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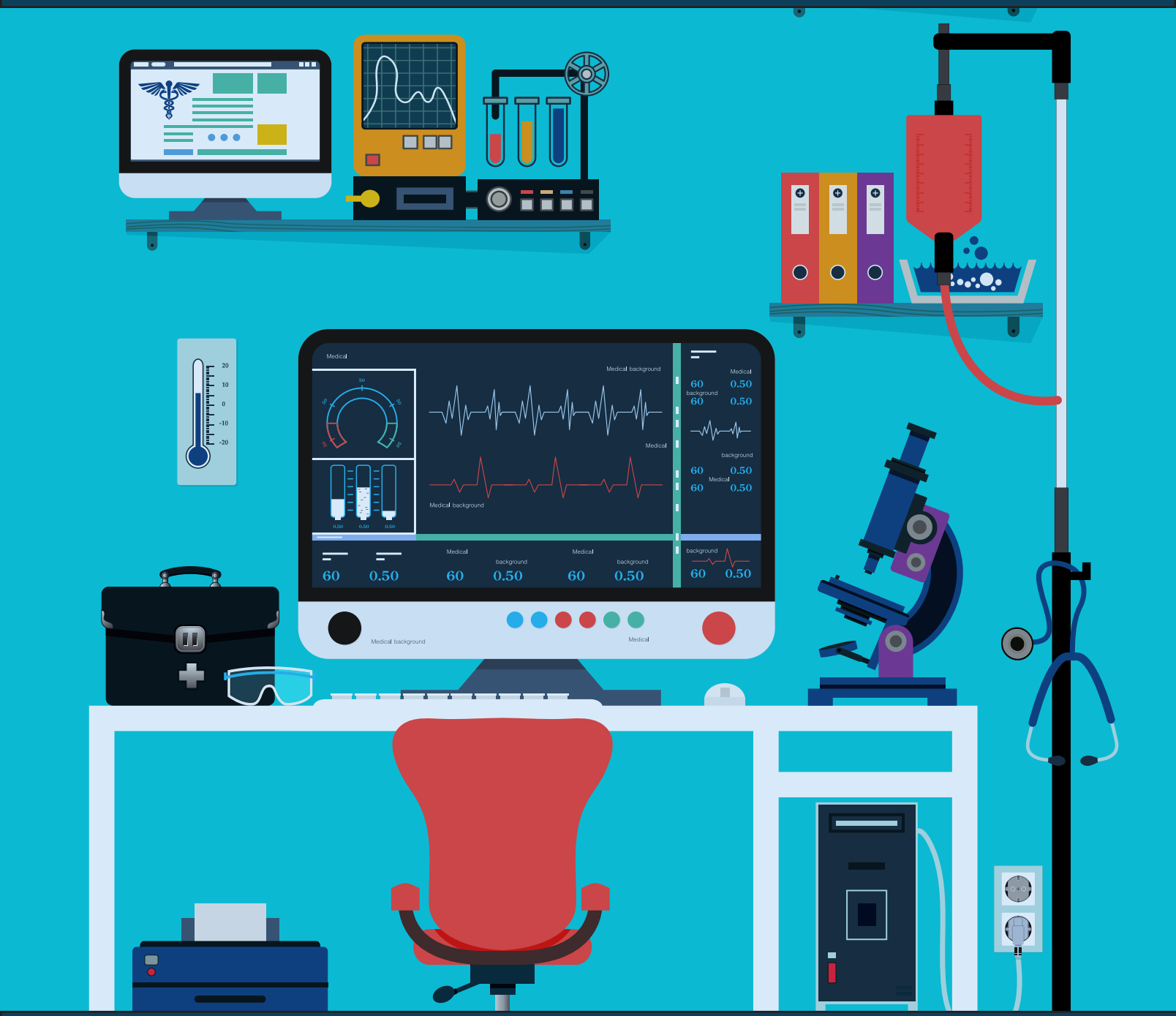

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by SANDRA JOHNSON, MD, FAAD

Proud to be an Arkansan

I am proud to be an Arkansan. Those are words I would not have predicted myself saying before 1995. I grew up in Ohio and planned to be a Buckeye for life. As a good young American, I was not very good at geography and was not even sure where Arkansas was on the map. However, as Woody Allen says, "If you want to make God laugh, then tell him your plans." I moved to Arkansas in 1996, met the love of my life, and made Arkansas home. Fast forward to Easter weekend April 2020: I am saying the words "I am proud to be an Arkansan" regularly and writing an editorial telling anyone who would listen (or read) that I am proud to be an Arkansan.

We are in the midst of the Covid pandemic. We are social distancing. We are trying to flatten the curve. We are living a new normal. We are trying to provide essential medical care. We are rationing personal protective equipment. We are counting medical personnel and ventilators. We are paying less than $2.00 per gallon of gas. We are hoping to avoid a recession. We are seeing people lose their jobs. We are homeschooling our kids. We are living with our college-aged children who we picked up from college last month. We are all living a new normal. We are all in this together, but alone.

So, with this pandemic, why am I regularly thanking God that I am an Arkansan? Well, because we are all in this together, but alone. We have phenomenal leadership. We (Brad and I) enjoy watching the daily press conferences from our amazing governor. Gov. Asa Hutchinson is the bomb. He is navigating these uncharted waters with ease. He presents the details, creates the plans, encourages us to make good choices, and makes us all feel comfortable with this new normal.

Every day, the governor highlights another great Arkansan who is doing his or her part to help all of us. One of those great people is Heather Larkin, president and CEO of the Arkansas Community Foundation. She created an account and secured a lot of funds to help Arkansans in need. We have a selfless, hard-working director of the Arkansas Department of Health, Dr. Nate Smith. Dr. Smith is making hard choices and doing it skintastically. Many people from UAMS, the governor's office, ADH, the Legislature, etc., are leading by example in both word and deed. We are all wearing masks to limit spreading contagions and, as Gov. Hutchinson said, we are doing it with style (#fashionweek).

My favorite hero through all of this is my brother-in-law, Lee Johnson. I do not know how many hats he wears, but he wears them all well. He is an emergency physician, a legislator, director of emergency medical services for our community, and a local medical expert for the media. He rises to every challenge and does it with kindness and grace. It would be easier to count the hours he has not worked or volunteered during the past two months than to count the number of hours he has worked. He is an inspiration to me. His wife and family are also sacrificing and inspiring Arkansans, including me.

If my favorite daily show is the governor's press conference, my favorite nonprofit is Arkansas Community Foundation, and my favorite hero is Lee, then I should also state that my favorite source for information is the AMS Covid-19 Daily Update. We have all been bombarded with Covid information from so many sources, it became difficult for me to look at all of it. The AMS Covid-19 Daily Update provides clear, concise, correct information that is practical to me as a practicing physician and business owner. I appreciate the concise reviews with URL links to more information. This update helps me to navigate telemedicine, loan applications, PPE availability, number of Covid cases, and more.

I could elaborate for a long time about why I am proud to be an Arkansan and who my favorite Covid heroes are, but I have a word limit. I would enjoy hearing why all of you are proud to be Arkansans and who your favorite Arkansas heroes are during this Covid pandemic. As Mr. Rogers says, "Look for the helpers." The helpers are the true heroes. Thank you for being a helper and a hero. Not all helpers and heroes wear capes; some wear white coats and scrubs. Stay safe and healthy and skintastic.

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by SANDRA JOHNSON, MD, FAAD

So You Want to Open a Med-Spa?

"First do no harm" is the oath we take as physicians. We are all so blessed to be physicians. Personally, I am honored to be a dermatologist. I love skin and everything about skin. I especially enjoy both the art and science of dermatology. I am writing this article in response to being repeatedly asked by entrepreneurs, estheticians, physicians, and nurses, what the best way is to open a medi-spa or laser center. I have decided to assemble my thoughts and share them with all of you. My first reaction is flattery. I wonder if you want to do this because it looks so easy and so fun. Is it because at Johnson Dermatology, the only people who do the injections are two board-certified dermatologists and a nurse practioner who works very closely with me? Is it because we have more than 15 laser, light, and energy devices? Is it because we are one of the top 150 practices in the country for Allergan, and we do not use breast implants or cool sculpting or cool tone? Is it because we are in the top 1% of practices in the country for Evolus? Is it because we are the #1 practice in Arkansas for Colorescience and the #7 practice in the U.S. for Skin Medica?

After I stop being flattered and prideful, the first question I want to know is why? What is your motivation? Do you love skin? Do you enjoy doing these procedures? Is it for the money? Are you unhappy with what you are doing and think this will make you happy? Whatever your reason, I encourage you to understand the state laws. Since I am fortunate to practice in the great state of Arkansas, I will limit the following to what I understand about our state laws. I will also state that I do not understand the laws from the dental board (their website is https://www.asbde.org/) or the nursing board (their website is https://www.ark.org/bon/licensure/status.php). Station THV11 in Little Rock addressed this issue on November 25, 2019, and stated that the Arkansas Dental Board does not have guidance on this matter. Both the Arkansas State Medical Board and Arkansas State Board of Nursing are currently trying to address these important issues with new regulations. I will gladly share these proposed and currently active regulations with anyone who wants to contact me.

The Arkansas State Medical Board is the organization that has given me my medical license and that has given the facility where I practice a medical license. The American Board of Dermatology has given me board certification in 2000, 2010 and 2020. The Arkansas Medical Act and Regulation was last revised by our Arkansas State Medical Board in August 2018. It clearly states that in our great state, all medical practices must be owned by a medical doctor, according to Regulation 4-29-305. For example, I need and have two medical licenses for the state of Arkansas. One is for me personally to practice medicine, and one is for Johnson Dermatology for the practice of medicine to occur at our location. This means that a nurse, entrepreneur, or esthetician cannot own a medical practice in the state of Arkansas since they cannot apply for the practice of medicine at their location as I understand the law.

If you are a physician who wants to open and run a medi-spa in the state of Arkansas, there are a few other rules and laws that I have learned. One is that every patient must be seen and evaluated by a licensed medical provider such as a physician, nurse practioner, or physician assistant before a laser treatment or injection. I do not know what the law is for dentists. This means that realistically I could not own a practice in Mena where an esthetician is injecting Botox Cosmetic (a medical procedure) or performing laser hair reduction (a medical procedure) on a patient while I was seeing patients at Johnson Dermatology in Fort Smith. I also legally should not purchase fillers for a registered nurse to inject them at another location where I do not see the patient first. Also, a registered nurse cannot inject or laser a patient unless that patient is first evaluated by a physician or physician assistant or nurse practitioner. Evaluating a patient for injectables or laser is considered the practice of medicine, and registered nurses do not have this in their scope of practice.

Another interesting fact I learned many years ago is that most lawsuits for laser procedures are performed by non-core physicians or their delegated person. The article on the subject is titled "Common Causes of Injury and Legal Action in Laser Surgery," by H. Ray Jalian, MD; Chris A. Jalian, JD; Mathew M. Avram, MD, JD in JAMA Dermatol. 2013;149(2):188-193. Before you decide to add these procedures to your practice, you may want to confirm that you will have malpractice insurance coverage, if that is important to you. I am not sure what the law is for this, but I do know that I am covered for these procedures from our malpractice insurance carrier since the 1990s, State Volunteer Mutual Insurance Company.

The American Med Spa organization also has clear guidelines about the practice of medicine within a medical spa. Here is a link to an article from their website: https://www.americanmedspa.org/news/169407/Want-to-be-a-Medical-Director-at-a-Medical-Spa-Beware.htm

The American Academy of Dermatology has a position statement about medi-spas that reads, "Medical spas are facilities that offer a range of services, including medical and surgical procedures, for the purpose of improving an individual's well-being and/or appearance. The distinguishing feature of medical spas is that medicine and surgery are practiced in a non-traditional setting. Procedures by any means, methods, devices, or instruments that can alter or cause biologic change or damage the skin and subcutaneous tissue constitute the practice of medicine and surgery. These include but are not limited to the use of: scalpels; all lasers and light sources, microwave energy, electrical impulses, and all other energy emitting devices; thermal destruction; chemical application; particle sanding; and other foreign or natural substances by injection or insertion. Any procedure that constitutes the practice of medicine, including but not limited to any procedure using a Food and Drug Administration (FDA)-cleared or regulated device that can alter or cause biologic change or damage, should be performed only by an appropriately-trained physician or appropriately-trained non-physician personnel under the direct, on-site supervision of an appropriately-trained physician in accordance with applicable local, state, or federal laws and regulations."

As a dermatologist, I was trained during and after my four-year dermatology residency about skin and procedures affecting the skin. I like my scope of practice. I do not choose to treat the common cold or broken bones. Anything outside of dermatology is outside of my scope of practice. Even though I was trained to perform Mohs surgery and blepharoplasty in dermatology residency, I choose not to include these procedures in my scope of practice because I do not regularly perform them. I also choose not to collaborate with a nurse practioner or physician assistant for anything outside of my scope of practice. I am a board-certified dermatologist, which means I am trained to treat healthy and diseased skin. I love skin.

One final thought: the longer I practice, the more I learn that outcomes and experience matters. My mother-in-law humbled me many years ago; after I have been injecting and practicing for about five years, she told me in a matter-of-fact way that she thought I was finally getting good – that my injections were hurting less and the results better. There is definitely a learning curve to everything. I have also learned that any injector can have complications and problems. I pray that I never have a serious complication. However, if I do, I hope I will be able to stay with that patient, understand and identify the issue, and do my best to make it right. If you are still considering adding injections or lasers or other cosmetic treatments to your scope of practice or are considering working with a medi-spa, below are some links to other websites you may want to peruse. If you are considering being a patient who gets cosmetic treatments, I encourage you to make sure you are going to a licensed facility and being treated by a trained, licensed professional. You will want to know how many years they have been performing those procedures and how many of those procedures they perform per day. You will also want to know how any complications will be managed, how you will contact them for any complications, etc.

My final wish is that you always feel and stay skintastic. I wish every patient health and happiness. I wish you happiness and peace. Thank you for allowing me to share some of the facts and regulations and some of my opinions with you.

- https://www.aad.org/Forms/Policies/Uploads/PS/PS-Medical%20Spa%20Standards%20of%20Practice.pdf
- https://www.surgery.org/consumers/consumer-resources/consumer-tips/patient-safety-advisory-on-medi-spas
- https://www.naplesnews.com/in-depth/news/special-reports/2019/01/30/miami-doctors-plastic-surgery-empire-becomes-floridas-deadliest-clinics/2462068002/
- https://www.americanmedspa.org/news/169407/Want-to-be-a-Medical-Director-at-a-Medical-Spa-Beware.htm
- https://www.littlerockcosmeticsurgery.com/medi-spa-arkansas/
- https://www.hairfacts.com/wp-content/uploads/sites/5/2010/04/aada-state-regulation-of-medical-spa-facilities-toolkit.pdf

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The following physicians have joined AMS in March and April 2020.

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Cynthia Morgan, MD
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Do you know other physicians who are not a members? We encourage you to reach out to your peers and tell them what the Arkansas Medical Society means to you and why they should join you.

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BY MUSA YILANLI, MD;[1] APARNA DAS, MD; [1] VERONICA RANEY, MD; [1] NIHIT KUMAR, MD [1]

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Treatment with Hydroxyzine for Paradoxical Vocal Cord Dysfunction

Abstract

Paradoxical vocal cord dysfunction (VCD) is associated with several psychiatric conditions. Early intervention for ongoing anxiety problem is reported to be beneficial for patients in reducing symptoms of VCD. While there is extensive literature supporting the use of psychological interventions, the evidence for the use of medications is limited. The aim of this case report is to expand the evidence base about the medication options that can be used to treat VCD. We present a case with paradoxical vocal cord dysfunction who was successfully treated with hydroxyzine and psychoeducational intervention.

Introduction

Paradoxical vocal cord dysfunction (VCD), or paradoxical vocal-fold motion, (PVFM) is an insufficiently understood medical condition. It was first described by Patterson et al. in 1974, who named it Munchausen's stridor[1]. It is a functional disorder of the vocal cords that leads to acute upper airway obstruction.[2] The presentation of the disease frequently mimics an episode of asthma as patients experience several similar symptoms including coughing, breathing problems, or inspiratory stridor[2]. The exact incidence and prevalence is not known as the condition is frequently misdiagnosed as asthma or other medical condition, or remains undiagnosed. Based on the available literature, certain trends are noted; there is greater prevalence in females[2, 4, 5], and there is wide age range from 14.5 to 33 years for the occurrence of this disorder[2].

It is important to get a detailed physical history with physical examination and extensive work-up to rule out laryngeal causes of VCD such as paralysis, granulomas, or airway malacia. Once the organic causes have been ruled out, the triggers for PVFM may be broadly divided into psychological factors (accounting for 70% of the cases) and hypersensitivity reaction or other neurological disorders (accounting for the remaining 30% of the cases).[2,5] A multidisciplinary approach is essential to the management of PVFM/VCD dysfunction. Most teams have pulmonologists, otorhinolaryngologists, speech and language pathologists, and psychologists or psychiatrists.[5] From a psychiatric standpoint, it is important to do a comprehensive psychiatric evaluation taking into account and thoroughly investigating a history of clinical depression, history of abuse and personality disorder, and other somatoform disorders.

Treatment for VCD begins with accurate diagnosis and subsequent family education about the disease. Alleviating symptoms is highly recommended before dealing with disease-related stressors because the new medical diagnosis and poor symptom control may easily cause significant anxiety. There is extensive evidence for several psychotherapeutic treatment options including biofeedback, hypnosis, and cognitive-behavioral therapy (CBT). However, there is dearth of literature regarding the use of medications for the management of PFVM. We present a case of paradoxical VCD that was successfully treated with hydroxyzine along with early psychoeducational intervention.

Case Report

A 16-year-old Caucasian male with history of seasonal allergies and sports-induced asthma presented to Emergency Department (ED) with intermittent choking spells, coughing, and difficulty swallowing. Patient reported that his symptoms had started as daily dry cough and then he had started experiencing choking episodes that lasted about a minute. During these episodes, patient felt he could not breathe and his throat was closing up. His symptoms had gradually worsened, and he had started to fear drinking and eating. After the first emergency visitation, esophagogastroduodenoscopy (EGD) was done that showed erosive changes and the presence of an esophageal stricture, which was dilated during EGD. Patient was diagnosed with Gastro esophageal reflux disease (GERD) and was started on omeprazole, sucralfate, and hyoscyamine. After the EGD, patient continued to avoid eating and drinking due to fear/feeling of food getting stuck. Patient then started to experience choking episodes at night time that woke him up; these episodes were accompanied by intense fear. Patient presented back to ED when Otolaryngologist/ENT was consulted and flexible scope done, which revealed healed pharyngeal lesions with significant inflammation and post cricoid edema. Vocal cords were mobile and the supraglottic anatomy was normal. Patient was sent home with reassurance. The following day, patient presented to a different ED with complaint of obstruction on the left side of his throat that he could feel when swallowing. Patient was admitted to general pediatrics for further investigation. He was put on clear liquid diet briefly and his upper gastrointestinal study revealed normal anatomy. Patient was then switched to regular diet and psychiatry was consulted for evaluation of anxiety as a potential contributor to his symptoms. During evaluation, patient endorsed somatic symptoms as well as symptoms of social anxiety. He stated he did not want to lie down supine on the bed, which could exacerbate his spasmodic episodes. He was interested in taking an as-needed medication to help him deal with his fear and so was started on hydroxyzine 25 mg at bedtime for anxiety and sleep. On follow-up the next day, patient stated he was able to sleep better with this medication and he only had one mild episode during the night. Speech therapist worked with the patient on breathing and relaxation techniques to help during laryngospasm episodes. During hospitalization, patient and family education with constant reassurance was provided by psychiatrist. Patient was discharged after three days of hospital stay. Patient was contacted over the phone a week after his discharge for follow up and he denied having any further episodes.

Discussion

Our patient received a new diagnosis of GERD with a prior history of sports-induced asthma and seasonal allergies. Even after being started on treatment of GERD, he continued to have intermittent choking episodes that would wake up him up from sleep, and his condition continued

to worsen. ENT evaluation ruled out local causes of obstruction, and speech pathology diagnosed him with VCD. It appears that GERD perhaps acted as a trigger for paradoxical VCD in this case, but it did not explain the worsening choking spells. Eventually, the psychiatry team assessed him and started him on hydroxyzine. Patient was also provided psychoeducation about the disease and the possible relationship with anxiety and sleep problem by psychiatry. In general, the management of VCD requires a multidisciplinary approach, which helps to provide appropriate diagnosis and treatment of the disease. Treatment team usually consists of an allergist, gastroenterologist, otorhinolaryngologist, a pulmonologist, a speech language pathologist, and a psychologist and/or psychiatrist. It is important to evaluate for and treat co-morbid conditions like GERD or asthma. Once organic/anatomical factors are ruled out, the patient should be evaluated for psychiatric conditions.

On review of literature, it was found that the prevalence of psychological/psychiatric comorbidity in patients with VCD has been reported to be as high as 75%.[6] Some of the psychiatric conditions associated with VCD are depression [7], anxiety[8], post-traumatic stress disorder, conversion disorder, and primary and secondary gain related to somatoform disorders[8]. In a retrospective study involving 160 patients, Dietrich et al.[9] analyzed the distribution and frequency of perceived stress, anxiety, and depression in patients who presented to their voice disorders clinic with various voice disorders (including VCD). The authors reported that VCD patients had the highest prevalence of stress, anxiety, and depression. Most voice disorders were more common in females as compared to males, but males with VCD had a much higher rate of comorbid anxiety and depression.

Many studies have supported the use of various types of psychological interventions/ psychotherapies like cognitive-behavioral interventions, hypnotherapy, biofeedback, speech therapy, and psychotherapy[2,4,5,6]. Psychotropic medications have been used but sparingly, mostly for the management of underlying psychiatric comorbidity. Benzodiazepines has proven successful in some patients with VCD who have underlined anxiety problem[3]. In treating 62 patients with VCD, Varney et al.[5] reported generally positive experience with low-dose amitriptyline, required mean dose was 20 mg, in conjunction with psychotherapy and behavioral therapies. Brown et al. [7] reported an adult with VCD along with depression and psychogenic amnesia who was treated with psychotherapy and oral desipramine. One case report by Thurston et al.[6] reported that one patient was successfully treated by a single psychiatrist using a combination of psychotherapy and high dose of venlafaxine with lithium augmentation.

Our patient reported some anxiety symptoms during the interview. Patient also complained of anticipatory anxiety related to difficulty swallowing food and multiple episodes of breathing difficulty at night. The frequent ED visits were an indicator of severe impairment. Patient had significant sleep initiation problem. Hence, after reviewing the overall condition, it was decided to initiate hydroxyzine to help with sleep and anxiety. Hydroxyzine is a first-generation antihistamine that has anticholinergic antihistaminic, antiemetic, antispasmodic, and anxiolytic properties.[10] Its selective anti-histamine profile leads to the sleep-promoting benefits of H1-receptor blockade with minimal anticholinergic side effects.[10]

Although the exact mechanism of action is unknown, hydroxyzine also plays a role in the management of anxiety. Our case report show that hydroxyzine can be a safe anxiolytic option. The adverse reactions profile of the hydroxyzine, well described in the literature, is limited, with dizziness, drowsiness, blurred vision, dry mouth, stomach upset, and headache. The most prominent side effect is sedation.[10]

Conclusion

Paradoxical VCD is a functional disorder whose management requires a multidisciplinary approach. Evidence on the use of psychological interventions exists, but there is paucity of literature on the use of psychotropic medications. At this time, there are no established guidelines to treat patients with VCD. Our case report indicated that hydroxyzine could be a good treatment option for VSD and can be used with minimal side effects. More research is needed to explore the use of psychotropic medications in VCD.

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A CLOSER LOOK AT QUALITY

Pediatric Osteoporosis Management is Critical

LAURA J. HOBART-PORTER, DO, FAAPMR

Osteoporosis is a condition commonly ascribed to elderly people, but it can also be a serious condition in children. The incidence of pediatric osteoporosis is not known, but there are several disease processes that place children at higher risk. Appropriate management of pediatric osteoporosis is critical, given that 90% of bone density is determined by puberty, and consequences for missing it can be debilitating and life long.[1]

Bone development begins in fetal life and is influenced by maternal factors including substance exposure (smoking, caffeine, alcohol), vitamin D deficiency, activity level, intrauterine growth restriction and diabetes, all of which adversely influence bone development. As a child develops, prematurity, genetics, diet, mobility, chronic disease and medications also convey an increased risk of lower bone density.[2,3]

The primary means of assessing bone density in children remains pediatric dual-energy X-ray absorptiometry (DXA). For children, studies should be ordered at a facility capable of controlling for age and stature for a more accurate measurement. Children should be able to lie flat on an exam table. Significant contractures, hip dislocation and the presence of orthopedic instrumentation limit the data that DXA can collect. There are no reliable norms for children under age 3. Children weighing less than 20 kg. or smaller than 115 cm. may not have references available for their size, rendering data of little utility. Consistent measurement approaches from year to year allow reliable monitoring of disease progression. Measure DXA annually in at-risk populations.[3]

There are other means of assessment beyond DXA, but lack of age and stature-related norms makes interpretation difficult. Plain radiographs can be used to estimate bone density in younger children, but this is a highly subjective method without normative data. Quantitative computed tomography, quantitative ultrasonography and magnetic resonance imaging offer an assessment of bone structure in addition to bone density itself.[3] However, these are most often employed in research settings. Lack of normative data is a limitation in the pediatric population.

In 2013, the International Society of Clinical Densitometry published a Pediatric Position Statement, delineating the differences between adult and pediatric osteoporosis. Osteopenia in adults is defined as DXA Z-score between -1 and -2.5; osteoporosis is defined as DXA less than 2.5. In children, a Z-score less than -2 is called "low bone density," not osteopenia. Z-scores less than or equal to -2 with clinically significant fracture(s) meet criteria for osteoporosis. The Society defines clinically significant fractures as one or more vertebral compression fractures, two or more long bone fractures by age 10, or three or more long bone fractures up to age 19.[2]

When low bone density (LBD) is identified, it is important to address the etiology. Laboratory tests of alkaline phosphatase, osteocalcin, type I procollagen, calcium, phosphate and vitamin D-25OH can help determine if LBD is related to a modifiable risk factor. Low vitamin D levels are common within pediatric populations. Levels less than 20 ng/mL are considered low; borderline low is less than 30. Children with disabilities that contribute to LBD may need vitamin D levels ranging

from 60-100 ng/mL. Vitamin D level should be assessed every six months in children receiving targeted supplementation or those with LBD. Recommended daily allowance is 400 IU per day for infants <12 months and 600 IU per day for older children. The vitamin D dose may need to be several thousand units daily to achieve target serum levels in children with clinically significant LBD.[4,2]

Additional modifiable risk factors related to bone density include soda consumption and weight-bearing exercise.[1] Soda consumption can interfere with absorption of vitamin D and calcium. If children are taking these supplements, they should not drink soda. Weight-bearing exercise is more challenging in children with mobility impairments (such as cerebral palsy). Only minimal gains are reported with use of a static standing frame in this population. It should not be utilized as the sole method to improve or maintain bone density in those with impaired mobility.[4] Exercise is important to improve bone density, but it comes with increased risk for those with LBD. Even getting dressed can result in fracture. Range of motion exercises should be done cautiously with LBD children. The child's therapists and other professionals should be informed of LBD, as this can impact care plans. A physical medicine and rehabilitation specialist (PM&R) can provide detailed recommendations to therapists.

Osteogenesis imperfecta is known to cause fracture-prone bones. Other pediatric conditions can result in osteoporosis,[2,1] including connective tissue disorders, such as Marfan's syndrome and Ehlers-Danlos. Inflammatory conditions, such as juvenile rheumatoid arthritis, lupus and inflammatory bowel disease can decrease bone production and increase resorption. Impaired absorption of calcium and vitamin D can occur in children with chronic kidney or liver disease, milk allergy, cystic fibrosis, short gut or celiac disease. Hormonal imbalances can adversely impact bone health, including abnormal corticosteroid production, hypogonadism, hypothyroidism, Turner syndrome, idiopathic short stature and exercise-induced amenorrhea. Those with prolonged immobility, asthma, hemophilia and anemia are at risk for LBD due to lack of activity. Children with impaired mobility related to cerebral palsy, spina bifida, spinal cord injury, brain injury or muscular dystrophy are at high risk of LBD.[5,4] Pediatric cancer is strongly associated with LBD, due to prolonged immobility and side-effects of essential medications. A fracture in any of these populations can be devastating, impacting mobility and function in an already impaired child.

Medications are a frequent iatrogenic cause of pediatric osteoporosis. Often, these are unavoidable as the conditions they treat (cancer, seizures, etc.) are more detrimental than osteoporosis. Corticosteroid use is often required to treat inflammatory or autoimmune conditions, and care must be taken to address additional modifiable risk factors. Some medications may be substituted, particularly in individuals at high risk of fractures. For example, the use of depot medroxyprogesterone for menstrual suppression or birth control is ill-advised in a child who has spina bifida.[6,7] Other medications that may lower bone density include seizure medications, proton-pump inhibitors, loop diuretics and oral anticoagulants.

In those with LBD, medication management and exercise recommendations are essential.[4] PM&R specialists can help with activity and exercise recommendations. If vitamin D supplementation is insufficient, refer to an endocrinologist. Bisphosphonates, which can be helpful in the adult population, are not as well studied in pediatrics. The choice to use these medications is best left to a specialist in bone health. ▲

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Unrecognized Baclofen Withdrawal Secondary to Inaccurate Medication Reconciliation in a Spinal Cord Injury Patient with an Intrathecal Baclofen Pump

Abstract

Baclofen therapy is often utilized to treat spasticity in patients that have suffered neurologic insult. In this case, a 39-year-old man with spastic quadriplegia presented with fever, abdominal pain, nausea, and hematemesis 24 hours after intrathecal baclofen (ITB) pump replacement. Initial work-up was negative, and ITB pump was confirmed to be functioning properly. Later, it was discovered that the patient had been taking oral and intrathecal baclofen, and was in acute baclofen withdrawal. Baclofen withdrawal has a non-specific array of symptoms and can easily be misdiagnosed. Review of dosing habits can prevent adverse drug events and prolonged hospitalizations.

Case Diagnosis

A 39-year-old man with spastic quadriplegia secondary to complete spinal cord injury presented to the emergency department (ED) 24 hours after intrathecal baclofen (ITB) pump replacement with complaints of fever, abdominal pain, nausea, and coffee ground emesis. Routine work up for infection and abdominal pain was notable for a urinary tract infection (UTI) and bowel impaction. Due to the severity of symptoms, he was admitted and started on empiric antibiotics. Despite treatment with antibiotics, his fever, hypertension, and tachycardia did not improve. The surgical site was evaluated, and no wound infection or surgical complication was found. The patient then underwent bowel disimpaction for suspected autonomic dysreflexia, but this also failed to alleviate his symptoms. Within 24 hours of admission, he had developed mental status changes with hallucinations. The Physical Medicine and Rehabilitation team was consulted to interrogate the ITB pump for possible pump malfunction, and it was found to be functioning appropriately. On review of the patient's medication list, it was noted that he had an outpatient prescription for oral baclofen as needed (PRN). The outpatient ITB pump clinic notes were then reviewed, and it was discovered the patient had been taking oral baclofen scheduled rather than intermittently. Because his outpatient prescription for oral baclofen had been documented as PRN, oral baclofen was not scheduled during his admission for ITB pump replacement, nor had it been administered during this readmission. Oral baclofen therapy was reinitiated, and within 12 hours patient had resolution of cognitive impairments and significant improvement in spasticity, blood pressures, and heart rate confirming that a significant contribution to his presenting symptoms was baclofen withdrawal.

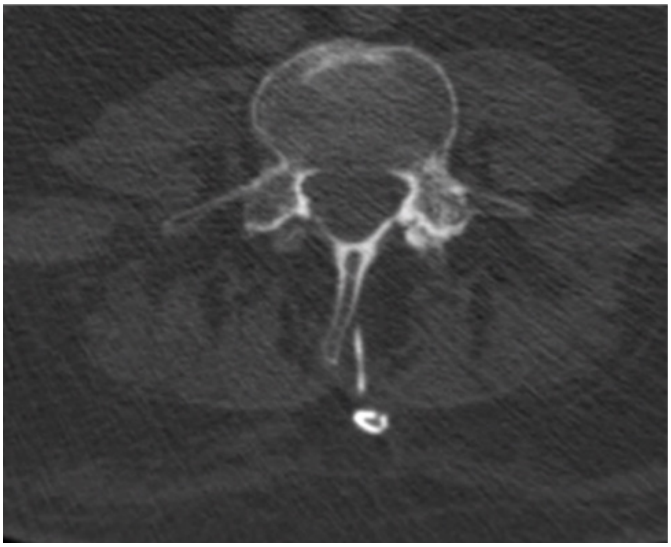

Figure 1. Baclofen pump catheter entering the spinal canal at L3- 4 level and terminating at the T6-7 level without discontinuity or kinking.

Discussion

We present a case of unrecognized baclofen withdrawal resulting in a complicated post-operative course and prolonged hospital stay. Baclofen is commonly used for the treatment of spasticity. It is a GABA-B receptor agonist in the central nervous system, and achieves its therapeutic effect via prevention of calcium influx in presynaptic neurons, which reduces presynaptic neurotransmitter release.4 It has been shown to improve spasticity in 70-96% of patients.[4] Baclofen is administered either orally or intrathecally. Administration of baclofen intrathecally has been found to be effective for patients with spasticity that is not well-controlled with the maximum dose of oral baclofen therapy. Intrathecal baclofen therapy allows for higher concentrations of drug in the cerebrospinal fluid than can be achieved with oral baclofen therapy.[6]

Withdrawal from baclofen can be a life-threatening condition and has a wide variety of signs and symptoms such as high fever, pruritis, sedation, weakness, altered mental status, rebound spasticity, and muscle rigidity.[2] Baclofen withdrawal may be treated with IV hydration, administration of oral or intrathecal baclofen, or even dantrolene and IV benzodiazepines.[7] The consequences of untreated baclofen withdrawal are severe and have been seen to progress to rhabdomyolysis, neuroleptic malignant syndrome, disseminated intravascular coagulation,[3] multi-organ system failure, and death.[8] Due to the non-specific sequelae associated with baclofen withdrawal, work-up for a different cause is often sought. There are case reports of baclofen withdrawal initially being misinterpreted as sepsis, myocardial infarction, and pulmonary embolism due to the often-complicated presentation of acute baclofen withdrawal.[8,9]

Table 1. Differential Diagnoses to consider when working up suspected Baclofen Withdrawal
Sepsis
Myocardial Infarction
Pulmonary Embolism
Autonomic Dysreflexia
Delirium
Meningitis
Encephalitis
Non Convulsive Status Epilepticus
Hypocalcemia
Malignant Hyperthermia
DIC
Rhabdomyolysis

Due to the severe consequences, a high index of suspicion should be maintained to avoid overlooking a case of baclofen withdrawal, especially in patients with an ITB pump. In these patients, a logical etiology of baclofen withdrawal is a pump malfunction, thus it is important to have physical medicine and rehabilitation interrogate the ITB pump in a timely manner to ensure proper pump functioning. There are many causes of ITB pump malfunction, ranging from battery failure, catheter migration, catheter kinking, and baclofen refill errors.[1]

In our patient, the ITB pump was interrogated and confirmed to be functioning properly, making the diagnostic picture even more clouded. It is important to note that a properly functioning ITB pump does not rule out baclofen withdrawal. If the ITB pump is functioning as intended and baclofen withdrawal is still suspected, careful review of dosage habits of all medications should be further reviewed.

Medication withdrawal is often due to communication errors that most frequently occur during transitions in the setting of the patient's health care such as the peri-operative period or transitions to and from skilled nursing facilities.[10] Additionally, many patients in need of ITB pump suffer from neurologic conditions rendering them unable to communicate their medication regimen. In these circumstances, collateral history from family or care givers and thorough chart review is useful in determining the most up-to-date information regarding the patient's medication and dosing habits. In our patient, detailed chart review of outpatient ITB pump notes revealed that the patient had been taking oral baclofen on a scheduled basis rather than PRN as had been previously charted, resulting in the patient being in baclofen withdrawal despite receiving the intended dose of baclofen from a functional ITB pump.

Adverse drug events such as withdrawal are the second-most-common complication during hospitalization.5 In complicated cases, medication withdrawal can be masked by other conditions, making the true etiology of symptoms difficult to ascertain. Previous charting can also contribute to confusion due to the inherent ambiguity arising from PRN medications. Thus, in every patient encounter, it is important to review dosing habits of all medication in order to make fully informed decisions and minimize errors.

Conclusion

Baclofen withdrawal is a potentially lethal complication that can be due to medication error or ITB pump malfunction. These patients often suffer from a nonspecific array of symptoms and often have many comorbidities that cloud the diagnostic picture, leading to delay in detection and treatment of withdrawal. This case supports the importance of reviewing dosing and compliance for all medications with the patient to ensure full and complete understanding of the patient's medications at every encounter. Resuming baclofen dosing often leads to complete resolution of symptoms. Thus, early recognition and treatment is critical to minimize complications and a prolonged hospital stay. In this case, early medication reconciliation may have reduced or eliminated the complications experienced during hospitalization.

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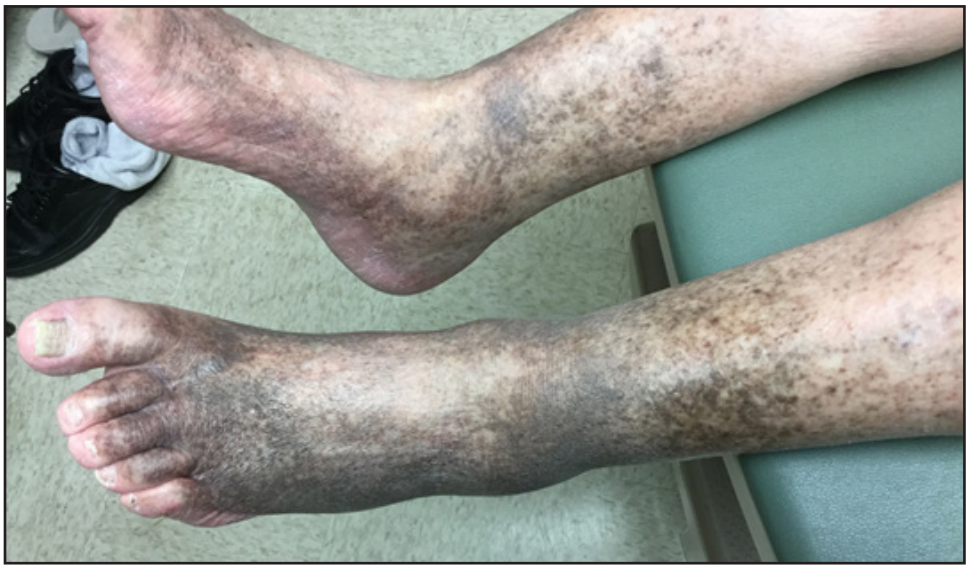
by SHELBY WEBB, MD CANDIDATE, CLASS OF 2020
UAMS DEPT OF DERMATOLOGY

An 82-year-old previously healthy man initially presents to the clinic with new-onset scalp folliculitis and rosacea. The patient was started on doxycycline and was to follow up in four to six weeks. The patient was lost to follow up for 10 months, during which time another physician switched him from doxycycline to continuous minocycline to try and achieve better control of the folliculitis.

Eight months later, he now presents with severe gram-negative scalp folliculitis and new blue-gray hyperpigmentation of bilateral lower extremities, as well as persistent rosacea. The patient has no reported history of renal, cardiac, lung, or hepatic disease. He denies other prescription medications, over-the-counter medications, or herbal supplements. The patient also denies consuming any products containing silver. The patient does not report fluctuations in symptoms related to sun exposure. He is started on isotretinoin for his recalcitrant rosacea and scalp folliculitis.



Considering the patient history given and clinical image provided here, what is likely the cause of his new-onset blue grey pigmentation, and what appropriate steps should be taken?

A. This is venous stasis, with hemosiderin related pigmentation. Obtain Ultrasound Doppler to assess for venous stasis and advise the use of compression stockings bilaterally.

B. This is bronze-pigmentation of hemochromatosis. Obtain measurements of serum transferrin saturation and ferritin, along with genetic testing for HFE mutations to rule out hereditary hemochromatosis.

C. This is drug-induced hyperpigmentation from long-term use of minocycline. Immediate discontinuation of the minocycline may result in resolution of hyperpigmentation.

D. This is likely argyria, and the patient is not sharing his full medication list. Obtain a skin biopsy to evaluate for silver granule deposition.

Answer: C

While the differential diagnosis of new-onset skin hyperpigmentation includes the diseases listed above, the patient history and presentation are most consistent with drug-induced hyperpigmentation, which is most commonly noted to be due to minocycline, amiodarone, various chemotherapy agents, prostaglandins, oral contraceptives, and antimalarial drugs. The patient has been on minocycline for almost approximately eight months now, raising the suspicion that the skin hyperpigmentation is from long-term minocycline use. This drug turns black when oxidized, resulting in discoloration of skin, as noted here. There are three types of cutaneous hyperpigmentation induced by minocycline. Type I is the most common type and presents as blue-black pigmentation of previous areas of inflammation or scarring. In Type II, there is characteristic blue-gray discoloration of previously normal skin of the legs, as is present in this patient. Type III is the least common type and presents as diffuse muddy-brown pigmentation of sun-exposed areas. Early recognition and discontinuation of the offending agent is key in promoting resolution of skin hyperpigmentation, though such resolution may take months to years.

Chronic venous stasis can result in stasis dermatitis as venous blood pools in the legs and causes local endothelial damage of the microvasculature. This may occur in one or both legs and rarely develops in other areas. Skin discoloration can range from scaly, pruritic plaques and irregular shaped areas of hyperpigmentation to ulceration. Topical corticosteroids can help to control acute dermatitis, but treating the venous stasis using compression stockings is the mainstay of treatment.

Hemochromatosis is one of the most common inherited errors of metabolism. It is a condition of increased absorption and storage of dietary iron. The skin discoloration in this disorder is characterized by a slate-gray or bronze hyperpigmentation in sun-exposed areas due to iron and melanin deposition. Disease onset generally is between 30 to 50 years of age. This condition is treated with repeated phlebotomy to decrease iron stores.

Agyria is a rare skin condition related to chronic exposure to silver-containing products that deposit diffusely in the skin, conjunctiva, oral mucosa, and nailbeds. The skin discoloration in this condition is often more pronounced in sun-exposed areas, especially the hands and face. This is related to the reduction of colorless silver in the dermis upon exposure to sunlight. Hence, discontinuation of silver-containing products, avoidance of sun and use of sunscreen is encouraged to prevent further skin discoloration.

It is also important to note that as the minocycline is discontinued due to adverse effects, we must also address the worsening gram-negative folliculitis. Treatment options include Isotretinoin or other various antibiotics directed against gram-negative bacteria.

by AMANDA EDERLE, MD CANDIDATE, CLASS OF 2020
UAMS DEPT OF DERMATOLOGY

A 67-year-old female with medical history significant for end stage renal disease, diabetes mellitus, and morbid obesity presents to the emergency room with a five-to-six-day history of livedo reticularis and exquisitely painful, firm, stellate purpura of the thighs, lower legs, abdomen, and buttocks. The patient also has a history of secondary hyperparathyroidism and an elevated calcium-phosphate product. Physical examination revealed the findings seen in the image..

The therapeutic intervention most likely to result in long-term control and decreased morbidity/mortality is:

A. Irrigation and vigorous debridement

B. Intravenous sodium thiosulfate

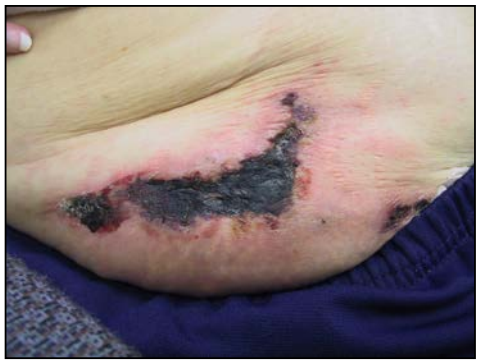
C. Reassurance and observation

D. Peritoneal hemodialysis

E. Intravenous vitamin K

Answer: B - IV sodium thiosulfate

The patient has calciphylaxis, a relatively uncommon disorder occurring primarily in the setting of end stage renal disease, which is characterized by painful ulcerations and necrosis of the skin and subcutaneous tissue. Mortality is high, and often



the result of sepsis. Though the pathogenesis of calciphylaxis is not fully understood, clinical findings suggest it results from ischemia and necrosis caused by calcification and subsequent thrombus formation within arterioles of the skin and subcutis. Risk factors for calciphylaxis include end stage renal disease, female sex, obesity, hypercalcemia, hyperphosphatemia, elevated parathormone level, and concurrent use of warfarin. Areas of the body with high fat content, such as the lower extremities, abdomen, and buttocks, are typically affected. Deep-wedge biopsy demonstrates calcification within the media of small- and medium-sized arterioles (which may be visualized with plain radiography), intimal hyperplasia and fibrosis, and intraluminal microthrombi. In addition to the painful skin lesions, some patients will have concurrently elevated calcium, phosphorus, or parathyroid hormone; thus, phosphate binders, low calcium bath dialysis, bisphosphonates, calcimimetics, and even subtotal parathyroidectomy may be employed. The six-month mortality rate of this disorder is approximately 50%, and mounting evidence suggests that IV sodium thiosulfate, a potent antioxidant, is effective in solubilizing calcium. Meticulous wound care, pain management, and treatment of secondary microbial infections are also important aspects of treatment. Sodium thiosulfate therapy is typically continued for several months, and response is monitored by a decrease in the number, size, or pain of calciphylaxis ulcerations/eschars.

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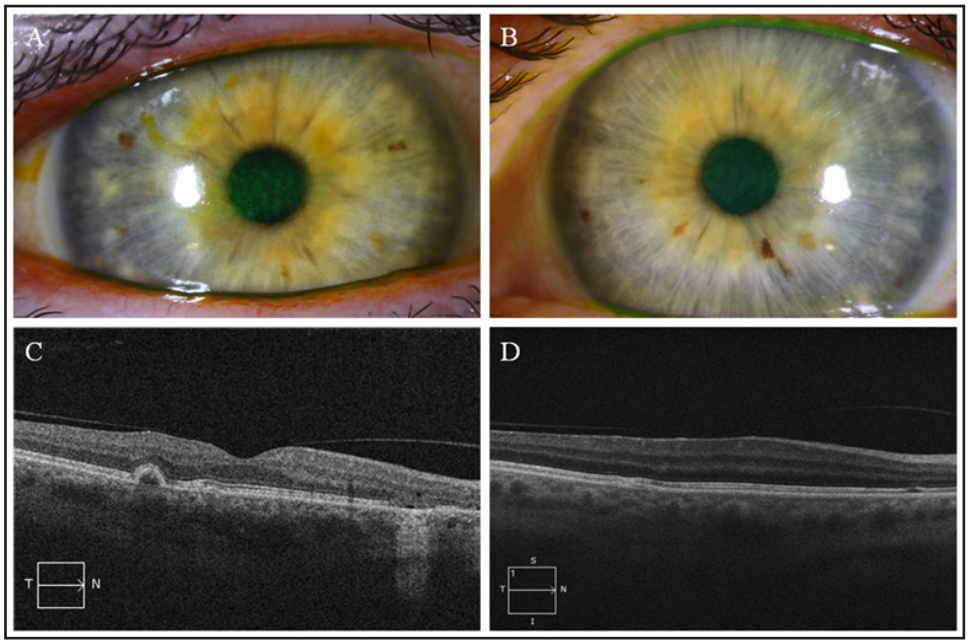
Program Death-1 Inhibitor-Induced Pigment Epithelial Detachment and Keratoconjunctivitis Sicca

Abstract

Purpose: To describe of retinal pigment epithelium detachment (PED) and keratoconjunctivitis sicca (KCS) in a patient treated with nivolumab, an anti-programmed death-1 inhibitor (PD-1) antibody.

Methods: Case report of a patient referred by oncology for severe eye irritation and blurry vision while undergoing nivolumab therapy for metastatic cutaneous melanoma.

Conclusion: This is the first described case of PED in a patient on nivolumab therapy. Although KCS is readily reported in the literature, aggressive and early treatment can completely resolve ocular surface disease. Drugs in the anti-PD-1 family require extremely aggressive treatment for ocular surface disease, and we suggest screening for potential retinal involvement.



Figures A-D. A-B, Severe keratoconjunctivitis sicca in right and left eyes, respectively, showing dense fluorescein dye uptake centrally with some lid margin staining on the right eye. Figure C, High-definition optical coherence tomography (HD-OCT) highlighting a perifoveal pigment epithelial detachment (PED) [arrow]. Figure D, HD-OCT showing resolution of PED.

Introduction

Retinal pigment epithelium detachment (PED) is the separation of the retinal pigment epithelium (RPE) from Bruch's membrane. Pigment epithelial detachment is a prominent feature in retinal diseases such as age-related macular degeneration (AMD).[1] Here we describe a case with an apparent association between nivolumab, a treatment option for metastatic melanoma, and PED. Nivolumab is an anti-programmed cell death protein-1 (anti-PD-1) monoclonal antibody that inhibits T-cell check points that normally allow tumor cells to evade the host's immunity.[2-4] Several published cases have reported the association of ocular surface disease (OSD) with use of nivolumab.[2-6] Herein, we present a case of PED associated with nivolumab for metastatic melanoma.

Case

A 59-year-old male with metastatic cutaneous melanoma on treatment with nivolumab was referred to the ophthalmology clinic for bilateral red, painful eyes for about one month with noticeably decreased vision over two weeks. At the time of referral, the patient was undergoing his tenth cycle of nivolumab therapy. He denied any history of dry eye disease. On initial evaluation, the patient had best corrected visual acuity of 20/200 in both eyes. Ocular examination revealed dense punctate epithelial erosions with epithelial edema, diffuse conjunctival staining, and severe papillary reaction in both eyes. Dilated fundus examination revealed a dome-shaped elevation in the right macula. High-definition ocular coherence tomography (HD-OCT) was then performed, which revealed right perifoveal PED. (Figures A-D) The patient was started on cyclosporine 0.05% with preservative-free-artificial tears every two hours while awake and lubricating ointment three times daily for severe keratitis. The PED was managed with observation. Subsequent visits at one-month and three-month follow-up showed some improvement in corneal surface with mild improvement in symptoms. The PED remained stable. Due to persistent epitheliopathy, autologous serum tears were initiated for OSD and continued observation for the PED was again elected. Three months of serum tears resulted in tremendous improvement in signs and symptoms and the patient's uncorrected visual acuity increased to 20/25 OU. Meanwhile, after his twelfth round of nivolumab, oncology recommended stopping therapy due to systemic adverse effects and opted for serial imaging and dermatologic exams. At his next follow-up appointment of six months, the patient maintained stable vision with resolution of all signs and symptoms of ocular surface disease. Ocular coherence tomography of the macula showed nearly 50% reduction in PED in size after stopping nivolumab.

Discussion

The advent of new drugs to harness the ability of one's immune system to treat cancer has led to promising results, specifically for metastatic melanoma. Programmed death 1 (PD-1) protein is a T-cell inhibitory receptor that allows circumvention of the body's natural T-cell immunity. Programmed death ligand 1 (PD-L1) is the primary ligand that is upregulated in solid tumors that propagates this circumvention. Nivolumab binds the PD-1 receptor and disrupts the inhibitory pathway, allowing for T-cell upregulation and, thereby, tumor regression.[2-4] Nivolumab has been reported to cause bilateral uveitis with keratitis5 and corneal perforation secondary to OSD.6 However, to the best of our knowledge, PED in association with nivolumab has not been described previously.

In a multicenter phase 1 trial of 207 patients treated with nivolumab, 5% of patients were reported to have dry eye.[2] Zimmer et al[3] report 1.6% of patients with ocular adverse events including uveitis with macular edema while using a similar anti-PD-1 drug. The OSD associated with anti-PD-1 therapy is readily documented. In our case, the patient had only minimal response to cyclosporine 0.5% and had significant relief once autologous serum tears were initiated. In combination with the halting of immunotherapy, the patient had complete resolution of the OSD. Although the patient did not have an OCT showing the lack of a PED, there was spontaneous reduction in size soon after discontinuation of nivolumab. Fortunately for our patient, the lesion is not problematic visually, but a PED involving the fovea can be visually significant. Our case reiterates the need for aggressive treatment for OSD in patients taking nivolumab with either cyclosporine 0.5% or autologous serum tears. It also highlights the importance of complete evaluation of the eye as there may be an association of retinal pathology in patients receiving anti-PD-1 therapy.

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Marshallese Pacific Islander Maternal Health Context

Abstract

Arkansas birth records (n=2,488) show that Marshallese in Arkansas experience a high rate of maternal and infant health disparities. Early and consistent prenatal care is strongly associated with positive birth outcomes and is a state health priority. However, Marshallese are less likely to receive prenatal care in the first trimester compared to other racial/ethnic groups. UAMS has engaged with community health care partners and the Marshallese community to overcome these barriers and address the prenatal and perinatal health disparities through the development of a Healthy Start program.

Marshallese Pacific Islander Maternal Health Context

The Pacific Islander population in the U.S. increased by 40% from 2000 to 2010, making it the second-fastest growing population in the U.S. Majority of the growth occurred in the South (66%), specifically in Arkansas (252%), where majority of Pacific Islanders are Marshallese.1 Arkansas has the largest population of Marshallese Pacific Islanders living in the continental U.S. (~14,000 people).[2-6]

Pacific Islanders residing in the U.S. have disproportionally higher rates of preterm birth (<37 completed weeks) and lower birthweight infants (<2,500 grams).[7-9] Additionally, the Pacific Islander population is more likely to experience preeclampsia, primary cesarean delivery, excessive gestational weight gain, gestational diabetes mellitus (GDM), and low exclusive breastfeeding initiation and duration at six months compared to other racial/ethnic minorities, and the U.S. population in general.7-9 Pilot analysis of Arkansas birth records (n=2,488) has shown that Marshallese in Arkansas experience a high rate of maternal and infant health disparities: 15% of Marshallese women received no prenatal care (compared to 1.6% women nationally); 19% of Marshallese infants were born preterm (compared to 9.6% nationally), and 15% of Marshallese infants were low birthweight (compared to 8.3% nationally).[9] Early and consistent prenatal care is strongly associated with positive birth outcomes and is a state, national, and global health priority. However, Pacific Islanders are less likely to receive prenatal care in the first trimester compared to other racial/ethnic groups, and are thus at a higher risk for maternal and infant health disparities.[7]

Qualitative data demonstrate that Marshallese women report numerous structural and social-cultural barriers that constrain obtaining prenatal care.[10] Structural barriers include challenges negotiating health insurance, transportation, and language. Social-cultural barriers include lack of understanding of the importance of seeking early and consistent prenatal care, perceived discrimination from prenatal care providers, and an overall fear of the health care process.[10]

Development of a Healthy Start Program

UAMS has engaged with community health care partners and the Marshallese community to overcome these barriers and address the prenatal and perinatal health disparities through the development of a Healthy Start program, translated "Jined ilo Kobo" in the Marshallese language. "Jined" means mother, and "Kobo" refers to the concept of preserving, preparing, shaping, safekeeping, protecting, and warmth from the mother's breast. The program builds upon the Marshallese's matriarchal culture in which mothers are recognized as the givers and sustainers of life. In the traditional oral stories, Marshallese mothers held positions of power as the leaders who established new clans (jowi) and new lineages (bwij). The term "Jined ilo Kobo" is used to describe a position of power where a mother holds her child close to her breasts for warmth and protection. It was believed that as a Marshallese mother held her child close and guided her/his development, the child would grow up and continue to thrive and serve the Marshallese community. Jined ilo Kobo signifies the highest position in the Marshallese culture, and all mothers who attain Jined ilo Kobo are revered.

The Jined ilo Kobo program seeks to ensure access to culturally sensitive, community-based health care and social services for Marshallese women, pregnant mothers, infants, and families. UAMS will work with community service providers to improve coordination. Community coordination will be accomplished through the use of bilingual Marshallese Care Coordinators who will help women, pregnant mothers, infants, and families 1) sign up for health insurance; 2) understand how to utilize their insurance; 3) access culturally-appropriate health care and social services; and 4) ensure pregnant mothers receive early prenatal care.

Marshallese Care Coordinators will help Marshallese families connect with resources such as the Special Supplemental Nutrition Program for Women, Infants and Children (WIC), the Children's Safety Center, and other community programs supporting families. Bilingual Marshallese Care Coordinators will also support pregnant women and families with translation and health navigation throughout their pregnancy, regardless of which health care provider women are using. Jined ilo Kobo will partner with the community to promote health education and activities. One of the primary objectives of Jined ilo Kobo will be to provide parenting education that leverages Marshallese cultural values. The program will also engage fathers and partners in the process and provide fatherhood support services. In short, Jined ilo Kobo focuses on supporting pregnant mothers, infants, and families with the key resources they need to thrive.

Community partners involved in the Jined ilo Kobo program include: Arkansas Coalition of Marshallese (ACOM); Marshallese Education Initiative (MEI); and Family Network. Health care providers involved in the Jined ilo Kobo program

include the Community Clinic, Washington County Department of Health, Arkansas Children's Hospital Northwest, and Boston Mountain Rural Health Clinic.

UAMS believes that through this program Arkansas can reduce the maternal and child health disparities experienced by Marshallese over the next five years by 1) reducing preterm births by half, from 19% to 9.5%; 2) reducing the number of low birthweight infants from 15% to 8.5%; and 3) reducing the number of pregnant women who receive no prenatal care from 15% to less than 5%.

Funding for the Jined ilo Kobo program is provided by local private foundations and a grant from the Health Resources and Services Administration's Maternal and Child Health Bureau Division of Healthy Start and Perinatal Services. For more information about the Jined ilo Kobo Health Start program, please email mch@uams.edu.

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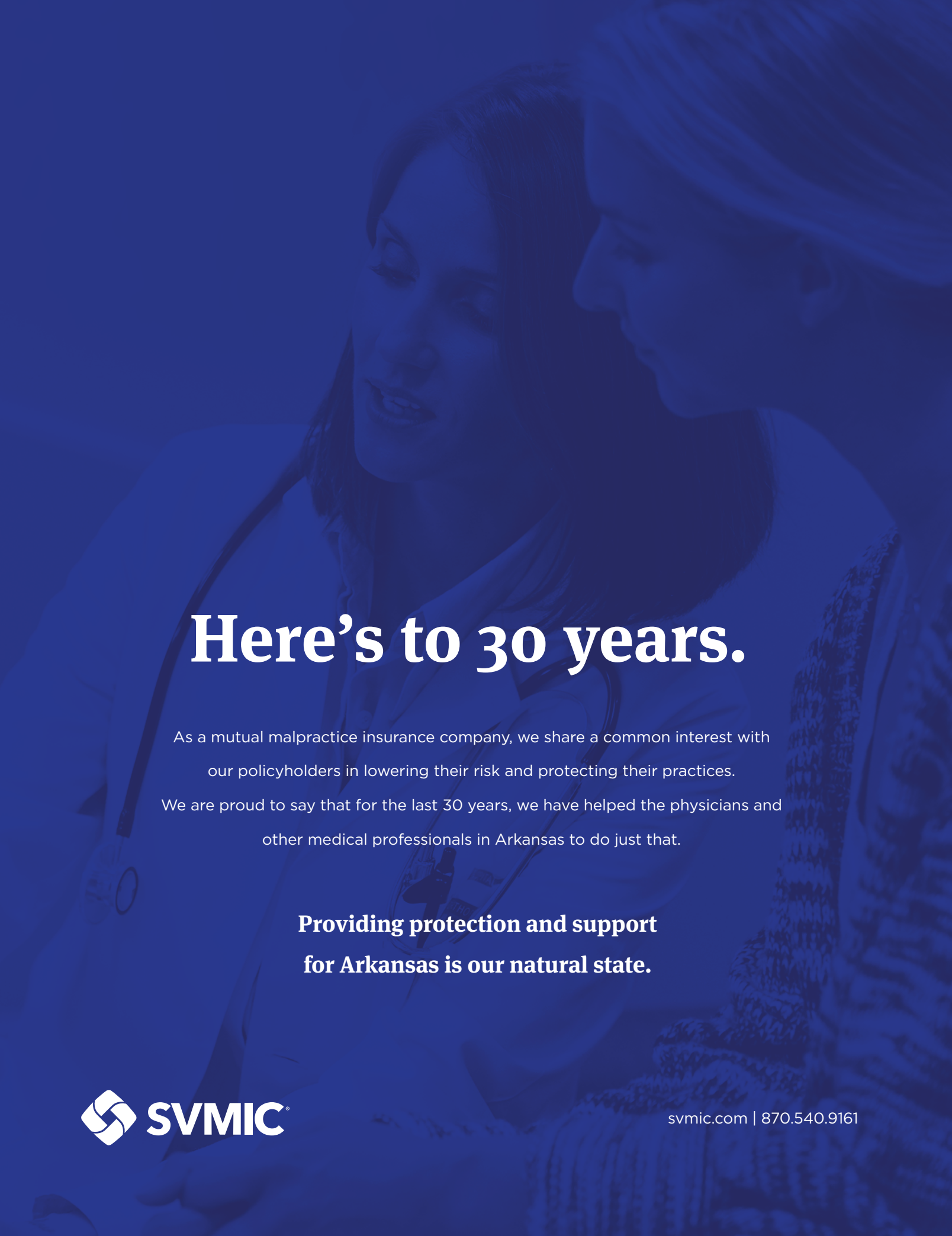

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