR has recently become available with varying results. Recent studies have shown a specificity of 74% and a sensitivity of 88% for diagnosis of *Nocardia* infection via PCR in respiratory samples.[9] As PCR sensitivity does not capture 100% of infections, it is recommended that

clinicians also obtain specimens for culture to confirm PCR accuracy as well as for antibiotic sensitivity data for guiding treatment.

Conclusion

Nocardia paucivorans is a rare disease in humans that can infect immunocompromised individuals occasionally leading to dissemination. Moreover, it does not often respond to the empiric antibiotics used in the hospital setting, resulting in delayed treatment and poor outcomes in disseminated disease. Cultures can take at least 2 weeks to turn positive, often resulting in delayed diagnosis although newer PCR techniques may help speed up diagnosis. It most commonly presents with pulmonary symptoms including pleural effusions, cough, dyspnea, hemoptysis, and dissemination to other body systems including the CNS, soft tissue, and rarely causing infective endocarditis. Treatment of *Nocardia* is not well established, however TMP/SMX is the most utilized treatment. Additional clinical trials are needed to establish treatment guidelines and the necessity of prophylactic treatment in immunocompromised individuals.

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Suspected Pseudocholinesterase Deficiency During Left Thyroid Lobectomy and Isthmusectomy: A Case Report

Jennifer Boleyn, MD; Madison McLaury, MD; and Stephanie Wieman, MD

Abstract

Background: Pseudocholinesterase (butyrylcholinesterase) deficiency is an acquired or inherited condition in which decreased plasma levels of the pseudocholinesterase enzyme lead to an inability to metabolize the neuromuscular blocking agents succinylcholine and mivacurium, prolonging their paralytic effects. This often results in delayed extubation and additional intensive care requirements in the postoperative period.

Case Description: We describe a case of suspected pseudocholinesterase deficiency in a previously healthy 59-year-old female who underwent a left thyroid lobectomy and isthmusectomy. The patient received 120 mg of succinylcholine chloride before intubation. The patient did not meet extubation criteria following the completion of the procedure approximately two hours after receiving succinylcholine chloride. The patient was transferred to the ICU for respiratory support and for the medication to clear from the patient's system. The patient regained muscle control approximately four hours after receiving succinylcholine chloride and was extubated without complication. The patient shared post-extubation that she had a blood relative with the diagnosis of pseudocholinesterase deficiency.

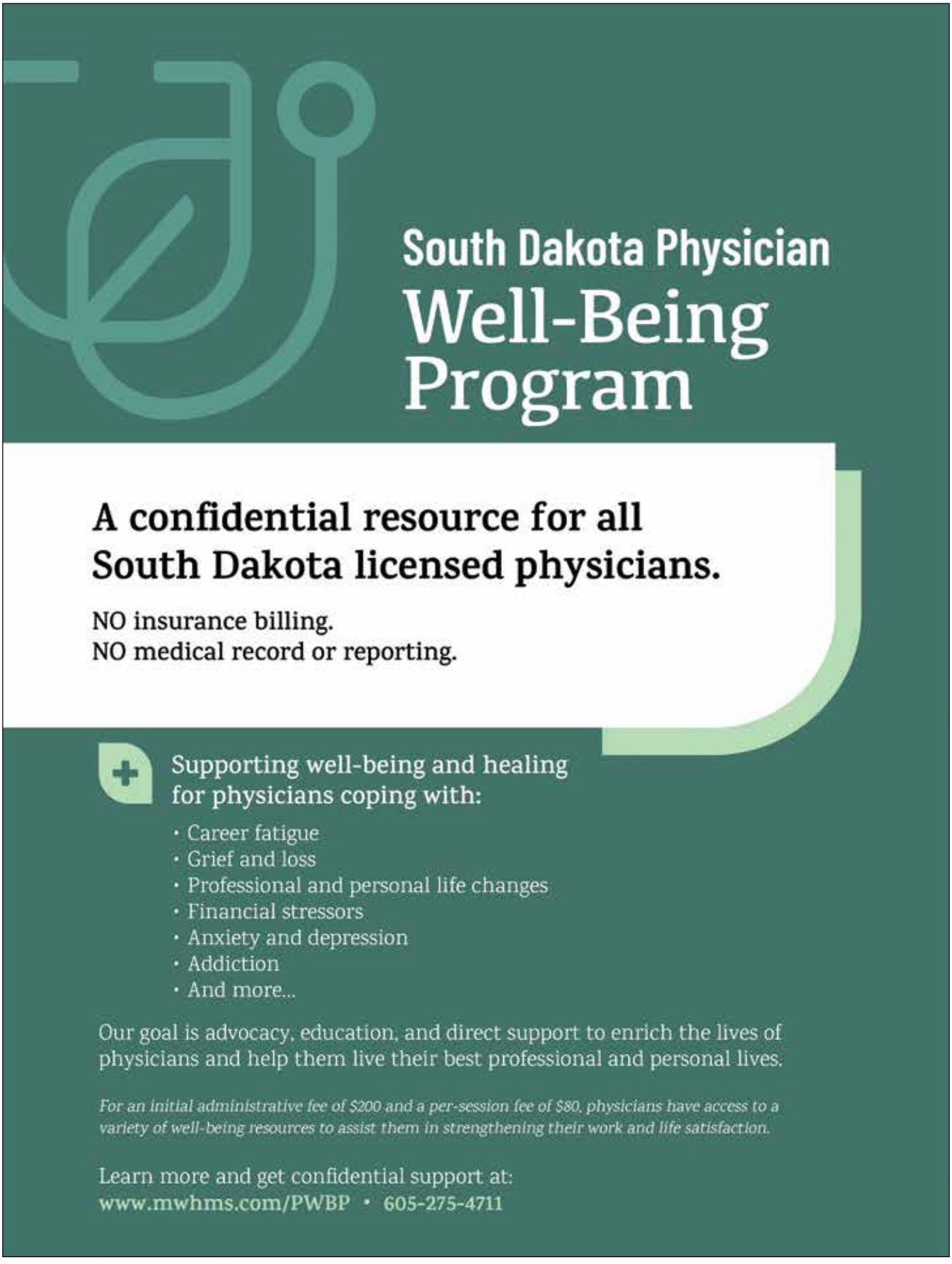
Conclusion: Pseudocholinesterase deficiency is rare but can result in potentially serious complications following the administration of succinylcholine chloride, mivacurium, or ester local anesthetics due to reduced metabolism and subsequently increased pharmacodynamic effects. Given the widespread use of succinylcholine chloride as a neuromuscular blocking agent, such as in this case, providers must be aware of the presentation, pathophysiology, diagnosis, and management. Additionally, this case demonstrates the importance of thoroughly inquiring about any personal or family history of anesthetic complications during a preoperative assessment.

Introduction

During preoperative evaluations, anesthesiologists assess for numerous conditions and comorbidities that can complicate anesthesia care and patient safety. Some of the well-known conditions providers watch for include malignant hyperthermia and allergies to medications. One condition that is uncommon but important for providers to be able to identify is pseudocholinesterase (butyrylcholinesterase) deficiency. Pseudocholinesterase (PChE) deficiency is a condition in which pseudocholinesterase plasma levels are decreased or absent. This enzyme is responsible for the metabolism of succinylcholine, mivacurium, and local ester anesthetics (procaine, tetracaine, cocaine).[1] Succinylcholine and mivacurium are neuromuscular blocking agents that are commonly used in clinical anesthesia practice as muscle paralytics to optimize intubation and surgical procedures. Paralysis for succinylcholine is observed within 60 seconds of intravenous administration and continues for four to six minutes for normal individuals[2]. Due to its rapid onset and short half-life, succinylcholine is often used for rapid sequence intubation and in procedures where muscle paralysis is undesirable, such as when nerve monitors are required.[3] Decreased PChE activity leads to prolonged paralytic effects, delayed extubation, and potential postoperative complications. The purpose of this case report is to highlight a suspected case of PChE deficiency discovered at the time of surgery and to educate healthcare professionals on the presentation, pathophysiology, diagnosis, and management of the condition.

Case Presentation

A 59-year-old, previously healthy female initially presented to an otolaryngologist for the workup of a multinodular thyroid. Fine needle aspiration of the left thyroid revealed a follicular neoplasm of uncertain behavior. The patient was subsequently scheduled for a left thyroid lobectomy and isthmusectomy with intraoperative recurrent laryngeal nerve monitoring. Past surgical history included colonoscopy and a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cesarean section with tubal ligation. She denied any trouble with monitored anesthesia care (MAC) or spinal anesthesia during either of these cases. She had never undergone general anesthesia or received a neuromuscular blocking agent. She denied any family history of malignant hyperthermia or adverse reactions to anesthesia. She had no known drug allergies. Her medications included over-the-counter zinc, vitamin C, vitamin D, vitamin E, fish oil, magnesium, and a daily multivitamin. The patient was hemodynamically stable during the preoperative period and her electrocardiogram revealed sinus rhythm. The patient was 5' 4", weighed 72.1 kg, and had a class II mallampati score and an American Society of Anesthesiologists (ASA) physical status score of two. The anesthetic plan included general endotracheal anesthesia.

Induction medications included 2 mg midazolam, 100 mcg fentanyl, 60 mg lidocaine, 200 mg propofol, and 120 mg succinylcholine chloride. She was successfully intubated without complication. The patient remained hemodynamically stable throughout the surgery. Of note, while the recurrent laryngeal nerve was easily identified by the primary surgeon during the procedure, it did not stimulate with nerve stimulus. This was initially thought to be due to a malfunctioning nerve monitor.

Following completion of the surgery, the patient had difficulty regaining muscle movement and did not meet the criteria for extubation approximately two hours after receiving succinylcholine chloride. At this point, a diagnosis of pseudocholinesterase deficiency was suspected. The patient, still intubated, was admitted to the intensive care unit (ICU) for ventilatory support where she remained hemodynamically stable and sedated with propofol. While in the ICU, she regained consciousness, followed commands, and moved all four extremities. She was successfully extubated after a pressure support trial approximately four hours after the induction of anesthesia and had a full recovery. After an in-depth discussion with the patient, she shared that she has a nephew with an "anesthesia issue" that she couldn't remember the name of. After asking additional questions, she revealed it was pseudocholinesterase deficiency. The patient explained that she was told her family history of pseudocholinesterase deficiency was not relevant during a preoperative discussion before a colonoscopy. The patient understood this as meaning her family history of an adverse reaction to anesthesia was not relevant to any surgical case when the anesthesia team was likely only referring to the fact that paralytics would not be required during a colonoscopy. The misunderstanding of the pertinence of her family history and lack of other family members with adverse effects to anesthesia led to her omitting the information during her preoperative examination the morning before this case.

Succinylcholine was added to her allergy list. The complication was included in both the anesthesiologist's and surgeon's intraoperative and post-operative documentation. Educational materials were shared with her and her husband. The patient was encouraged to tell every future surgeon and anesthesiologist of this diagnosis and inform family members of a genetic risk of pseudocholinesterase deficiency. She was also provided information on the closest center for genetic testing and counseling.

Discussion

Pseudocholinesterase deficiency, while uncommon, is an important condition for all anesthesiologists to be aware of and capable of managing. The case discussed here is a classic example of an unidentified PChE deficiency that led to a complicated postoperative course. This case depicts the difficulty in detecting this condition in the preoperative period and iterates the importance of neuromuscular monitoring throughout procedures. If a nerve monitor malfunctions intraoperatively, further investigation should occur.

Pseudocholinesterase deficiency can present as either an acquired or inherited condition. The inherited form arises from a mutation on the butyrylcholinesterase (BCHE) gene (located on chromosome three) and transfers in an autosomal recessive fashion, with the degree of enzymatic function decreasing with each abnormal variant of the gene received. Heterozygotes present with ~30% increase in paralysis time to a standard dose of succinylcholine when compared to patients with normal gene variants. Homozygotes can experience paralysis for greater than two hours.[4] In a literature review of 40 case reports, the paralysis length in patients with PChE deficiency ranged from 50 minutes to 10 hours.[5] The duration of the blockade is proportional to the level of pseudocholinesterase deficiency.

If a strong family history is not present when extended paralysis following the administration of succinylcholine or mivacurium occurs, it is important to consider an acquired deficiency. Acquired PChE deficiency can arise secondary to numerous comorbidities including liver disease, hypothermia, burns, dialysis, HELLP syndrome, MI, or kidney disease. It can also occur in specific physiologic states (pregnancy, postpartum, and advanced age), as well as from certain

drugs (steroids, cytotoxic medications, MAO inhibitors, organophosphate insecticides, and anticholinesterase medications).[4]

Options for diagnostic testing include the qualitative dibucaine test or quantitative enzyme level measurement via colorimetry of plasma samples. Dibucaine is an amide local anesthetic that inhibits the pseudocholinesterase enzyme. The dibucaine test involves the addition of dibucaine to a patient's blood sample to determine the percent of pseudocholinesterase enzyme inhibited. Abnormal gene variants lead to less inhibition, and therefore small dibucaine numbers. Homozygous normal variant displays about 80% inhibition of the pseudocholinesterase enzyme, with a dibucaine number (DN) of 70-80. Heterozygosity with an abnormal variant displays inhibition of the enzymatic activity by 50-60%, with a DN of 50-60. Homozygotes with two abnormal gene variants have DN of less than 30.[6]

There are no treatments or alternative enzymes currently available to overcome the prolonged effects of succinylcholine in these patients. Management includes maintenance of sedation, and continuation of monitoring and mechanical ventilation until motor function returns. The extended duration of the paralytic effects often leads to patients being sent to the intensive care unit following surgery.

Given the postoperative complications that can occur, providers need to have a high suspicion of this condition if intraoperative nerve monitoring is inadequate or if the return of motor function is delayed upon the emergence of anesthesia. While this case highlights the importance of taking an in-depth personal and family medical history in the preoperative setting, it also emphasizes the importance of educating patients appropriately on the condition and iterating the necessity of notifying healthcare providers before future procedures.

Conclusion

This report describes a case of suspected pseudocholinesterase deficiency presenting after the use of succinylcholine chloride. Pseudocholinesterase is an enzyme involved in the metabolism of succinylcholine chloride, mivacurium, and ester local anesthetics. While rare, an inherited or acquired pseudocholinesterase deficiency can cause increased pharmacodynamic effects of these medications because of slower removal of the medication from the body. Providers should be aware of the presentation, pathophysiology, diagnosis, and treatment when using these medications.

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Post-Intensive Care Syndrome – An Opportunity For Collaborative Transition Between the Intensive and Primary Settings

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Abstract

Critical care advancements have allowed clinicians to discover the many functional disabilities that survivors suffer. Recent research has focused on improving the long-term outcomes of critical illness survivors and optimizing their functional recovery. Post-intensive care syndrome (PICS) describes the disability that remains in those surviving critical illness following discharge from the intensive care unit (ICU). This comprises impairment in cognition, neuropsychiatric health, and physical function of the ICU survivor. Consequent to this, the health of family members of the survivor may also be affected adversely, termed PICS-family. PICS is defined as a new or worsening impairment in physical (ICU-acquired neuromuscular weakness), cognitive (thinking and judgment), or mental health status arising after critical illness and persisting beyond discharge from the acute care setting.

Introduction

Due to the advancements in critical care medicine and subsequent improvements in acute illness survival rates, clinicians have begun to uncover the significant functional disabilities experienced by critical illness survivors.[1] Recently, research has been dedicated to enhancing the long-term outcomes and functional recovery of critical illness survivors.[2,3]

The five most common ICU admission diagnoses for adults are respiratory failure with required mechanical ventilation, acute myocardial infarction, cerebral infarction or intracranial hemorrhage, status post percutaneous cardiovascular procedures, and septicemia with or without the need for mechanical ventilation. Mortality rates among patients in adult ICUs range from 10% to 29%, varying based on age, underlying comorbidities, and illness severity. Patients admitted to the ICU tend to face higher mortality rates in the following decade than people of the same age who have not been through such admissions.[4] Critical illness entails significant implications that extend beyond primary hospitalization. For patients who survive, targeted post-discharge care offers an opportunity for benefit.[5,3] Upon discharge, critical illness survivors seldom follow up with an intensivist; more often, they are discharged for follow-up to their primary care physician or, increasingly, an advanced practice provider.[6]

Each year in the U.S., around 5.7 million patients receive ICU care, 4.8 million will survive their ICU stay, and approximately 50% to 70% will develop post-intensive care syndrome (PICS) at some point after discharge.[6,7] PICS encompasses a range of complications that can persist after critical care. These include ongoing cognitive, psychiatric, and physical dysfunction. Notably, PICS generally does not include patients admitted with traumatic brain injury and stroke.[8] The identification of PICS typically occurs during the immediate period following critical illness. However, due to the persistence of symptoms and the condition's relative lack of recognition, there is no specific timeframe after critical illness when PICS may or may not occur. [6]

Physical Impairment

Physical impairments are primarily associated with pulmonary and neuromuscular system abnormalities. Patients experiencing PICS display various signs and symptoms of ICU-acquired weakness (ICU-AW), ranging from poor mobility to quadriparesis. At a minimum, many patients have persistent disabilities in their activities of daily living. Additional morbidities include contractures, loss of muscle bulk, compromised lung function, and malnutrition. ICU-AW is thought to arise from microvascular ischemia, which impairs neuronal mitochondrial function and causes demyelination. However, the pathophysiology is poorly understood.[7-11]

Cognitive Impairment

The BRAIN-ICU Study, published in 2013, followed the executive and cognitive function of 821 ICU survivors at 3 and 12 months following their discharge. 74% of the patients had experienced delirium during their ICU stay, that had resolved by the time of discharge. Twelve months after discharge, of the cohort of patients that followed up in clinic, 34% had cognitive testing scores similar to that of patients with moderate traumatic brain injury. 24% of patients had cognitive scores consistent with mild Alzheimer's disease.[12] This data likely underrepresents the degree of residual impairment, as only 328 patients out of the original 821 made it to the 12-month follow-up. The remaining patients either unfortunately passed away or were lost to follow up.

The severity of cognitive impairment in PICS ranges from mild to severe, with individuals experiencing challenges in various cognitive domains. PICS commonly affects specific areas of cognition, including attention/concentration, memory, mental processing speed, and executive function. These functions are crucial in carrying out a discharge plan, such as adhering to medications, following dietary restrictions, and managing appointments. Impaired cognition may also contribute to communication difficulties often observed in patients undergoing rehabilitation after a critical illness, further hindering recovery.[12,13,14,15]

Neuropsychiatric Impairment

Psychiatric complications following a critical illness can be severely debilitating. Mood disorders, anxiety, depression, and post-traumatic stress disorder (PTSD) are prevalent.[16] A recent meta-analysis revealed that approximately one in five adult ICU survivors develop PTSD within the year following ICU discharge.[17] This is comparable to PTSD rates observed among civilian survivors of war conflicts.[18] Considering that around 5.7 million patients are admitted to ICUs in the U.S. annually, and with mortality rates ranging from 10% to 29%, this meta-analysis indicates that approximately 1 million patients every year may develop PTSD following ICU admission.

Sexual dysfunction is also a prevalent issue, particularly in those with symptoms of PTSD. A prospective observational study of 127 ICU patients who spent more than three days in the ICU found sexual dysfunction in 44 percent of patients.[19] This dysfunction was strongly associated with symptoms of PTSD and was unrelated to age, sex, length of stay, or degree of respiratory intervention such as the use of mechanical ventilation or tracheostomy.

A retrospective study of over 420,000 consecutive ICU survivors reported a higher risk of suicide and self-harm than non-ICU hospital survivors. Factors associated with suicide or self-harm included previous depression or anxiety, previous PTSD, invasive mechanical ventilation, and the use of renal replacement therapy.[20]

Diagnostics

No consensus exists on the instruments for evaluating and diagnosing PICS. Clinical experts recommend a two-step set of expert-validated PICS outcome instruments. The assessment process involves an initial screening and a comprehensive assessment if needed. Outpatient providers should consider including qualified physical and occupational therapists in evaluating these patients.[21,22]

An initial screening consists of five brief tests covering the three clinical domains of PICS. These tests are freely available and collectively take approximately 20 minutes per patient. These include the Patient Health Questionnaire-4 (PHQ-4) and health-related quality-of-life scales (EQ-5D-5L). These questionnaires can be given to patients after rooming, prior to their visit with the provider in a similar manner to how the Patient Health Questionnaire-9 (PHQ-9) or General Anxiety Disorder-7 (GAD-7) is administered in many outpatient clinics. The PHQ-4 and EQ-5D-5L can be obtained free of charge online. Administering these forms prior to the appointment with the provider saves in-person examination time for the physical and cognitive assessments.

Diagnostics: Physical Impairment

Physical function is evaluated with the Timed Up-and-Go (TUG) test, which measures functional mobility, and handgrip strength measured with a dynamometer. The TUG test assesses a patient's ability to rise from an armchair, walk 3 meters, pivot, and return to the seated position. Handgrip strength quantitatively measures muscle strength in the hand and forearm and has been linked to mobility, cognition, functional status, and mortality in older adults. If concerning findings arise from the TUG test or handgrip dynamometer, the Lawton Instrumental Activities of Daily Living Scale can be used for further evaluation. The Lawton Scale covers eight activities instrumental to independent function. It is considered more sensitive to complex skills necessary for community living. It takes 10 to 15 minutes to complete and can be administered before the face-to-face visit.[23-25]

Diagnostics: Cognitive Impairment

Cognition can be evaluated using the Montreal Cognitive

Assessment (MoCA), Modified Mini-Mental State Examination (MMSE), Mini-Cog, and the animal naming test. While the MMSE and Mini-Cog are widely recognized and extensively studied in the general population, their ability to predict cognitive impairment at six months in ICU survivors is uncertain. The Society of Critical Care Medicine (SCCM) advises the MoCA to evaluate patients with suspected cognitive dysfunction following critical illness. The MoCA incorporates an assessment of executive function abilities, making it a more sensitive test for detecting mild impairment. Executive dysfunction is common among ICU survivors and may respond to rehabilitation and compensation strategies. A MoCA score of <26 indicates mild cognitive impairment, while a score of <18 indicates moderate to severe cognitive impairment consistent with dementia. The MoCA is available in various versions and languages, requiring approximately 10 minutes for complete administration. However, it necessitates the completion of a paid training and certification course to ensure proper administration, interpretation, and scoring of the results.

The Mini-Cog is a brief assessment that takes around 3 minutes to administer, with minimal language content, and is available in multiple languages. It comprises a 3-item recall and a clock drawing component. Initially designed as a screening tool for hepatic encephalopathy, the animal naming test involves verbally naming as many animals as possible within 1 minute. For healthy control subjects, naming less than 15 animals in 1 minute is considered an abnormal result and an indication of cognitive impairment.[26-29]

Diagnostics: Neuropsychiatric Impairment

Initial mental health assessment should involve using Patient Health Questionnaire-4 (PHQ-4), a four-item scale screening for anxiety symptoms. Respondents rate their anxiety, worry, depression, and other feelings over the preceding two weeks. For expanded assessment, the Hospital Anxiety and Depression Scale (HADS), which is slightly longer at 14 items, is recommended.[28,30,31]

The EuroQol-5D-5L can also be used. It assesses health-related quality of life across five dimensions, focusing on self-perceived physical and mental illness. The PTSD assessment, while not part of the primary screening, is suggested using the Impact of Event Scale–Revised (IES-R).[32-33]

Management

Follow-up Schedule

Due to limited data, the recommended frequency of outpatient clinic visits after hospital discharge is poorly defined.[36] Delaying the follow-up visit beyond 30 days limits the ability to screen for PICS symptoms that were potentially unrecognized at discharge. Some PICS symptoms require urgent intervention to prevent further decline. It is, therefore, essential to assess critical care survivors in an outpatient setting as soon as possible. The Society of Critical Care Medicine (SCCM) suggests an initial assessment 2 to 4 weeks following discharge. However, the timing of these visits can be challenging, particularly if patients need help with their ability to travel, for example, those being discharged to a care facility or residing in a rural area.[33-35]

A survey by the Critical and Acute Illness Recovery Organization (CAIRO) Post-ICU Clinic Collaborative found a range of visit lengths in ICU aftercare programs, from 31-60 minutes to over 2 hours.[36] The initial post-discharge outpatient visit requires extensive assessment and an exam by the provider. Recommended screening alone requires a minimum of 20 minutes to complete. Therefore, an extended visit duration is necessary over the standard 15-20 minute return visit. The follow-up frequency has also yet to be well established. The SCCM recommends "serial" assessments of unclear duration. An outpatient visit frequency of 1-, 3-, 6-, and 12 months post-discharge has been suggested. The frequency of outpatient visits should be tailored to meet each patient's needs.[7]

Management: Physical Impairment

Post-ICU patients often suffer from chronic anemia following discharge. During their ICU stay, many patients receive myelotoxic drugs. Risk factors for developing anemia include sepsis, malignancy, female sex, older age, and malnutrition. Many of these cases are detected during the ICU stay. The threshold for ordering a complete blood count, reticulocyte count, and iron panel (iron, ferritin, total iron-binding capacity, transferrin) should be low in the outpatient setting, particularly when patients complain of dyspnea or generalized fatigue.

Risk factors for myopathy include prolonged ICU stays, poor premorbid health, and a longer duration of mechanical ventilation. A graded exercise program can help accelerate recovery and get patients back to their pre-hospitalization abilities. Common risk factors for neuropathy include sepsis and multiorgan failure. Neuropathic pain medications, such as gabapentin (Neurontin), anticonvulsants, capsaicin cream (Zostrix), Venlafaxine (Effexor), selective serotonin reuptake inhibitors (SSRIs) can be considered. However, no studies have examined their therapeutic effect in critical illness polyneuropathy.

Many patients are exposed to bone-demineralizing drugs in the ICU and experience limited weight-bearing for prolonged periods.[37] Currently, no evidence suggests that post-ICU patients should be screened for osteoporosis and Vitamin D deficiency differently than other populations. Physicians should consider prolonged immobility a pertinent risk factor in women older than 60 and consider earlier screening in select patient populations.[38]

The preventive use of bisphosphonates is beneficial for patients meeting clinical, T-score, or FRAX criteria. Weight should be closely monitored and compared with preadmission values. The threshold for nutritionist referral should be low to ensure proper caloric and vitamin intake.

ICU patients who had required tracheostomy for airway management should be evaluated for potential dyspnea and stridor. A bronchoscopy or computed tomography should be considered if complications are suspected.

Management: Cognitive Impairment

For patients with mild cognitive impairment (MCI), screening and treatment for vascular risk factors, especially hypertension, is essential. Modification of these risk factors may help reduce the progression of cognitive decline. While less compelling evidence exists for the efficacy of other treatments like antiplatelet therapy, statin therapy, smoking cessation, and diabetes management in preventing dementia, they are still recommended for additional health benefits. Regarding cognitive interventions, various cognitive rehabilitation methods, such as memory training, external memory cues, and organizational aids, have shown promise in improving cognitive function in healthy older adults. However, it's uncertain whether individuals with MCI would benefit from similar programs, as the results from studies have been mixed, showing only modest short-term improvements in cognitive domains compared to control conditions.

Further research is needed to better understand the potential benefits of cognitive rehabilitation methods for patients with MCI.

Management: Neuropsychiatric Impairment

Risk factors for PTSD in the post-ICU setting include prolonged mechanical ventilation, ARDS, use of sedatives, and neuromuscular blocking.[39] SSRIs should be considered in patients who meet the criteria for or show significant symptoms of PTSD. Trauma-focused therapies, such as cognitive-behavioral therapy (CBT), exposure therapy, and eye movement desensitization and reprocessing therapy, are recommended as the first-line treatment for most adults with PTSD. Additionally, patients should be monitored closely for signs and symptoms of depression in this setting and pharmacologic and behavior therapy should be initiated when appropriate. More progressive treatment for anxiety disorders in these patients includes tours of the ICU several months following discharge and psychotherapy focused on recalling factual memories.

Conclusions

Currently, ICU care success is measured by patient survival, transfer to the general medical floor and eventual discharge. However, it is evident that ICU survivors experience far-reaching negative health consequences impacting patients' quality of life and persisting long after discharge. Improving outcomes in patients after critical illness requires collaboration between practitioners and researchers in both the inpatient and outpatient settings. Unfortunately, the best strategy to improve these adverse outcomes has yet to be determined. Thus, additional research is crucial in establishing data-driven post-ICU syndrome screening and assessment tools for practitioners in clinical settings.

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Raoultella planticola Urinary Tract Infection Presenting as Hyperbilirubinemia in a 3-Day-Old Infant

David Jensen, MD; Anthony Drazick, MS IV; Rochelle Boote, MD, FAAP; and Peter Paul Lim, MD, FAAP

Abstract

Raoultella planticola is a Gram-negative, aerobic, nonmotile bacterium that is ubiquitous in the environment usually implicated in opportunistic infections. There have been very few reported cases of *Raoultella planticola* infection in the pediatric population. Most of these reports have been in cases of neonatal septicemia. This case report describes a case of a 3-day-old Hispanic full-term male that presented with recalcitrant hyperbilirubinemia despite maximal phototherapy found to have urinary tract infection with *Raoultella planticola* on multiple cultures. The patient's hyperbilirubinemia appropriately responded to treatment of the UTI. This report highlights that, albeit rare, neonatal UTI can present as recalcitrant hyperbilirubinemia. *Raoultella planticola* is a rare organism that is normally found in the environment but may be a bona fide etiologic agent in neonatal UTI.

Introduction

Hyperbilirubinemia often manifested by jaundice is a common presenting medical condition among the neonatal population. While often benign, hyperbilirubinemia can have detrimental neurologic effects on a neonate if significantly elevated unless treated appropriately.[1] In most cases, the hyperbilirubinemia is physiologic due to an imbalance between bilirubin production and bilirubin clearance. Bilirubin clearance is dependent on the conjugation of bilirubin by the liver enzyme uridine diphosphate glucuronosyltransferase (UGT) and a newborn has 1% of the activity compared to a typical adult. The enzyme reaches adult activity at approximately 3 months of life.[1] Pathologic causes such as blood type incompatibility, hepatic enzyme defects, metabolic issues, hepatic anomalies, and infections could be harbinger of a more serious issue.[1,2] Most often, neonates who have a UTI present with fever and poor feeding but may be associated with jaundice. UTIs were discovered in 6 to 18% of full-term or preterm infants who presented with prolonged or worsening jaundice.[3] *Escherichia coli* (*E. coli*) is the most common isolated pathogen associated with neonatal UTI. Other causes of UTIs in neonates are *Enterococcus*, Group B streptococcus (GBS), and *Klebsiella sp.*[3,4] An emerging pathogen, *Raoultella planticola (R. planticola),* a bacterium ubiquitously found in the environment has recently been implicated across heterogenous infectious syndromes.

R. planticola is a Gram-negative, aerobic, nonmotile bacterium prevalent in water and soil and usually implicated in opportunistic infections.[5] Most presentations of *R. planticola* infections have been reported in the adult population, where the pathogen has been implicated in bacteremia, cholangitis, abscesses, pneumonia, endocarditis, and UTIs.[6] Many pediatric infections involving *R. planticola* have been in neonatal septicemia, including 4 cases where the diagnosis was made at 9-27 days of life.[4] There have only been a few cases of pediatric UTI from *R.planticola* in the literature.

The case presented stresses the importance of prolonged hyperbilirubinemia as a presenting picture of neonatal UTI. It also sheds light on the possibility of an emerging pathogen, *R. planticola,* as a possible bona fide infectious agent of UTI in this age group. The patient presented may be one of the youngest to have been diagnosed with a UTI from *R. planticola* from limited literature review of PubMed (Figure 1).

Figure 1. Patient presenting with jaundice.

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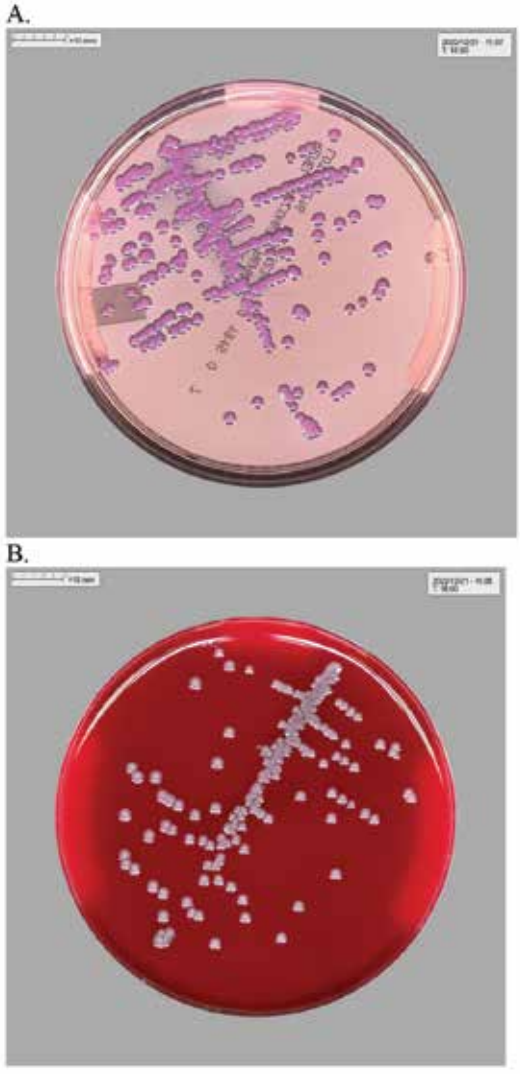
Case Presentation

A 3-day-old term, 3075-gram non-circumcised male infant with normal newborn screen presented with jaundice and scleral icterus concerns to his pediatrician. The maternal prenatal course was unremarkable. Delivery was complicated by positive maternal group B streptococcus culture as part of screening and postpartum chorioamnionitis. The neonate's Coomb's test was negative. Total serum bilirubin (TSB) at 29 hours was 9.9 mg/dL (reference range 0-12.0 mg/dL) prompting discharge from the hospital after birth. The remainder of the history obtained at presentation was unremarkable, including appropriate numbers of feeding, bowel movements, wet diapers, with no fever reported. Baby's intake composed of both formula and breast milk. Vital signs were within normal limits. His weight was down 7.5% from birth. Physical examination was pertinent for jaundice, scleral icterus, diaper rash, and an uncircumcised penis. Initial TSB was 16.2 mg/dL at 72 hours of life on presentation. TSB recheck the following day revealed an increase to 20.2 mg/dl. Transcutaneous phototherapy was done and recommendations for increased feeding were then initiated. At follow-up at six days of life, physical exam showed worsening jaundice on the whole body with scleral icterus. Patients weight on follow up was now 8.3% down from birth weight. TSB level at this time showed an increase to 25 mg/dL, prompting admission.

Maintenance IV fluids were started along with a nasogastric tube for continuous feed. Sepsis workup was initiated despite the absence of fever and other symptoms because of the baby's age and increasing bilirubin levels despite maximal phototherapy. Phototherapy was continued with two lights at admission. His laboratory workup included TSH, G6PD, blood smear, CBC, urine CMV, blood culture, LDH, DAT, CMP, and CRP, which were all reassuring and within normal limits except for bagged urine analysis that showed 3+ leukocyte esterase and significant leukocyturia, which eventually grew Gram-negative rods. This warranted repeat catheterized urine collection. He was empirically started on ampicillin and gentamicin. His blood culture was negative. Both bagged and catheterized urine samples grew >100,000 CFU/mL of *Raoultella planticola* resistant to ampicillin (Figure 2). After the culture results, he was placed on ceftazidime. He was then switched to cefazolin per susceptibility profile (Figure 4), and his hyperbilirubinemia started to decrease as shown in Figure 3. The repeat urine culture was negative after 5 days of cefazolin. The patient was discharged from the hospital 10 days after receiving 7 treatments of IV antibiotic. The patient was sent home on oral cephalexin and completed 14 days of total therapy. Renal ultrasound and voiding cystourethrogram (VCUG) were performed to check for congenital anomalies for susceptibility to UTIs and both came back normal. Outpatient follow-up at 27 days of life showed improvement in jaundice and substantial weight gain to 28% above birth weight. Patient has been well and had no further complications at their 6-month well child visit.

Figure 2. *R. planticola* culture from patient grown on MacConkey agar (A) and blood agar (B).



Discussion

While neonatal hyperbilirubinemia is often a benign condition among neonates, it can become life-threatening if in excessive levels with theoretical possibility to cause kernicterus that implicate some chronic neurologic harm. Typically, bilirubin levels peak in the first 3 to 5 days of life and decline over the following weeks.[7] Neonates are

Figure 3. Timeline of events for evaluation and treatment of patient being presented.

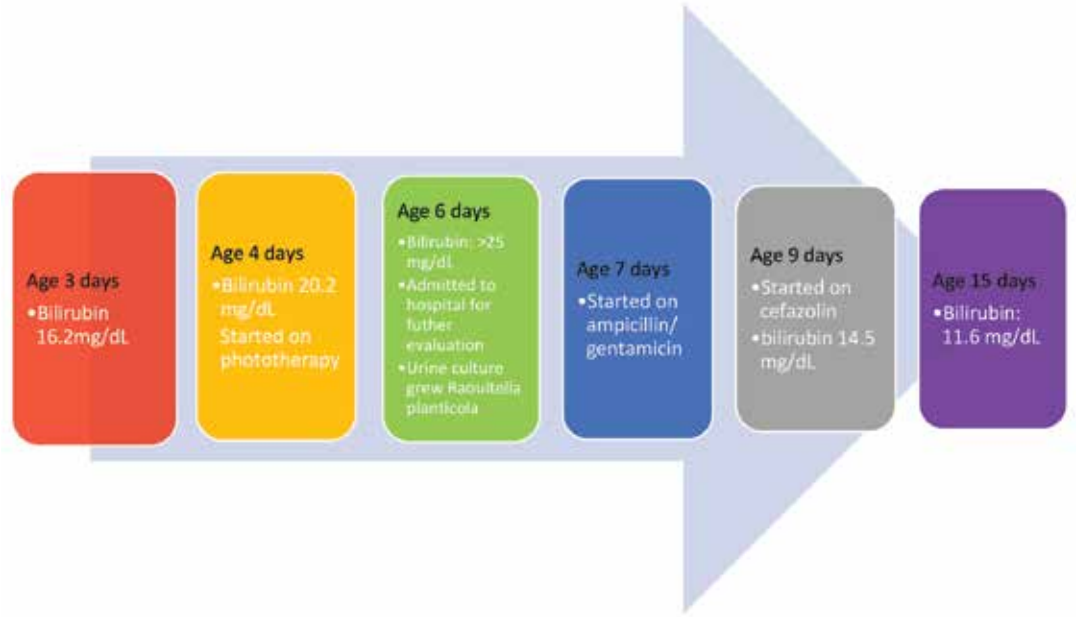


Figure 4. Susceptibility panel for this patients *R. planticola* infection.

Urine Culture Final

Organism 1	Raoultella planticola
Colony Count	>100,000 CFU/mL

	Rao plant M.I.C.	RX
Ampicillin	>=32	R
Ampicillin/Sulbactam	4	S
Cefazolin	<=4	S
Cefepime	<=1	S
Ceftazidime	<=1	S
Ceftriaxone	<=1	S
Ciprofloxacin	<=0.25	S
Gentamicin	<=1	S
Levofloxacin	<=0.12	S
Meropenem	<=0.25	S
Nitrofurantoin	<=16	S
Piperacillin/Tazobactam	<=4	S
Tobramycin	<=1	S
Trimethoprim/Sulfamethoxazole	<=20	S

not considered to have physiologic jaundice if total serum bilirubin levels reach over 17 mg/dL and are not at risk for kernicterus until levels reach over 20 or 25 mg/dl dependent on their risk factors.[7] Bilirubin, at high enough levels, is able to cross the blood brain barrier if unconjugated and can be toxic to the brain causing acute bilirubin encephalopathy resulting in hypotonia, poor suck and eventually progressing to hypertonia and retrocollis. If bilirubin levels are not reduced, long term damage of the basal ganglia and brainstem nuclei primarily possibly causing symptoms such as choreoathetoid cerebral palsy, and an upward gaze paresis.[7]

Up to 80% of infants will have some degree of jaundice.[8,9] Various studies have shown that 5.8% to 21% of neonates with jaundice have a UTI.[2,4,10,11] Hyperbilirubinemia is most commonly treated with phototherapy when the total serum bilirubin levels pass the threshold on the Bhutani curve.[1] The threshold used is determined by neurotoxic risk factors such as gestational age, hemolytic disease of the newborn and fetus, and sepsis.[1] Phototherapy is used with the goal to decrease the likelihood of further increases in TSB concentration that would require an escalation of care[8] Phototherapy duration and rebound hyperbilirubinemia were more prevalent in neonates with UTI in the first two weeks of life.[11] Current treatment recommendations for neonatal UTI are ampicillin and gentamicin because most common agents are enteric pathogens.[4] This patient was empirically started on ampicillin and gentamicin but improved after switching to targeted therapy once susceptibility profile resulted.

R. Planticola is a rare organism to cause UTI in the pediatric population. *Raoultella spp.* is found in soil, water, and plants but is now becoming an emerging pathogen in humans especially among the immunocompromised. An estimated 9 to 18 % of the human population is thought to be colonized by the bacteria. They are known to inhabit the respiratory and gastrointestinal tract.[12,13] *Raoultella* was within the genus *Klebsiella* until 2001, when molecular analysis revealed differences in the 16S ribosomal RNA and RNA

polymerase beta subunit encoding genes instituting the new genus *Raoultella*. Both genera belong to the *Enterobacteriaceae* family. Both species are very similar and have made them almost indistinguishable. A unique feature of *Raoultella* is the enzyme histidine carboxylase, enabling it to produce histamine.[14]

The first case reported of the species was in 1984 in a patient with sepsis.[6,14] With the emergence of the bacteria, over 70 instances of *R. planticola* have been reported, mostly in adults.[14] Infections such as bacteremia, endocarditis, conjunctivitis, oral mucositis, neutropenic fever, and UTIs have been reported in the pediatric population summarized in Table 1.[15] Of the UTIs, three other pediatric cases have been reported in the literature. Similarly, to other pathogens complicated in UTI, *R. planticola* infection risk was associated with advanced age, immunocompromised conditions like cancer and diabetes, and impaired renal function.[10] Two pediatric UTI reports involve predispositions such as congenital urinary tract anomalies and bladder rhabdomyosarcoma.[5,16] Our patient had normal renal ultrasound and VCUG. The third pediatric UTI case was an otherwise healthy 2-month-old who presented with fever, hematochezia, diarrhea, and decreased urine output.[17] The patient discussed had a normal renal ultrasound and VCUG, but being a neonate, he was more susceptible to infections due to a developing immune system and scant immunological memory due to limited exposure to microorganisms while in utero.[18]

Treatment of the three pediatric UTIs and the one presented here all

Table 1. Summary of *R. planticola* cases in the pediatric population.

Author/year of publication	Age of patient	Source of infection (e.g. blood, urine, csf, etc)	Treatment done and duration (e.g. antibiotics used)	Outcome of patient (e.g. recovered, died, etc)
Howell et al. 2017[17]	2 months	Urine	Ceftriaxone x1 dose cefalexin (10d)	No additional fevers and improved oral intake prior to discharge. Full resolution anticipated.
Fukuda et al. 2021[5]	4 months	Urine	IV cefotaxime day 1. Cefazolin days 2-6. Discharged on cephalexin on day 7. Continued low does (10 mg/kg) Cephalexin for 6 months after discharge for prophylaxis	Follow up a week after discharge showed complete resolution of UTI. MRU revealed hydronephroureter with marked dilation of the right ureter, but no ectopic ureter. Dynamic renal scintigraphy showed obstruction of urine in the right kidney. Normal VCUG.
Chen et al. 2020[4]	Post natal day (PND) 26	Blood culture	Cefipime (14d)	Extubated on PND 32 with clinical improvement.
Chen et al. 2020[4]	PND 27	Blood culture	imipenem (21d) meropenem (14d), Amphotericin B(10d)	Full recovery
Chen et al. 2020[4]	PND 20	Blood culture	Meropenem (14d), piperacillin tazobartan (7d), benzacillin (7d), fluconazole(10d)	Was being treated for pneumonia prior to *R. planticola* infection and worsened on day 20. Requiring intubation. Improved on 2nd day of antibiotic therapy.
Chen et al. 2020[4]	PND 9	Blood culture	Meropenem (14d)	Full recovery
Yoon et al. 2015[16]	16 months	Urine	IV cefotaxime and ampixillin/sulbactam (6d) and subsequent oral cefpodoxime (4d)	Three days after initiation of antibiotics, urinalysis was normal with no evidence of pyuria, hematuria, or bacterial growth. Patient was receiving chemotherapy for bladder rhabdomyosarcoma. 6 weeks later had further cystitis due to *E.coli*. He was treated successfully and had no recurrence of UTI after surgical tumor resection for at least 2 years after.
Atici et al. 2018[19]	PND 34, PND 45	Conjunctival swab, blood culture	Netilmicin eye drop (7d) Empiric vancomycin and meropenam. De-escalation to piperacillin tazobactam (14d)	Leukopenia and thrombocytopenia resolved after 48 hours of antibiotic therapy. Extubated on third day of therapy.
Pacilli et al. 2019[15]	8 years	Peritoneal fluid	IV antibiotics (5d) and oral trimethoprim-sulfamethoxazole (5d) after discharge.	Asymptomatic and well at 6 month follow up.
AlSweed et al. 2018[20]	4 years	Blood culture	Ceftriaxone and gentamycin (length not stated)	Patient left during antibiotic therapy (endocarditis case)
Bardellini et al. 2017[21]	16 years	Swab of oral lesions	Amikacin and ceftazidime (8d)	Complete healing of lesions and resolution of symptoms were reached. He was able to complete antineoplastic therapy without further complications. Alive and well at 1 year follow up.

involved using cephalosporins with complete resolution. In three out of 4 cases, *R.planticola* was resistant to ampicillin, and the other was pan-sensitive. *Raoultella spp* contains beta-lactamase, making them innately resistant to penicillin alone.[14] There have been other reports where *Raoultella* has had multidrug resistance species, including carbapenems.[4,22]

Conclusion

In conclusion, we present a case of a neonate with protracted hyperbilirubinemia recalcitrant to phototherapy found to have a *R. planticola* UTI. To our knowledge, this case reports the youngest patient in the literature with a UTI caused by *R. planticola*. *R. planticola* is a potential emerging pathogen in the pediatric population, mostly in the immunocompromised population, with strains that may demonstrate multidrug resistance. This calls for a high index of suspicion among clinicians given the variability of its resistance pattern. This case highlights that a ubiquitous organism such *R. planticola*can be a bona fide pathologic agent for neonatal UTI. It further shows that neonatal UTI can present with resistant hyperbilirubinemia even in a well-appearing baby.

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Children's Nebraska Accredited as Level 4 Epilepsy Center by National Association of Epilepsy Centers

Content submitted by Children's Nebraska

Children's Nebraska, the region's pediatric health care leader, has been accredited as a Level 4 Pediatric Epilepsy Center for 2024 and 2025 by the National Association of Epilepsy Centers (NAEC). Recognized as the highest level of accreditation for epilepsy care, the designation is earned by programs that offer clinical expertise, facilities and capabilities necessary to provide advanced medical and surgical evaluation and treatment. The accreditation is the latest achievement for Children's growing Pediatric Neurosciences team, which offers the region's most comprehensive pediatric epilepsy program and is dedicated to improving the lives of children and families experiencing epilepsy and seizures.

"The level 4 epilepsy center designation attests to our ability to provide the highest levels of comprehensive care to our young patients with epilepsy," said Sookyong Koh, M.D., Ph.D., Children's division chief of Pediatric Neurology. "Children and their families no longer need to travel far out of the state of Nebraska to seek specialty care to adequately control their seizures. I am most grateful for our Neurosciences team for their dedication and effort to achieve this honor and recognition."

Level 4 centers such as Children's provide routine care as well as specialized services to treat the most complex forms of epilepsy, providing intensive neurodiagnostic monitoring; extensive medical, neuropsychological and psychosocial diagnosis and treatment; complete evaluation for epilepsy surgery and a broad range of innovative, leading-edge surgical interventions for uncontrolled seizures.

"The rigorous standard required for level 4 epilepsy certification attests the ability, heart and resolve of Children's Nebraska to provide the highest echelon of treatment and support for children and their families suffering from epilepsy," said Arnett Klugh III, M.D., Children's division chief of Pediatric Neurosurgery. "Harnessing expertise in stereotactic EEG, advanced neuroimaging, robotic assisted surgery, 3D 4K microscopy, neurostimulation and laser therapy, we stand poised to directly impact the quality of care for pediatric epilepsy patients. Our strength resides in collaboration across multiple disciplines, propelling us to serve and lead in pediatric epilepsy care. We are committed to delivering the excellence our patients deserve wrapped in compassion and understanding."

Children's Neurosciences team collaborates in its specialized epilepsy care from Children's Neurosciences Center, led by division chief of Pediatric Neurosurgery Arnett Klugh III, M.D., division chief of Pediatric Neurology Sookyong Koh, M.D., Ph.D., surgical director of epilepsy and pediatric neurosurgeon Afshin Salehi, M.D., M.S., and epileptologist Spriha Pavuluri, M.D. The team comprises three neurosurgeons, eight neurologists including three sub-specialized epileptologists, seven advanced practice providers, dozens of nurses and many more support team members. It partners with colleagues in the Epilepsy Monitoring Unit, Hospital Medicine, Neurodiagnostics, Neuropsychology, Neuroradiology, Nutrition, Surgical Services and more to deliver the best outcomes and experiences for children and families.

Take the Survey – Why Did You Pursue a Career in Healthcare?

Fran Rice
Executive Director, Health Connect

Health Connect of South Dakota recently received a Beyond Idea Grant from the SD Community Foundation to bring together educators, healthcare professionals, and youth to develop a framework for developing a Healthcare Careers Exploration Program for Out of School Time Programs (HCEP-OST) for South Dakota. The catalyst for this project is recognizing that there are healthcare disparities in South Dakota. People of color and rural populations face healthcare disparities and inequity due to obstacles based on race, ethnicity, socioeconomic status, education, access and geographic location. Multiple research studies show 1) a direct correlation between the lack of diversity in healthcare, racial and ethnic healthcare disparities in accessibility, and patient experience and satisfaction, as well as 2) a steady decline in the number of rural medical students and interest in practicing in rural settings.

Rural, indigenous, and youth of color or ethnicity generally do not have access to structured opportunities to explore healthcare careers, a contributing factor for the lack of diversity in healthcare. It is well known that youth begin planning their professional pathways during their K-12 years. Career exploration opportunities are common for 6-12 grades and rare at the elementary level. Due to curricular demands, it is very challenging to provide in-depth healthcare career exploration programs in the formal classroom, especially at the elementary level. Out of school time programs are in a unique position to fill this gap. They can serve as a link to healthcare professionals and institutions that enables youth to explore healthcare careers in a safe setting. To our knowledge, there is not an evidence-based healthcare careers education program specific for OST, with a focus on grades 3-5.

Through literature review, and surveying and small group discussions with multiple populations - healthcare professionals, out-of-school time professionals, youth, and the general public - we seek to:

1) Learn of barriers and challenges to pursuing a healthcare career
2) Learn of support systems that enable youth to pursue a healthcare career
3) Learn what inspires individuals to pursue a healthcare career
4) Learn if and how South Dakota out of school time programs are integrating healthcare related activities and pathways
5) Learn what support is needed for out of school time programs to implement a healthcare careers exploration program

Information gathered from the focus groups, surveys, and literature review will contribute to the creation of a HCEP - OST framework. This framework will be used to evaluate existing programs, resources, curriculums, recommend adaptations for the OST setting, and identify gaps to address when developing a coordinated HCEP-OST program.

Gaining insight into what drives younger youth to choose healthcare as a career will dovetail quite nicely with other state initiatives that are mainly focused on youth in middle or high school.

From this data, we aim to have these questions answered. Where does the excitement for healthcare begin? What helps it to begin? During play time? Within school hours? After school hours? What is a great age to begin the discussion? Who will be the motivator at an OST program for youth? Can we begin a cohort of healthcare dreamers at younger ages?

As healthcare professionals your input into this research is welcomed. Please take this 3-5 minute survey and let us know about your journey into healthcare and how you are able to share your love of healthcare!

Healthcare Professionals

https://www.surveymonkey.com/r/OSThealthprofessionals

This page is sponsored by Health Connect via a grant from the South Dakota Community Foundation. The grant aims to develop a framework for developing a Healthcare Careers Exploration Program for Out of School Time Programs (HCEP-OST) for South Dakota.

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South Dakota State Medical Association Political Action Committee

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Read coverage of the 2023 SDSMA Annual Leadership Conference in next month's issue of *South Dakota Medicine*, online at sdsma.org, and in email updates.

Jen Tinguely, MD, MPH Sworn in as SDSMA President

Jennifer J. Tinguely, MD, MPH, of Sioux Falls became the 143rd president of the SDSMA on May 31, 2024. She is a family medicine physician and chief medical officer of Falls Community Health in Sioux Falls. She has been involved in leadership in healthcare for many years. She has held positions on the SDSMA's Board of Directors, Policy Council, committees, her local District 7 Medical Society, and is a member of the American Medical Association. Dr. Tinguely is also a graduate of the SDSMA's Health Leadership Institute. During the state legislative session, Dr. Tinguely has volunteered for the SDSMA's advocacy efforts by participating in the Doctor of the Day program at the state capitol in Pierre and providing testimony on bills related to gun safety. Prior to her medical training, she received a Master of Public Health degree from Boston University with a focus on maternal and child health and Native American health disparities. She worked for four years in the public health arena at Columbia University Mailman School of Public Health in New York City before moving back to South Dakota to receive her medical degree from the University South Dakota School of Medicine and completed her residency with the Sioux Falls Family Medicine Residency Program. Dr. Tinguely completed additional training on buprenorphine prescribing and is a strong advocate of medication assisted treatment for patients with substance use disorders. She is part of a post-overdose response team in Minnehaha and Lincoln counties. In April 2024, she was appointed to the South Dakota Board of Addiction and Prevention Professionals. Dr. Tinguely is a clinical associate professor

in the Department of Family Medicine at the University of South Dakota Sanford School of Medicine and is the course director for the Pillar 2 Cultural Immersion course.

Legal Brief Highlight: Limitations of Actions

A medical malpractice action may be brought against either individual physicians or professional corporations within two years after the alleged malpractice occurred, with a few exceptions.

A person making a claim based on an alleged error or omission that occurred while he or she was a minor must commence an action within two years of the alleged malpractice or before his or her 19th birthday, whichever period is longer. Because a minor need not bring a malpractice action until after they become an adult, all records pertaining to the treatment of minors should be kept beyond their 19th birthday. This rule should apply even if the physician-patient relationship has been terminated prior to that time.

Exceptions exist for continuing injury to the patient, continuing treatment of the condition, and if the physician intentionally conceals the condition or the cause of the condition.

For more information, download the SDSMA legal brief *Limitations of Actions* at sdsma.org. The SDSMA provides legal briefs for member physicians on a variety of rules and regulations that apply to the medical profession. For non-members, legal briefs are available for purchase.

Member News

For Your Benefit:

Membership Services from the SDSMA

SDSMA Membership Services staff work 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 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 SDSMA's monthly update on programs and services available to physicians through their affiliation with the SDSMA.

USD SSOM Graduation

Sixty-one new physicians graduated on May 3 from the University of South Dakota Sanford School of Medicine in the class of 2024. At the ceremony, SDSMA President Denise Hanisch, MD, led the Affirmation of the Physician, where this group of dedicated doctors pledged their unwavering commitment to uphold the core values of the medical profession. Wishing the graduates the very best in their journey ahead!

Keep Your Information Up-to-Date: Log on Today

In order for the SDSMA office to provide members with timely information, it is important that members regularly review their contact information on file with the SDSMA. Have you changed practice locations? Is your email correct? Is your mail going to the right place?

Visit sdsma.org and log into your secure online account. Please take a few minutes to review your profile and make any necessary updates. Updating your secure account keeps your information up to date and notifies the SDSMA of any changes so you are accurately listed in the member directory and ensures that your membership materials, emails and renewal notices are sent to the appropriate mailing and email addresses.

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

Help Shape the Future of Medicine in South Dakota

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

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

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

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