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A Year of Transition

By Tim M. Ridgway, MD, FACP
SDSM A President

I t has been almost one year since my induction as SDSMA president, and I cannot help but be reflective and philosophical. I hit the ground running in regard to our newly developed Health Leadership Institute. A fair amount of time was spent speaking with various constituencies about the value leadership training could bring. As expected, there were some bumps in the road, but I am pleased to state we have now hired an excellent lead facilitator and plans are under way to begin our first cohort of leadership training. This promises to be an excellent way to add value to your membership.

The next piece of important business was overseeing the change in our governance structure. Change can be difficult, and this is no exception. Our Council of Physicians has changed its name to the Policy Council, and will meet biannually instead of quarterly. It is hoped that by streamlining this process, we can conduct business more efficiently. This will work, however, only if we have committed involvement by our membership.

Perhaps the biggest issue of the year related to the sale of DAKOTACARE. Our CEO, Barb Smith, and your Executive Committee have put in many long hours to ensure we could maintain the excellent relationship the SDSMA and DAKOTACARE have shared over the years. I am pleased to say that we have come a long way in achieving that goal. Stay tuned for more information on that front.

The 2016 legislative session again proved to be very busy, and our organization was again at the forefront of the issues. Medicaid expansion was a very hot topic, which our organization supported. While slowed negotiations with CMS prevented the introduction of legislation to expand Medicaid during the 2016 session, a coalition established by the governor will continue its efforts to develop ways to expand both coverage and services to those currently without. Highlights of bills passed (which the SDSMA supported) included HB 1110, an act to provide medical care for certain unborn children, SB 28, an act to require meningococcal immunization for school entry, HB 1079, an act to permit the prescription and possession of an opioid antagonist in certain instances, and HB 1029, an act to make an appropriation to the Department of Health to fund a rural residency program. This legislation will appropriate the necessary funds to the South Dakota Department of Health to support the development of a rural family medicine residency track for two additional medical students per year. There were many other accomplishments this session, and I refer you to the SDSMA website which highlights all our accomplishments in this arena.

Of course, Barb and I embarked on our yearly presidential district visits in the fall and wrapped them up in January. Each year we hear from past presidents how wonderful and fulfilling these visits can be, and they certainly lived up to the hype. After meeting with you in your home districts, I gained a fresh and wonderful perspective on the incredible work you are doing for the health of your patients and community. These experiences make me very proud to be called a South Dakota physician!

Over the past year, I have heard from some of you about concerns as to who the SDSMA truly represents-independent physicians versus system physicians versus others. It is my fervent belief the organization should represent ALL physicians. When the SDSMA was initially formed, and really up until the recent past, most physician's in the state practiced independently. Times have changed, however, and now the majority of South Dakota physicians are employed by health care systems. With this change, our organization too must change. I would urge all of you-independent and health care system physicians alike, to find a way to become active and be a voice for your constituency. We have abundant resources to advocate for physicians and patients, but to do this effectively, we require active participation from members who represent each demographic of the organization.

On the evening of my induction as your president, I referenced an article describing ethical and practical guidelines for professional medical associations. The main point of the article was to emphasize that physicians in these associations should associate to improve the care of the sick, to advance the health of the public, and to ensure that their fellow associates are faithful to that mission. In addition, they need to provide mutual professional support for their colleagues. We need to keep these principles in mind. This indeed has been a year of transition, and it is up to us to determine what the future holds for our association. Thank you for allowing me the privilege to serve as your president this year, and in closing I leave you with this quote from Buddha, "In the face of adversity, be grateful, for such opportunities do not come by often."

REFERENCES

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We know children.
In this month’s issue of South Dakota Medicine, we have a number of thought provoking manuscripts. In this issue Wookey and McKean discuss the importance of evaluating for distress in our patients with a cancer diagnosis. In their cross-sectional study they demonstrated a higher rate of distress in these subjects which presented with depression, fatigue, nervousness, loss of interest in normal activities and other negative symptoms. This manuscript highlights the importance of evaluating patients with a diagnosis of cancer for these common symptoms and providing appropriate support for these patients to improve their quality of life.

One aspect of cancer therapy especially for our young patients which can be a source of great distress and has been receiving increased attention is the possibility of post-therapy infertility or sterility. With improved survival rates, fertility after cancer therapy is becoming progressively more important for our younger patients. The term Oncofertility is a new branch of medicine that deals with preserving fertility in patients who are preparing to undergo lifesaving cancer treatments; including surgery, radiation and chemotherapy. For post-pubertal males cryopreserving gametes is a reasonable option with minimal risk. For female patients fertility preservation is a more complicated process requiring more intensive treatment options. Currently medically accepted fertility preserving options for females include in vitro fertilization with egg or embryo cryopreservation. For patients preparing to undergo pelvic radiation one can perform an oophoropexy, where the ovaries are moved out of the radiation fields and labeled with a clip so that the radiation oncologists can adjust their fields to lower exposure to the gonad. Other options include cryopreserving ovarian tissue, inducing a hypogonadotropic hypogonadal state with GnRH analogues and donor eggs/embryos. Cryopreserving ovarian tissue and inducing a hypogonadotropic state should still be considered experimental. The Oncofertility Consortium (http://oncofertility.northwestern.edu) is a nationwide group of clinicians, basic scientists and clinical researchers with an interest in helping cancer patients preserve their ability to reproduce after lifesaving treatment for cancer.

Drs. Huber and Vogt continue their discussion on “How to be an Author: A primer on writing for publication in the medical literature II: manuscript submission.” In part one the authors discuss the importance of having a well thought out plan on writing the manuscript prior to putting pen to paper (or fingers to keyboard). In this issue they discuss the process of preparing and submitting your manuscript to a medical journal. These two manuscripts are very helpful for the novice and expert medical writer.

Childbirth is usually a joyous event for the parents and family. This momentous event can be scarred by complications, most of which are uncommon. In this issue of the journal, Potu et al explore an unusual, potentially devastating complication of pregnancy – peripartum cardiomyopathy. This diagnosis can be difficult to make in its early stages, as normal pregnancy can mimic some of the symptoms. Since early diagnosis and treatment is vital to successful management, a high level of suspicion is warranted. In this manuscript the authors explore in detail the diagnosis and management of this life-threatening illness.

In this issue we also have three interesting case reports discussing cases of exanthemous pustulosis, central diabetes insipidus and hereditary pyropoikilocytosis. I hope you find this issue as interesting and informative as I did.
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Acute Generalized Exanthematous Pustulosis Secondary to Nifedipine

By Anup Shrestha, MD; and Vincent Rizzo, MD

Abstract
Acute generalized exanthematous pustulosis (AGEP) is an acute pustular reaction that occurs as a severe cutaneous adverse reaction to certain group of drugs. It has distinct clinical and histological features compared to other drug skin reactions as Stevens-Johnson syndrome or drug reaction with eosinophilia and systemic symptoms (DRESS). In this case report, we are presenting a 52-year-old female who underwent treatment for hypertensive urgency few weeks prior was readmitted in the hospital because of innumerable, fine pustules covering the whole body. Her discharge medications included nifedipine, labetalol. Her skin biopsy revealed subcorneal and intraepithelial pustules with neutrophilic collections, consistent with AGEP. She also sustained acute kidney injury and cerebellar stroke while remaining inpatient. After 18 days of inpatient conservative treatment her kidneys recovered, skin replenished and stroke symptoms improved. There is no single case report of such serious drug reactions along with other comorbidities due to nifedipine to date.

Introduction
In 1980, Beylot and colleagues introduced the term, “acute generalized exanthematous pustulosis” (AGEP) to describe acute pustular reactions with distinct clinical and histological features.1 AGEP is one of the severe cutaneous adverse reactions. We present an interesting case of AGEP due to nifedipine. No case report of AGEP with nifedipine has ever been reported to date.

Case Report
This is a 52-year-old female with past medical history of hypertension, psoriasis, and newly diagnosed diabetes type 2, discharged three weeks prior from the hospital after treatment for hypertensive urgency. She came in with the chief complaint of acute development of generalized small pustules all over the body over the period of three days. Her last discharge medications included nifedipine ER, labetalol and metformin. She reported no fever, headaches, or recent sick contacts. Initial vital signs were blood pressure 164/99, pulse 99 BPM, RR 18, T: 98.5 degrees, oxygen saturation of 99 percent on room air. Physical examination was positive for diffuse generalized rash over the neck, extremities, torso, axilla, non-blanchable, very dry scaly skin, and scattered fine pustules throughout the whole body. Initial lab findings were significant for leukocytosis (white blood cell of 18.3K) with bandemia. Her renal function was normal on admission (Table 1).

Initial impression of AGEP versus pustular psoriasis was made. She was treated with IV fluids and vancomycin initially. Dermatology service was consulted and skin biopsy was done. It revealed subcorneal and intraepithelial pustules with neutrophilic collections. The epidermis showed mild spongiosis with accompanying superficial dermal edema, which are consistent with AGEP. Her four sets of blood cultures were negative for any growth. Skin cultures failed to grow any organism. Streptococcal throat cultures were not done.

Table 1. Patient’s Renal Function Over the Period of Hospitalization

<table>
<thead>
<tr>
<th></th>
<th>Na</th>
<th>K</th>
<th>Cl</th>
<th>CO2</th>
<th>BUN</th>
<th>Cr</th>
<th>Glucose</th>
</tr>
</thead>
<tbody>
<tr>
<td>On admission</td>
<td>135</td>
<td>4.3</td>
<td>100</td>
<td>24</td>
<td>16</td>
<td>0.8</td>
<td>202</td>
</tr>
<tr>
<td>Day 9 (peak)</td>
<td>138</td>
<td>4.9</td>
<td>108</td>
<td>16</td>
<td>61</td>
<td>7.4</td>
<td>174</td>
</tr>
<tr>
<td>Day 18 (on the day of discharge)</td>
<td>132</td>
<td>5.4</td>
<td>104</td>
<td>23</td>
<td>27</td>
<td>1.9</td>
<td>159</td>
</tr>
</tbody>
</table>
On the sixth day of admission, the patient developed acute kidney injury (AKI). AKI work-up revealed normal C3/C4 level, serum osmolality of 298, urine osmolality 342, urine Na 71, K 20.8, chloride 49, negative urinary eosinophils, negative viral hepatitis panel. Her renal function over the period of hospitalization is tabulated in Table 1.

Renal ultrasonogram showed the right kidney at 12.4 x 5.4 cm, the left kidney at 13.1 x 6 cm; both kidneys were in normal shape and configurations with no evidence of hydronephrosis. Metabolic monitoring was done, and her AKI resolved spontaneously without the requirement of dialysis. Renal biopsy was not done in the patient as per the nephrologist’s recommendation.

In the morning of the ninth day of admission, she developed slurred speech. A head computed tomography (CT) showed interval development of subacute to acute infarct in the lateral right cerebellum, with increasing patchy white matter ischemic change in the high left frontal region. Carotid doppler showed zero to 19 percent diameter reduction bilaterally. A brain magnetic resonance imaging (MRI) showed acute to subacute infarcts in the right cerebellum and left frontal regions and remote infarcts in the left basal ganglia/corona radiate. An MRA showed a small focal millimeter-sized area of signal dropout in the proximal left A1 segment and also in the right MCA trifurcations, decreased flow related signal intensity along the course of the distal right vertebral artery – likely atherosclerotic in nature as well, and no MR detectable aneurysms. Transthoracic echocardiogram showed no patent foramen ovale and normal valves with ejection fraction of 60 percent. Neurology service was consulted and recommendations for aspirin and statins were made along with blood pressure control.

After 18 inpatient days, the patient had new skin with natural tone. Her acute tubular necrosis has resolved and speech has improved. She was discharged with follow-up appointments in medical and dermatology clinics in a week period. All her prior hypertension medications were stopped and replaced with another group of medications, which included clonidine and minoxidil.

**Discussion**

AGEP is a very rare but severe cutaneous adverse reaction that is usually caused by drugs. Antibiotics are the causative agents in more than 90 percent of the cases. A test can be conducted one month after the resolution of the disease. CD4 and CD8 cells are involved in the pathogenesis. CD8 cell initial influx results in apoptosis of keratinocytes and formation of sub corneal vesicles. The influx of drug-specific CD4 cells and the presentation of the drug bound to major histocompatibility complex (MHC) class II by...
keratinocytes results in the release of CXCL8 and granulocyte-macrophage-colony stimulating factor (GM-CSF) by both CD4 cells and keratinocytes and the migration of neutrophils into the epidermis, transforming the subcorneal blister into a sterile pustule. The treatment of AGEP is mostly supportive care. Corticosteroid has been used in the treatment in various studies. There was no difference in the course and duration of the disease between the different regimens. The entire self-limiting episode lasts up to 15 days. AGEP has a favorable prognosis, although secondary infection in patients with other medical comorbidities may result in poor outcomes. In previous studies, liver and kidney injury were reported. Thirty-two percent of patients with AGEP were found to have disturbed renal function in a retrospective analysis. Acute interstitial nephritis is a well-established mechanism of adverse renal drug reaction. Some cases of renal failure in AGEP may find an explanation in concomitant leukocytoclastic vasculitis (20 percent of cases) as hypersensitivity vasculitis may affect renal function. Our search in PubMed with AGEP leading to AKI revealed only one article, and no article with AGEP causing stroke. Patch testing was not done in our patient with the fear of recurrence of similar disease.

Conclusion

This is an unusual case of AGEP secondary to nifedipine, the only calcium channel blocker used in the patient. It’s even rare to see a patient with frank AGEP with AKI and ischemic cerebellar and left frontal stroke. The report is limited in terms that definite patch testing for particular drugs were not carried out with the fear of reproduction of symptoms. It was also not entirely clear if the AKI occurred due to the drug reaction itself or vancomycin, but our hypothesis is that it was probably because of a drug reaction as the medication was stopped abruptly after three dosages.

REFERENCES


Please note: Due to limited space, we are unable to list all references. You may contact South Dakota Medicine at 605.336.1965 for a complete listing.

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Permanent Central Diabetes Insipidus as a Complication of S. pneumoniae Meningitis in the Pediatric Population

By Hannah G. Statz, MS III; and Benson S. Hsu, MD, MBA, FAAP

Abstract
Diabetes insipidus is a rare but recognized complication of meningitis. The occurrence of diabetes insipidus has been previously attributed to Streptococcus pneumoniae (S. pneumoniae) in a handful of patients and only once within the pediatric subpopulation. We present the clinical course of a previously healthy 2-year, 8-month-old male child ultimately diagnosed with central diabetes insipidus (CDI) secondary to S. pneumoniae meningitis. Permanent CDI following S. pneumoniae meningitis is unique to our case and has not been previously described. Following the case presentation, we describe the etiology, pathophysiology, diagnosis, and treatment of CDI. The mechanism proposed for this clinical outcome is cerebral herniation for a sufficient duration and subsequent ischemia leading to the development of permanent CDI. Providers should be aware of CDI resulting from S. pneumoniae meningitis as prompt diagnosis and management may decrease the risk of permanent hypothalamo-pituitary axis damage.

Introduction
Diabetes insipidus (DI) results from the decreased production or absent response to arginine vasopressin hormone (also known as antidiuretic hormone, or ADH). ADH is synthesized by the magnocellular neurons in the hypothalamus, stored and secreted by the posterior pituitary, and involved in the regulation of water balance through receptors located on the kidneys.

ADH is released in two primary circumstances. The first is in response to an increase in the body’s plasma osmolality. The second is in response to hypovolemia, such as during hemorrhage or shock. Small variations in plasma osmolality and/or relatively large variations in volume status trigger ADH release. When properly working, ADH increases free water resorption in the kidneys. Absence of ADH decreases free water resorption resulting in polyuria and dilute urine. Compensatory polydipsia occurs as a result of thirst centers in the brain attempting to increase total body water.1

DI can be categorized as one of two types: central or nephrogenic. Central diabetes insipidus (CDI) occurs when ADH production or release is decreased or absent. Nephrogenic diabetes insipidus (NDI) occurs when the kidneys do not respond to ADH. The common results of both CDI and NDI are polyuria and polydipsia. Whether or not the problem occurs at the level of the kidneys differentiates the pathophysiology and treatment of these two entities. In CDI the receptors for ADH, which are located in the collecting tubules of the kidneys, are functioning. Therefore treatment with exogenous vasopressin will correct CDI.1 In comparison, patients with NDI do not respond to vasopressin, whether endogenous or exogenous. NDI may be hereditary; this is due to dysfunctional receptors normally responsible for water resorption. Other causes of NDI include drug induced such as might occur with lithium toxicity, or electrolyte disturbances including hypercalcemia or hypokalemia.1

Causes of CDI are referenced in the discussion.

This case report will focus on central diabetes insipidus (CDI). We discuss the various etiologies of CDI while noting that suspected bacterial causes have only been
described in sporadic case reports. Specifically, our report is only the second case in the current literature of CDI occurring due to *S. pneumoniae* meningitis within the pediatric population. Understanding the pathophysiologic etiology and recognizing the possibility of CDI secondary to meningitis is vital for the provider as prompt diagnosis and appropriate treatment are essential to decrease the risk of hypothalamo-pituitary axis damage.

**Case Presentation**

The patient is an otherwise healthy, fully immunized, 2-year, 8-month-old male who presented to a local primary care clinic with symptoms of fever, emesis, and general discomfort despite use of over the counter ibuprofen and acetaminophen. He was diagnosed with an unspecified viral illness but in the subsequent 24 hours continued to have persistence of symptoms with new onset decrease in activity and verbalization, yet was moving all extremities and continued to have appropriate response to environment. He was again seen in the local primary care clinic with recommendations of continued symptomatic treatment. The following day he was noted to have spells of being “unresponsive and shaking” per parents’ report. Although these spells were self-resolving, his parents felt that he was getting worse and becoming less responsive as he was no longer able to focus on his environment. With a third visit to his primary physician, he was admitted for concerns of seizures and possible meningitis, three days after initial presentation.

His exam at time of hospitalization revealed a febrile and lethargic male. His neurologic exam demonstrated a minimally responsive patient (Glasgow Coma scale not calculated) with reactive pupils bilaterally. Following admission, blood cultures were obtained and he was started on ceftriaxone for concerns of meningitis. Parents reported progressive neurologic decline following admission resulting in a complete lack of response to environment by evening, just hours after admission. On routine nursing rounds, he was found to have an unresponsive left pupil at 2 a.m., the morning after admission.

A regional pediatric intensive care center was contacted and following recommendations, the patient was emergently intubated, started on hyperosmolar therapy of mannitol and hyperventilated for concern of increasing intracranial pressure leading to herniation. His antibiotic coverage was expanded as he was noted to have a gram positive blood culture, later identified to be *S. pneumoniae*. Air medical transport to the pediatric intensive care unit (PICU) was uneventful.

Upon arrival to the PICU, he was hemodynamically stabilized through aggressive volume resuscitation and use of vasopressors. He was noted to have minimal neurologic response to stimulation without sedation. Electroencephalogram (EEG) noted diffuse slowing
bilateral. Left pupil had persistence of dilation and unresponsiveness.

Although his initial serum sodium was 144 mEq/L, within less than 24 hours following admission to the PICU, his sodium increased to 174 mEq/L with brisk urine output. Serum osmolality was found to be 351 mOsm/k with urine osmolality at 77 mOsm/k. Due to concern of diabetes insipidus, he was immediately started on vasopressin with strict fluid control for a goal of gradual decrease in serum sodium over the following 48 hours (Figure 1). Unfortunately, despite achieving a gradual decrease in sodium over 48 hours, he had another acute herniation event resulting in a dilated and unresponsive right pupil. Reversal of right-sided pupillary changes was accomplished through immediate hyperventilation, aggressive sedation, and hyperosmolar therapy.

On admission to the PICU, magnetic resonance imaging (MRI) noted leptomeningeal enhancement consistent with meningitis. Additionally, there was diffuse swelling in the cerebral hemisphere along with several foci of restricted diffusion in the cerebral hemispheres bilaterally (including the splenium of the corpus callosum and tectal plate of the midbrain) suggestive of ischemic changes (Figure 2). Emergent head computed tomography (CT) scan performed at time of right pupillary dilation and unresponsiveness revealed worsening brain swelling, consistent with increased intracranial hypertension, and multiple evolving areas of ischemia. This worsening of intracranial swelling was confirmed on a follow-up MRI showing downward transtentorial and cerebellar tonsils herniation (Figure 3).

The patient required a long course of supportive therapy within the PICU to allow the acute swelling from the infectious insult and consequent inflammatory response to subside. Aggressive treatment for intracranial pressure during the time of acute swelling included keeping the patient sedated and intubated, starting anti-epileptic therapy and utilizing hyperosmolar therapy. By time of transfer from the PICU, the patient was weaned off all positive pressure ventilation and oxygen support. However, he continued to demonstrate a need for vasopressin for free water homeostasis and was transitioned to desmopressin acetate (DDAVP) tablets orally. Evaluation of pituitary function revealed normal thyroid studies with expected adrenal suppression secondary to steroids given for airway swelling at time of extubation. By discharge, growth hormone function was yet unclear. Given significant neurologic and motor delays including being blind, deaf, and unable to ambulate, he was transferred to a long-term inpatient rehabilitation unit. Over time, he has recovered the majority of his motor skills with improvement in hearing through the use of a cochlear implant. However,
to this day, he continues to need DDAVP.

Discussion

The most common etiology of central diabetes insipidus (CDI) is idiopathic and accounts for 30 to 50 percent of cases. It has been proposed, however, that undiagnosed autoimmune processes might be responsible for a percentage of the idiopathic cases. Other infiltrative diseases such as Langerhans cell histiocytosis or intracranial tumors such as a craniopharyngioma are thought to be the second most common causes of CDI, accounting for 38 percent of cases in one study. Less than 10 percent of cases of CDI were found to result from neurosurgery, trauma, or familial disease.

Our patient reflects an infectious etiology of CDI with only rare case reports previously documented, predominately in children. One retrospective study from 1986 found an 11 percent incidence rate for post-central nervous system (CNS) infection induced CDI in children. This relatively high incidence rate suggests that infectious etiologies of CDI might be more common; however, we were unable to identify further studies reflecting similar results.

Infectious causes of meningitis leading to CDI include various viruses and bacteria. Viral infection such as with Herpes simplex type 2, Coxsackie virus, or Cytomegalovirus can cause CDI and is documented in a limited number of case reports. The proposed mechanism for CDI is the “viral destruction of vasopressin producing neurons.” While CDI following bacterial meningitis has rarely been described in the literature, the majority of bacterial causes resulted as a late complication from tuberculous meningitis (TBM). To a lesser degree, non-TB infectious meningitis from GBS, H. influenzae, E. coli, Cryptococcus, Neurobrucellosis, and S. pneumoniae has been found to induce CDI.

The focus of this paper is S. pneumoniae meningitis as an etiology of permanent CDI, and its rarity within the pediatric population is portrayed in a recent retrospective study from 2013. Levy et al. studied 79 children admitted for CNS-related infection between 2007 and 2010, three of whom had S. pneumoniae meningitis. Interestingly, not a single child with meningitis developed CDI. It is also important to note that while there are many cases referencing infectious causes of pituitary hormone deficiencies (such as adrenocorticotropic hormone, luteinizing hormone, thyroid stimulating hormone, or follicle-stimulating hormone) this rarely extends to solitary or simultaneous deficiency of ADH. For example, in one study of 19 patients with CNS infection, six patients developed either isolated corticotropic or borderline gonadotropic deficiency but none developed CDI. Our case represents the second documented pediatric case of CDI from S. pneumoniae meningitis. To our knowledge S. pneumoniae as the causative species of CDI is similarly rare in the adult population, having only been reported three times.

CDI due to bacterial infectious etiologies results from damage to the hypothalamo-pituitary area of the brain and/or the tracks that run between these two regions. For example, the mechanism for TBM induced CDI has been reported as a result of tuberculomas with associated dense exudate and sellar or suprasellar calcification, infection predisposed to the area of the basal cisterns, or progressive damage or scarring of the hypothalamo-pituitary region after TB infection of the CNS. Damage from non-TB bacterial infections has been proposed to occur by infiltration of bacteria into the pituitary stalk, intracranial hypertension, exudate causing basal arachnoiditis and inflammatory irritation, hypothalamic infarction, or ischemic damage or lesions located in this region of the brain. In addition, it has been noted that S. pneumoniae causes a vasculitis with inflammation resulting in transient CDI.

The course of CDI can be both short or long term. Commonly, CDI is transient and resolves spontaneously. However, CDI may also develop via the “triphasic response,” often leading to permanence. The first phase occurs within 24 hours to about a week after hypothalmo-pituitary damage and is essentially acute CDI. In the second phase, or interphase, a reserve of ADH is released from the degenerating pituitary or damaged hypothalamus. This is thought to be independent of CNS control. Interphase is similar to the syndrome of inappropriate secretion of ADH (SIADH) in which the polyuria and polydypsia of CDI disappear. Rarely a third phase occurs in which CDI develops permanently. In absence of the third phase, the typical sequelae is a return to physiologic fluid regulation. In our case, CDI developed acutely and permanently without going through a triphasic “polyuria – antidiuresis – polyuria” response. To this day our patient is on desmopressin therapy for CDI. Interestingly, all three
cases of adult *S. pneumoniae* meningitis were transient. Whether CDI was permanent or transient in the earlier pediatric report was not specified.\(^5\)

We suspect permanent CDI may have developed in our patient due to a combination of factors. Permanence may be due to the specific location of injury, rate of tissue damage, or relative percentage of ADH producing tissue lost. CDI is rare. It is even less likely to develop if damage is isolated to the posterior pituitary. In cases with slower destruction of the posterior pituitary, ADH production persists from the hypothalamus, and a shift of ADH release to a supra-pituitary site, such as the median eminence, may occur.\(^1\) Additionally, ADH production in the magnocellular neurons of the hypothalamus is greater than what is required for water homeostasis.\(^2\) If as little as 15 percent of ADH producing tissue is left intact, water and serum sodium imbalance may only be transient.\(^3\)

Thus, we suspect that damage in our patient must have exceeded 85 percent. Additionally, the rather rapid course of *S. pneumoniae* meningitis may not have provided an adequate time interval for the axis of ADH release to "shift" to a more superior location.

These mechanisms play a key role in the pathophysiology of CDI. However, we believe that essential to the clinical outcome in our patient was cerebral herniation for sufficient duration and subsequent ischemia leading to the development of permanent CDI. Findings related to excessive intracranial pressure were confirmed on MRI and evidenced by his development of dilated and non-reactive pupils. Intracranial hypertension and consequent ischemic damage is a mechanism of disease reported in a previous case of *S. pneumoniae* meningitis induced CDI as well.\(^2\)

### Conclusion

In conclusion, we present a case of CDI that developed after *S. pneumoniae* meningitis in a 2-year, 8-month-old male child. The development of transient CDI following *S. pneumoniae* meningitis has been reported in three adult cases. Only one case of CDI has been reported in the pediatric population, and whether this was permanent or transient was not specified. Ours is the first documented case of permanent CDI in the pediatric population.

An essential mechanism proposed for the development of permanent CDI in our patient is intracranial hypertension resulting in cerebral herniation for a sufficient duration, and associated ischemic change. Associated signs of cerebral herniation such as non-reactive pupils should raise suspicion for potential sequelae of CDI in patients with *S. pneumoniae* meningitis. This is a unique case that should increase provider awareness of CDI resulting from *S. pneumoniae* meningitis, and prompt diagnosis and management may decrease the risk of permanent hypothalamo-pituitary axis damage.

### References


Please note: Due to limited space, we are unable to list all references. You may contact South Dakota Medicine at 605.336.1965 for a complete listing.
Hereditary Pyropoikilocytosis: A Rare But Not Uncommon Disease

By Vasantha L. Gali, MD; and Douglas W. Lynch, MD

Background
The literature describes hereditary pyropoikilocytosis (HPP) as a severe form of hereditary elliptocytosis (HE). It is a rare red cell membrane defect which causes hemolytic disease in infancy. This condition was first described in 1975 by Zarkowsky and coworkers. HPP is an autosomal recessive disorder seen mostly in African families, although it has also been described in Caucasian whites and Arabs. Per the Genetic and Rare Diseases (GARD) Information Center, the prevalence of HE in the U.S. is not greater than 2.5 to 5 per 10,000. In both HPP and HE, mutations in spectrin, the principal protein of the erythrocyte membrane skeleton impair conversion of spectrin heterodimers into tetramers. This accounts for the mechanical fragility of HPP erythrocytes and high number of red cell fragments seen in the peripheral smear. The purpose of this case report is to describe the red cell morphology and presentation of HPP and to highlight this condition as a diagnostic consideration when evaluating/treating neonates with anemia.

Case Description
A full term neonate was noticed to be pale shortly after birth. A complete blood count revealed a low hemoglobin (Hgb = 9.6 g/dL; Reference range: 14.5-22.5), which had further dropped (Hgb = 9.1 g/dL) after a few hours. However, the bilirubin was within the reference range for age. The neonate was clinically stable without edema or respiratory distress. Placental exam was normal, and no acute blood loss was reported during labor. Kleihauer-Betke and Coombs tests were negative. Peripheral smear examination revealed an anemia with marked anisopoikilocytosis. Hemoglobin electrophoresis was suggested as a hemoglobinopathy was a diagnostic consideration. The patient's parents were immigrants from northern Iraq. Two of the patient's older sisters were healthy. However, an older brother was diagnosed with hemolytic anemia at birth requiring multiple blood transfusions and eventually a splenectomy. At 1 week of age, the red cells displayed marked anisopoikilocytosis, hypochromia, with mild to moderate fragmentation, target cells, teardrop cells, and rare ovalocytes (Figure 1, 2). The leukocytes and platelets were unremarkable. Based on the findings, the diagnosis of a hemolytic anemia, most consistent with RBC membrane protein defect, possibly hereditary pyropoikilocytosis was made and red cell transfusion was recommended at 2 weeks of age.

A workup at 6 months of age demonstrated a slightly elevated hemoglobin F with no abnormal variants on hemoglobin electrophoresis, normal RBC enzyme panel and an increased osmotic fragility.

Outcome
Our patient is now 3 years old and received transfusions approximately every three months since the time of diagnosis with a treatment plan for transfusions if the patient was symptomatic or had a hemoglobin less than 6 g/dL.

Discussion
HPP is a rare autosomal recessive red cell membrane disorder which acquired its name because the erythrocyte morphology resembles that seen in thermal injury. Red blood cells in HPP are susceptible to additional budding and fragmentation upon heating to 46 degrees Celsius, whereas normal RBCs are unaffected at temperatures below 50 degrees Celsius. This entity was originally described by Zarkowsky et al. and is predominantly seen in people of African descent, although it has been found in patients of other ethnic groups. Spectrin is the principal component of the red cell cytoskeleton membrane protein which is responsible for resistance and elastic deformability of the red cells. Mutations in the spectrin gene result in impaired conversion of spectrin heterodimers into tetramers. This accounts for the mechanical instability of HPP erythrocytes and if severe enough leads to RBC fragmentation, microcytosis, and hemolysis. Depending on the severity of the spectrin deficiency, the clinical phenotype is variable from asymptomatic to those with
severe hemolysis as early as the neonatal period. If the mother carries the gene, the newborn should be monitored for symptoms of hemolytic anemia, RBC fragmentation, poikilocytosis, elliptocytosis, and microspherocytosis as evident on peripheral blood smears. Complications of severe anemia, including splenomegaly, growth retardation, frontal bossing, and early gallbladder disease are common. Mild to moderate cases are managed with folate supplements and transfusions as necessary if the hemoglobin is less than 6 g/dL. The National Institutes of Health recommends splenectomy only for severe cases with complications. A clear discussion about the possible overwhelming infections with encapsulated organisms should be made with the patient’s family members. Diagnostic workup algorithms for neonatal anemia have been published in the past and include detailed history, physical, complete blood count with reticulocyte count, and direct antiglobulin test (DAT). Positive DAT points to immune mediated hemolytic anemias, where as negative DAT cases may require further workup with hemoglobin electrophoresis, red cell enzyme studies, and peripheral blood smear studies to arrive at an exact etiology. The diagnosis of HPP is based primarily on clinical suspicion and striking anisopoikilocytosis, microspherocytes and red cell fragmentation on the peripheral smear. A positive family history and a hematological evaluation of the family members is helpful in making the diagnosis in the appropriate clinical setting. Increased RBC thermal sensitivity and osmotic fragility will aid in the diagnosis. A direct antiglobulin test may be done to rule out autoimmune hemolytic anemia. Confirmatory molecular genetic testing is also offered for genetic counseling but is not usually required for diagnosis.

REFERENCES


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Douglas W. Lynch, MD, Department of Pathology, University of South Dakota Sanford School of Medicine.

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12 p.m. SDSMA PAC Lunch: Working Together to Combat Human Trafficking in South Dakota – Kevin Koliner, U.S. Attorney Sioux Falls
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Evaluating Symptom Distress in Cancer Patients

By Vanessa Wookey, MD; and Heidi McKea, MD

Abstract

Background: Distress in cancer patients negatively affects emotions and coping abilities and can reduce treatment adherence, quality of life, and survival rates. The prevalence of distress in cancer patients has been reported at 35.1 percent and 37.8 percent, but is frequently undiagnosed. Previous studies have produced conflicting results regarding reported symptoms. This study aims to help health care providers identify symptoms correlated with distress to improve recognition and treatment.

Methods: A cross-sectional study was performed via medical record review of 40 adult cancer patients at the Avera Cancer Institute. Responses were compared using Pearson’s chi-square test and the t-test.

Results: The average age of participants was 56.8 ± 12.0, and 65 percent had breast cancer. The mean overall score for distress was 4.6 ± 2.7 points on a 0-10 scale (95 percent CI 3.71 – 5.45). Twenty-four patients (60 percent) reported clinically significant distress. Females were more likely to report sadness. Specific symptoms with a statistically significant association with a higher overall distress included: fears, depression, sleep, worry, fatigue, nervousness, eating, and loss of interest in normal activities.

Conclusions: Although our sample size was small and homogeneous, the results demonstrated statistically significant associations between overall distress and the symptoms of fears, depression, sleep, worry, fatigue, nervousness, eating, and loss of interest in normal activities. These findings can increase awareness of symptoms associated with distress and allow clinicians to recommend specific interventions. Though many oncology clinics screen for distress, distress remains an important factor affecting quality of life and warrants further investigation.

Background

Distress is an “unpleasant experience” that can be caused by a variety of physical, social, or psychological/emotional factors and manifests itself in various ways in cancer patients, negatively affecting emotions, coping abilities, and cancer treatments, in addition to symptoms unique to the individual patient. Distress can reduce adherence to treatments, quality of life, and patient survival rates. Therefore, routine screening for distress is now recommended in oncology clinics so patients can receive the necessary interventions and referrals to reduce distress and improve their quality of life.

Although the prevalence of distress in cancer patients has been reported at 35.1 percent and 37.8 percent, clinically significant distress is frequently undiagnosed and goes unrecognized in more than half of cancer patients with distress. The results of previous studies on symptom distress have produced conflicting results regarding reported symptoms and age and gender differences. This study aims to improve the recognition and treatment of distress in cancer patients by identifying the prevalence of overall distress and the symptoms associated with more severe distress levels.

Methods

A cross-sectional study was performed via medical record chart review, collecting demographic data, cancer diagnosis, and patient responses to the distress management surveys collected by social workers at the Avera Cancer Institute.
for clinical purposes. The pre-determined study duration of one year was the primary determinant of the sample size. All patients 18 years old or older who completed surveys (n=40) between January 2012 and December 2012 met inclusion criteria and were included in the study. Patients meeting inclusion criteria were patients undergoing chemotherapy teaching who completed the distress management survey on their first visit to their oncologist after their first chemotherapy session, controlling for differences in stage of treatment. Patients ranked their overall distress using a 0-10 scale, 10 representing the most distress, and identified specific symptoms causing the distress, including practical, family, emotional, spiritual, and physical issues. SPSS was used to compare responses across demographic groups, cancer type, and cancer stage at diagnosis using Pearson’s chi-square test. A t-test comparing the mean distress in patients endorsing vs. not endorsing a symptom was performed for each of the specific symptoms. Two participants did not report an overall distress level so means were calculated using the reported overall distress from the other 38 participants. A p value of less than 0.05 was used as a threshold for statistical significance. This study was approved by the Avera Institutional Review Board.

Results

During the study period, 40 patients completed the distress management survey. Table 1 shows the characteristics of participants in the study. The average age of participants was 56.8 ± 12.0. The mean overall distress for the 38 participants reporting an overall distress was 4.6 ± 2.7 (95 percent CI 3.71 – 5.45), and the median was 5.0. Females reported a higher mean overall distress (4.8 ± 2.8, 95 percent CI {3.7, 5.8}) than males (3.9 ± 2.8, 95 percent CI {1.9, 5.9}), but the difference was not statistically significant. Twenty-four patients (60 percent) reported clinically significant distress, defined by the National Comprehensive Cancer Network as an overall distress level of four or greater. Females were more likely than males to report sadness (p = 0.022). There were no other statistically significant differences in overall distress or specific symptoms compared across gender, age groups, marital status, cancer stage, or cancer type.

Table 2 demonstrates the mean distress for patients endorsing a symptom compared to those not endorsing the symptom. None of the 40 study participants reported difficulties with fevers, bathing/dressing, or mouth sores. Specific symptoms with a statistically significant association with a higher mean overall distress included: fears (t = -4.32, p < 0.001), depression (t = -3.84, p < 0.001), sleep (t = -3.56, p = 0.001), worry (t = -3.14, p = 0.003), fatigue (t = -2.92, p = 0.006), nervousness (t = -2.53, p = 0.016), eating (t = -2.35, p = 0.025), and loss of interest in normal activities (t = -2.26, p = 0.003). No other symptoms had statistically significant correlations with overall distress.

Discussion

This study assessed the prevalence of overall distress in cancer patients and factors, such as demographic characteristics and symptoms, influencing distress. Previous studies have found the overall prevalence of distress in cancer patients to be 35.1 percent and 37.8 percent. Twenty-four patients reported clinically significant distress, an overall distress level of four or greater, corresponding to a prevalence of 60 percent in our sample. However, this number likely includes patients with an appropriate adaptive response after a cancer diagnosis and may not represent a clinically significant problem, providing the distress resolves.
Symptom distress has been found to be more prevalent in women; however, other studies report no gender differences. Studies have found distress to be more prevalent in younger age groups, and in patients older than 80 years. Other research reports no differences between age groups. Symptom distress also differs between cancer types; the prevalence has been reported as 43.4 percent in lung cancer patients, but 29.6 percent for gynecological cancers. The types of cancer producing the greatest distress in other studies were lung, pancreatic, brain, and Hodgkin’s lymphoma. Gynecological, prostate, and colon cancers produced the lowest distress. Although our data did not show any statistically significant differences with regards to gender, age, marital status, stage or cancer type, these results should be interpreted with caution because of a small and homogenous sample population. Though we did not find any significant differences between cancer stages, only five patients had metastatic disease, so our results may not accurately represent distress in patients with incurable disease. It has been reported that the prevalence of distress has been reported to be higher in those with a previous

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history of a psychiatric disorder, but, interestingly, is not significantly related to medical comorbidities; however, we did not analyze the effect of medical comorbidities or pre-existing psychiatric disorders on patient distress in our study.

Fears, depression, sleep, worry, fatigue, nervousness, eating, and loss of interest in normal activities all were found to have a statistically significant relationship to overall distress, which suggests that patients who report these symptoms are more likely to have a higher distress level. A variety of symptoms with a significant relationship to distress have been reported in other studies. One study reported nausea as the most common symptom, followed by worry, nervousness, fatigue, and anorexia. Another found the most common distressing symptom to be fatigue, followed by pain, stress, depression, and anxiety. Overall, the studies on symptom distress have been inconsistent, and more research should be done.

The study results are clinically relevant because distress is a predictor of mortality in cancer patients and has a significant impact on perceived dignity, although distress did not affect cancer progression. A meta-analysis reported a 26 percent increase in mortality in patients with depressive symptoms and a 39 percent increase if diagnosed with major depression, even after controlling for known risk factors associated with poorer prognoses.

Randomized control trials in breast cancer patients have shown encouraging results in the benefits of treating distress. Psychosocial interventions reduced the risk for breast cancer recurrence, increased the disease-free interval, and increased survival compared to controls. For patients with stage 4 breast cancer whose depressive symptoms improved during the first year, the effect on increased survival was shown to persist even after 14 years. Median survival of the patients whose symptoms improved was 53.6 months compared to 25.1 months in those whose scores increased in the first year, even after adjusting for prognostic indicators. Interestingly, the increase in survival only depended on symptom improvement, with or without intervention, reinforcing the importance of effective treatment for distress.

Recognizing distress is only the first step in the treatment of distress. An Italian study found that the implementation of a distress screening tool similar to the one used in this study, increased the recognition of distress and referral to appropriate psycho-oncological services by 79 percent compared to clinical judgement of oncologists. However, less than half of the patients referred actually utilized the service, suggesting there are significant barriers other than recognition that impact the treatment of distress. Referring patients based solely on clinical judgement not only decreases the number of cases of clinically significant distress referred, but also increased the number of patients without clinically significant distress that were referred.

All adult patients who completed surveys were included in our sample, and surveys were administered to each patient at the same point during treatment, controlling for differences in distress over the treatment course. Patients completed the survey at a single cancer center, controlling for potential differences in survey administration or patient education at different locations.

Several limitations of this study deserve mention. First, our sample size was small, mostly female, largely Caucasian, and almost two-thirds of patients in our sample had a diagnosis of breast cancer; therefore, the results cannot be generalized to other populations or all cancer types and should be interpreted with caution, as the potential for unidentified confounding factors is high. Patients were not screened for pre-existing clinical depression because positive screening at presentation could be due to normal responses to a cancer diagnosis, and lack of a clinical diagnosis would not rule out the presence of prior depression. However, pre-existing depression or other psychiatric diagnoses could represent another confounder. Socioeconomic status was not directly assessed, but all of the patients in the sample had some form of insurance, which is a potential confounding factor. The subjective nature of symptoms and reporting bias are other potential confounding factors. However, the biggest limitation involves the timing of survey administration. The survey was administered for clinical purposes before each patient’s second chemotherapy infusion and does not account for differences in symptom distress at different stages of treatment and may misrepresent the influence of symptoms that appear later in the treatment course. Our results could be confounded by the potential that initial distress could be a healthy adaptive response to a cancer diagnosis rather than a clinically significant problem. As such, distress should be measured at multiple points in time during therapy and further investigation with prospective studies is warranted. Randomized control trials would also be important to evaluate the effectiveness of potential interventions to reduce distress.
Acknowledgements
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REFERENCES


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Peripartum Cardiomyopathy: Literature Review

By Kalyan Potu, MD; Amol Raizada, MD; Ketineni Sujithasree, MBBS; Adam Stys, MD; and Maria Stys, MD

Abstract
Peripartum cardiomyopathy (PPCM) is a rare cause of heart failure that affects women during the last month of pregnancy to the first five months after delivery. The disease occurs in about one in 1,000 births in the U.S. Risk factors include advanced age, multiparity, twin pregnancy, African origin, preeclampsia or preexisting hypertension, and severe anemia. Heart failure in PPCM is treated similarly to heart failure from other causes, bearing in mind the pregnancy and lactation status. In this review article, we discuss the background, etiology, clinical evaluation, treatment, and natural history of peripartum cardiomyopathy, with a major emphasis on treatment.

Background
Peripartum cardiomyopathy (PPCM), also called pregnancy-associated cardiomyopathy, is a rare cause of heart failure that develops during the peripartum period, which encompasses the last month of pregnancy to the first five months after delivery in women with no known cardiovascular disease.¹ The worldwide incidence of PPCM varies from as high as one in 300 to as low as one in 40,000, emphasizing the involvement of genetic and cultural factors.¹² As per a recent nationwide, population-based study in the U.S., the overall peripartum cardiomyopathy incidence rate was one in 968 live births for the years 2004 to 2011.¹ Seven much higher incidences have been reported in places like Africa and Haiti.² In Africa, incidences range from one per 100 to one per 1,000.³ In Haiti, a five-year prospective study identified the incidences to be around one per 300 live births.³

Etiology
The etiology and pathogenesis of PPCM are still unclear. However, several hypotheses have been postulated, including viral myocarditis, prolactin toxicity, autoimmune mechanisms, malnutrition, and hormonal changes associated with pregnancy against a susceptible genetic makeup.⁶ Animal studies have shown evidence of an increased incidence of myocarditis in pregnant mice due to Coxsackie and echoviruses.⁷ In a published series of 17 patients with postpartum cardiomyopathy, three patients (17 percent) had a positive family history of peripartum cardiomyopathy.⁸ A 16-kDa derivative of prolactin has been shown to exhibit proinflammatory and cardiotoxic properties.⁹ This may explain the efficacy of D2 receptor agonists like bromocriptine (that reduce the secretion of prolactin from the anterior pituitary) in the treatment of PPCM.¹⁰

Risk factors for PPCM, on the other hand, are better defined and include:¹¹

1. Advanced age;
2. Multiparity;
3. Twin pregnancy;
4. African or Hispanic origin;
5. Preeclampsia or preexisting hypertension; and
6. Severe anemia.

Clinical Evaluation
Symptoms and signs of PPCM are typical for heart failure. Clinical features of PPCM include dyspnea (shortness of breath), orthopnea (difficulty breathing when lying flat), pitting edema, jugular venous distention, excessive weight gain, cough, palpitations, and chest pain.

The National Institutes of Health suggests four diagnostic criteria to define PPCM:¹²

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1. The development of heart failure in the last month of pregnancy or within five months after delivery;
2. No other identifiable cause of heart failure;
3. No recognizable heart disease prior to the last month of pregnancy; and
4. Left ventricular systolic dysfunction demonstrated by echocardiography (LV ejection fraction less than 45 percent and/or fractional shortening less than 30 percent on echocardiogram.

Treatment
PPCM patients have considerable morbidity and increased mortality risk, with PPCM necessitating inpatient admission. Heart failure in PPCM can develop very rapidly. It should be treated similarly to other patients with heart failure according to guidelines on acute and chronic heart failure.14

Medical Management of Heart Failure in PPCM
The medical management of heart failure is extrapolated from clinical trials that have looked at non-pregnant patients with reduced ejection fraction (EF). There are no randomized, controlled trials evaluating drug therapy in patients with PPCM. Drug therapy, however, needs to take into consideration the risk to the fetus in utero or during lactation.

During pregnancy, angiotensin-converting enzyme inhibitors (ACEIs), angiotensin II receptor blockers (ARBs), renin inhibitors, and aldosterone antagonists are contraindicated because of fetal teratogenicity.15-17 Hydralazine and nitrates can be used instead of ACEIs/ARBs. Certain ACEIs (e.g., benazepril, captopril, enalapril) have been tested in breast-feeding women and deemed safe for the babies.18 When ACEIs need to be prescribed during breast-feeding, benazepril, captopril, or enalapril are recommended.18

Beta-blocker treatment is indicated for all heart failure patients, as tolerated.19 Carvedilol, bisoprolol, and metoprolol succinate are recommended beta-blockers, as these have been evaluated in patients with heart failure with reduced EF.20

Diuretics should be used only if pulmonary congestion is present, due to the risk of decreased blood flow to the placenta.1 For women with PPCM who have delivered and are not breastfeeding, heart failure management is as per standard therapy.

Cardiac Resynchronization Therapy and Implantable Cardioverter Defibrillator
Cardiac resynchronization therapy (CRT) with an implantable cardioverter defibrillator (ICD) is indicated in patients with moderate to severe heart failure (NYHA class III or IV), despite optimal medical management, left ventricular ejection fraction (LVEF) less than or equal to 35 percent, QRS duration greater than or equal to 120 ms, and in sinus rhythm.21 CRT is also indicated in patients with mild heart failure (NYHA class II), despite optimal medical management, LVEF less than or equal to 35 percent, QRS duration greater than or equal to 150 ms, and in sinus rhythm.18 CRT reduces all-cause mortality, improves symptoms, improves exercise capacity, and reduces re-hospitalization rates for heart failure.22-24

Anticoagulation Therapy in PPCM
Anticoagulation therapy after delivery – but once bleeding has stopped – should be considered in patients with very low EF because ventricular thrombi and cerebral embolism occur frequently in PPCM patients.1 Anticoagulation is recommended in patients with evidence of intracardiac thrombus or systemic embolism, as well as in patients with atrial fibrillation.14,25 Subcutaneous low molecular weight heparin (LMWH) is recommended during pregnancy.25 After delivery, the patient can be transitioned to vitamin K antagonists if there is a continued need for anticoagulation.

Delivery in PPCM
Urgent delivery irrespective of gestation period with Cesarean section should be considered in women with advanced heart failure and hemodynamic instability despite treatment.26 Vaginal delivery is preferable if the patient is hemodynamically stable and there is no indication for Cesarean section.

Bromocriptine in PPCM
Bromocriptine, a dopamine agonist that blocks prolactin, is being experimented with as a novel, disease-specific therapy for PPCM. In a randomized clinical trial of 20 patients with peripartum cardiomyopathy, patients who received bromocriptine in addition to standard heart failure therapy regained a significant amount of left ventricular function at six months compared with the control group treated with standard heart failure therapy alone.27

Natural History
Reported mortality rates with PPCM vary widely between zero and 19 percent in the U.S., while heart transplantation rates have ranged from 6 to 11 percent.28 Left
ventricular function deterioration is reported in up to 50 percent of cases despite optimal medical management.29

Recurrence in Subsequent Pregnancies
The major concern about postpartum cardiomyopathy is the potential risk of recurrence with subsequent pregnancies, even if left ventricular function returns to normal. A subsequent pregnancy carries a risk of recurrence of 30 to 50 percent.30 An analysis of more than 100 women with peripartum cardiomyopathy in the U.S. demonstrated PPCM recurrence with their second or third pregnancy in more than half the cases.31 In light of the aforementioned data, even if the EF is normalized, the patients should be strongly counseled regarding the substantial risk of recurrence with subsequent pregnancies. If the EF does not normalize, subsequent pregnancy should be discouraged.

Concluding Summary
PPCM patients have considerable morbidity and mortality risk, and patients must be admitted to the hospital once this diagnosis is first established (a cardiology consultation should be sought as soon as a diagnosis is made). Heart failure in PPCM should be treated similarly to other patients with heart failure, bearing in mind the pregnancy and lactation status. Diuretic therapy is useful in the early decompressed phase especially with pulmonary edema, and beta-blockers are safe to use for long-term therapy in both pregnancy and lactation. Angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, renin inhibitors, and aldosterone antagonists are contraindicated during pregnancy, but certain ACEIs (benazepril, captopril, enalapril) are safe for the babies being breast-fed. A combination of hydralazine and nitrates can be used instead of ACEIs/ARBs in the last trimester. Subsequent pregnancy carries a significant risk of recurrence, even if the EF recovers. All patients with a history of PPCM should be counseled regarding this.

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So You Want To Be An Author: A Primer On Writing For Publication In The Medical Literature Part II: Manuscript Submission

By Victor C. Huber, PhD; and H. Bruce Vogt, MD

Abstract
Completing a draft of a manuscript that demonstrates the impact of your research within the current literature is the first step toward publication. The next step involves a review process that will allow your peers to provide feedback on the written document, with the goal of improving the presentation of your work. To complete this process, an author will have to be willing to accept constructive criticism of his or her work, as presented, and to modify the manuscript based on the feedback received. This peer-review process will ultimately shape the final draft of your manuscript, and here we provide some points to consider as you navigate the submission and review process.

Introduction
Publishing the findings of a research project requires a well-written story that describes how the data collected tests a hypothesis and, subsequently, advances the field of study. The first part of the process involves assembling the data into a cogent narrative that will improve the potential for publication. The second part includes the submission and review process. This article has been written to highlight the key steps in this process, and to provide some tips for completing this process, in a manner that will benefit both new and established authors.

Target Journal – Things to Consider
The first step in the submission process is to select an appropriate journal for publishing your article. When considering journals for possible manuscript submission, authors should strive to submit their work to one which is peer-reviewed. This process can either be “double-blinded,” or the journal may only maintain the anonymity of the reviewers. A peer-reviewed journal will customarily use outside sources of expertise in the field to determine whether the authors have presented an idea with the potential to contribute to the literature, and will provide feedback that will improve the experimental design and/or presentation of the data. Feedback from peers is important to the intellectual integrity of the scientific study and to advancing the field.

Peer-reviewed journals can be of a broad interest that appeal to the general scientific community and the public, or the interest may be limited to researchers and clinicians within specialized research areas. One attempt at defining the importance of a journal to the scientific community is the impact factor. The impact factor reports the frequency with which a journal’s recent articles (within the previous two years) have been cited in the current year’s publications (i.e., a ratio), but it does not necessarily reflect the importance of data published within either the journal or submitted manuscript. The impact factor can be higher due to editorial policies/practices of the journal. As examples, the journal may publish a lot of review articles (common sources cited); may invite senior scientists to submit manuscripts (a greater likelihood that their papers will be cited); or may decline articles that are less likely to be cited (e.g., case reports). When selecting a journal for potential publication, the authors should strongly consider the true impact that they believe their findings have within the scientific community, and submit to an appropriate journal. Proper attention to the level of interest from the scientific community in a particular topic, as well as to the journal’s readership as defined by the editors, will reduce the number of submissions.
rejected based on these factors and help authors improve their path to publication.

**Manuscript Submission**

**The Review Process**

The majority of journals have adopted an online, electronic submission process, which requires the author to assemble and upload manuscript documents and related materials such as tables and figures. In addition, authors must submit a cover letter to the editor. This letter should summarize one to two key points of the research, with specific emphasis on how these findings are relevant to the readers of that particular journal. This letter should contain statements related to Institutional Review Board, Institutional Animal Care and Use Committee, and Institutional Biosafety Committee approval, as relevant to the study, and this letter should describe any potential conflicts of interest that the authors may have. Finally, this letter should indicate that all authors listed have read and approved the final manuscript, and that the manuscript contains original work that has not been previously published and is not under consideration by another journal. The journal that ultimately accepts the manuscript has the ability to control the copyright of the previously published and is not under consideration by another journal. The journal that ultimately accepts the manuscript has the ability to control the copyright of the published article, and submitting the same article to two journals at the same time violates this process.

Once the manuscript has been submitted, an editor will be assigned, and reviewers will be identified. It is reasonable to expect that the review process will take four to six weeks, unless the article is rejected without review. The decision to reject without review is usually communicated in a shorter timeframe. Two examples of reasons for rejection without review include failure to follow the guidelines within the “Information for Authors” or a decision by the editor that the article does not fit within the scope of the journal. When such decisions are made by the editor, feedback is generally limited. When an editor makes the decision to subject the manuscript to full review, the article will commonly be forwarded to a minimum of two expert reviewers. From this peer review, one of three decisions is generally communicated. The editor may indicate that the manuscript has been rejected; has been accepted (typically pending attention to minor details); or may offer a willingness to subject the paper to review again pending a major revision. When a manuscript has been rejected or a major revision is requested, feedback may be quite extensive. Authors should use the feedback received to revise the manuscript for re-submission to the initial journal or to a different journal, possibly one with a more appropriate focus.

**Responding to Reviewer Comments**

Most manuscripts require at least minor revisions, and the feedback may include comments relative to major concerns (e.g., experimental design or interpretation of data), a need for clarification, or grammar and punctuation issues. Regardless of the feedback from the reviewers, there are two things to remember when responding to these comments. The first thing is that the authors are responsible for any mis-interpretation of the manuscript. Secondly, all points from the reviewers need to be addressed during the re-submission phase. This is typically provided within an accompanying cover letter that provides individual responses to each comment raised, including how it was addressed and/or reasons for not addressing the comment (e.g., limits of study, experimental models, etc.). It is critical that the authors respond to comments from the reviewers using polite, respectful language that reflects appreciation for the suggestions to improve the manuscript. If a point raised by a reviewer cannot be addressed, authors can state this limitation in the cover letter detailing the response to the reviewers. To improve the likelihood of publication, very few items should remain unaddressed. In addition to this detailed description of how comments from reviewers were addressed, authors should use this cover letter to thank the editor for the reviews provided, and indicate that they have responded to all comments and suggestions. At this stage, the editor and the reviewers have indicated interest in the manuscript and the project presented, and careful attention to the comments in the revised manuscript will likely yield an article that is accepted for publication. If unclear about a recommendation, authors should contact the editor asking for clarification.

**Manuscript Acceptance**

Once a manuscript has been accepted for publication, authors will receive a letter indicating the same and that prior to publication proofs will be generated and forwarded for author approval. At this stage further modifications of the text, tables, and figures are limited. In fact, in most situations the only revisions that can be made at this stage are ones that correct grammatical errors, including those inadvertently created during the editorial stage. Authors will also receive a “transfer of copyright” document to sign. In the interim between acceptance and actual publication of the manuscript, authors can cite the article in their curriculum vitae as “In Press.”
Summary
Drafting a manuscript that presents scientific data that test a novel hypothesis is the first step toward publishing a scientific research paper. The next step involves a peer review process that relies on feedback from selected experts in the field to help evaluate the impact of a study, and to improve its presentation. Throughout the process, an author can expect to have revisions requested, even if the article is well-written at the time of submission. Throughout this article, we have described the expectations of the review process, as well as tips to improve overall publication success.

REFERENCES

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H. Bruce Vogt, MD, Professor Emeritus and Former Chair, Department of Family Medicine, University of South Dakota Sanford School of Medicine.
While guidelines for the development of an institutional antimicrobial stewardship program have been available since 2007, the recent emphasis placed upon antimicrobial stewardship by government agencies may push hospitals, clinics, and other medical facilities to get more serious about implementing such programs. In 2014, the Centers for Disease Control and Prevention (CDC) released a checklist of “core elements of hospital antibiotic stewardship programs.” These core elements include: leadership commitment, including formal statements of support and resources allocated for the effort; accountability and drug expertise, comprising of physician and pharmacist leadership, preferably with infectious disease/antimicrobial stewardship training; action, including implementation of at least one recommended antimicrobial stewardship activity targeting optimal antimicrobial use, such as facility-specific treatment guidelines and prospective audit/feedback of antibiotic prescriptions; tracking and reporting the process and outcomes; and education, addressing antimicrobial prescribing data and current resistance patterns. Later in 2014, President Obama issued Executive Order 13676, directing the Department of Health and Human Services (HHS) to “review existing regulations and propose new regulations or other actions, as appropriate, that require hospitals and other inpatient healthcare delivery facilities to implement robust antimicrobial stewardship programs that adhere to best practices, such as those identified by the CDC.” As result, The Joint Commission has proposed a standard for antimicrobial stewardship and many expect the Centers for Medicare and Medicaid Services (CMS) will require hospitals and long term care facilities to implement evidence-based antimicrobial stewardship programs by the end of 2017 as a condition of participation.

Large hospitals and health systems are likely to secure the resources they need to implement evidence-based antimicrobial stewardship programs (ASPs) given these expected mandates. One might expect small to medium-sized facilities to encounter more difficulty, however, due to limited resources. Common questions for these institutions may include: Is there leadership commitment and financial support for an antimicrobial stewardship program? Does the facility have a physician willing to lead the program and a pharmacist with antimicrobial expertise? Can technology and other resources be obtained to implement antimicrobial stewardship activity, track/report progress and provide the education necessary to facilitate and sustain improvements?

Collaborative partnerships may facilitate the development of effective antimicrobial stewardship programs through pooling of resources. For example, small to medium sized hospitals may wish to partner with larger hospitals or health systems to gain regular access to infectious disease physicians and pharmacists with infectious disease expertise. Combine these resources with technology such as electronic order sets, web-based conferencing, and decision-support/data tracking tools, and you have the framework for an antimicrobial stewardship team with a wide reach. Avera Health implemented this type of antimicrobial stewardship program (ASP) in Fall 2015. Avera’s system-wide ASP was developed and implemented using the existing infectious disease physicians/infectious disease pharmacists based in Sioux Falls and pharmacy staff from each of the six Avera regional hospitals. As a team, the Avera Health ASP developed electronic order sets for common infectious diseases and standardized intravenous to oral conversion policies and renal adjustment guidance for antimicrobial use throughout the system. The ASP’s most recent innovation is the establishment of a daily web conference call used to review individual patient cases through screen-sharing technology in order to provide antimicrobial stewardship recommendations for patients receiving care at Avera hospitals participating in the ASP. Pharmacists present the patient cases to an infectious disease physician and
pharmacist during this call, a recommendation is formulated, and the recommendation is communicated to the patient’s provider by the pharmacist located at the patient’s facility. Initial results from the Avera Health ASP have been promising; suggesting pooling of resources may be a viable approach to system stewardship without the need to add personnel.

Are you ready for antimicrobial stewardship? Reviewing the CDC checklist of “core elements” is a good place to start answering that question. Review the checklist. Ask yourself what ASP resources currently exist at your site? What additional resources might you need? Consider seeking out advice or even partnerships with existing ASP programs to address your needs. Each of the three largest health systems in South Dakota has an ASP. Seek their advice and collaboration. Develop a plan and move forward to combat antimicrobial resistance.

REFERENCES


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SDSMA Member Open Forum

Friday, June 3, 2016

The SDSMA wants your input on important issues impacting the practice of medicine.

An SDSMA Member Open Forum will be held on Friday, June 3, 2016 as part of the SDSMA 2016 Annual Leadership Conference. The Open Forum will provide physician members an opportunity to bring issues to the attention of the SDSMA. In-turn, you will be given the opportunity to have your issue discussed among SDSMA membership and to hear from others across the state.

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Please download the form (Microsoft Word doc) and return it to the SDSMA office to bring forward an issue or concern with which SDSMA action may be needed. Each submitter will be asked to present their topic to SDSMA membership during the Open Forum.

The deadline for submissions is May 11, 2016. Submissions received after the deadline will not be considered at the 2016 Annual Leadership Conference.
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 Hopefully April showers (and high winds) have subsided in your area by now, revealing the beginnings of “May flowers” in your life and/or garden.

I may sound like a broken record (or depending on your genre: cassette, 8-track tape, or CD), because my message this month recalls a familiar theme from one of my first articles in this publication from 2008 and mentioned briefly last month—in-network referrals. DAKOTACARE prides itself on having the most comprehensive physician network in South Dakota and in the region. Nearly every physician specialist in the state is a participating DAKOTACARE provider, leaving few if any gaps in specialties. In fact, we estimate 95 percent of all health care needs can be met by the physicians and hospitals in South Dakota. So, when your patients require a referral, please refer to a DAKOTACARE member. DAKOTACARE is a home-grown South Dakota company made stronger through your support.

Good for Patients
Keeping patient referrals in South Dakota is good for our patients. Health care costs can be challenging enough without the added expense of travel costs and lost time at work for family members. Not to mention other service deficits we see as physicians, like duplicated tests/services and care coordination gaps, which result in more costs without any improvement in quality of care.

Good for Physicians
Despite the fact that our physicians and other clinical professionals often work within competing health systems, keeping patients within our state is good for physician collegiality and cooperation, even if it means referring to a competing entity. Keeping referrals in state when possible benefits not only the patient, but the very profession of medicine in South Dakota, as well as the communities we serve by keeping our dollars local.

Good for DAKOTACARE
Finally, in-network referrals are beneficial to DAKOTACARE. It helps us provide better oversight of medical costs, ultimately resulting in lower premiums and improved affordability of health insurance. Unlike many large national health plans, we have long prided ourselves in being a compassionate guardian of the health care dollar, constantly striving for efficiencies and improved value through improved quality of care, not just cost-cutting measures. Using in-network (i.e., in-state) physicians helps to achieve this goal.

In your individual Provider Agreement from DAKOTACARE, there are two important excerpts that I believe require special mention as a reminder of our mutual commitment:

• “DAKOTACARE and the Physician mutually desire to provide quality health care services in a cost-effective manner that is consistent with accepted medical practice.”

• “Except when medically contraindicated, as determined by the Physician, and approved by DAKOTACARE, the Physician agrees to refer DAKOTACARE Members for Covered Services only to Participating Providers contracted with DAKOTACARE.”

Along with Dr. Jim Engelbrecht and Dr. Mike Pekas, as medical directors for DAKOTACARE our job is to assist physicians and their patients in obtaining the appropriate health care efficiently, while maintaining focus on the cost-effectiveness of that care. We ask for your assistance to achieve this lofty goal and thank you for your professionalism in working through individual cases.

Our founding premise from 30 years ago remains the same today: DAKOTACARE is an advocate for patients and physicians. We hope that when contemplating a referral, you will respect our company and seek care for your patient(s) within our network whenever possible. When it is not possible, please understand that our scrutiny and questions are in the interest of everyone—patients, physicians, hospitals, and communities. Thank you.
Join us Friday, June 3

Membership Mixer
6:00 pm

Awards Banquet & Scholarship Recognition
7:00 pm

The Membership Mixer and Awards Banquet are sponsored by the following Diamond Sponsors:

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Transforming Clinical Practice Initiative

By Stephan D. Schroeder, MD, Medical Director, South Dakota Foundation for Medical Care

The Transforming Clinical Practice Initiative, or TCPI, is designed to help clinicians achieve large-scale health transformation. This initiative is one part of a strategy advanced by the Affordable Care Act to strengthen the quality of patient care and spend healthcare dollars more wisely. It aligns with the criteria for innovative models set forth in the Affordable Care Act:

- Promoting broad payment and practice reform in primary care and specialty care,
- Promoting care coordination between providers of services and suppliers,
- Establishing community-based health teams to support chronic care management, and
- Promoting improved quality and reduced cost by developing a collaborative of institutions that support practice transformation.

This initiative is designed to support more than 140,000 clinician practices over the next four years in sharing, adapting, and further developing their comprehensive quality improvement strategies. It is one of the largest federal investments uniquely designed to support clinician practices through nationwide, collaborative, and peer-based learning networks that facilitate large-scale practice transformation.

The Practice Transformation Networks are peer-based learning networks designed to coach, mentor and assist clinicians in developing core competencies specific to practice transformation. This approach allows clinician practices to become actively engaged in the transformation and ensures collaboration among a broad community of practices that creates, promotes, and sustains learning and improvement across the health care system.

Great Plains Quality Innovation Network (QIN) is working in support of the COMPASS Practice Transformation Network, established by the Iowa Healthcare Collaborative, and the National Rural Accountable Care Consortium for their TCPI work in Kansas, Nebraska, North Dakota, and South Dakota. This work started in December 2015 and runs through July 2019. Our role is to assist with the assessment of practices, identifying areas of opportunity as well as high performers, and providing quality improvement guidance to help clinicians achieve large-scale health transformation.

Great Plains QIN will be assessing practices to identify where they fall in the transformation phases continuum. The five phases of transformation are: set aims; use data to drive care; achieve progress on aims; achieve benchmark status; and thrive as a business via pay-for-value approaches. The QIN/QIO will then provide education and collaboration along with the Practice Transformation Networks to guide the provider practices through all phases of transformation.

For more information, please contact Great Plains QIN/South Dakota Foundation for Medical Care at 605.336.3505 or email me at stephan.schroeder@area-a.hcqis.org.

REFERENCES

https://innovation.cms.gov/initiatives/Transforming-Clinical-Practices/
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The American Medical Association Economic Impact Study, completed in conjunction with the South Dakota State Medical Association, shows how much physicians add to the economic health of South Dakota.

Check the effect physicians have on the U.S. economy by viewing the national report from the AMA, as well as highlights from the South Dakota study, at ama-assn.org/go/eis.

Please activate your 2016 AMA membership through the South Dakota State Medical Association by calling (605) 336-1965.
Is equality between the sexes good for men and women and society?

Of course, men and women are different in very important ways! Yet some people exploit the differences to make subservient generalizations about women. I’ve heard it said that women are, or should be: shy, passive, want-to-please, adoring, doting, and happy as homemakers serving their children and husbands. Sigmund Freud the psychoanalyst said, "Nature has determined woman’s destiny through beauty, charm, and sweetness... in youth an adored darling and in mature years a loved wife." Freud even went so far as to suggest that women who wanted careers were neurotic and imbued with "penis envy."

As Betty Friedan observed in 1963, Freud’s ideas had become especially popular in the mid 1940s, suggesting women are only fulfilled as housewives and mothers, and unhappy having a career. She decries Freud’s error-in-judgement in her book The Feminine Mystique. Friedan noted that in the early 1900s there had been grand advances made for the rights of women, especially for higher education, for pursuit of career, and for the right to vote. Except for the right to vote, much of this went away after World War II, when men came home from war and took away jobs from women who had been employed to support the war.

The returning soldier longed for a comfortable home where he was the bread-winner and his wife the house-keeper who raised the children. Friedan said this caused great unhappiness in some households when women who wanted a career were strongly discouraged to do so. Injustice and inequality beget disharmony.

Since then, due to the effort of Friedan and many others, women have choices and are coming close to equality in career work. Currently new physician graduates are about 50-50 women to men. Studies show satisfying careers and/or choices for all the adult members of a family results in happier children. Outside-the-home careers are not for everyone but the operative word is “choice.” Equality begets family and societal harmony.

Take it one step further; experts state that empowering and educating women in developing countries, or where there is great poverty, allows for the most effective way to bring the region in the direction of prosperity. Empowered and educated young women (better yet mothers and fathers,) raise girls and boys to become more effective and respectful members of society. Equality between men and women does not subtract from the success of men; it increases success for everyone.

When women and men are equals, society becomes healthy.
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**Legal Brief Highlight: Limitations of Actions**

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

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

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

For more information, download the SDSMA legal brief *Limitations of Actions* or access the risk mitigation resources at www.sdsm.org. Through the SDSMA Center for Physician Resources, the SDSMA has developed more than 50 legal briefs that are available to members. In addition, the Center develops and delivers and programs for members in the area of practice management, leadership and health and wellness.

*Source: SDSMA staff*

**The Issue Is...**

**CMS Outlines Plans for the Remainder of 2016**

The Centers for Medicare & Medicaid Services (CMS) says that its 2016 agenda is a busy one and is focused on physician input and a collaborative future that provides better care for patients. The agency laid out focus areas that it will apply to its work with the physician community throughout the rest of the year:

**Listening to physicians.** CMS must “get a better and more direct feel for what is happening on the front lines of care delivery,” CMS Acting Administrator Andy Slavitt said. “It is clear from listening to physicians there is fatigue,” he said, “with change, with measurement, with new requirements that come from the outside that aren’t simple to implement.”

**Simplifying.** Reducing burdens and giving physicians back more time to spend with patients is an area in which CMS says it plans to improve.

**Supporting change in care delivery.** For example, last year, Medicare began paying for advanced care directive conversations.

**Technology.** CMS says it will be sharing details and inviting comment as it rolls out proposed regulations this spring implementing the Medicare Access and CHIP Reauthorization Act. The proposed regulations will include changes to the Meaningful Use program.

*Source: AMA staff*

*“The Issue Is” is the SDSMA’s monthly update on key policy issues of importance to physicians.*
Registration Deadline Approaching for the 2016 SDSMA Annual Leadership Conference

The 2016 SDSMA Annual Leadership Conference – with a new schedule and new events – heads back to the Hilton Garden Inn, Downtown Sioux Falls on Friday, June 3. The May 20 deadline to register is quickly approaching.

With presentations, discussions, networking opportunities and social events, the Annual Leadership Conference is a great time to share ideas and learn from fellow members.

Check out the new schedule and new events! Do you have a policy issue you’d like to bring to the SDSMA’s attention? Schedule a time to address the Council by contacting the SDSMA office at 605.336.1965 or visit www.sdsm.org. Members have until May 11 to submit issues for consideration.

The Annual Leadership Conference is a benefit of your membership. For the latest details about exciting events taking place during the 2016 SDSMA Annual Leadership Conference, visit www.sdsm.org. Buy your tickets for the banquet and SDSMA PAC lunch online by May 20.

Those who need a hotel room may call the Hilton Garden Inn Downtown Sioux Falls at 605.444.4700 and ask for the South Dakota State Medical Association block.

Source: SDSMA staff

New Schedule & Events! Register at sdsm.org

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
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<tbody>
<tr>
<td>8:30 a.m.</td>
<td>Membership Meeting with presentations from AMA President Dr. Steven Stack and SSOM Dean Dr. Mary Nettleman</td>
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<tr>
<td>9:30 a.m.</td>
<td>Medical Marijuana Panel Discussion</td>
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<tr>
<td>11 a.m.</td>
<td>Open Forum</td>
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<tr>
<td>12 p.m.</td>
<td>SDSMA PAC Lunch: Working Together to Combat Human Trafficking in South Dakota – Kevin Koliner, U.S. Attorney Sioux Falls</td>
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<tr>
<td>1:30 p.m.</td>
<td>Council Meeting</td>
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<tr>
<td>6 p.m.</td>
<td>Membership Mixer</td>
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<tr>
<td>7 p.m.</td>
<td>Awards Banquet &amp; Scholarship Recognition</td>
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Human Trafficking is Topic of SDSMA PAC Lunch

During the SDSMA Annual Leadership Conference, the SDSMA PAC will feature a speaker on the topic of human trafficking.

The SDSMA PAC lunch presentation, “Working Together to Combat Human Trafficking in South Dakota,” will be held from 12-1:30 pm Friday, June 3 at the Hilton Garden Inn, Downtown Sioux Falls. The speaker will be Kevin Koliner, U.S. Attorney – Sioux Falls.

Lunch tickets are $40 and are available at www.sdsm.org. Please purchase your tickets by May 20.

It’s not too late to register for all of the activities taking place during the Annual Leadership Conference – find more information at www.sdsm.org.

Source: SDSMA staff

Membership Mixer is Networking Opportunity

Members are invited to the SDSMA’s Membership Mixer from 6-7 pm Friday, June 3 at the Hilton Garden Inn in Downtown Sioux Falls during the 2016 SDSMA Annual Leadership Conference.

Enjoy a unique and fun opportunity to meet SDSMA leaders and network with your peers. Meet other physicians in the state to expand your professional network and share ideas. The event is sure to be an all-around great time! Spouses are welcome to attend. Drinks and hors d’oeuvres will be served.

The mixer is free of charge. Tickets are required for the Awards Banquet & Scholarship Recognition following the mixer. To buy banquet tickets, please visit www.sdsm.org or call 605.336.1965.

Source: SDSMA staff

Don’t Miss the Awards Banquet & Scholarship Recognition

Enjoy a fun-filled evening of celebration Friday, June 3 at the 2016 SDSMA Annual Leadership Conference at the Hilton Garden Inn, Downtown Sioux Falls.

Start the evening with the Membership Mixer at 6 p.m., and continue the celebration at the Awards Banquet and Scholarship Recognition, where H. Thomas Hermann, Jr., MD, will be sworn in as SDSMA president, awards will be bestowed upon fellow colleagues, outgoing and incoming officers will be celebrated and welcomed, and medical student scholarship recipients will be recognized.

The mixer is free of charge; banquet tickets are $65 and must be purchased in advance. To buy tickets and register for other annual meeting activities, visit sdsm.org. Please purchase tickets and register by May 20.

Source: SDSMA staff

“Anatomy of a Medical Malpractice Lawsuit” Practice Support Presentation – 7 pm CT June 14

The SDSMA Center for Physician Resources brings you its Practice Support Series on Risk Mitigation at 7 p.m. CT Tuesday, June 14 with the presentation, “Anatomy of a Medical Malpractice Lawsuit.” To register for this free webinar, find a link on the homepage calendar at www.sdsma.org. The remaining programs in this series include the following:

• 7 p.m. CT June 14 – Anatomy of a Medical Malpractice Lawsuit
• 7 p.m. CT Sept. 13 – Physician Resiliency: Healing the Healer

This program will provide key advice on the litigation process and how to get through a malpractice claim both personally and financially.

The USDSOM designates this activity for a maximum of 1 AMA PRA Category 1 Credit(s). Physicians should claim only the credit commensurate with the extent of their participation in the activity.
## CME Events

Continuing Medical Education events which are being held throughout the United States (Category 1 CME credit available as listed)

<table>
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<tr>
<th>May 2016</th>
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<td>Internal Medicine Grand Rounds</td>
<td>USD Women in Medicine and Science</td>
<td>Pediatric Grand Rounds</td>
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<td>Surgery Grand Rounds: Evolution of Hepatic Surgery</td>
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### DO YOU HAVE A CME EVENT COMING UP? WOULD YOU LIKE TO HAVE IT LISTED HERE?

Contact: Elizabeth Reiss,  
South Dakota Medicine,  
2600 W. 49th Street, Suite 200,  
Sioux Falls, SD 57105  
Phone: 605.336.1965  
Fax: 605.274.3274  
Email: ereiss@sdsma.org

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