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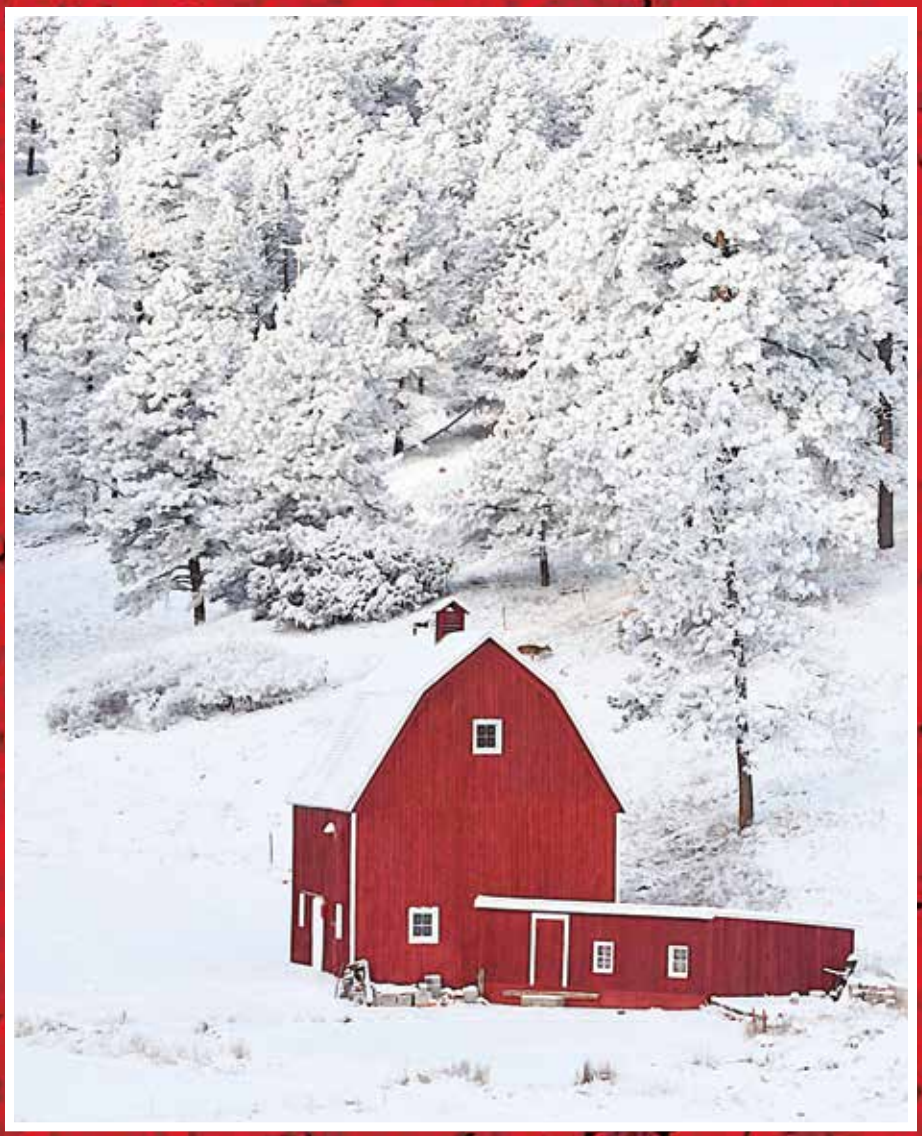

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February Issue Highlights

Keith Hansen, MD
Editor, *South Dakota Medicine*

Welcome Welcome to the February 2025 issue of *South Dakota Medicine*. In this issue we have a number of manuscripts written by medical students and residents from the University of South Dakota Sanford School of Medicine.

In this issue, Dr. Lim presents an Images in Medicine demonstrating the presentation of Hydroa vacciniforme. The purpose of this feature is to demonstrate images such as pictures of dermatologic conditions, ultrasounds or radiographs or histopathology of common or unusual medical conditions. Hopefully, these images will assist the clinician in their daily practice when a patient presents with similar complaints. If in your daily practice you discover a case with a classic or unusual image please consider submitting it to the Images section in *South Dakota Medicine*.

In *Observing Siblings of Children with Complex Medical Needs*, Barwari et al. discuss the effect on children of having a sibling with complex medical needs. The students, with Dr. Schimelpfenig as their mentor, took advantage of a volunteer opportunity at Give Kids the World in Orlando, Florida, to observe the siblings of children with complex medical needs. They then evaluated their data and have added to the literature on the effect on siblings of children with complex medical needs. This article highlights one of the many volunteer activities that students at USD SSOM are involved with despite their very busy academic schedules.

Also in this issue are a number of interesting case reports to describe unusual presentations. We also have a letter to the editor further discussing the difficult and sometimes conflicting ideas on the management of cirrhotic ascites with midodrine use in this condition. I hope you find this issue of *South Dakota Medicine* as interesting as I have.

Conflicting Evidence Regarding Midodrine Use to Improve the Tolerability of Diuretics and Outcomes in Patients with Cirrhotic Ascites

Dawood Shehzad, MD; Mustafa Shehzad, MD; and Dawlat Khan, MD

To the Editor,

Conflicting evidence exists regarding the use of midodrine to enhance natriuresis, diuresis, and outcomes in patients with cirrhotic ascites. Ascites is a significant complication of cirrhosis and an important prognostic indicator. Patients with advanced cirrhosis and ascites often require hospitalization for optimization of their diuretic regimen, large-volume paracenteses, and consideration for transjugular intrahepatic portosystemic shunt (TIPS).[1]

The circulatory and cardiac compromise in cirrhosis has been well documented.[2] Portal hypertension is induced by an increased resistance to flow secondary to distorted sinusoidal architecture.[2] Portal hypertension results in splanchnic and systemic vasodilation that causes endothelial dysfunction, releasing nitric oxide and vasoactive molecules. Low systemic vascular resistance results in a hyperdynamic state with increased cardiac output and heart rate. Systemic vasodilation decreases effective arterial blood volume, activating the renin-angiotensin-aldosterone system (RAAS). This leads to volume expansion via sodium and water retention in the setting of already poor oncotic pressure.[3] Despite volume expansion, ongoing portosystemic shunting, and splanchnic vasodilation, effective arterial blood volume remains low. A lower mean arterial pressure (MAP) of 60-65 mm Hg is tolerated in compensated and stable cirrhotic patients, which is usually sufficient to maintain end-organ perfusion. Any disruption to this system can result in significant hypotension and decompensation.[4] Volume overload due to the above mechanism is an important landmark in the natural disease course of cirrhosis.

The usual diuretic regimen in these patients consists of spironolactone and furosemide.[4] However, some individuals become resistant to diuretic therapy (diuretic-resistant ascites), or, more commonly, develop diuretic-related complications such as hypotension and acute kidney injury, thereby limiting safe diuresis. Aggressive diuresis poses a therapeutic challenge frequently encountered in clinical practice.

Midodrine is converted into desglymidodrine, an α1 receptor agonist that raises vascular tone and blood pressure. It has no β-adrenergic receptor activity and has minimal impact on the central nervous system due to poor blood-brain barrier penetration. Therefore, it improves systemic and renal hemodynamics in nonazotemic cirrhotic patients by counteracting splanchnic vasodilation.

Angeli et al. conducted a randomized control trial in 1998, in which 25 [17 without hepatorenal syndrome (HRS) and 8 with type 2 HRS] patients with cirrhosis and ascites were studied.[5] All patients received a one-time dose of 15 mg of midodrine. In patients without HRS, within 3 hours of midodrine administration, there was an increase in MAP (89.6 ± 1.7 vs. 81.80 ± 1.3 mm Hg; $p < 0.0001$), (GFR) glomerular filtration rate (93.1 ± 6.5 vs. 77.0 ± 6.7 mL.min-1; $p < 0.025$), and urinary sodium concentration (92.7 ± 16.4 vs. 72.2 ± 10.7 micro Eq/L; $p < 0.025$). No significant improvement was observed in GFR, renal plasma flow, or urinary sodium in the patients with HRS at 3 and 6 hours.

Kalambokis et al. conducted another randomized control trial in 2007.[6] 39 cirrhotic patients were randomized to a 7-day treatment course with midodrine 10 mg thrice daily or placebo. (11 patients without and 12 with ascites) or placebo (8 patients without and 8 with ascites). In patients with ascites treated with midodrine, there was a significant increase in GFR from 79.7 ± 23.2 ml/min to 92.2 ± 26.9 ml/min at seven days, $p <0.01$. Urine

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volume and urine sodium also showed a statistically significant increase. In patients without ascites treated with midodrine, GFR and urinary volume changes were non-significant. There was a statistically significant increase in urinary sodium excretion in those treated with midodrine.

In 2010 Misra et al. conducted a randomized, double-blind, placebo-controlled, cross-over study of 15 non-azotemic cirrhotic patients with ascites patients to intravenous furosemide with either placebo or midodrine 15 mg (a one-time dose was used).[7] It was hypothesized that midodrine co-administration would enhance the natriuretic effects of furosemide in these patients due to midodrines favorable effects on renal hemodynamics. Urine sodium was measured at 0, 0.5, 1, 1.5, 2, 2.5, 3, 4, 6 and 8 hours. The GFR was calculated using iothalamate clearance. The six-hour GFR rate, urine sodium excretion, and urine volume were not statistically significant between the two groups. The six-hour total urine volume in the midodrine-furosemide group was 1770 ± 262 mL compared to the placebo-midodrine phase 1962 ± 170 mL, p = 0.25.

In 2012, Singh et al. demonstrated that midodrine at a fixed dose of 7.5 mg thrice daily significantly improved 40 patients with refractory/recurrent ascites.[8] Twenty patients received midodrine and standard medical therapy (furosemide and spironolactone), and twenty received standard medical treatment alone. MAP, GFR, urine output, MELD (Model for End-Stage Liver Disease), urine sodium, and other parameters were measured at 1, 3, and 6 months. At 6 months, the differences between the MAP and GFR between the groups were not significant. The urine output was significantly higher in the midodrine group at 1 and 3 months by close to 600ml/day; however, at 6 months follow-up, the increase in urine output/day did not remain statistically significant. Urinary sodium excretion significantly increased in the midodrine group after treatment at 1 and 3 months, but again was not significant at 6 months. Baseline MELD scores (~ 14.8 ± 4.6) were similar in both treatment groups (p = 0.140). MELD score significantly from baseline in the standard medical therapy group and was 16.1 ± 5.6, 17.3 ± 4.3, and 19.5 ± 5.1 at 1, 3, and 6 months, respectively, but did not differ significantly from baseline in the midodrine group (13.9 ± 4.1, 13.5 ± 3.3 and 14.5 ± 3.7) at 1, 3 and 6 months respectively.

In another study in 2017, 78 cirrhosis patients were randomized to receive midodrine at a dose of 12.5 ± 2.5 mg/day (based on tolerability, n = 41) or standard medical therapy (n = 37).[9] Midodrine increased the MAP, and urinary volume at one month. There was no change in heart rate (in contrast to results noted by Kalambokis et al. in their study). There was no decrease in urine sodium at 1 month (in contrast to results noted by Singh et al.). There was also no change in MELD scores at 1 month, in contrast to that found by Singh et al.

In 2021, Anand V. Kulkarni et al. randomized 45 patients with newly diagnosed acute on chronic liver failure to midodrine (n = 21) to a mean dose of 22.5 mg/day for 22.3 ± 8.8 days.[10] Midodrine significantly improved the MAP ($p < 0.001$) and urinary sodium excretion (p = 0.05). The reduction in weight at 1 month trended towards significance but did not reach it (p = 0.08). The change in urine sodium (p = 0.06) and urine output (p = 0.77) were also reported at 1 month. The midodrine group tolerated higher doses of diuretics and had fewer diuretic-related complications. There was no significant mortality difference, likely due to the fact that the average MELD-Na was 28 for the study group.

Smaller trials have shown that terlipressin improves renal function in patients with cirrhosis and ascites without hepatorenal syndrome.[11] Terlipressin is a vasopressin analog that reduces splanchnic circulation and is increasingly used in patients with HRS. Midodrine, octreotide, and albumin are used in areas where terlipressin is unavailable.

As mentioned above, in the absence of HRS, the use of multiple doses of midodrine to increase urinary sodium excretion, GFR, and MAP and decrease the incidence of diuretic-related complications has yielded conflicting results. The effect on mortality has been varied.

The potential benefits of vasoconstrictor therapy in non-HRS patients with cirrhosis and ascites should be explored further. Conflicting results make it difficult to definitively conclude what role midodrine should play

in this patient population. Midodrine is orally administered, cheap, easy to titrate, and has few adverse effects. In clinical practice at our institution, we see an expanding role for midodrine in patients with peri-stable blood pressures requiring diuresis. Large, well-designed randomized control trials in this area are needed.

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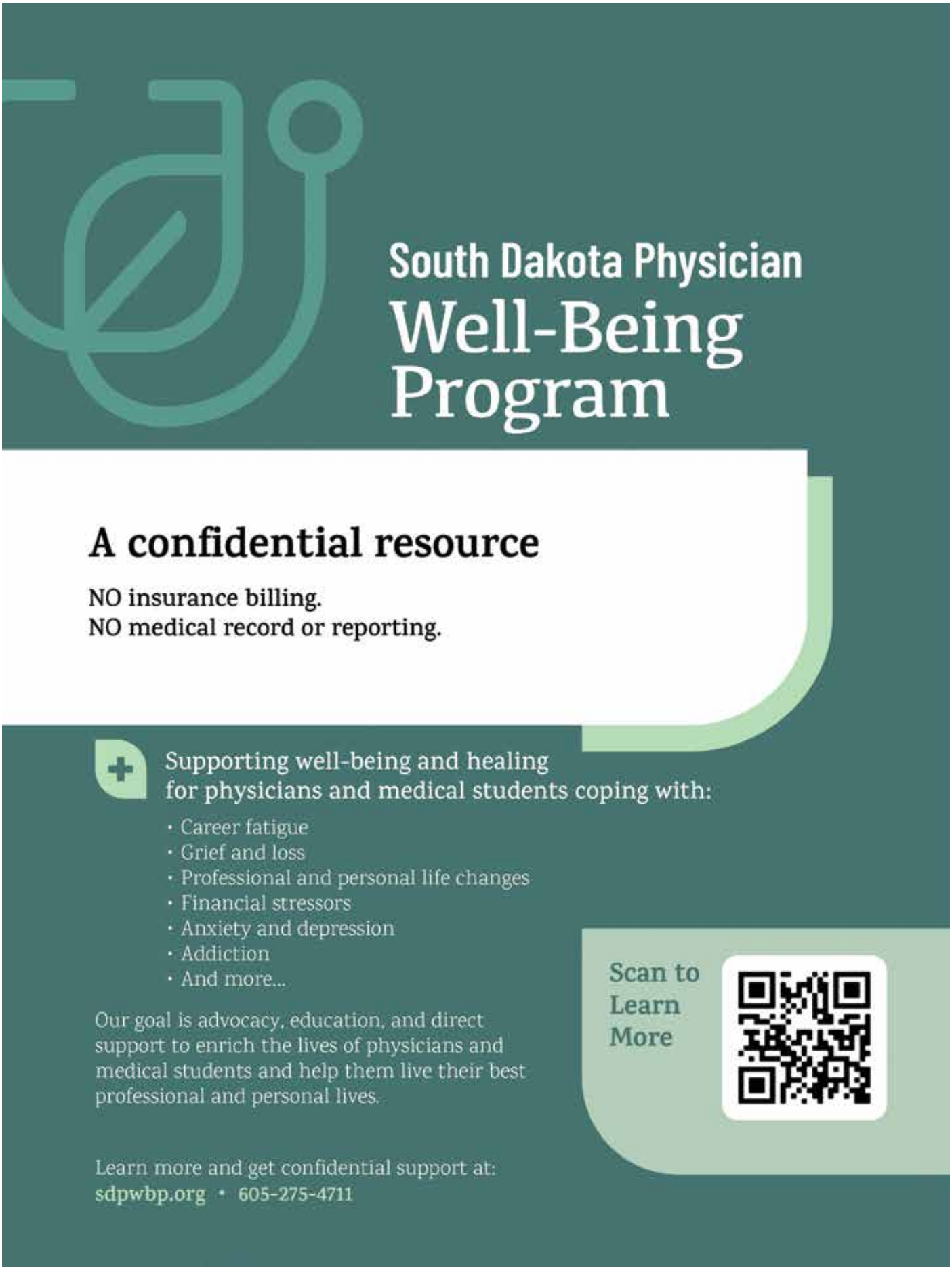
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Time for a Buzzkill

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

March Madness is just around the corner. This is a perfect time to start sharing the unwelcome news that alcohol consumption (of any type — wine, beer and spirits) is linked to at least seven different types of cancer, including cancers of the breast, colorectum, esophagus, liver, oral cavity, pharynx, and larynx. This was the message recently released by U.S. Surgeon General Dr. Vivek Murthy entitled *Surgeon General's Advisory on Alcohol and Cancer Risk.*

This isn't breaking news by any stretch — the scientific evidence for this connection has been well known by those in the research community and medical fields for decades. However, less than half of Americans recognize alcohol use as a risk factor for cancer. This is very concerning because *a lot* of Americans consume alcohol on a regular basis. A Gallup survey from July 2023 found that 62% of Americans reported drinking any amount of alcohol and of those who reported drinking alcohol, 69% of them said they had drunk alcohol in the past week. And while most of our patients would likely say that drinking alcohol in heavy amounts on a regular basis is dangerous for a multitude of reasons, most of them probably don't understand that drinking alcohol in even small amounts can increase their cancer risk.

Cancer risk increases as alcohol consumption increases. However, for certain cancers, like breast, mouth, and throat cancers, evidence shows that the risk of developing cancer may start to increase at less than one drink per day. For breast cancer specifically, 16.4% of total breast cancer cases are attributable to alcohol consumption. Annually in the U.S., there are about 100,000 alcohol-related cancer cases and about 20,000 alcohol-related cancer deaths. For comparison, there are approximately 13,500 alcohol-associated traffic crash fatalities per year in the U.S. Again, this is one of those facts that might surprise our patients. No one is going to question that drinking alcohol and driving is dangerous. But they will likely question the cancer association or dismiss it outright. But our job is to remain steadfast in our messaging and to always share the most accurate scientific evidence we have at the time.

Alcohol is third in line for being a preventable cause of cancer. Tobacco is the leading preventable cause of cancer and obesity is the second leading preventable cause. Messages regarding the risks of alcohol consumption will soon be spread more broadly when the existing Surgeon General's health warning label on alcohol-containing beverages will be updated to reflect the increased cancer risk that consuming alcohol causes. This updated warning will be similar to the warning label seen on tobacco-containing products. The warning label on cigarettes was added in 1965 and it's not hard to imagine that the populace took some time to truly start believing that message. The same will likely be true for alcohol and it will take repeated messaging from all of us in our respective fields.

It is absolutely necessary that we screen all our patients for alcohol use and will need to keep in mind that we're not just looking for those patients who have a diagnosable alcohol use disorder but are screening for any alcohol use (just as we should be asking about any amount or type of tobacco use). If you have patients who are struggling with excessive alcohol use and are interested in cutting back or stopping alcohol altogether, consider prescribing naltrexone. This medication has been approved for alcohol use disorder for over 30 years and yet is greatly underutilized. Other FDA-approved medications for alcohol use disorder include acamprosate and disulfiram, but naltrexone is easier for patients to take from a dosing standpoint and doesn't have all the unpleasant side effects of disulfiram.

As a primary care provider who cares for patients who have a wide range of alcohol consumption habits, I can already foresee the challenges. Many patients are going to harken back to the days when they were told that that a glass of red wine with dinner might have some cardiac benefit. And many patients are going to feel like their alcohol use isn't "excessive" but rather just a normal part of their routine, whether it's that glass of wine at dinner or a couple of drinks on the weekend. Alcohol use has become so normalized in today's society and this is going to make it that much more difficult to convince patients of the known cancer risk. This will not be a message that most patients are going to receive easily. And while nobody wants to be a buzzkill, we as physicians don't want to see our patients killed by something preventable.

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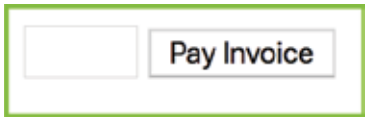

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Nilotinib Induced Large Pericardial Effusion in a Patient Being Treated for Chronic Myeloid Leukemia

Muhammad Hasan, MD; and Melanie LaVoie, DO

Abstract

Nilotinib, a second generation tyrosine kinase inhibitor, has remarkably improved outcomes in patients with newly diagnosed Philadelphia chromosome positive (Ph+) chronic myelogenous leukemia by targeting BCR-ABL1 fusion protein. Despite the safety and efficacy of these agents, adverse side effects can occur due to unintended inhibition of off-target kinases. While the clinical ramifications of serosal inflammation – including pleural and pericardial effusions - have been a common occurrence in patients on older tyrosine kinase inhibitors (TKI), the effect of nilotinib causing pericardial effusion is rare. This case report highlights a rare occurrence of pericardial effusion while being treated with nilotinib for chronic myeloid leukemia.

Introduction

Chronic myeloid leukemia (CML) is a myeloproliferative disorder characterized by the presence of the Philadelphia chromosome and the resultant BCR-ABL1 fusion protein, which plays a crucial role in the pathogenesis of the disease. Tyrosine kinase inhibitors (TKIs), notably imatinib and nilotinib have revolutionized the treatment of CML, significantly improving patient outcomes. Nilotinib, a second-generation TKI, has emerged as a frontline therapy for patients with newly diagnosed Philadelphia chromosome-positive (Ph+) CML due to its enhanced potency and selectivity against BCR-ABL.[1]

While TKIs have demonstrated remarkable efficacy in controlling CML, they are not devoid of adverse effects. Serosal inflammation, including pleural and pericardial effusions, has been reported as a known complication of TKI therapy.[2] The occurrence of pericardial effusion secondary to nilotinib, in particular, remains a rare phenomenon with limited documented cases in the literature.

Case Presentation

We present a 47-year-old man with a complex medical history including end-stage renal disease, hypertension, type 2 diabetes mellitus, and chronic myeloid leukemia treated with nilotinib. He presented to the emergency department complaining of sudden onset shortness of breath, which began four hours prior while he was resting comfortably in bed. Upon arrival, he denied any associated symptoms such as cough, fever, chest pain, or chills. Upon examination, the patient was found to be vitally stable and alert, oriented to person and place, with clear bilateral chest sounds and normal heart sounds (S1 plus S2). Initial investigations, including a chest X-ray and EKG, revealed a widened mediastinum and diffuse low voltage sinus tachycardia, respectively (Figures 1 and 2).

Figure 1. EKG showing Sinus tachycardia with low voltage.

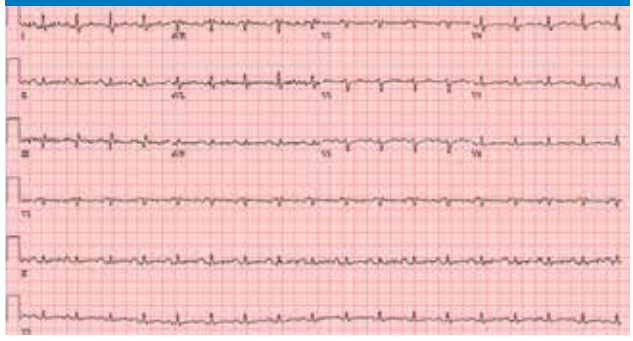


Figure 2. Chest X-ray showing widened mediastinum.

Further imaging with computed tomography angiography (CTA) confirmed a large pericardial effusion (Figure 3). Subsequent transthoracic echocardiography revealed tamponade physiology, prompting emergent pericardiocentesis (Figure 4) with placement of a percutaneous drainage catheter and aspiration of 300 cc of serosanguineous fluid. Extensive laboratory investigations, including a comprehensive respiratory panel, autoimmune workup, and fluid analysis, were largely unremarkable except for the presence of 2700 RBC and 1300 WBCs (80% neutrophils and 12% lymphocytes) in the pericardial fluid. Flow cytometry and cytopathology were negative for lymphoproliferative disorders or evidence of chronic myeloid leukemia. A repeat echo post-procedure confirmed complete drainage. Over the next three days his drain expelled an additional 100 cc of fluid. Pigtail catheter was removed after repeat echocardiogram showed trivial pericardial effusion on day three. Given the exclusion of other etiologies, the pericardial effusion was attributed to nilotinib, and it was decided to discontinue his nilotinib due to this association. A follow-up echocardiogram performed one and three months later showed no recurrence of the effusion

Figure 3. CTA chest showing a 3.2 cm pericardial effusion (blue arrows) without RV strain.

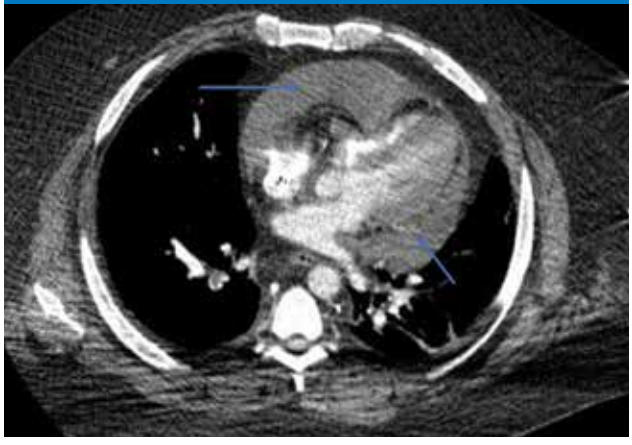
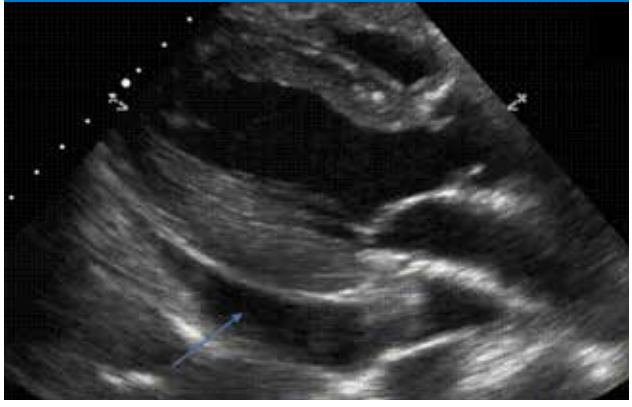


Figure 4. Parasternal long axis view showing pericardial effusion (blue arrow).



Discussion

CML is classified as a hematopoietic stem cell (HSC) disorder characterized by a specific translocation known as t(9;22)(q34;q11), which results in the fusion of the BCR and ABL1 genes, thereby generating the pathogenic BCR-ABL1 oncogene.[3] Treatment of CML involves the use of BCR/ABL TKIs. While serositis is a rare but notable side effect associated with TKIs, it has been documented in clinical trials. The incidence of serositis varies, with dasatinib, in particular, showing rates ranging from 10% to 48% and being frequently associated with pleural effusions. Other TKIs such as imatinib, nilotinib, and bosutinib exhibit a lower incidence of serositis (<1%).[4] While the exact mechanism underlying TKI-related pleural effusion remains incompletely understood, it is believed to be linked to the inhibition of PDGFRβ. Nilotinib is a second-generation tyrosine kinase inhibitor which not only targets ABL but also inhibits KIT and PDGFR. When comparing nilotinib to imatinib and dasatinib, it's more specific for ABL than KIT or PDGFR. The decreased specificity for PDGFR could potentially explain the rarity of pericardial effusion caused by nilotinib.[5]

This vignette emphasizes on the awareness of pericardial effusion caused by nilotinib. Further studies are warranted to identify patient-specific risk factors for pericardial complications, enabling tailored management for higher-risk populations, while also exploring preventive strategies for cardiac side effects.

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Advanced Care for Children in All Stages of Heart Failure

Content submitted by Children's Nebraska

The Advanced Pediatric Heart Failure & Transplant Program at Children's Nebraska received full approval from the Centers for Medicare and Medicaid Services in November 2024, is active in the United Network for Organ Sharing system and is actively listing patients for heart transplantation. Led by surgical director Camille Hancock Friesen, MD, and medical director Jason Cole, MD, the program provides comprehensive care for children with heart failure, serving patient families in the region and beyond.

Leading-Edge Care

Children's Advanced Pediatric Heart Failure & Transplant Program offers compassionate, family-centered care, ensuring patients and families are well-informed throughout the care process. The multidisciplinary team comprises specialists from various pediatric fields, including Cardiology, Cardiothoracic Surgery, Cardiac Critical Care, Anesthesiology, Nutrition, Child Life, Social Work, Pharmacy and more, with consultations available from other specialties. Care is provided in Children's 32-bed, acuity-adaptable Cardiac Care Unit (CCU) in the state-of-the-art Hubbard Center for Children, where patients receive continuous, specialized monitoring and treatment.

Recently, Children's Advanced Pediatric Heart Failure & Transplant team performed its first-ever placement of a durable mechanical circulatory support in the form of ventricular assist device (VAD), a significant milestone. The life-saving device can serve as a bridge to heart transplantation or, in some cases, act as a long-term solution for children with heart failure. The ability to offer VAD implantations is a testament to Children's commitment to providing the most advanced pediatric cardiac care.

Exceptional Care Closer to Home

Children's Advanced Pediatric Heart Failure & Transplant Program is a major step in expanding access to care. Advanced pediatric cardiac therapies are available close to our patients' and families' homes. Patients and families traveling more than 90 miles outside Omaha can access the Carolyn Scott Rainbow House, a 56-room overnight guest house for a patient's immediate family.

Camille Hancock Friesen, MD, Jason Cole, MD and Ram Kumar Subramanyan, MD, PhD, performing Children's first ever ventricular assist device (VAD) placement.

To learn more about Children's Advanced Pediatric Heart Failure & Transplant program, visit ChildrensNebraska.org/HeartTransplant or scan the QR code. To refer a patient, call 402.955.4350.

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Mystifying Masquerade: Renal Cell Carcinoma Associated Arteriovenous Fistula Presenting as High-Output Heart Failure – A Case Study

Jaswanth Rao Jasti, MD; Thabuna Sivaprakasam, MD; Randall Lamfers, MD; and Orvar Jonsson, MD

Abstract

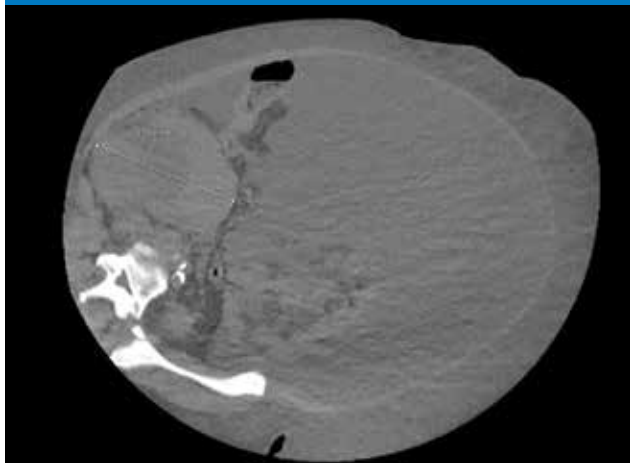
We detail an uncommon presentation of high-output heart failure (HOHF) triggered by an arteriovenous fistula (AVF) associated with renal cell carcinoma (RCC) in a middle-aged woman with widespread anasarca. The rarity of HOHF linked to malignancies posed significant diagnostic challenges. This case emphasizes the intricate and thorough diagnostic process required to elucidate the connection between RCC and HOHF. The utilization of advanced abdominal imaging, right heart catheterization, and precise blood gas analysis from both the inferior vena cava and renal vein was pivotal in establishing the diagnosis. This comprehensive diagnostic regimen was crucial in identifying the RCC-associated AVF as the causative factor for HOHF, highlighting the importance of a detailed investigative approach in cases of unusual cardiac presentations associated with malignancy.

Case

A 59-year-old woman with a history of chronic kidney disease (stage 3b) and hypertension sought care at our emergency department, reporting a three-month history of progressive dyspnea, abdominal distension, bilateral lower extremity edema, generalized fatigue, and an unintentional weight gain of 60 pounds. Physical exam was remarkable for diffuse anasarca but stable hemodynamics on room air. Labs showed acute kidney injury with a creatinine level of 2.34 mg/dL (baseline: 1.35-1.45 mg/dL), BNP at 1,164 pg/mL, and a slightly elevated troponin at 0.034 ng/mL. Electrocardiogram and Chest X-ray were largely unremarkable except for low voltage leads and enlarged cardiac silhouette respectively. Computerized tomogram (CT) of the abdomen and pelvis was pursued in view of significant abdominal distension that showed cirrhotic appearance of liver with substantial ascites and an incidental large right renal mass suspicious for renal cell carcinoma (RCC) (Figure 1).

Given the clinical presentation, the differential diagnosis focused on heart failure, cirrhosis, renal failure, particularly with the discovery of a renal mass and liver nodularity on CT imaging. Diagnostic paracentesis indicated cardiac ascites with a serum ascitic albumin gradient (SAAG) of 1.3 g/dL and an ascitic total protein of 3.9 g/dL. Transthoracic echocardiogram (TTE) revealed a normal left ventricular ejection fraction (LVEF) of 55-60% without any wall motion

Figure 1. CT scan of abdomen/pelvis without contrast showing a 13 cm right renal mass.



abnormalities however the right atrium and ventricle were severely dilated with a right ventricular systolic pressure (RVSP) of 59 mm Hg (Figure 2). A right heart catheterization confirmed this diagnosis, suggesting high-output heart failure secondary to an arteriovenous fistula within the renal mass (Table 1), a hypothesis supported by venogram and blood gas analyses showing high oxygen saturations in the right renal vein and inferior vena cava (IVC) (Figure 3 and Table 2). Additionally, portal hypertension was ruled out with a normal hepatic venous pressure gradient (HVPG) of 1 mmHg on the transjugular hepatic venogram (Table 3).

Figure 2. TTE showing interval resolution of diastolic septal flattening and reduction in size of pericardial effusion (left – at presentation, right – after diuresis).

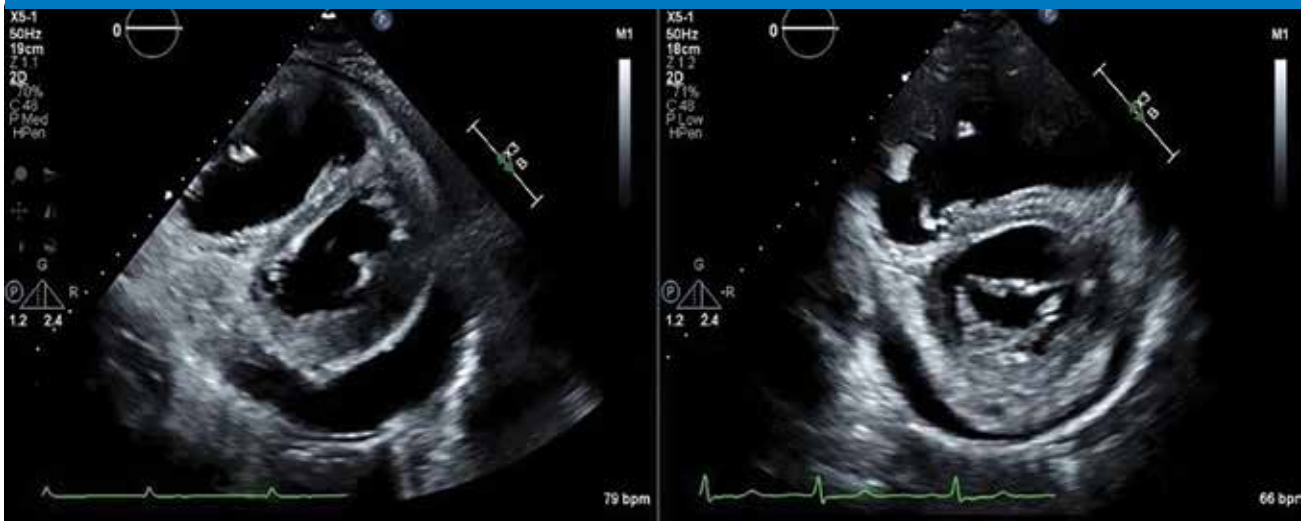


Table 1. Hemodynamic findings on right heart catheterization.

		Reference range
Right atrial pressure	8 mmHg	1-5 mm Hg
Mean pulmonary arterial pressure	52 mmHg	<20 mmHg
Pulmonary capillary wedge pressure	24 mmHg	<15 mmHg
Cardiac output/cardiac index	9.86 L min^{-1} / 4.5 L min^{-1} m^{-2}	4.4 – 8 L min^{-1} / 2.5 – 3.9L min^{-1} m^{-2}
Pulmonary vascular resistance	2.8 Woods units	1-3 Woods units
Systemic vascular resistance	6.6 Woods units	10- 15 Woods units

Figure 3. Venogram. Left – dilated suprarenal IVC, right – infrarenal IVC (green asterisk), dilated right renal vein (yellow asterisk) and dilated suprarenal IVC (red asterisk).

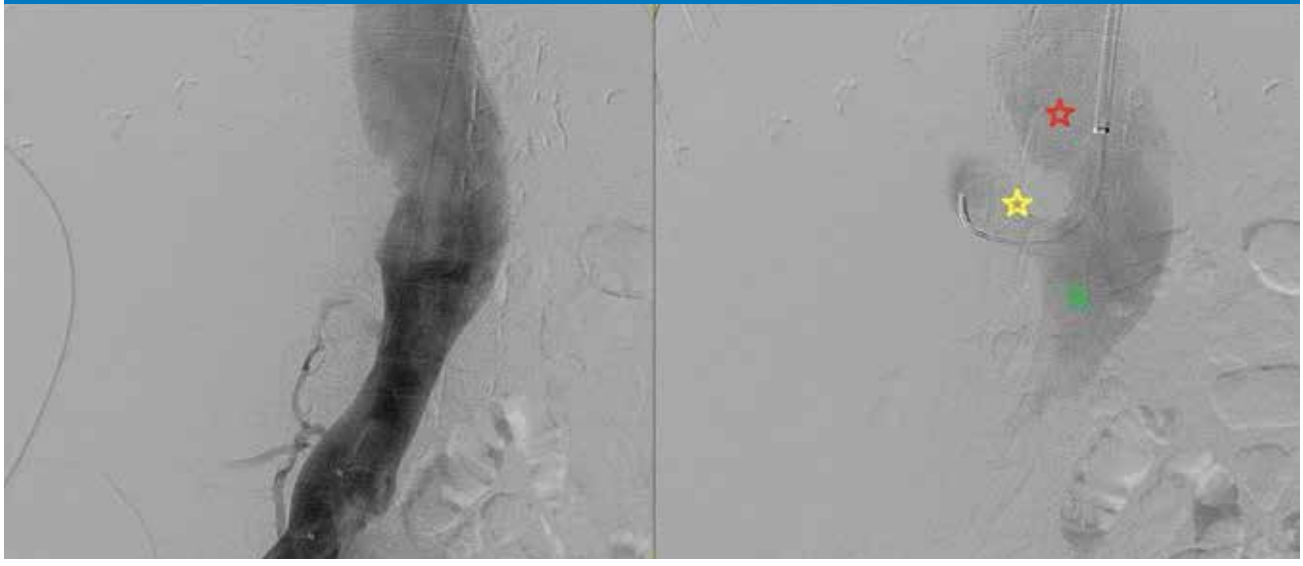


Table 2. Oxygen saturation (%) 'step-up' on inferior caval venogram.

Infrarenal inferior vena cava	79%
Right renal vein	96%
Suprarenal inferior vena cava	87%

Table 3. Transjugular hepatic venogram with pressure (mmHg) measurements.

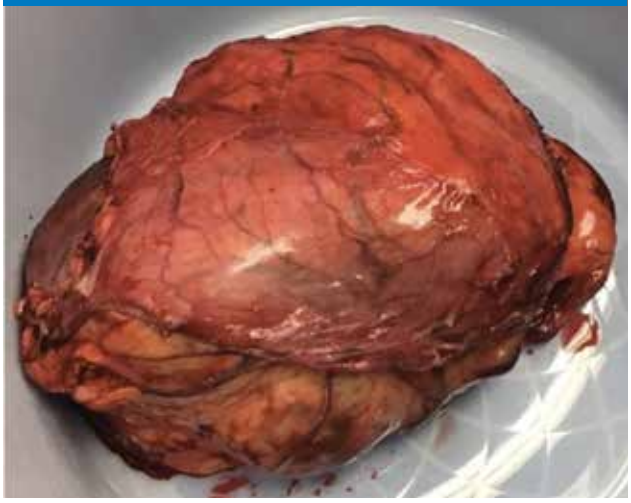
Free hepatic vein pressure (FHVP)	18
Wedged hepatic vein pressure (WHVP)	19
Hepatic venous pressure gradient (HVPG = WHVP-FHVP)	1

Management included volume optimization with aggressive diuresis and therapeutic paracenteses throughout the hospital course prior to right radical nephrectomy (Figure 4). Pathology of the specimen noted WHO grade 3 and AJCC stage III clear cell RCC. Postoperatively, the patient showed marked improvement in symptoms and cardiac function, highlighted by reduced dyspnea and edema. Following surgery, BNP levels decreased significantly, and renal function returned to baseline. Postoperative echocardiography illustrated decrease in pericardial effusion and right ventricular size (Figure 2). Cardiac MRI and stress test were unremarkable prior to planned immunotherapy for stage III RCC.

Discussion

The incidence and prevalence of HOHF are unknown; however, it is estimated to account for 1-2% of all heart failure cases.[1] The pathophysiology of HOHF varies depending on the underlying cause, which can be divided into two categories: (1) as a result of peripheral vasodilation, and (2) as a result of arteriovenous (AV) shunting.[2] Beriberi, thyrotoxicosis, sepsis, and chronic severe anemia are all notable cases of peripheral vasodilation. AV shunting on the other hand can be acquired (traumatic or iatrogenic), congenital (hereditary hemorrhagic telangiectasia), due to Paget disease of the bone, or related to an underlying malignancy.[3] AVF and HOHF associated with cancer are uncommon presentations. The most common underlying malignancy associated with AVF appears to be renal cell carcinoma.[4] Clear cell carcinoma is frequently associated with a mutated Von-Hippel-Lindau (VHL) tumor suppressor gene, which causes an increase in the production of hypoxia inducible factors (HIF), which promotes angiogenesis and hypervascularity.[5] Imaging, such as enhanced CT or MRI, as well as an angiogram, are frequently used to demonstrate hypervascularity or an AV shunt. A thorough examination and a step-by-step investigation plan, which included an ascitic fluid analysis, echocardiogram, and right heart catheterization confirmed HOHF to be the cause of our patient's presentation. An IVC and renal venogram with blood gas sampling were critical to establish AV shunting with the mass.

Figure 4. Gross specimen showing right kidney mass after nephrectomy.



Conclusions

Right heart catheterization is the gold standard to confirm a high-output state. AV shunting should always be considered in the workup of high-output heart failure. The presence of an arteriovenous fistula can be evaluated using imaging. Contrast enhanced CT or MRI are the most commonly used imaging modalities to identify a tumor-related AVF. When imaging is not helpful, a venogram with blood gas sampling could be considered to establish an RCC-associated AVF as seen in our case.

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Observing Siblings of Children with Complex Medical Needs

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Abstract

This study examined the behaviors of siblings of child(ren) with complex medical needs (CCMN). Previous research indicates that these siblings may develop resilience and empathy, but also face risks like depression and poor social skills. To gain further insights, we completed surveys after observing family interactions.

We reviewed 96 surveys, finding that siblings provided physical care 42.6% of the time and emotional care 32.5% of the time. Reactive outbursts were seen in 22.1% of cases, and 55.2% of siblings spent time alone, apart from their CCMN sibling. One-on-one time with parents was noted in 50% of observations, and siblings joined activities in 91.7% of cases. The siblings were mostly happy (73.7%), with some neutral (22.1%) and sad (4.2%). Conflicts occurred in 53.1% of cases, usually with mothers addressing the conflict.

These findings show that siblings of CCMN often take on significant caregiving roles, which can affect their emotional and social development. Although the study was set in a vacation environment, it highlights the importance of recognizing the unique challenges these siblings face. More research is needed to understand how age and the severity of the medical condition influence sibling experiences.

Introduction

We conducted an observational study on siblings of children with complex medical needs (CCMN) during a volunteer experience at a non-profit venue where families vacation. This setting provided an invaluable opportunity for the families to engage in rich experiences.

Previous research on siblings of CCMN unveils a complex interplay. Interestingly, multiple studies have demonstrated evidence of increased resilience and empathy among these siblings, suggesting that their unique family roles can foster significant personal growth.[1,2] Conversely, other studies indicate that siblings of CCMN are at increased risk of depression, low self-acceptance, poor social skills, and worse academic performance when compared to age-controlled participants with healthy siblings.[3,4] However, the work by Emerson and Giallo indicates that these negative impacts are minimal and are eliminated when adjusting for low socioeconomic status.[5]

Haukland et al. explain these contradictory findings by hypothesizing that many siblings simultaneously experience positive and negative emotional reactions to their situation.[6] Additional studies propose that these different experiences and reactions may be attributed to cultural discrepancies,[7] unique challenges associated with the CCMN,[8] and socioeconomic status.[5]

Methods

We conducted a survey to gain insight into our observations of CCMN and their families. This study does not meet the criteria for the NIH definition of human subject research because the data collected is from observations of public behavior without interference from observers. Questions included in the survey were designed to allow no direct interaction with families or observed parties. The questions were designed by the investigators with a goal to observe basic interactions focusing on siblings of CCMN. The survey was intended as a pilot study to provide information that could direct further studies. Prior to observing families and conducting surveys, participants were given an overview on types of behaviors, how to classify behaviors, identification of subjects, collection of data, and how to fill out the survey. We conducted surveys after every observation of CCMN and their siblings and parents. Observations were only

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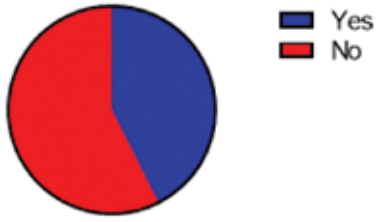
conducted on families that included siblings of CCMN. Nearly 200 families were present on location on any given day. The survey was administered via an electronic link with Google Forms, consisting of 10 questions, focusing on the encounter we witnessed. We were asked yes or no questions about whether or not the healthy sibling was performing physical cares (i.e. holding hand, wiping face), emotional cares (i.e. consoling an outburst), if the healthy siblings had reactive behaviors, if we observed one-on-one time with healthy sibling and parent, and if we saw a healthy sibling interact with another family. The survey also included observations on conflicts and stressful situations involving a CCMN, which family member intervened in the conflict (Mom, Dad, and/or siblings), and if we observed any healthy siblings spending time alone versus if they were always with the CCMN sibling. Additionally, the survey included space to describe the affect of the siblings (sad, happy, neutral). The survey was anonymous. Answers were automatically uploaded and recorded into an electronic database. None of the questions of the survey were required to be answered for the survey to be submitted and all blank or missing data was not included in the final analysis. At the end of the survey, there was a place for comments on anything else interesting the medical student saw and wanted to report. Data was analyzed using GraphPad Prism v.8.

Results

We completed a total of 96 surveys over six days. We documented siblings performing physical cares in 42.6% of observed interactions and emotional cares in 32.5% of observed interactions. Reactive outbursts were noted in 22.1% of surveys. Siblings were seen spending time alone, away from the CCMN 55.2% of the time. One-on-one time with healthy siblings and parents was seen in 50% of documented observations. Siblings of CCMN were seen participating in activities in 91.7% of survey responses. The most commonly observed affect of the healthy siblings was happy (73.7%), followed by neutral (22.1%), and sad (4.2%). The siblings were rarely seen interacting with other families at the resort (16.5%). Conflict was observed in 53.1% of survey responses. When there was conflict, the most common family member to be seen responding was the mom alone (25.4%), or the mom, dad, and sibling together (25.4%). The next most common response was the mom and dad together (21.6%), followed by the dad alone (13.7%) and the sibling alone (13.7%). There was one response of the dad and a sibling together.

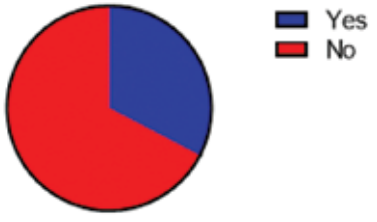
Figure 1. (A-H) Summary of observations of behaviors and relationships between CCMN, accompanying siblings, and parents. (I) Observed instances of conflict between CCMN and family members, as well as observed family members involved in resolving conflict.

1A. Observed physical cares.



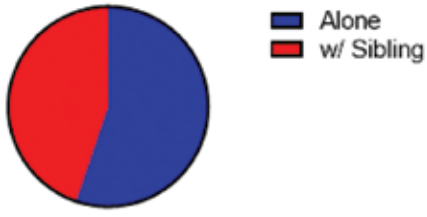
Total=94

1B. Observed emotional cares.



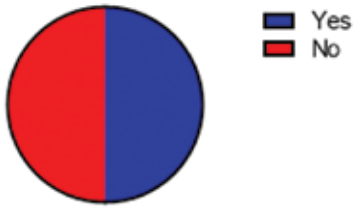
Total=95

1C. Observed sibling alone.



Total=96

1D. One-on-one time with parents.



Total=96

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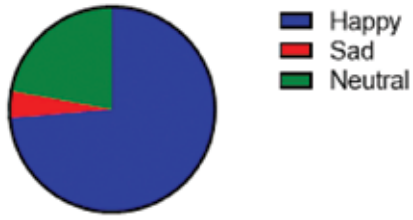
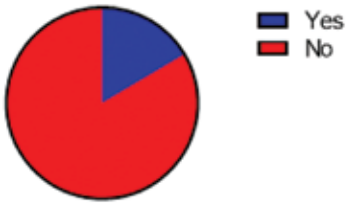
Figure 1. (A-H) Summary of observations of behaviors and relationships between CCMN, accompanying siblings, and parents. (I) Observed instances of conflict between CCMN and family members, as well as observed family members involved in resolving conflict.

1E. Observed sibling affect.



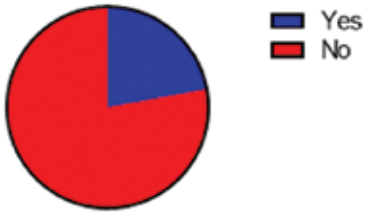
Total=95

1F. Interaction with other families.



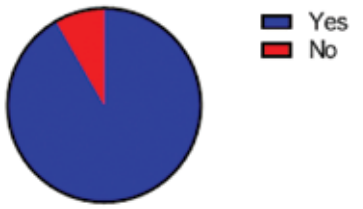
Total=91

1G. Observed reactive behaviors.



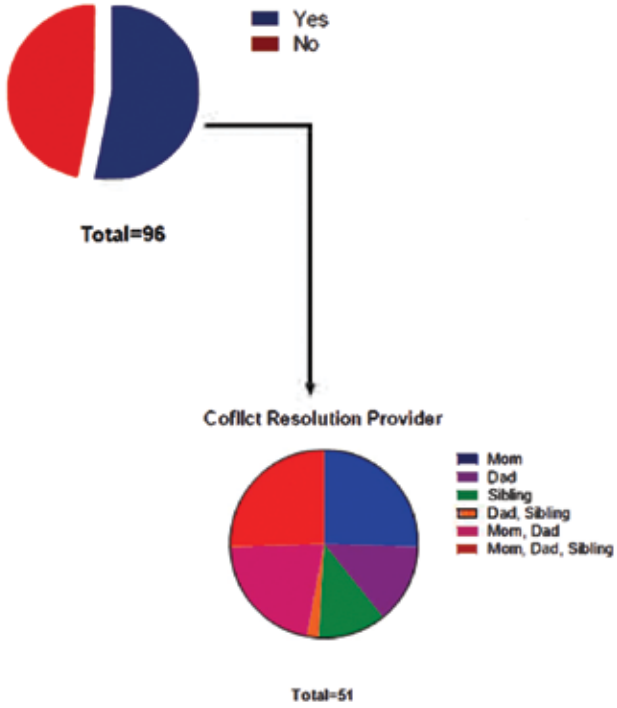
Total=95

1H. Sibling present in activities.



Total=96

1I. Observed instances of conflict between CCMN and family members, as well as observed family members involved in resolving conflict.



Discussion

Previous studies have demonstrated the psychosocial impact of complex medical needs on CCMN and their parents, including increased incidence of depression, anxiety, and decreased social engagement.[9] This study redirects the focus to examine the relationship between CCMN and their healthy siblings.

Our observations show that a significant proportion of observed healthy siblings performed emotional or physical cares for CCMN. Additionally, there were times when CCMN were left alone in the care of their siblings. Thus, these individuals are expected to have a large amount of responsibility for the well-being of their medically complex siblings. As reported by Kittelsen et al. siblings might even be exposed early to conversations and decisions about end-of-life care, creating additional emotional and physical stressors on healthy siblings.[10] While our observations of these relationships were limited, this responsibility could have a constant impact on siblings for the duration of their lives.

While these observations are valuable, they do come with inherent biases and limitations. Our observations occurred

in a setting which provided accommodations for children with complex medical needs. Additionally, the CCMN and their siblings were observed while in a vacation setting. These conditions could have provided a skewed perspective on the normal behaviors of siblings and provided an underestimation of the typical stress they experience day-to-day. Further work to identify how various factors, including the age of the sibling and the severity of the medical needs, would need to be conducted to better appreciate the impact of these stressors.

Conclusion

Our observations revealed that siblings of CCMN frequently help with physical and emotional care in addition to regular participation in activities with the rest of the family. The observations of siblings of CCMN provide evidence as to why these children may have qualities such as empathy and resilience later in life as reported in the literature. On the other hand, the presence of conflict was seen in approximately half of the interactions which follows the idea that these siblings may develop worse social skills and poor self-acceptance. Despite the limitations of this current study, we conclude that healthy siblings of CCMN are an underappreciated and unrecognized population with psychosocial stressors that need to be appropriately identified by providers. Based on our conducted study, we believe that further research is essential in fully understanding how age and the severity of the medical condition influence sibling experiences.

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South Dakota Adopts
Universal Blood Lead Testing
at 12 and 24 Months

EFFECTIVE JANUARY 1, 2025, the South Dakota Childhood Lead Poisoning Prevention Program is implementing universal blood lead testing for all children at 12 and 24 months of age.

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- **UNIVERSAL TESTING:** Blood lead level tests at 12 months and 24 months for all children, regardless of risk factors.
- **CATCH-UP TESTING:** Any child without two blood lead tests by age 3 years should receive testing.
- **CONTINUED RISK-BASED TESTING:** Annual testing for children aged 6 months to 6 years based on verbal risk assessment.

As a trusted physician, your role is critical in ensuring this initiative's success. Help ensure every child receives the testing they need by integrating universal testing into your routine care for young children. Let's work together to protect the health and potential of every child in South Dakota.

QUESTIONS:

Contact the program at 605-773-3737 with questions regarding case management of children with lead in blood.

HOW TO REPORT RESULTS:

- Fax to 605-773-5509
- Website reporting form at sd.gov/diseasereport,
- By mail to: Infectious Disease Surveillance, Department of Health, 615 East 4th Street, Pierre, SD 57501; marked "Confidential Disease Report"

All blood lead level results must be reported to the South Dakota Department of Health

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Cytomegalovirus Ventriculoencephaltis Post-Temozolomide Use For Glioblastoma Multiforme

Dawood Shehzad, MD; Helean S. Barwari, MS IV; Mustafa Shehzad, MD, and Garcia Alfredo, MD

Abstract

We report a case of a male in his 60s with a past medical history of glioblastoma status post chemoradiotherapy and surgical resection. He had completed neoadjuvant temozolomide and was 26 days post-chemotherapy on presentation. He presented with fever, weakness, and confusion. Further evaluation revealed cytomegalovirus (CMV) ventriculoencephaltis. He was started on valganciclovir, but his hospital course was complicated by a pulmonary embolism. Repeat imaging showed tumor progression, and he was a poor surgical candidate. This case highlights the complexities of managing patients with opportunistic infections post-chemotherapy. It adds to the growing literature of disseminated, invasive, and often lethal CMV infections in non-HIV patients post-use of temozolomide. We urge the need for vigilant monitoring and consideration of prophylactic measures in individuals deemed at risk for invasive CMV.

Introduction

Human cytomegalovirus (human herpesvirus-5) is a dsDNA member of the viral family Herpesviridiae. It is a ubiquitous virus that infects almost all individuals, and around 40%–100% of healthy individuals have positive serology (CMV antibodies).[1] Seroprevalence is higher in resource limited countries.[2]

CMV transmission occurs via blood products, breastfeeding, close-contact viral shedding, perinatally, and sexually.[3] Infection can occur as a primary infection, reinfection, or reactivation. Primary CMV in immunocompetent individuals usually has an asymptomatic or subclinical course. Mononucleosis is the most prevalent presentation of primary CMV, characterized by fever, rash, and leukocytosis. A definitive diagnosis is rarely required in immunocompetent patients, and the diagnosis of CMV cannot be made solely based on clinical examination. Clinical diagnosis is challenging due to similarity with Epstein-Barr virus.

After primary infection, CMV remains dormant in myeloid cells. Secondary disease results from reactivation or reinfection, common in immunosuppressed patients (e.g., HIV, organ transplant). This can present with specific organ diseases, such as encephalitis, retinitis, esophagitis, myo/pericarditis, pneumonitis, hepatitis, and colitis.[4]

Case Presentation

A male in his 60s with a past medical history of atrial flutter status post ablation, glioblastoma WHO stage 4 status post chemoradiotherapy and surgical resection presented after transfer from an outlying swing bed facility for one day of fever, weakness, and confusion. At his facility, he was noted to have waxing and waning confusion and a temperature of 102.8 °C. On admission, he was unable to provide much history and reported he felt 'fuzzy.' He had a temperature of 102.3°F, heart rate of 80 beats/minute, respiratory rate of 23 breaths/minute, blood pressure of 92/58 mm of Hg, and was saturating 97% on room air. On physical exam, he was alert and oriented to name and person but not place. He was ill-appearing and diaphoretic. His neurological exam was unremarkable, and he had no meningeal signs. His cardiopulmonary exam was remarkable for an irregularly irregular rhythm. His labs were remarkable for a WBC count of 3.7, Hgb 11.2, Plt 160, Cr 0.59, and Lactic acid of 1.5. A urinalysis was unremarkable, and a chest X-ray revealed no acute findings. A head CT showed evidence of a prior left parietal craniotomy, and encephalomalacia was otherwise unremarkable for any acute pathology (Figure 1). He was given intravenous fluid boluses for his hypotension, started on ampicillin, vancomycin, and ceftriaxone, and admitted for further management.

Five months prior, he had been diagnosed with glioblastoma multiforme via a brain biopsy after an MRI brain revealed an intra-axial lesion suspicious for malignancy. (Figure 2). Biopsy showed glioblastoma grade WHO grade 4, isocitrate

Figure 1. Noncontrast CT of the head showing Left parietal craniotomy with left parietal encephalomalacia.

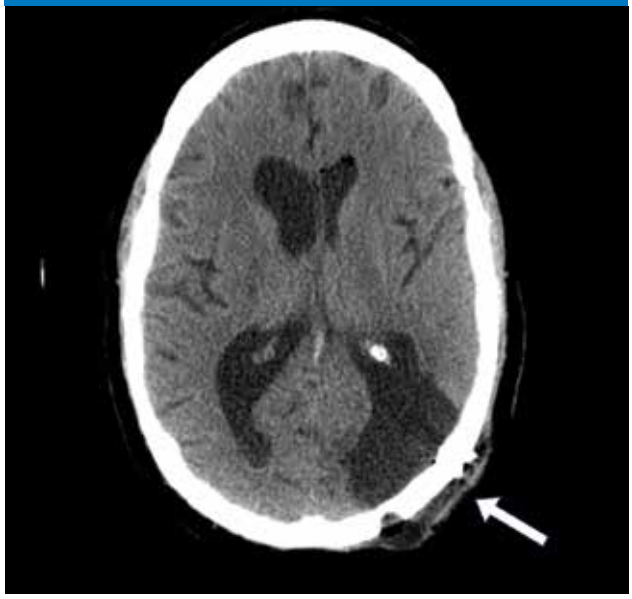
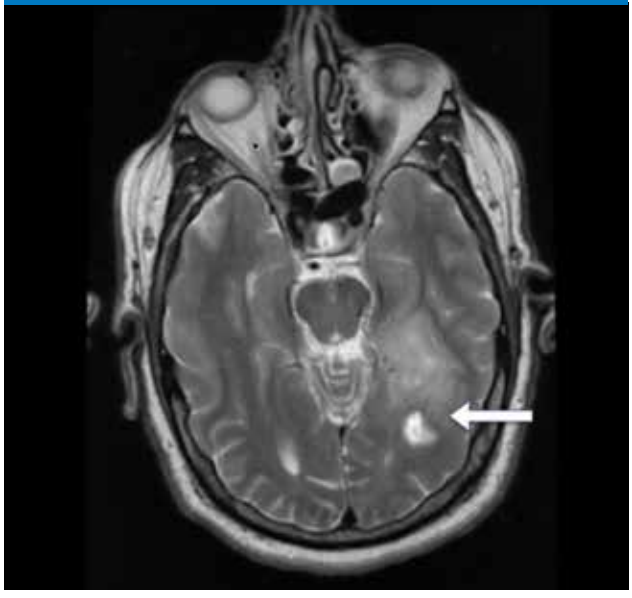


Figure 2. MRI of the brain showing an enhancing lesion in the left occipital lobe with irregular peripheral enhancement and central nonenhancement/necrosis. This favors a diagnosis of a glial neoplasm with the enhancing components possibly representing degeneration into a higher-grade component (glioblastoma).

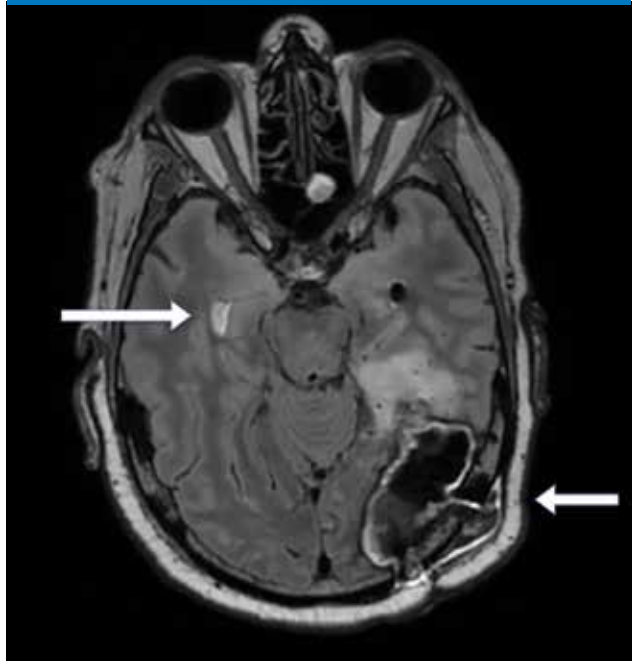


dehydrogenase (IDH) wild type, methylation status negative. He underwent gross total resection and left craniotomy and received a short course of dexamethasone prior to surgery. His postoperative course was complicated by the dehiscence of his craniotomy wound, a CSF leak, and a surgical site infection. He was treated with antibiotics and underwent wound revision one month later, which healed well. He did not receive any steroids post-operatively.

Post-operatively he started adjuvant external beam radiotherapy and temozolomide. During this time, he remained on trimethoprim-sulfamethoxazole (TMP-SMX) for Pneumocystis jiroveci (PJP) Pneumonia prophylaxis and was doing well. Twenty-six days after his last temozolomide dose, he became confused and febrile and was transferred to our facility.

At our facility, a lumbar puncture (LP) revealed an opening pressure of 20 cm H2O, pale yellow cerebrospinal fluid (CSF) with a WBC count of 106/microliter (82% neutrophils), glucose of 37 mg/dL, and protein of 258 mg/dL, indicating a central nervous system infection. Aseptic meningitis from TMP/SMX was also considered. Given his prior surgical site infection, for which he was treated with a short course of antibiotics, nosocomial bacterial meningitis was also a possibility; hence, he was continued on vancomycin, ceftriaxone, and ampicillin. A meningitis/encephalitis panel tested positive for CMV, and he started on a three-week course of intravenous ganciclovir. An MRI showed abnormal signals within the ventricles, suggesting ventriculitis (Figure 3). A CMV quantitative PCR on the CSF was positive for 619 IU/ml of CMV DNA.

Figure 3. MRI Brain showing the Left parieto-occipital surgical cavity with thin peripheral enhancement in communication with the left occipital horn, similar to prior exams. There is non suppression of CSF in the right occipital horn as well as the right frontal horn on FLAIR images, raising suspicion for intraventricular tumor spread vs ventriculitis.



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He continued to spike fevers, and during his hospitalization, he developed hypotension and acute hypoxic respiratory failure. A computed tomography angiography (CTA) of the chest showed acute bilateral pulmonary thromboembolism with a large clot burden. A lower extremity duplex showed non-occlusive deep vein thrombosis in the bilateral femoral veins. An echocardiogram revealed right heart strain, and he underwent pulmonary artery thrombectomy and was started on a heparin drip; he did well for a few days. Unfortunately, he became more confused and developed increasing episodes of twitching in his upper extremity. His infectious workup remained negative. A repeat MRI of the brain showed an increase in restricted diffusion in the lateral ventricle, compatible with pus from his ventriculitis. The residual glioblastoma involving the left temporal lobe was larger. He was evaluated by neurosurgery, who felt there was no role for further operative management. Ultimately, his family decided to pursue hospice care, and all antibiotics/virals and anticoagulation were discontinued.

Discussion

CMV ventriculitis typically presents with subacute alteration in alertness, cranial neuropathy, gaze-evoked nystagmus, and features of raised intracranial pressure. Other presentations of CMV-associated CNS infection, particularly in HIV-infected individuals, include necrotizing polyradiculomyelitis, a Guillain–Barre-like syndrome of ascending weakness with hyporeflexia, motor predominant neuropathy, and vasculitis.[5]

Diagnosing CMV infection typically involves demonstrating a cytopathic effect, characterized by cytomegalic cells containing intranuclear and intracytoplasmic inclusions resembling 'owl's eyes'.[6] While our patient didn't show this effect, their clinical presentation post-chemotherapy, MRI findings, and positive CMV PCR supported the diagnosis. Neuropathological imaging reveals periventriculitis with ependymal and subependymal necrosis, and hydrocephalus due to CSF flow obstruction.[7,8] MRI shows variable white matter signal intensities and periventricular enhancement, aiding in differentiation from other meningoencephalitis causes.[8] Additionally, CSF PCR is highly sensitive and specific for CNS CMV infection.[9]

Our patient had a recent diagnosis of glioblastoma and received near-total gross resection external beam radiotherapy in addition to temozolomide. The use of surgical resection along with adjuvant chemoradiotherapy remains the standard of care for the treatment of glioblastoma multiforme.[10,11] He developed cytomegalovirus ventriculitis as an opportunistic infection while receiving chemotherapy with temozolomide.

The EORTC-NCIC trial (2009) showed a survival advantage with temozolomide plus radiotherapy post-surgery followed by temozolomide alone versus radiotherapy alone in glioblastoma patients. Two-year survival rates were 27.2% versus 10.9%, and five-year rates were 9.8% versus 1.9%. MGMT promoter methylation status strongly correlated with survival. [12]

In the trial, 12/287 patients experienced grade 3–4 lymphopenia during temozolomide therapy. A meta-analysis on 39 patients advanced neuroendocrine tumors revealed a 46% incidence of grade 3–4 lymphopenia after four months of temozolomide treatment, with four cases of opportunistic infections (1 case of disseminated VZV, 1 case of disseminated CMV, and 2 cases of PJP).[12,13] Our patient developed grade 4 lymphopenia for 4 days, 28 days post-temozolomide therapy. Temozolomide-linked opportunistic infections are complicated by concurrent steroid use in the literature, though our patient received only a brief course of dexamethasone. [14]

For patients receiving temozolomide during radiation therapy, PJP prophylaxis with TMP-SMX is recommended due to observed lymphopenia and impaired T-cell immunity in initial phase 2 trials, although PJP pneumonia incidence is low and occurs in those with additional infection risks. Selective prophylaxis, continuing until lymphocyte count exceeds 800/microL, may be considered, with estimated numbers needed to treat and harm of 288 and 34, respectively. [15]

There is no recommendation for prophylaxis against other opportunistic infections, such as viral or fungal infections. Our case and others in the literature demonstrate an association between CMV infection and temozolomide.[16-19] The optimal approach to the antiviral treatment of CNS cytomegalovirus infection is not clearly defined. Response to treatment in patients who have cytomegalovirus encephalitis/ventriculitis with ganciclovir or foscarnet alone has not improved survival; even prophylaxis with ganciclovir or foscarnet, at doses used for maintenance in cases of cytomegalovirus retinitis, does not guarantee protection against development of cytomegalovirus encephalitis. [20] Because CMV encephalitis almost always develops in the context of profound suppression of cell-mediated immunity, the treatment approach should include attempts to decrease immunosuppression whenever possible. Preemptive therapeutic administration of valganciclovir or ganciclovir in patients who are receiving temozolomide treatment can be considered. Prophylactic administration of ganciclovir/valganciclovir has not demonstrated efficacy in

enhancing outcomes related to CMV reactivation in critically ill patients. Findings from a phase II trial indicate a reduction in CMV reactivation incidence; however, the trial failed to find significant results on incidence of secondary infections, duration of intensive care unit (ICU) stay, or levels of interleukin-6, a proinflammatory cytokine associated with mortality in ICU populations.[21]

Conclusion

This case highlights the complexities of managing opportunistic infections in immunocompromised individuals and malignancy. It adds to the growing literature of disseminated, invasive, and often lethal CMV infections in non-HIV patients post-use of temozolomide. We urge the need for vigilant monitoring and consideration of prophylactic measures in individuals deemed at risk for invasive CMV.

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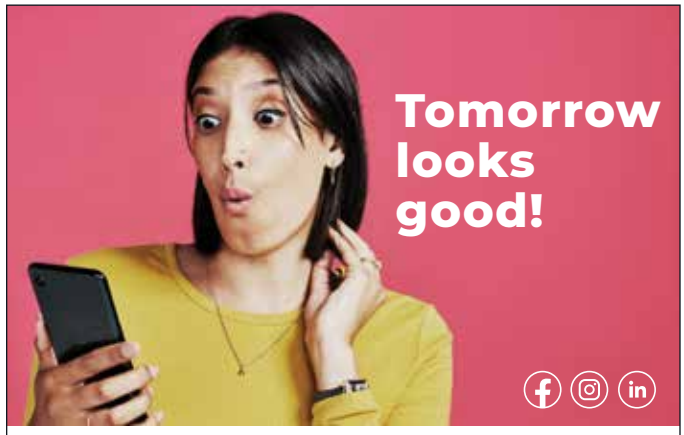

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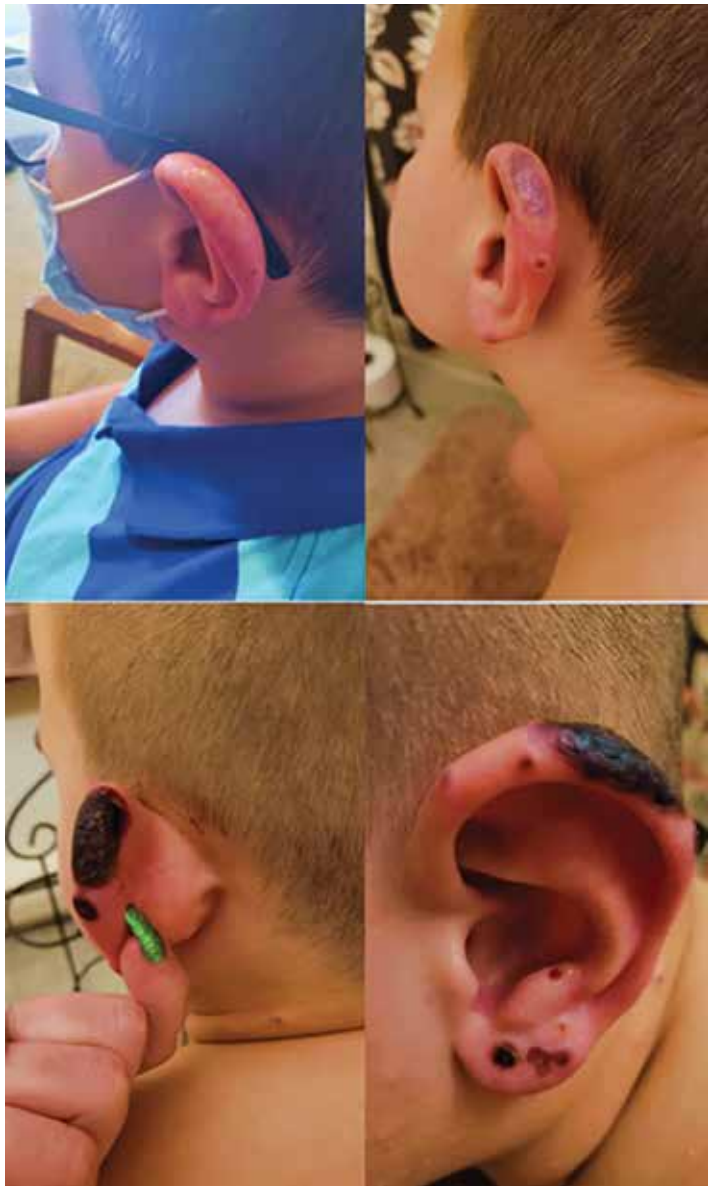
Hydroa vacciniforme: An Unusual Dermatologic Complication of Epstein-Barr Virus Infection

Peter Paul C. Lim, MD

A previously healthy 7-year-old boy presented with a one-year history of relapsing and remitting dynamic rash exclusively on sun-exposed areas, often in the helix of the ears. The rash would start as an erythema with edema and overlying vesicles that would evolve into eschar with underlying necrotic plaques and eventually heal with varioliform scarring with depressed atrophic macules (Figure). Skin biopsy showed spongiosis and interface dermatitis with perivascular lymphocytic infiltrate and papillary edema consistent with hydroa vacciniforme (HV). Whole blood Epstein-Barr virus (EBV) viremia was 221,508 IU/mL (108,538 copies/mL). Plasma viral load was 2,351 IU/mL (1.151 copies/mL). Mesenteric lymph node biopsy showed paracortical hyperplasia with reactive follicles at variable stages of maturation with scattered EBV cells with no evidence of lymphoma. Serum protein immunifixation test was normal and immunologic work up did not identify primary error of immunity. Recurrence of lesions was prevented by strict photorestriction. EBV is associated with variable cutaneous lesions and HV is a rare dermatologic complication of EBV.[1,2] This is usually associated with remitting and relapsing cutaneous eruption on sun-exposed areas of the skin that evolves from a vesicle with underlying erythema or crusted papules to eschar formation that often leads to varioliform scarring and pigmentation changes. Skin and lymph node biopsy are necessary to establish conclusive diagnosis.[1,2] Further evaluation for EBV-associated lymphoproliferative disorder or an underlying immunity impairment is imperative in the work up.



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Fighting For You and Your Patients

The SDSMA serves as your vehicle for advocacy for your patients and the art and science of medicine through lobbying at the state and federal levels, grassroots activity, and legal initiatives.

SDSMA PAC is your grassroots avenue that works to impact public policy decisions through bipartisan political participation. SDSMA PAC supports and elects pro-medicine candidates on the state level. Members of the SDSMA and their spouses can join SDSMA PAC.

The SDSMA's motto is "Values, Ethics, Advocacy." We take our advocacy role to heart. With your help, SDSMA and SDSMA PAC have the opportunity to dramatically impact the political and legislative process to create meaningful changes in South Dakota's current health care system:

- Improving health and access to care in rural areas;
- Increasing Medicaid reimbursement;
- Promoting Medicare physician payment reform and stopping reimbursement cuts;
- Working to improve clinical quality and patient safety;
- Partnering with state agencies to tackle regulatory, socioeconomic, public health and scientific policy issues;
- Advocating for public health immunizations;
- Promoting adequate funding for medical education;
- Stopping inappropriate expansion of non-physician scope of practice;
- Defending the patient-physician relationship; and
- Reforming medical liability.

If you would like to become involved in any of our advocacy programs, call 605.336.1965 or visit sdsma.org.

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

Help Shape the Future of Medicine in South Dakota

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

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

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

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

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

Don't Let Your SDSMA Benefits Expire – Renew Today!

Thank you for your membership in the SDSMA. Association renewal is annual and runs on the calendar year from January-December. If you haven't yet, please visit the link that was sent to your email to renew. There is still time!

We look forward to another great year of providing physicians with valuable benefits. SDSMA membership gives you access to networking and leadership opportunities with your local colleagues in your District Medical Society, a monthly subscription to South Dakota Medicine, your name and practice information in the physician directory, and advocacy on behalf of patients and the medical profession.

When you renew, you can choose to renew your AMA dues and sponsor a student's SDSMA membership dues for four years of medical school with one, easy transaction.

- **Advocacy.** Participate in opportunities to have your voice heard. The SDSMA advocates at the state and federal levels on key health care issues impacting patients and physicians.
- **Professional growth.** Grow leadership skills through the SDSMA Health Leadership Institute, committee involvement, collaborative efforts, and events.
- **Education.** Access education programs on current topics and publication opportunities in the peer-reviewed medical journal, *South Dakota Medicine*.
- **Well-Being.** Navigate the stressors of today's ever-changing health-care landscape and prioritize wellness with the South Dakota Physician Well-Being Program.

Thank you to those who have already renewed. If you haven't yet, please complete your renewal at sdsma.org today!

Have you recently retired? Please let us know by contacting the SDSMA office at membership@sdsma.org or 605-336-1965.

Please note: if you are a life member, your membership is continuous. You do not need to take any steps to renew.

Nominations Being Accepted for SDSMA Leadership Positions

TThe SDSMA Nominating Committee will meet on March 5 to nominate officer candidates for 2025-26 to be voted on by the membership. Those positions include President-Elect, Vice President, Secretary-Treasurer, and AMA Alternate Delegate. This committee also nominates candidates for three At-Large members of the Board of Directors and a Policy Council Chairperson from among (or such persons must be) members of the SDSMA Policy Council.

Those SDSMA members who are eligible to run for any of these positions and have an interest must submit their name to one of the members of the Nominating Committee listed below by February 24. The Nominating Committee member will present your name at the committee meeting.

To be eligible to run for an At-Large member or Policy Council Chairperson position, a member must be a member of the Policy Council and have attended at least four meetings as a Policy Councilor or Alternate Policy Councilor during the three years prior.

Those who have questions about the nominating or election process for SDSMA leadership positions may contact the SDSMA office at 605-336-1965.

Nominating Committee members:

- Rochelle S. Christensen, MD – jensrochelle@gmail.com
- Denise S. Hanisch, MD – dr.mom83@gmail.com
- Dawn M. Hill, MD – dawnhillmd@gmail.com
- Mary Jo Olson, MD – maryjo.olson@icloud.com
- Tim M. Ridgway, MD – tim.ridgway@usd.edu

SAVE THE DATE

2025 SDSMA Annual Leadership Conference is May 30

The 2025 SDSMA Annual Leadership Conference is Friday, May 30 at the Hilton Garden Inn Downtown Sioux Falls.

The theme of this year's conference is artificial intelligence. With presentations, discussions, networking opportunities and the awards banquet, the Annual Leadership Conference is a great time to share ideas and learn from fellow members.

Do you have a policy issue you'd like to bring to the SDSMA's attention? Schedule a time

to address the SDSMA Policy Council during the Membership Open Forum. Watch sdsma.org for policy submission information to be posted soon.

The Annual Leadership Conference is a benefit of your membership – there is no registration fee for members. Tickets are required for some events and meals, and will be available for purchase at sdsma.org.

Call For Abstracts – Student Poster Session

The South Dakota State Medical Association announces the Call for Abstracts for presentation at the 2025 Annual Leadership Conference being held on Friday May 30, 2025 at the Hilton Garden Inn Downtown Sioux Falls.

The committee welcomes submissions of abstracts from USD SSOM medical students. Early submission of abstracts is appreciated. The abstract may be from basic science, clinical research or the Humanities.

SUBMISSION

Abstracts must be submitted to
Keith.Hansen@sanfordhealth.org and ereiss@sdsma.org.

DEADLINE – Friday, March 7, 2025.

Please prepare your submission in accordance with the requirements listed below:

- Abstracts must be submitted in a Word document using Times New Roman Font with a font size of 12 and no more than 300 words (not including title and authors' names), single-spaced. Do not indent headings.
- Abstracts should be proofread prior to submission. Abstract data cannot be altered after the submission. Title and authors may not be changed.
- Abstracts must be original work and should not have been previously published.
- The initial aspect of the abstract should contain the title and the author(s) name and respective site(s) of the study. The author(s) MUST include a faculty member.
- The text should be structured to describe the objective of the study and results of your work. It should include the following headings:
  - **Introduction:** a brief statement about the purpose of the study and the current state of research in the field.
  - **Methods:** the method(s) of study or data collection used.
  - **Results:** a summary of the study results including sufficient details to support your conclusions.
  - **Conclusions:** a statement explaining the significance of your work and the implications for further research.
- In the title, please include type of study: case report, retrospective cohort study, etc.
- Case reports should be structured as: Introduction, case report, and conclusion.

Read *InSession* for Weekly Legislative Updates

For legislative updates from the SDSMA during session, watch your email for SDSMA's newsletter, InSession.

This weekly newsletter keeps members informed about SDSMA's legislative activities and key health-related bills and action alerts, providing a timely look at the actions of the state legislature. The 2025 state legislative session began on January 14 and continues through March 31.

2025 SDSMA Awards – Nominate Someone!

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

Nominations are being accepted at sdsma.org until February 28 for the following awards:

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

Are there people that come to mind who deserve to be recognized? Nominate them today! Award recipients will be honored at the awards banquet in Rapid City during the SDSMA Annual Leadership Conference on May 30, 2025.

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

Follow us on Social Media!

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

SOUTH DAKOTA
BOARD OF MEDICAL & OSTEOPATHIC EXAMINERS

101 N. Main Ave., Suite 301 Sioux Falls, SD 57104
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www.sdbmoe.gov sdbmoe@state.sd.us

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- **Licenses and regulates about 11,500 licenses within ten different medical categories**
- **Establishes regulations by proposing legislation and adopting administrative rules**
- **Provides Safe Haven, supports, and promotes confidential monitoring of its healthcare providers**

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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.

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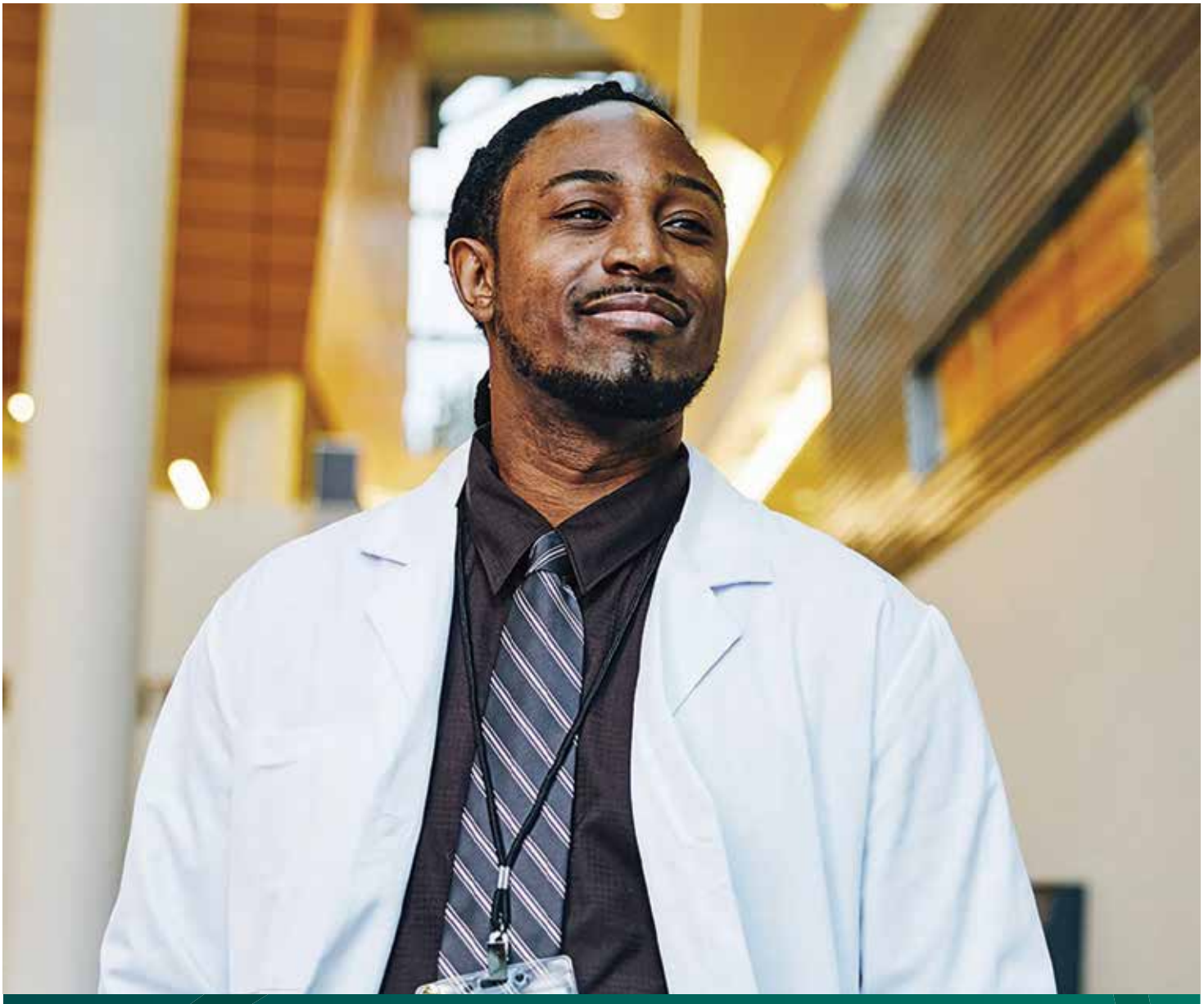

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Copic is proud to be the endorsed carrier of the South Dakota State Medical Association. SDSMA members may be eligible for a 10% premium discount.

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