PANHANDLE HEALTH

A QUARTERLY PUBLICATION OF THE POTTER-RANDALL COUNTY MEDICAL SOCIETY

Summer 2020 | VOL 30 | NO. 3

Summertime Illnesses and Conditions

A Message To Texas Physicians

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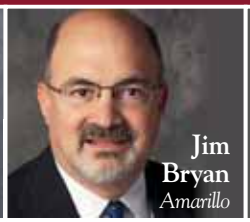
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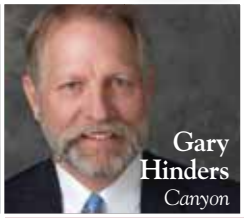
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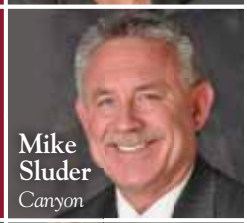
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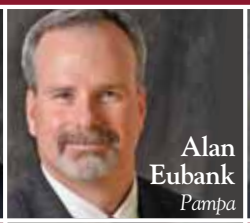
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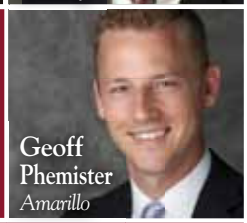
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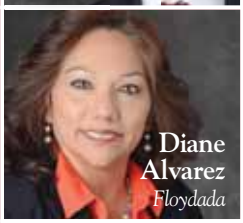
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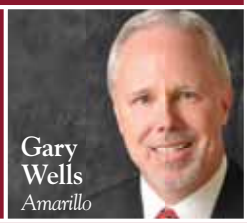
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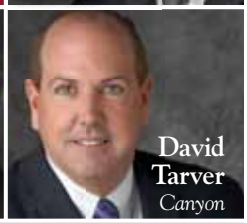
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On The Cover: ***Bluebonnets*** by Marsha Clements

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A Brief Note from the President

by Neil Veggeberg, MD

It has certainly been a strange year due to the COVID 19 virus. Nothing close to this has happened in my medical career. As best I've read in the media, the approach to this pandemic is self-isolation and identification of cases and contacts. I would never have expected so many people to voluntarily self-isolate. There have been exceptions, but it has overall been pretty well accepted. As health professionals we make so many recommendations such as stopping smoking, losing weight, eating well, and exercising that people do not heed. The social distancing has been embraced greater than I would have thought.

I've always enjoyed the quote "It is not the strongest of the species that survives, nor the most intelligent; it is the one most adaptable to change"

The next quote is, "Insanity is doing the same thing over and over again and expecting different results"

The first was attributed to Charles Darwin and the second to Albert Einstein. Neither one of them said them but we sort of wish they had.

If ever there was a time to be adaptable and to not do the same thing over and over again it is now. If ever there was a time for accurate data recording and scientific analysis it's now.

I have to give recognition to all the pulmonologists, ER docs, and hospitalists on the front line for their hard work and long hours. With elective procedures being put on hold, the workload has not been spread evenly. Drs. Scott Milton, MIchael Lamanteer, Brian Weis, Sheryl Williams, and Todd Bell, along with a cast of thousands who we can't even begin to name, have all risen to the occasion to take on leadership roles.

The world will survive this pandemic-it just will be a different place.

This quarterly issue is about summertime activities. I enjoy spending time down in Palo Duro Canyon. It's such a large expanse that social isolation is not a problem. In my time down in the Canyon there does not seem to be a limit to the things that can be problematic.

The problems stem from the weather, the terrain, and the wild animals.

From a weather perspective, I have experienced blizzards, hail storms, flash flooding, lightning strikes, wind gusts, and temperatures from below 0 degrees to above 116.

I rode one afternoon. A snowstorm was supposed to come in later that evening. It came in early and hit pretty hard. At the end of my ride the Ranger was there just checking to make sure I made it. Another ride was in excess of 110 degrees. At the end of my ride the Ranger asked me who I had seen on the trail, where they were, and how they looked. Dehydration is a significant problem since there are no water sources on the trails. The lighthouse trail which is the most popular is about 5 ½ miles long. Hiking at a reasonable pace it can be completed in about 3 hours. The last hour of the hike is exposed and heat can build up quickly. Many hikers do not anticipate the water needs and can get dehydrated quickly.

The Canyon consists mostly of loose material. It's good to stay on the trail. The sandstone rocks seem solid but can break without warning. They can give way while standing on ledges or can spontaneously come tumbling down the wall.. The contour of the Canyon is in a constant state of change. Heavy rains can wash out parts of the trail which can be treacherous if encountered around a blind corner while mountain biking or hiking.

There are wild animals to contest with. No direct sightings of mountain lions have been reported but rumors have it they're out there. Bobcats are plentiful but they are usually pretty shy and will try to avoid you. Coyotes will leave you alone usually but they're not shy in the Canyon. Rattlesnakes will do their best to avoid you but if startled could potentially jump out and bite you. I had a run-in with a porcupine. I was only worried that he might puncture a tire with a quill. Deer can startle easily and knock you off your bike. Tom turkeys will try to defend their ground. There's no end to it.

The outdoor environment is very pleasant to be in. Just watch for the dehydration, terrain changes, and run-ins with local animals.

Editor's Message:
Past Favorites and Current Pandemics

by Scott Milton, MD, FACP

In my previous article concerning COVID, I discussed the likely origins of COVID 19 as well as the current state of the pandemic from a more international perspective. At that time there were only about 15 cases identified in the US. My what a difference 10 weeks makes when in the middle of a pandemic! Amarillo has not only been affected by the pandemic but is now receiving national attention for the exploding number of cases in this area. Several factors have contributed to our rise in cases, as I will discuss later in this article.

Amarillo documented its first cases on March 18. Local community spread was clearly present at that point in time. Generally speaking, we have doubled our cases weekly and, at the time of this writing, have well over 3,200 cases documented in the community. The New York Times listed us as the fourth fastest growing city in the nation concerning COVID 19. With this, we have had 37 deaths. The health department believes that over 19,000 tests have been performed locally at the time of this writing. Social distancing measures including "Amarillo All In" were initiated within a few days of learning that COVID 19 was circulating in our community. Local government and the health department initiated orders for people to stay at home. Nonessential businesses were asked to close. Social distancing measures as well as masking while in public were strongly encouraged. These orders were initiated on March 30 and then renewed two weeks later. On April 27 we recorded 65 new cases in the community, our highest number to date. On the same day our governor ordered the state to be reopened on May 1. It is safe to say that the local government and local health department were very concerned with the timing of this order by the governor. It was also it very clear in the governor's order that any local order was overruled by the governor's actions.

The first cases of COVID 19 admitted to our local hospitals occurred around two weeks following a basketball tournament in Levelland Texas. Dozens of individuals became ill following this event. Within a short period of time, other hotspots were identified. Specifically, meat packing plants and individuals confined to the Texas Department of Corrections were soon becoming ill. Concerning the former, the Texas Panhandle has always been an ideal place to raise cattle. The great Southern bison herd of the 19th-century, which numbered in the millions, predated our current domestic herds of cattle. Because of our wide-open spaces, abundant grass and relatively mild climate, bovine animals have always outnumbered humans in this area. Currently thousands of cattle are processed every day through plants positioned in various parts of the Texas Panhandle. This industry is one of the most important industries in our area and employs thousands of individuals who work in these plants. This type of employment is grueling and requires large numbers of individuals to work shoulder to shoulder for hours on end in an enclosed environment. Most of these employees are immigrants, and many live in small houses or apartments that are not conducive to social distancing. Further, many individuals do not have their own transportation and must rely on busses to get to the jobsite. Many individuals also have difficulty speaking English, which further complicates efforts to reduce or mitigate transmission of COVID 19. In addition to meatpacking plants, several facilities in the Texas Department of Corrections have had multiple cases. Efforts to mitigate the spread of COVID-19 in this vulnerable population have been ongoing for weeks. Northwest Texas hospital continues to be impacted by this population.

As demonstrated by events described in the previous paragraph, it is clear that COVID-19 is not only highly contagious but is also spread more efficiently in areas where people are confined in an enclosed setting. COVID-19 is also spread more efficiently by the individual's behavior while in that confined space. For example, our first hospitalized cases occurred after attending a basketball game. In that setting hundreds of individuals are closely packed in a gymnasium. Most fans are boisterous and likely are hugging, yelling, and singing while cheering their team. Games usually last for more than an hour, which further enhances the likelihood of transmission. Another example is that of a choir in Washington state which was heavily afflicted by COVID-19 during a choir practice. It seems that the act of singing and/or yelling in close proximity to other individuals enhances spread. Meatpacking plants and prisons are places that confine large numbers of individuals densely in an enclosed space. The spread of COVID 19 has occurred rapidly in the setting of individuals working shoulder to shoulder for hours at a time.

Testing for COVID 19 has rapidly become more available, mainly through commercially available testing that has been brought to market. It is more than fair to say that the ability to test for this new infection initially was woefully inadequate. Apparently, the test initially developed by the CDC was faulty and hampered the production of tests available to the state and local governments. This test is a nasal swab PCR test and is currently still the only test available through our health department and state. The turnaround time for this is typically

| *continued on page 8*

24 to 48 hours at present. Further, testing is only provided to symptomatic individuals. More recently, an antibody test has become available to the public through commercially available kits. Initial tests produced confusing results many times. More recent, testing appears to be much improved and will likely prove useful in the future in determining when herd immunity may be achieved.

The treatment of the severe pneumonia that can be caused by COVID-19 is many times a difficult and lengthy process. The critical care teams, pulmonologists, ICU nurses and respiratory therapists at both hospitals have done an outstanding job in caring for these individuals. There is currently no FDA approved drug for the treatment of COVID-19. But there are several treatment protocols in use at our hospitals and across the country. One technique is "proning". Patients suffering from ARDS secondary to this novel virus are placed on their stomachs while intubated. It appears that proning allows for expansion and therefore more efficient use of lung tissue. Oxygen saturation can immediately improve when patients are placed in this position.

Hydroxychloroquine is an antimalarial and anti-inflammatory drug used for the treatment of malaria as well as some forms of inflammatory arthritis. There are some conflicting studies that have suggested benefit with the use of this drug in combination with azithromycin. More recent evidence suggests potential toxicity associated with these drugs, and it appears that most critical care specialists have shied away from use of this combination. Plasmapheresis is a technique that's also been utilized with some anecdotal success in our hospitals and across the country. Finally, the use of convalescent antibody or plasma that is collected from individuals who've recovered from COVID 19 and then administered to those critically ill has also been utilized. We are participating in a nationwide clinical trial in conjunction with the Mayo Clinic involving convalescent plasma.

The Panhandle area has hosted teams from the Center for Disease Control and the United States Department of Agriculture, specifically concentrating on the packing plants throughout the region. Screening efforts of thousands of employees working at these plants has been performed, as well as continued efforts to mitigate and stop transmission within the facilities. These resources will be used in conjunction with those from the state and local levels.

I was asked to provide an update just prior to print, and there are two other important events that I would like to mention. First, our community received remdesivir, an antiviral drug that has some evidence of providing benefit to individuals with severe complications of COVID 19. We administered this drug today to a patient and hope to see improvement soon. Second, preliminary testing of the Tyson packing plant suggests 20% of the workforce are infected, suggesting a high degree of asymptomatic transmission.

COVID 19 has expanded into an international crisis and will likely forever change life throughout the world. The Texas Panhandle is in the midst of a rapidly expanding epidemic as documented by our exploding numbers. The Texas Panhandle is somewhat isolated geographically, and it appears that COVID is affecting us later than most areas of the country and the state. The governor's statement that the numbers were slowing was not true in the Texas Panhandle at the time of his statement. I believe our numbers will continue to rise in the following weeks and, markedly so, when final test results become available from local packing plants. The current state of our hospitals and staff is good, and the likelihood of our resources becoming overwhelmed is relatively low, although still possible. Our local authorities remain in close contact with state officials, and we have been assured that resources are available if they are needed and could be distributed immediately. Our team at the health department has been working long hours trying to mitigate spread in our community. The health care professionals, and specifically, the internal medicine physicians and critical care specialists at both hospitals are working day and night caring for those severely ill from COVID 19. Our unified goal has always been to reduce illness and to save lives, and all of us will continue to focus on this goal as this pandemic unfolds.

Executive Director's Message

by Cindy Barnard, Executive Director

We usually associate diseases and ailments with the winter months, but there are a large number of summer-specific diseases. We all welcome summer—a time to be outside, picnicking and barbecuing, swimming, hiking, and simply enjoying the warm and sunny days. However, summer does bring a variety of seasonal diseases such as mosquito and tick-borne infections, sunburn, melanomas, heat stroke, firearm accidents, poison ivy, and many more. Our summer issue of Panhandle Health addresses some of these ailments indigenous to this season in the hope of educating you to some of summer-specific health concerns.

Of course, we are all more than aware of the Corona Virus, and we, at the Medical Society, have PPE supplies should you need them. Please call our office with your requests (806-355-6854).

A notice to our member physicians – we must fill vacancies on our Board of Directors and our Panhandle Health Editorial Board. Our Board of Directors meets every other month and the Editorial Board every month. Our meetings do not last over an hour and include dinner. If you have more questions about what these positions entail, or simply want to fill a vacancy on either Board, again, please call our office. We hope to hear from you soon. Thank you.

We extend our support to all who are on the front lines during the ongoing Covid-19 pandemic.

We wish to thank all Healthcare Workers, Law Enforcement, Firefighters and EMT's, for your tireless efforts in helping the people of the Texas Panhandle.

PANHANDLE HEALTH

A Publication of the Potter-Randall County Medical Society

Editorial Policy and Information for Authors

Purpose *Panhandle Health* strives to promote the health and welfare of the residents of Amarillo and the Texas Panhandle through the publication of practical informative papers on topics of general interest to most physicians while maintaining editorial integrity and newsworthiness.

Spectrum *The Journal* seeks a wide range of review articles and original observations addressing clinical and non-clinical, social and public health, aspects as they relate to the advancement of the state of health in the Texas Panhandle. Pertinent letters to the editor, news submissions, and obituaries listings are accepted pending editorial review. The Editorial Board accepts or rejects submissions based on merit, appropriateness, and space availability.

Submission process Material should be e-mailed to the editor at prcms@suddenlinkmail.com or mail a hard copy to Cindy Barnard, PRCMS, 1721 Hagy, Amarillo, TX 79106. A recent photograph of the author (optional) and a curriculum vitae or a biographical summary are also to be submitted.

Conflict of Interest Authors must disclose any conflict of interest that may exist in relation to their submissions.

Journal Articles Manuscripts should be double-spaced with ample margins. Text should be narrative with complete sentences and logical subheadings. The word count accepted is generally 1200 to 1500 words. Review articles and original contributions should be accompanied by an abstract of no more than 150 words.

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Illustrations Illustrations should be black and white only with complete-sentence legend.

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Letters Letters will be published at the discretion of the editor and editorial board. The length should be within 400 words. References should not exceed five. All letters are subject to editing and abridgment.

News News should be e-mailed prcms@suddenlinkmail.com or mailed to Cindy Barnard, PRCMS, 1721 Hagy, Amarillo, TX 79106.

Obituaries Listings of deceased members of PRCMS with highlights of their contributions are published when adequate information is available.

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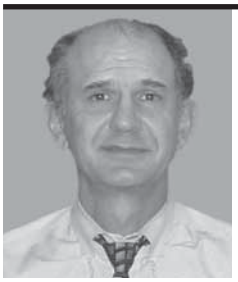

Heat Stroke and Other Heat-Related Illnesses

By Steve Urban, MD

Every summer our Panhandle emergency doctors and hospitalists manage patients with heat-related illnesses. Cases range from mild heat cramps all the way to life-threatening heat stroke in an ill-prepared hiker in Palo Duro Canyon or an elderly debilitated patient whose air conditioner gives out in the middle of a heat wave. The extremes, like our weather patterns, are dramatic. Untreated heat stroke often leads to death or permanent neurological damage, and yet, with prompt treatment, the outcome is usually good. This article will review the physiology, clinical manifestations, treatment, and prevention of heat-related illnesses.

Both resting metabolism and muscular contraction are exothermic reactions. Resting metabolism produces about 60 kcal of heat per hour; heat generation can increase 10 to 20 fold with strenuous exertion. The blood draining exercising muscles of experimental animals often exceeds 107^{0}F. To dissipate this heat energy, the body depends primarily on radiation, convection and evaporation. Temperature regulating neurons in the hypothalamus control blood flow to the skin (radiation, convection) and sweating (evaporation) to promote heat loss. If these mechanisms are overwhelmed—either by intense muscular activity or by impaired cooling—serious heat illness can supervene.

Milder forms of heat illness occur when fluid and electrolyte losses associated with sweating are under-replaced. Sweat is about ¼ normal saline; so both volume loss and free water loss can occur. Sports drinks can replace these losses, but inadequate fluid intake can lead to hypovolemia, muscle cramping or even syncope due to hypotension. Occasionally, excessive free water intake in the absence of sufficient volume replacement can lead to hyponatremia; although potentially serious (especially in marathon runners), this is not related directly to the heat stress and will not be further discussed here.

Incidence and predisposing factors

Mild forms of heat illness are very common and rarely lead to medical encounters; statistics about their occurrence are hard to come by. Classic heat stroke is associated with prolonged heat waves, especially in poor or crowded circumstances with limited access to cool surroundings. Exertional heat stroke is most commonly associated with 3 settings: (1) intense military training, especially of unacclimatized recruits (2) late summer and fall football practice ("two-a-days") and (3) recreational activities such as long distance running or hiking in hot and/or humid conditions. We occasionally see exertional heat stroke in day laborers (e.g. roofers, diggers), but they are usually acclimatized by daily activity in the heat. The U.S. Army reported 730 cases of severe heat illness between 2014 and 2018 (this despite the fact that the U.S. military pioneered the study of heat illnesses and strives to make drill sergeants aware of the data). Exertional heat stroke is the third leading cause of death in young athletes. In Saudi Arabia, when the Hajj falls in summer months (as it has lately), heat illness is a major public health concern.

Predisposing factors. An important feature of many cases of exertional heat stroke is lack of **acclimatization**. Heat acclimatization is a process by which the body increase the efficiency of heat loss. Remember that acclimatization requires exposure to a hot environment and is not the same thing as athletic conditioning. With acclimatization, cardiac reserve increases, sodium loss in sweat decreases (a process partly mediated by aldosterone) and total sweat available for evaporation increases. An acclimatized athlete can produce 1-2 liters of sweat per hour. Acclimatization takes at least two weeks to develop and is lost after another 2-4 weeks post cessation of heat exposure. The risk of exertional heat stroke in military recruits is greatest within this 2 week window, although some increased risk persists up to 2 months. Other factors that increase the risk of serious heat illness are obesity, diabetes mellitus, smoking, advanced age, impaired cardiovascular reserve, and use of certain drugs (amphetamine-like drugs, medications with anticholinergic properties, opioids and possibly NSAIDs). A previous history of severe heat illness (heat exhaustion or heat stroke) increases subsequent risk fourfold.

Environmental factors are, of course, important as well. Outside temperature is important, but so is humidity. Evaporative heat losses are minimal when the humidity is above 70%. The best way to assess heat risk is with a wet bulb globe thermometer, which takes humidity and sun exposure into account. Tables indicating unsafe WBGT readings are widely available (our athletic trainers at WTAMU use WBGT readings to assess safety of workout conditions). In addition, excess clothing and gear impair evaporative loss and should be avoided. For this reason, both the American College of Sports Medicine and the College Athletic Trainers Society provide specific recommendations for transition from light workouts to full pad and helmet football practices in the summer and early fall.

Clinical features:

Mild heat illnesses. Heat edema is a mild and self-limited swelling (often of the hands and feet) caused by vasodilation and a temporary increase in vascular permeability. Heat cramps are common, especially with inadequate electrolyte replacement, but usually last no more than 5 minutes after exercise. Prickly heat is a mild slightly pruritic erythema, often in occluded areas, that resolves in hours to days. Heat syncope is thought to relate to vasodilation and perhaps to limited cardiac reserve in the unacclimatized athlete. It often occurs just after the exercise is completed, and responds to elevation of the legs and, occasionally, to IV fluids. The most important issue in heat syncope is excluding a cardiac etiology (e.g. hypertrophic cardiomyopathy, which often causes post-exertional syncope).

Heat exhaustion is a more serious condition that can progress to heat stroke if not addressed. The main symptoms are

fatigue and exertional intolerance. GI symptoms such as vomiting and even diarrhea can occur, and the patient may be mildly hypotensive. An important finding is elevated body temperature, usually between 101°F (38.3°C) and 104.9° F (40.5°C). If exercise is discontinued and the patient divested of excess clothing and moved to a cool environment, hyperthermia and symptoms usually resolve within 30 minutes. The exercise session should be discontinued; some patients continue to complain of exercise-induced weakness and fatigue for days to weeks thereafter.

Classical heat stroke. Classical heat stroke occurs in elderly, often debilitated persons exposed to high ambient temperature and humidity for days to weeks, i.e. in a heat wave. These patients often have diabetes or chronic cardiovascular disease and are usually on medications that impair sweat loss (e.g. anticholinergics). CNS signs (confusion, agitation, obtundation, or coma) predominate, and core temperature will be above 40.5°C. This is the classic scenario where, despite the intense hyperthermia, sweating is often absent. As the hypothalamus shuts down, heat dissipation ceases and the temperature skyrockets. Prognosis is poor because the condition may go unrecognized for hours before cooling measures are instituted. If the patient survives, permanent brain damage, especially debilitating cerebellar ataxia, often follows. I was an intern at Parkland Hospital during the Dallas heat wave of 1978 when hundreds of poor people died. I cared for one elderly woman who had a core thermistor temperature of 114°F; she did not survive.

Exertional heat stroke is the more common form nowadays; it carries a significantly better prognosis because the inciting exertional event is usually recognized. These patients have hyperthermia above 40°C but remain moist. The hypothalamus is still functioning; so the patient continues to sweat. It's just that the amount of heat generated by exercising muscles overwhelms the body's ability to dissipate it. Again, recognizing CNS changes is critical. Sometimes the mental status changes are subtle—aggressiveness, irritability and mild confusion are usually the first signs (these can be hard to distinguish from just your usual football player). The skin is damp but clammy. Exertional heat stroke often causes rhabdomyolysis (CKs of 100,000 or more), elevated liver-related enzymes, DIC, and acute kidney failure, processes which may take days to weeks to resolve after euthermia has been achieved.

Management

I will confine my remarks to the most serious cases of heat illnesses, as the milder forms usually resolve without medical intervention. Occasionally patients with heat exhaustion will have hypovolemia with mild kidney injury or CK elevation and may rarely require overnight observation in the hospital. Full-blown heat stroke, however, is an acute medical emergency, with 20-50% mortality (if treatment is delayed). It must be managed aggressively. Temperature above 40.5°C in an adult should always suggest heat stroke. Your hypothalamus has enough sense not to raise your temperature to protein-denaturing levels in response to normal stimuli such as infection! Other entities to consider should include thyroid storm, neuroleptic malignant syndrome, anesthesia-induced malignant hyperthermia, CNS conditions that damage the hypothalamus, and certain drug intoxications (especially with cocaine or amphetamines). The clinical setting should distinguish these from heat stroke.

| *continued on page 12*

Initial measures are important: removing all unnecessary clothing and gear, transferring the patient to a cool shady environment, and obtaining an accurate core temperature reading (ideally by rectal thermometry).

Prompt core cooling (i.e. cooling of the blood going to the brain) is of paramount importance in suspected heat stroke. Cooling measures are often started in the field. Our EMTs are directed to start rapid core cooling in patients with suspected heat stroke—i.e. core temperature above 40.5^{o}C and any CNS changes. If cooling measures are available on site (see below), they will be instituted. If not, ice packs will be applied to axillae, posterior neck and groin, cold IV fluids will be administered and the patient taken to an ER post haste.

Probably the best place to have heat stroke is at an athletic training facility. Our athletic trainers at WTAMU are trained to recognize and to start treatment while awaiting the EMTs. Excess clothing will be stripped and, if core temperature is extremely high, ice water immersion started at the facility. If core cooling can be achieved in 30 minutes, mortality rates approach 0 and neurological sequelae are rare. In the military, they use ice sheets (frozen wet sheets) with good results. One problem locally is that accurate instruments to measure core temperature (i.e. rectal thermometers or probes) are rarely available or practical for use at the training facility.

There is an extensive literature on the best methods for rapid core cooling. Probably the most expeditious method is employed in Saudi Arabia. Emergency rooms in Mecca are equipped with large fans and misting devices that don't induce shivering or vasoconstriction and can decrease body temperature as rapidly as 0.3^{o}C/min (for comparison, the rate of cooling in an air-conditioned room is 0.05^{o}C/min). Most emergency facilities in the U.S. do not have such equipment.

The next best way to lower core temperature is with cold water or ice water immersion. This will lower core temperature approximately 0.2^{o}C/minute. Shivering is a problem and can be blocked with benzodiazepines. Patients with significant CNS changes may have seizures while in the ice bath; these patients are usually intubated prior to immersion. Once the core temperature drops below 101-102 F (this usually takes 15-30 minutes), the patent should be removed from the water bath and passive cooling continued. Ice water baths are actually more commonly found in athletic training facilities than in EDs. In all but the largest EDs, cooling will be accomplished by ice packs and cold IV fluids. Cooling blankets work too slowly; invasive cooling methods (such as those used for post-cardiac arrest hypothermia) take too long to deploy.

Ancillary measures include ventilation if respiratory depression or aspiration are risks, fluid hydration (average volume deficit=1.5 liters), prevention of shivering, and subsequent management of DIC, rhabdomyolysis, kidney injury, and electrolyte disturbances, if present.

Prevention:

The American Academy of Sports Medicine and the National Collegiate Athletic Association have published specific performance guidelines to minimize the risk of heat stroke. They offer specific recommendations about duration of workouts and use of pads and helmets. Atmospheric conditions for workouts should be monitored (with wet bulb globe thermometry, if possible). Regular breaks are important; fluids should be available at all times, not just with breaks. Preworkout hydration with at least 17 oz of water is recommended. Sports drinks can be used for hydration for workouts >1 hour (vitamin drinks and energy drinks are not recommended). The athlete's weight should not decrease more than 2% with each workout. Athletic trainers are taught to recognize subtle mental status changes, and the opinion of the trainer to excuse the athlete from further exercise should be unchallenged by the coaching staff.

According to Luke Kasper, director of sports medicine at WTAMU, one of the most important preventive measures is the pre-participation evaluation by the medical or training staff. Asking questions about conditioning, heat or exercise induced symptoms, screening for sickle cell trait, and any prior episodes of heat illness can go a long way toward the prevention of exertional heat stroke.

One important question is return to exercise in patients who have had prior heat illnesses. Minor manifestations such as heat edema, heat cramps, or heat syncope (assuming that cardiovascular conditions have been considered) do not preclude next-day activity. Patients with more severe heat illness should not exercise for at least 1 week until they have been cleared of all symptoms and lab abnormalities by a doctor. Activity then can be gradually increased over 2-4 weeks. Resumption of activities in the heat can be considered after this time if all manifestations have resolved, realizing that the risk of heat related illness will always be elevated in such a patient.

In summary (no pun intended), summer heat in the Panhandle can be both good and bad. On the one hand, it encourages people to run around without many clothes on, which in selected cases improves scenic beauty of our region. On the other hand, inattentiveness to hydration, acclimatization, and proper duration of exercise can put one at risk of heat-related illness. Prompt identification and treatment of environmental heat illnesses improves outcomes and helps prevent long-term restrictions and disability.

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Foodborne Illness: Diagnosis, Management, and Prevention

By Ganesh Maniam, MSIII and Nicole Lopez, MD, FAAFP

As summer approaches, outdoor social gatherings become a meaningful way to socialize and share food in the warm weather – but with these picnics and barbeques comes the risk of a foodborne illness! Foodborne illnesses affect approximately 48 million Americans annually. Over a hundred thousand patients require hospitalization each year due to foodborne disease, resulting in an estimated 3000 deaths annually in the United States (1). In general, foodborne disease is classified as either foodborne infection or foodborne intoxication (1). Eliciting a complete patient history including relevant dietary and risk factors is necessary for diagnosis and management of foodborne illness. A physical exam should make note of orthostatic changes, assessment of skin turgor, abnormalities in the abdominal exam, and hydration status. Stool cultures and stool PCR testing can provide a definitive diagnosis and aid in management and treatment recommendations, though stool cultures are positive in < 40% of cases (2).

For acute episodes of foodborne illness, identification of the causative pathogen is often not necessary unless the patient presents with severe inflammatory diarrhea or neurologic manifestations. However, identification of the causative agent may assist the patient in preventing future episodes of food poisoning. Many community related outbreaks are caused by *Salmonella* spp. or *Campylobacter* spp., and these agents should be suspected in patients with symptoms presenting 1 - 4 days after ingestion of contaminated food (3). Most states require reporting of outbreaks to local, county, state, or national agencies; education of the public through the news media is an important strategy to prevent further outbreaks in the community. Vomiting as the primary symptom of gastritis or gastroenteritis suggests a viral etiology or consequence of preformed toxins from *Staphylococcus aureus* or *Bacillus cereus* (4). Diarrhea, either inflammatory (bloody) or noninflammatory (watery), is another presentation of foodborne illness. Noninflammatory etiologies can be caused by almost any enteric pathogen, while inflammatory diarrhea is caused by more invasive species such as *Shigella* spp. or *Campylobacter* spp (4). Persistent diarrhea lasting for at least 2 weeks necessitates diagnostic work-up for parasitic causes such as *Giardiasis, Cryptosporidium*, and *Entamoeba histolytica* (4). Indeed, the timing of symptoms is often helpful in identifying the causative agent of foodborne illness **(see Table 1)** (3).

Table 1. *Likely Etiology of Foodborne Illness based on Timing of Symptom Onset*

2 - 4 hours leading to predominantly nausea & vomiting	Suspect *S. aureus* preformed enterotoxins or *B. cereus* preformed emetic toxin
6 – 12 hours leading to predominantly abdominal cramps and diarrhea	Suspect *Clostridium perfringens* preformed toxins
8 – 16 hours leading to predominantly abdominal cramping and noninflammatory diarrhea	Suspect *B. cereus* preformed enterotoxins
12 – 48 hours leading to vomiting and noninflammatory diarrhea	Suspect norovirus (or another calicivirus)
24 – 96 hours leading to fever, abdominal cramps, diarrhea, and vomiting	Suspect *Salmonella* spp., *Shigella* spp. or *E. coli*
3 – 4 days leading to bloody diarrhea	Likely enterohemorrhagic *E. coli* or *Campylobacter* spp.
3 – 5 days leading to fever, vomiting, and noninflammatory diarrhea	Suspect rotavirus, astrovirus, or enteric adenovirus
3 – 7 days leading to fever, diarrhea, and abdominal pain	Suspect *Yersinia enterocolitica*
10 – 13 days leading to fever, headache, myalgia, and rash	Suspect *Toxoplasma gondii*
1 – 3 weeks leading to abdominal pain, diarrhea, constipation, and systemic symptoms	Suspect *Entamoeba histolytica*
1 – 4 weeks leading to gastroenteritis, periorbital edema, muscular pain, fever, chills	Suspect *Trichinella spiralis*
1 – 4 weeks leading to malaise, fever, vomiting, rose spots, constipation, and bloody stools	Suspect *Salmonella typhi*

Less commonly, neurologic presentations of foodborne illnesses can be caused by a number of uncommon illnesses such as botulism and scombroid poisoning, or even Guillain-Barré syndrome secondary to the diarrheal illness of *Campylobacter* spp. infection (4). Given that these illnesses may result in respiratory depression or cranial nerve palsies (4), a more intensive management approach is necessary and may even require hospitalization. Systemic presentations with a longer latency period suggest parasitic infections, such as ingestion of the larvae of *T. spiralis* in undercooked pork, which can, weeks later, lead to burrowing of the adult nematode into human muscle tissue to cause muscular pain and periorbital edema (3). Likewise, abdominal pain and weight loss occurring 3 months after exposure may be due to a tapeworm infection with either *T. saginata* (beef) or *T. solium* (pork) (3). These patients also put others at risk without proper handwashing, as the *T. solium* tapeworm eggs can cause fecal contamination of food or water to cause a devastating infection of the brain known as neurocysticercosis (3).

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Prevention of foodborne illness is through patient education. Such disease is often caused by poor hand hygiene, cross contamination, inadequate refrigeration, or insufficient cooking. The United States Department of Agriculture (USDA) has released food safety guidelines that patients should be encouraged to follow (5). When shopping, nonperishables should be purchased before any refrigerated and frozen items. Customers should also ensure that all meats are packaged without leaks and all foods are purchased before expiration dates (5). Washing hands and surfaces is necessary both before and after cooking. Separating foods until serving will help prevent cross-contamination (5). Proper storage of foods includes ensuring that the refrigerator is below 40°F and the freezer is below 0°F (5). Thawing is most safely done through the refrigerator, but other methods such as cold water thawing or microwave thawing are acceptable as long as the food is cooked immediately (5). Cooking animal products can be done safely through checking the internal temperature with a food thermometer **(see Table 2)** (5). Any food left out at room temperature for more than 2 hours should be discarded, but otherwise can be saved as leftovers if refrigerated immediately and used within 3 days (5).

Overall, the recognition and treatment of foodborne illness first requires obtaining a detailed patient history and exclusion of severe causes of abdominal pain, vomiting, or diarrhea. For the management of most patients, only supportive care through hydration and bowel rest is typically needed. Inflammatory diarrhea or a protracted course of diarrhea will require further diagnostic work-up, while neurological symptoms leading to dyspnea (such as Guillain-Barré syndrome) usually require hospitalization. Patients should be educated on the necessity of handwashing, cleaning cooking surfaces, and preventing cross-contamination of foods; USDA guidelines regarding shopping, storage, and cooking are also available for patients desiring further information.

Table 2. *Minimum Internal Temperatures for Animal Products*

Beef, pork, veal, lamb	160°F for ground products 145°F for steaks, chops, and roasts (and allow to rest for at least 3 minutes)
Chicken, turkey	165°F
Eggs	160°F
Fish, shellfish	145°F
Ham	145°F for fresh / smoked ham (and allow to rest for at least 3 minutes)
Reheating cooked animal products	165°F for reheating fully cooked ham 140°F for reheating fully cooked ham packaged in USDA-inspected plants 165°F for all leftovers

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UPCOMING EVENTS 2020-2021

TMA Fall Conference

Sept. 11-12
Hyatt Lost Pines
Austin Area, TX

TMA Advocacy Retreat

Dec. 4-5
Omni Barton Creek
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AMA House of Delegates Interim Meeting

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Manchester Grand Hyatt
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Firearm Injuries and Their Prevention: A Public Health Issue

By Taru Bharadwaj, Ravi Bharadwaj MD

Public health concerns are usually associated with problems like heart disease, stroke, obesity, and other such internal ailments that require medical intervention over long periods of time to treat. However, external issues that affect health are just as crucial to address. In particular, firearm injuries continue to grow more prevalent in the United States. More than 39,773 people die from firearms (CDC), and over 67,000 people are injured every year (Fowler et al., 2016) with a death rate of 12.2/100,000 (CDC, 2017). Firearm injuries continue to grow in the United States, and death and injury by gun violence constitute a public health crisis (Bauchner et al., 2017).

Though the second amendment of US constitution gives a right to bear arms to its citizens, Texas gun laws mostly focus on who can carry, how they can carry, and where they can carry, instead of who can own which types of guns. According to 2017 data, Texas has more than 1.2 million individuals who are active holders of concealed gun permits. (David Tarrant, 2017). In 2015, Texas signed "Open Carry" legislation, which allows licensed handgun holders to carry handguns openly on their shoulders or hip.

Gunshot wounds are the second leading cause of injury deaths, just after motor vehicle crashes (Hanna et al., 2015). Homicide deaths by firearms are four times greater than homicide deaths from other causes in the United states, and Texas trends of firearm-injuries closely follow the overall national trends ("Texas Injury Data Brief," 2015). According to another journal, 13,205 working-aged adults died from homicide by firearm (Sabbath, Hawkins, & Baum, 2020). This does not even take into account firearm deaths due to suicide or accidents. In fact, many accidental firearm deaths involve children, making children in the United States more at risk of accidental firearm death than children in any other developed nation (Hemenway & Solnick, 2015).

Note that rates of fatal and non-fatal injuries are not equally distributed in the population. Age, gender, ethnicity, and state of residence can affect firearm injury. Males account for about 86% of all firearm deaths. Between 2010 and 2012, the ratio of male to female firearm suicide rate was found to be 7:1. Similarly, young adults between 25 and 34 years are at highest risk for fatal firearm injury. Suicide by firearm peaks in ages 64 years and older. African Americans have the highest rate of homicide firearm death, and Caucasians have the highest rate of suicidal firearm deaths. Violent crimes as well as gunfire injuries are higher in metropolitan area as compared to non-metropolitan area.

The medical response to firearm injury is based on the nature and severity of the injury. Unintentional injuries are more common in arm and legs whereas assaults more commonly involve trunk and legs **(Table 1)**.

A typical firearm injury consists of an entry wound surrounded by abrasion and burn, a permanent cavity created by the projectile and many times an exit wound (Shrestha, Kanchan & Krishan, 2020). Extensive tissue damage depends on the projectile/bullet energy (mass and velocity of projectile), resulting in a temporary cavity that results from torn tissue or organ shattering. Likewise, the severity of a gunshot injury depends on the amount of blood loss, tissue damage, mass and velocity of the bullet, and even the design of the gun. This information, as well as complex research on the interpretation of bullet trajectory using radiology and imaging, is very helpful to the medical professionals (Hanna, Shuaib, Han, Mehta, & Khosa, 2015). The most important point when considering the outcome of a firearm injury is the location of the entrance wound (Maiden, 2009), followed by the assessment of the most vulnerable locations and organs of the body. Familiarity with these can help with prompt medical action.

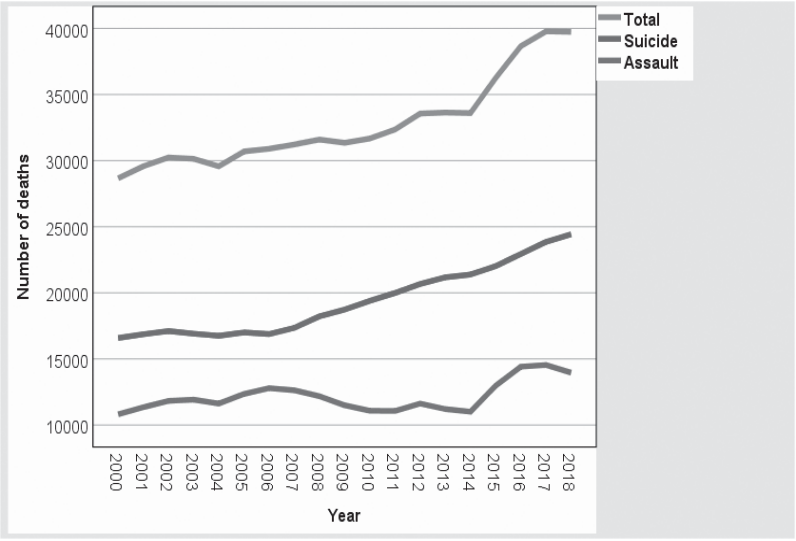

Figure 1: Number of deaths caused by firearm between 2010 and 2018 (data from NSC.org)

Tissue damage and blood loss are responsible for most early deaths from firearm injuries. A human body of 176 pounds has about 4.8 liters of blood, but if a bullet severs a large artery like the aorta, then it takes only 4.6 seconds to lose 20% of blood volume, enough to render the person unconscious (Maiden, 2009). Therefore, early recognition of physiological responses, especially blood loss, remains critical in saving lives, with immediate focus on reducing the amount of blood loss (Maiden, 2009). Additionally, factors like the amount of tissue damage, the resulting shock wave, age, and even the design of the bullet can all vastly affect the type of medical treatment required (Maiden, 2009).

Body region	Unintentional	Assault
Head and neck	10%	11%
Upper trunk	6%	20%
Arm and hand	34%	14%
Lower trunk	7%	19%
Leg and foot	43%	35%

Table 1: Primary body part injured in non-fatal in gunfire injury: unintentional vs. assault (Fowler, Dahlberg, Haileyesus, & Annest, 2015)

Typical complications of gunshot injuries such as airway obstruction, pneumothorax, and hemorrhage should be managed within 10 minutes when resources are available. Patients with internal bleeding should be given the priority for transportation, and hemorrhage from extremities should be speedily controlled with tourniquets or other available measures. Inpatient management is usually directed by damage-control surgery with highest priority to life threatening conditions (Franke et al., 2017). Medical professionals, especially trauma surgeons, are well-trained in these issues, but teaching this information to the general public could also save lives. If firearm injuries were recognized as a public health concern and communities could easily access this type of information (the same way they have easy access to information on fighting obesity or stroke), then perhaps there would be a higher chance of survival.

To help reduce firearm injuries, the American College of Physicians has called for action (Butkus, Doherty, & Bornstein, 2018). They reaffirmed their previous statements and added emphasis on appropriate regulation of the purchase of legal firearms, firearm owner's best practices to reduce accidental injuries, and legislation to control semiautomatic firearms and to temporarily remove firearms from individuals who are determined to be at imminent risk of harming themselves or others (Butkus et al., 2018).

Along with information on immediate action, like any other public health initiative, prevention mechanisms should also be widely known. According to the Harvard Injury Control Research Center, based on data and analysis over nineteen years in 50 states, more guns yield more unintentional firearm deaths. In fact, the mortality rate was seven times higher in the four states with the most guns as compared to the four with the fewest (Miller, Azrael, Hemenway, & Vriniotis, 2005). However, another large factor is simply unsafe gun storage, leading to easy access for children, who are highly vulnerable to unintentional firearm deaths (Matthew et al. 2001). Furthermore, a scientifically driven understanding of risk and protective factors is another important mechanism of prevention (Fowler et al., 2015). Based on the available epidemiological data, states and communities can take action to prevent firearm violence in certain groups. This is especially true for the state of Texas, which has a high rate of firearm suicide, firearm homicide, and unintentional firearm death (Fowler et al., 2015). If preventative action were taken, then families could make individual decisions to protect themselves and their children; this includes a decision to not purchase guns or at least to have secure and safe storage of guns. Firearm injury and death is also closely linked with mental health, alcohol and drugs. According to one study, suicide rates are higher in households with a handgun (Sorenson & Vittes, 2008). Again, safe storage or limited access to guns are some ways to counteract this. However, the most important way to prevent firearm suicide is to acknowledge mental health issues and to provide help to those who need it. Support is crucial in these cases to help someone who is considering using a firearm to hurt themselves.

Firearms have played a large role in history, changing outcomes of wars and even affecting geography. However, as a nation and as a state, we should have an open discussion about the role of firearms in our communities. If the taboo around the discussion of firearms as a public health issue were removed, more people would have access to preventive measures and would know some immediate medical action. Communities and families could make more well-informed decisions when it comes to firearms, the same way they might change their diet to battle heart disease. More public knowledge about the medical implications and preventive measures around firearms might lead to a significant reduction in avoidable firearm injury and death.

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What You Should Know About Skin Cancer: Signs, Diagnosis, Treatment, and Prevention

By Summer Clark, MD

Introduction

In the United States, the number of people diagnosed with skin cancer each year is greater than that for all other cancers combined (1,2). Approximately 4.3 million cases of basal cell carcinoma (BCC), the most common form of skin cancer, are diagnosed in the U.S. annually (3). More than 1 million cases of cutaneous squamous cell carcinoma (SCC), the second most common form of skin cancer, are diagnosed each year (3), with annual deaths due to SCC of approximately 15,000 people, more than double that of melanoma (primarily due to the increased incidence of SCC compared to melanoma). Melanoma is less common than BCC and SCC, but more deadly, with an estimated 6,850 deaths from melanoma in 2020 (1). With the incidence of skin cancer on the rise, it is important to be familiar with the signs of skin cancer as well as its diagnosis, treatment, and prevention.

Risk Factors and Signs of Skin Cancer

Risk factors for skin cancer include skin type, history of indoor tanning, history of sunburn and unprotected exposure to ultraviolet rays, genetics/family history, presence of multiple atypical nevi, and history of solid organ transplant. For risk factors that are modifiable, it is important to mitigate their influence as much as possible. Non-melanoma skin cancers occur most commonly on sun-exposed sites. In women, the most common location for melanoma is the legs; for men, the most common location is the back.

The morphology of cutaneous malignancies can vary widely, but there are some key features that can be helpful in identifying different types of cancers and guiding the decision to biopsy. BCC's are commonly pink and indurated, with a pearly or shiny quality. The most common location is the nose, and they frequently have a central erosion. SCC's are most commonly erythematous, indurated and crusted with hyperkeratosis. Size and induration of a lesion are helpful in differentiating SCC from actinic keratosis, a precursor lesion to SCC. Melanoma most often presents as a darkly pigmented macule, patch, papule, or nodule, often with variation of the pigment throughout the lesion and irregular borders. Not all melanomas are pigmented, however, and amelanotic melanoma occurs more commonly in very light skin types. Beware the growing pink papule.

Regarding patient counseling and symptoms, patients should monitor for any lesion that is growing, changing in shape or color, nonhealing, or bleeding. SCC's particularly may be painful. Any lesion of concern exhibiting such qualities should be evaluated. In recent years, the use of dermoscopy (viewing lesions through a magnifying loupe), has proven to be a helpful tool in determining which lesions require biopsy.

Diagnosis of Skin Cancer

Skin cancer is diagnosed through a skin biopsy. There are two commonly performed types of biopsies, shave biopsy and punch biopsy. When evaluating a lesion of concern for biopsy, the physician should choose the type of biopsy that is most likely to obtain the tissue needed to enable the pathologist to yield an accurate diagnosis. For most lesions of concern, a shave biopsy is the preferable method. For crusted lesions, it is important to sample the deeper, indurated portion of the lesion, not only the superficial crust. For lesions where melanoma is part of the differential diagnosis, a "saucerized" shave biopsy is a useful technique, where a deeper, scoop-style biopsy is performed with the goal of extending the biopsy to encompass the full depth of the lesion. Accurate assessment of depth,

| *continued on page 20*

known as Breslow depth for cases of melanoma, is the most important piece of information to obtain, as it is the most significant factor in determining treatment and prognosis. If the lesion of concern is relatively small, approximately 1.5 cm or less, it is helpful to attempt to biopsy the entirety of the lesion, both its surface and deep components, to decrease the risk of sampling error and provide optimal information on depth. For lesions that are highly clinically suspicious for melanoma, an excisional biopsy may be performed. Punch biopsies are more commonly used for rashes rather than neoplasms, though they may be utilized for small neoplasms concerning for melanoma that can be fully encompassed by a punch biopsy. They may also be used for diagnosis of deeper dermal or subcutaneous nodules, as in these cases a shave biopsy is less likely to provide an adequate specimen. Single or multiple punch biopsies of larger lesions concerning for melanoma are not generally recommended due to increased risk of sampling error. In such cases, a broad saucerized shave biopsy would be preferable. Patients with lesions concerning for SCC or melanoma should also undergo examination of the draining lymph node basin to assess for lymphadenopathy, potentially denoting metastasis.

Treatment of Skin Cancer

There are myriad treatment options utilized in managing skin cancer, including topical chemotherapy, locally destructive techniques such as electrodessication and curettage, traditional wide local excision, Mohs micrographic surgery, and systemic medications for locally advanced or metastatic cutaneous malignancies. For the three most common forms of skin cancer (BCC, SCC, and melanoma), the National Comprehensive Cancer Network (NCCN) provides useful guidelines to help clinicians manage these malignancies. The method of treatment chosen depends on the aggressiveness of the cancer, which is influenced by size, location, histologic subtype, and other patient factors such as history of solid organ transplant. For BCC's and SCC's occurring on high-risk locations such as the head, neck, hands, and feet, Mohs micrographic surgery offers the highest rate of cure. During Mohs surgery, a cancer is excised with a narrow margin, and, while the patient waits in the office, the specimen is processed en face rather than using the traditional breadloafing technique. If the margins are positive, further excision is performed at the location of residual tumor only, and this is repeated until the tumor is eradicated. En face processing allows for 100% evaluation of the margin, resulting in fewer false negative margin assessments and thus a decreased risk of recurrence. Additionally, Mohs surgery is tissue sparing, with the smallest amount of tissue resected in order to remove the cancer. In 2012, the Journal of the American Academy of Dermatology published appropriate use criteria to aid clinicians in deciding which tumors should be treated with Mohs surgery (4). The Mohs Surgery Appropriate Use Criteria App is available for smart phones and is a useful clinical tool in the management of skin cancer.

Prevention

Efforts to prevent damage to the skin from ultraviolet radiation are of primary importance in the prevention of skin cancer. The implementation of sun protective measures is most effective when begun in childhood; however, even in patients who have already developed skin cancer, protection from the sun decreases the risk of future skin cancer formation. Commonly recommended sun protection measures include use of sunscreen with SPF 30 or higher when exposed to the sun, use of a broad-brimmed hat and sun protective clothing, seeking shade whenever possible, and avoiding being outside between the peak hours of 10:00 am – 4:00 pm. Indoor tanning should be avoided entirely, and there is no safe amount of tanning – the idea of a "base tan" is a myth, as any increase in melanin production is a response to DNA damage. Adequate vitamin D levels can be achieved through oral supplementation. In addition to practicing sun protective measures, patients should also endeavor to become familiar with their skin and existing lesions, so they may be aware if changes occur. Monthly self-exams of the skin are often recommended for patients at high risk of developing skin cancer.

Conclusion

Skin cancer is an exceedingly common problem, particularly in our area of Texas where much of the population is engaged in agriculture, resulting in an abundance of time spent outside in the sun over the course of a lifetime. Prevention of sun exposure and the resulting DNA damage is of primary importance. When lesions of concern do arise, prompt diagnosis and treatment are necessary to yield the most favorable outcome.

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Summertime Injuries in Children

By Johnnie Faircloth, MD

Children in the United States die from preventable injuries more than from any other cause. The number of preventable unintentional injury and death cases in children rises during the summer months. According to Safe Kids Worldwide, each year around 60 percent of these cases occur from May through August. Drowning, bicycling, and falls are associated with a large number of these summer injuries. In this article I'd like to discuss these injuries and their prevention.

Water Safety

In the heat of the summer months, water activities can be refreshing and can provide children with fun and exercise but not without the potential risk for injury. The leading cause of death for children ages 1 through 4 years and the second leading cause of death for all children under age 14 years is drowning. Toddlers usually get into trouble around swimming pools while older children have more accidents in natural bodies of water. Many non-fatal drownings lead to hospitalization and lifelong sequelae such as brain injury.

Prevention of water related injuries requires minimizing modifiable risk factors. These risks include poor swimming ability, inadequate supervision and disuse or lack of safety devices. Children of all ages need direct supervision while swimming and engaging in water activities. Children may drown quickly and quietly; so adults who are supervising swimmers should have no other distractions or responsibilities. Swim lessons can be a fun family activity and may be started as early as a year of age. Learning to swim has been shown to decrease the risk of drowning in toddlers. Inflatable pool toys and floats are fun but not a replacement for life preservers. Children should wear properly fitted life jackets when on the water. Installation of a four sided fence around swimming pools reduces risk of drowning.

Bicycle Safety

Learning to ride a bicycle is a rite of passage in childhood. Bicycles afford children a modicum of freedom as well as healthy, efficient and fun travel with friends. Bicycles are also associated with many unintentional summertime injuries. Child bicycling deaths increase by 45 percent during the summer months. Modifiable risk factors which may help prevent bicycle associated injuries include increasing rider visibility, using protective equipment and following safety laws.

Many bicyclists are injured each year when they are struck by a motorist who failed to see them. Active lighting is one way to improve the visibility of bicyclists. White headlights and red taillights with reflectors on moving parts greatly increase visibility of the rider in low light settings. Reflective and light-colored clothing also helps drivers of motor vehicles see bicyclists. Well-fitted helmets with chin straps in addition to other padding and gloves can prevent injuries during a crash or a fall from a bicycle. The cyclist's adherence to traffic safety laws, including riding in bike lanes when available, may play a major role in preventing a majority of bicycle related deaths which can occur at non-intersections in roadways.

Falls and Playground Safety

As summer temperatures rise, so do the risks of fall associated injuries. Falls are a leading cause of injury in children each year, with deaths from falls increasing by 21 percent in the summer. In the hot summer months toddlers may fall from open windows or balconies, and energetic older children spend hours climbing on playground equipment. Some modifiable risks for fall injuries include inadequate supervision, improper use of playground equipment and trampolines, and failure to implement safety barriers.

Inquisitive toddlers are going to explore their environment. Keeping high chairs and other furniture away from open windows may help prevent a fall. Installing window guards or stops, which limit the ability to open a window past a safe point, will also help. Older children should be encouraged to use neighborhood playground equipment in a safe manner. Parents or supervising adults should check the equipment for defects

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and should make sure that rubber, mulch or other soft material surface is installed below the equipment. These surfaces work best to prevent injury when they are at least 1 foot in depth and surround the equipment by 6 feet.

Trampoline Safety

The safe use of trampolines could have been discussed in the previous section but deserves a little more attention. Proper use of trampolines provides a popular source of summertime fun and exercise. Improper trampoline use is associated with sprains and strains of limbs, fractured bones, concussions and potentially catastrophic head and neck injuries. The majority of these injuries occur on home trampolines, especially when more than 1 child is using the trampoline in a turn. Children under age 6 years are especially vulnerable. For these reasons and others, the American Academy of Pediatrics recommends against all home trampoline use.

If someone still wants to continue using or chooses to purchase a trampoline for home use, they should adhere to a few safety recommendations. After checking with their home owners insurance to see if trampoline injuries are covered, the trampoline owner should inspect the trampoline mat and springs for wear. Padding and barrier netting can be used to help prevent injury from landing on springs. Only one child should be allowed to use the trampoline at a time and somersaults should be discouraged as they increase the risk of neck injury if performed incorrectly.

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Summertime Safety

By Ross Shadbolt, Amarillo Fire Department

In light of the lockdown that all of us are facing, many cannot wait to enjoy summertime activities once again. Many people enjoy camping and hiking or social interaction in their own backyard, while others are fortunate enough to have property or access to a property that is located in a rural area. As people plan summer activities, they need to be aware of safety precautions to avoid fire.

When you are outside on trails or hiking, make sure you have packed enough water. According to the experts at University of Pittsburgh Medical Center, one should drink 14-22 ounces of water two hours prior to an activity. It is also recommended to drink 15-20 ounces every 15 minutes during outdoor activities. Instead of Sports drinks stick with water if your activity is under 60 minutes.

Lieutenant Carlos Castillo with the Canyon Fire Department states: "Those who go out for trail/day hikes underestimate the length and complexity of the trails and underestimate their water needs. Because of this, the Canyon Fire Department runs around 4-5 calls a month for heat related issues. They average one death a year for either heat-related issues or falls."

Many of those who enjoy hiking are also those who stay overnight in the campgrounds. Campers should know if there are any restrictions at the campsite for building campfires and if there are any fire rings or fire pits already in place. If there is not a pit, and pits are allowed, make sure you choose a site 15 feet from anything that could be flammable. Choose an area that is open and level but that is protected from wind gusts. Campers should clear an area 10 feet in diameter around the pit and dig it a foot deep, encircling the edge with rocks. When done, make sure that there is ample water to completely flood the fire. It must no longer make a hissing noise when water is applied. If it is too hot to touch, it is too hot to leave. According to the National Interagency Fire Center, in 2019 humans burned 202,918 acres of land in the southwest part of the United States. Due to this fact, during many times of the year (often 8 out of 12 months) the Texas Panhandle is in a burn ban. When this is the situation, the only burning allowed is a self-contained gas grill for cooking.

As the weather warms up from the cold winter months, grilling seems to be a great alternative to cooking in the kitchen. In the U.S., 7 out of 10 adults have a grill or smoker. Between years 2013-2017 fire departments averaged 10,200 home fires that were caused by grills or hibachis. Out of these fires 4,500 were structure fires and 5,700 were outdoor fires. These fires caused an annual average of 10 civilian injuries and $123 million in property damage. July is the peak month for grill fires (17%). May, June, and August are also high grill fire months.

Between the years of 2013-2017, an average of 19,000 patients per year went to the emergency rooms because of injuries involving grills. Half of the injuries were thermal burns, which included burns from fire, as well as from contact with hot objects. Five thousand two hundred thermal burns per year were caused by such contact or other non-fire events. Children under five accounted for an average of 2,000 (38%) of contact-type burns per year. These burns typically occur when someone, often a child, bumps into, touches or falls on the grill, grill part or hot coals.

Gas grills are involved in an average of 8,700 home fires per year. Eleven percent of gas grill structure fires and 23% of outside gas grill fires are caused by leaks or breaks, which are the primary problems with gas grills. Charcoal or other solid-fueled grills are involved in 1,100 home fires each year, including 600 structure fires and 500 outside fires annually.

Not all of these accidents can be avoided; however there are some basic ways to limit injuries or fires. Keep a fire extinguisher or garden hose handy when lighting up the grill. Don't leave the grill unattended for long periods of time, especially if you have small children. If there is a minor burn, run the injured part under cool water then clean the burn gently with soap and water. Have a first aid kit available. The first aid kit should contain petroleum jelly or aloe vera that can be applied to a minor burn. If needed, you can protect the burn from rubbing and pressure with a sterile non-stick gauze lightly taped or wrapped over it.

For those that are fortunate enough to have a home away from home, or access to a rural dwelling, there are some things to consider in order to keep you and your place safe before a wildfire threatens your area. To prevent embers from igniting your homes clear leaves and other debris from gutters, eaves, porches and decks. Remove dead vegetation and other items from under your deck or porch. These should be within 10 feet of the house. Screen or box-in areas below patios and decks with wire mesh to prevent debris and combustible materials from accumulating. Remove flammable materials, such as firewood stacks and propane tanks 30 feet away from your home's foundation and outbuildings, including garages and sheds. If it can catch fire, don't let it touch your house, deck or porch.

Wildfire can spread to tree tops. Prune trees so the lowest branches are 6 to 10 feet from the ground. Keep your lawn hydrated and maintained. If it is brown, cut it down to reduce fire intensity. Dry grass and shrubs are fuel for wildfire.

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Don't let debris and lawn cuttings linger. Dispose of these items quickly to reduce fuel for fire.

Inspect shingles or roof tiles often and replace or repair those that are loose or missing to prevent ember penetration. Cover exterior attic vents with metal wire mesh no larger than 1/8 inch to prevent sparks from entering the home. Enclose under-eave and soffit vents or screens with metal mesh to prevent ember entry.

It is wise to assemble an emergency supply kit and place it in a safe spot. Remember to include important documents, medications and personal identification. Develop an emergency evacuation plan and practice it with everyone in your home. Plan two ways out of your house and designate a meeting place.

Contact your local planning/zoning office to find out if your home is in a high wildfire risk area, and if there are specific local or county ordinances you should be following. If you are part of a homeowner association, work with your neighbors to identify regulations that incorporate proven preparedness landscaping, home design and building material use. Talk to your local fire department about how to prepare, when to evacuate, and the response you and your neighbors can expect in the event of a wildfire. Learn about wildfire risk reduction efforts, including how land management agencies use prescribed fire to manage local landscapes. In the event that there is a wildfire in your community, know how to respond.

In the event of a wildfire, stay aware of the latest news and updates from your local media and fire department. Get your family, home and pets prepared to evacuate. Place your emergency supply kit and other valuables in your vehicle. Move patio or deck furniture, cushions, door mats and potted plants in wooden containers either indoors or as far away from the home, shed and garage as possible. Close and protect your home's openings, including attic and basement doors and vents, windows, garage doors and pet doors to prevent embers from penetrating your home. Connect garden hoses and fill any pools, hot tubs, garbage cans, tubs, or other large containers with water. Firefighters have been known to use the hoses to put out fires on rooftops. Leave as early as possible, before you're told to evacuate. Do not linger once evacuation orders have been given. Promptly leaving your home and neighborhood clears roads for firefighters to get equipment in place to fight the fire, and helps ensure residents' safety. Learn how you can make a positive difference in your community by educating yourself.

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"Leaves of Three, Let Them Be"

By Hannah Wilkerson, MSIII & Ellen Hampsten, MD

Toxicodendron, literally meaning "poisonous tree," is a genus of plant with family members including poison ivy, oak, and sumac. The well-known contact dermatitis caused by these plants is highly prevalent, with about 50% of people exposed in nature having a reaction. Symptoms usually begin anywhere from a few hours to 4 days after contact and include intense itching and redness with development of papules and vesicles often in a characteristic linear pattern directly where the plant made contact.

Poison ivy, oak, and sumac can be found throughout the United States, with the exception of Alaska, Hawaii, high altitudes, and in the desert regions. Poison ivy can grow as a vine or a shrub, and each leaf has 3 leaflets, hence the term "leaves of three, let them be." Poison oak is similar, but usually more shrub-like. Poison sumac is a shrub with 7-13 paired leaves; this plant is usually found in swampy areas, particularly in the eastern United States.

What makes it "poisonous?"

The allergic component of poison ivy, urushiol, is present in the oils throughout the plant and can be released with minimal damage to the plant. Upon contact with skin, the allergen is taken up by Langerhans cells and is presented to T-cells. After clonal T-cell expansion, a second exposure will cause cytokine release that leads to the symptoms in 12-48 hours (delayed hypersensitivity). This is known as a cell-mediated Type IV hypersensitivity reaction. While a second exposure is more likely to cause a response, urushiol is known to be powerful enough to cause clinical symptoms even on the first exposure.

How can the reaction be prevented?

The easiest answer to prevention is to know how to identify these plants and simply to steer clear. Washing your skin with soap and water within about an hour of exposure can reduce the chance of developing a rash. Commercial washes that remove the oils from skin are also available. The oil can stay on clothing and tools for weeks to months, so washing contaminated items well can help prevent developing a rash next time you grab those tennis shoes or garden shovel. Don't forget the pets; fur can also be a culprit for transferring plant oils to your hands.

How is it treated?

Symptoms often will self-resolve in 1-3 weeks. As many of us know, that can be a pretty miserable 1-3 weeks. Calamine lotion, over the counter hydrocortisone creams, oral anti-histamines like diphenhydramine, and cool baths/compresses can all help to relieve itching in the meantime.

If the rash is widespread or involves sensitive areas such as the face or genitals, oral steroids are an option. The generally accepted treatment is 1 mg/Kg/day (maximum 60 mg per day) of oral prednisone, tapered over 14-21 days. Shorter courses of steroids may result in rebound dermatitis.

The most common complication of poison ivy dermatitis is a secondary bacterial infection. Watch out for signs of infection, as antibiotics may be warranted in some cases.

FAQs:

Can poison ivy spread from one part of the body to another, or from person to person?

No, not after the oil from the plant has been removed from your skin. The fluid from the blisters does not contain urushiol, so spreading this fluid is not going to spread the reaction. Lesions may show up at different times depending on the amount of urushiol initially present and the thickness of the skin involved, which can make it seem as though it is spreading.

Can poison ivy be inhaled in smoke?

Yes. If you're trying to rid your backyard of poison ivy, lighting it on fire is not the way to go. Exposure to the smoke can cause airway irritation and lung damage, so any difficulty breathing should prompt a trip to the doctor.

If I'm allergic to poison ivy, will I also have a reaction to the other plant species?

Yes, urushiol is the common denominator creating an allergic response in these plants, so if you're allergic to one you're allergic to all of them. In fact, there are also some foods that contain urushiol that can cause issues for people with a severe allergy to urushiol. These include mangoes, pistachios, and the shells of cashews.

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Snakebites in North America: What to Expect as Summer Approaches

By Dr. Rouzbeh K. Kordestani, MD, MPH

Snakebites are common in the United States. In the last decade, as many as 9,000 cases a year (average of 6,000) have been documented. This number is significant, but globally 1.2 million individuals are bitten by snakes each year. This much larger global number carries with it a tremendous degree of morbidity and mortality.

Most snakebites are actually dry, meaning that there is no discharge of venom into the bite. In addition, most snakes in the world are non-venomous. These facts downplay the dramatic morbidity and the mortality that can come along with snakebites.

In the United States, snakebites are more common than most people realize. Hospital records show a steady presentation of patients with snakebite throughout the year, with a higher number presenting when the weather is warmer. As for geography, more snakebites are seen in the southern arid/dry states, specifically Texas, Florida, and Arizona.

Even though the numbers of snake envenomations are high, mortality from snakebite is surprisingly rare. In fact, morbidity and mortality from snakebites are so rare that many young physicians in the United States and those in training have never cared for a patient with snake bite/envenomation. For this reason, there is a paucity of knowledge about the clinical presentation of this condition and how best to treat it. In an attempt to address this lack of knowledge, in 2013 the American College of Medical Toxicology (ACMT) founded the North American Snakebite Registry (NASBR). It was hoped that, by establishing such a registry, all data on snakebites would be enrolled into a centralized databank, allowing for the diagnosis, study and tabulation of snakebites on a national scale. Since its inception, the registry has met with some success. Since the enrollment is voluntary, however, many centers have chosen not to take part. In fact, Florida has very few centers that have enrolled. Therefore, the data can only be studied within its own context and cannot be extrapolated to the other parts of the country.

NASBR and its first window of data

The NASBR was first established in 2013. Even though participation in the registry is voluntary, the registry is carefully run by the ACMT. Data collection and enrollment are scrutinized by members of the ACMT prior to their listing in the database. For example, when an envenomation occurs, it cannot simply be reported over the phone. In their effort to keep the data exact and the registry clear of deficiencies, the examinations are actually completed with the toxicologists present. The diagnosis and the presentation are confirmed prior to the patient being enrolled into the registry. In this way, its data are reliable and can be tabulated into the system effectively. Follow-ups, labs results, and readmissions are also handled and carefully followed by the toxicologist and medical professionals. Because of this tight control, very little information is left out or missed.

In 2017, Ruha et al presented the first tabulation of the NASBR data, reviewing the first two years of data collection (from 2013 to 2015). Even though approximately 6,000 bites occurred on average during each of the collection years, the total number of snakebite cases registered into the database was only 450 (from 14 sites in 10 states; again Florida was not included;). Their presentation and conclusions are based on this data set.

NASBR and its initial 450 patients

Of the 450 snakebites registered through the NASBR from 2013-2015, almost all were due to pit vipers. This included rattlesnakes and copperheads. Cottonmouth snakes and other types of pit vipers were responsible for a majority of the additional bites. Marine species were conspicuously absent. Presumably this is because no centers in Florida were enrolled in the registry.

Of the 450 bites reported, a majority of victims were male. Interestingly, over a quarter were aged 12 and under. 54% of bites were on the lower extremity, and most bites were accidental or unexpected.

Almost all patients presented to their respective medical center or hospital for evaluation. Depending on their clinical presentation, most were admitted. A very small percentage stayed over 24 hours in the hospital. Their stay again was dependent on their clinical progression.

A majority (84%) were treated with antivenom, Crotalidae Polyvalent Immune Fab antivenom (CroFab). The amount of CroFab used was dependent on the patient's clinical presentation and the worsening of their symptoms.

Who gets bitten and where?

Based on the NASBR, the age of presentation was fairly broad, from 1 year of age to over 89. Of these, most bites (almost 70%) occurred in males. Only a small number occurred in children. Two hundred and forty-five of the bites were on the lower extremity. Most of these were not on the toes. Interestingly, 27% of patients who were bitten on the lower extremity were not wearing shoes. Of the group who were wearing some sort of footwear, it was most often sandals or flip-flops.

In the patient population from the registry, about 80% of those bitten noted that it was unintentional. This especially applied to women and children. In 19% of the total patients in the registry group (86 patients), the bite was intentional. This occurred specifically when a patient was playing with or reaching for the snake, be it a pet or in an attempt to grasp them. Almost this entire group of patients was male.

How Did They Present?

The clinical presentation of patient

with snakebites was variable. All demonstrated local tissue effects, such as swelling, ecchymosis and erythema. A small percentage presented with necrosis at the tissue site.

After the initial presentation, a significant number progressed to signs of systemic toxicity. Vomiting, diarrhea, hypotension, tachycardia, neurotoxicity, bleeding, respiratory failure and rhabdomyolysis were seen in patients as the snakebite effects become more generalized. These symptoms were more often seen in patients with previous or established underlying medical conditions. Systemic toxicity was more often seen in patients with rattlesnake (as opposed to copperhead or cottonmouth) bites (50%). Late signs of hematologic toxicity were also more frequent in patients with rattlesnake bites (40%).

Treatment Course

Almost all patients with snakebites were treated with Crotalidae Polyvalent Immune Fab (CroFab). Once envenomation was confirmed, they were treated with a starting does of 4 vials of CroFab. The treatment course was then adjusted and modified depending on the clinical progress of the patients. With most pit viper bites, across all patients, an initial treatment amount of 4 vials was enough. When the study patients were tabulated, an average of 10 vials was eventually needed for complete treatment. The greatest amount of CroFab was used to treat bites from rattlesnakes.

Most patients with snakebites were admitted to the hospital for observation. This was customary across all centers treating snakebite victims. Of the 450 patients in the database, 159 were discharged in less than 24 hours (with improving clinical picture). Only 27 patients (of the total 450) were kept over 72 hours. Almost all of these were patients with significant toxicity at presentation. Almost all of these patients (24 of 27) were victims of rattlesnake bites.

Once patients were deemed stable, they were sent home. Toxicologists from the registry then followed up with patients to ensure that they continued to improve. If there was any change in the clinical picture, the patients were encouraged to return to the hospital for further evaluation and possible treatment.

In Conclusion

In an attempt to better understand snakebites, their incidence, their clinical presentation, and the natural progression in their treatment, the NASBR was established in 2013. Since its inception, it has functioned as a central hub for data tabulation and collection for snakebites in the United States.

Even though enrollment in the registry has been voluntary, the NASBR data has shown that most venomous snakebites are due to pit vipers. The registry has shown that most bites occur in males, and most often are unintentional. Most snakebites are unexpected and are often on the lower extremities. Victims of snakebites will usually present with clinical signs of swelling, ecchymosis, and/or erythema.

The registry informs us that, once a patient presents and is treated with CroFab, most do well and can be discharged within 24 hours. The registry clarifies that, because of the possibility of clinical decompensation, the usual patient is admitted to the intensive care unit for monitoring. Patients with rattlesnake bites tend to do worse and to need more intensive treatment.

A Look to the Future

The NASBR has quickly established itself as a centralized information hub for the tabulation of snakebites in the United States. As enrollment in the registry continues to grow, it will serve as a nidus for understanding snakebites. Based on idata from the years 2013 to 2015, it has already furthered the understanding about the incidence of snakebites, the clinical presentation of snakebite victims, the needed clinical interventions, the response of patients, and the morbidity and mortality seen. It has outlined the best course of treatment. It has also shown us what to expect in regard to delayed presentations and how best to plan for these.

In the future, snakebites will be better understood and treated due to the data put forth by the NASBR. Even though we cannot change some of the behaviors that leads to snake bite, the NASBR data can help us plan a safer and more regimented treatment course for this all-too-frequent Panhandle summertime hazard.

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The Spanish Flu Pandemic of 1918

By Dr. Rouzbeh K. Kordestani, MD, MPH

It is estimated that the flu of 1918 touched up to 900 million people, almost 50% of the entire world population at that time. While it infected a vast many, the actual casualties were not as severe. Still, while the standard "seasonal" flu often adversely affected about 0.5% of a population each year, the flu of 1918 affected or killed as many as 5% of the world population.

In European countries, such as France and Italy, a full 3-5% of the population died. In India, records estimate that 13.88 million people died. In Japan, 23 million people were infected and about 400,000 died. In Iran, the mortality was exceptionally high, at 15% of the entire population. In China, more than a million people died. In Russia, more than 3 million people lost their lives. Isolated smaller regions were luckier. Smaller islands like Marajo and American Samoa were hardly affected, in part due to their isolation and in part due to the efforts of their governments to restrict travel. All in all, by the time the pandemic passed, it had claimed over 100 million human souls.

The flu of 1918 became known as the "Spanish Flu" for the simple reason that the Spanish press discussed it most openly. Even though the pandemic affected many European countries equally, Spain, a neutral country at the end of the conflict, along with its press, was openly discussing and noting the presence of the new respiratory illness. Other countries such as the Germany thought it would be bad for morale to discuss deaths from the disease. So, while the warring nations suffered most from the effects of the disease, the Spanish had the misfortune to claim the name.

How did the Spanish Flu start?

No theory or fact can clearly explain the origin of the virus. However, several theories exist as to how and when the pandemic began. One theory suggests that the pandemic started in Europe, in late 1917 to early 1918. This theory suggests that the flu first took hold in Europe and ravaged both the Allies and the Central Powers. It first affected the Central Powers of Germany and Austria. It then passed to the Allied armies. The theory hypothesizes that the flu may have affected the Central armies disproportionately and may have had a part in causing more deaths than in the Allied camps, simply because it took a foothold there first. The severe loss of life in the lines of the Central Powers may have led to their compromise and eventual defeat in the war. Unfortunately, the evidence in support of this theory is sparse because of poor record keeping at the end of the war.

Another theory suggests that the disease may have originated in the United States, in Haskell County, Kansas, at one of the Army bases. Records show early spread of a flu-like condition in early 1918. The first death was documented in early March. This theory holds that this death may have been the first death due to the virus. This is significant since this same army base was used as a farming center for chickens, pigs and rations for the troops. In the same period, in the same area, historical records show the emergence of both a new avian flu virus and a new swine flu virus. Records also show a rapid spread of this new flu within the army bases in Kansas. The theory goes that these infected troops, along with their stocks of infected chickens and pork, were transported to Europe with the American troops, thereby taking the disease to Europe in late March and early April of 1918. There is ample data to support this theory. However, conflicting documents suggest that the disease process was well in place in the battlefields of Europe prior to the arrival of American troops.

A third theory suggests that the origin of the Spanish flu is from China or East Asia. Genetic sequencing of the Spanish flu shows a possible link to China. This theory is supported by the lower death rates seen in the Chinese population. If the people of China had already been exposed to the disease, they would then have developed some immunity. This immunity in turn would explain the lower death rates. Unfortunately, accurate population and mortality records from this period in China are not available. The records that are available do not support such a hypothesis.

The most accepted theory places the origin of the disease around March or April of 1918, in Brest, France. Here, an influenza virus had its start. Brest was significant as it served as a confluence for the Allied troops. The troops were collected there to start their offensives. These Allied troops, Americans included, were amassed together in close quarters. In this setting, the flu began its spread. Furthermore, once the war was over, as the troops were being debriefed and readied to travel back to their home countries, the respiratory virus was firmly present in Brest, in these same staging areas. In such conditions with amassed armies, where there was malnutrition, overcrowding, and additional illnesses, the flu had a particular advantage. Most importantly, with the new modalities of travel, the physical barriers of distance and topography were overcome, allowing the disease access across the globe, to most populations. Much of the data collected on the troops and their movements support this explanation.

So why was the Flu of 1918 so deadly?

With the war raging in Europe for more than four years, the troops were often battered and sickly. In their fatigued states, poorly housed, poorly nourished, exposed, and in overcrowded areas, the soldiers were most vulnerable to respi-

ratory illnesses such as the flu. This confluence of factors placed the young population at a particular disadvantage. Moreover, many of the deaths seen in the time of the pandemic were actually due to secondary infections, superimposed on the flu. In thousands of cases, the flu was a gateway to bacterial pneumonia. This combination of a bacterial and a viral infection proved deadly. Also, mortality records show an increased incidence of tuberculosis. With the flu in place, secondary tuberculosis infection could and most often did lead to death.

The lack of therapeutic interventions in the early 20th century did not help those who were infected. Very few drugs were available to combat the virus or the bacterial superinfections. The disciplines of virology and bacteriology were in their infancies. Without access to antibiotics or antivirals, simple remedies were advocated as therapeutic interventions. These simple remedies unfortunately themselves often did harm. Looking through the journals and details of the period, one of the few drugs that was available was Bayer aspirin. It had been touted as the cure for a great many illnesses. When the Spanish flu surfaced, many groups including physicians advocated the use of high doses of aspirin. During the height of the outbreak, daily doses as high as 30 grams were routinely prescribed. This dosing led many individuals to die of aspirin toxicity and organ failure.

So why was the Spanish Flu so different?

Most influenza strains tend to affect the weaker in the exposed population. The strains mostly kill the youngest, those less than 2 years of age and the oldest, those above the (physiological) ages of 60-65. Along with this, the next population most adversely affected are the immunocompromised. The Spanish Flu of 1918, however, was different in that it affected the younger population, aged from 25-40, far more. Records show that a full 92% of mortalities seen with the Spanish Flu were below the age of 65.

Several theories have been put forth to explain this discrepancy. Two are of particular note. One theory suggests that the deaths were due to the emergence of a combination of a human and avian flu strains. Worobey and his evolutionary biology colleagues in Canada retrospectively studied the genetic sequence of the Spanish Flu and compared it to the H1N1 flu strain of 1900. The genetic sequence similarities between the two were significant. The theory is that the H1N1 flu strain of early 1918 (possibly left over in the population from the Russian flu of 1900) combined with an avian flu strain from the early 20th century. This combination then mutated and became the deadly Spanish Flu virus.

The other theory that is most often ascribed is that of a "cytokine storm." It is known that, when compared to the elderly, infants or the immunocompromised, younger individuals have a stronger immune system, As the younger patients became exposed to the virus, their immune system overreacted with an overwhelming, crippling response. This immune response caused pulmonary edema, and additional systemic overreactions. This in turn led to multi system organ failure and subsequent death. This theory is well supported. Darwyn Kobasa and his team were able to unearth bodies of soldiers from the period. They then isolated the Spanish Flu strain from their tissues. A genetic replicant was constructed and then used to infect healthy primate hosts. As proof of the cytokine storm, all of these primate hosts proceeded to go through the same sequence of over-reactive immune responses and systemic organ failures. All of the animals died in similar patterns to the young soldiers of 1918.

The disease came in three waves, the second being the most severe

The Spanish Flu attacked the populations of the world in three waves. The initial wave hit Europe in early 1918. Population and mortality records confirm this first wave. The first wave mirrored those of previous flu cycles. In this way, the Spanish Flu was thought to be another simple flu, following the typical pattern. The disease started with respiratory complaints and progressed from there. It initially affected the elderly, the sick, and the immunocompromised like the household flu.

By August of 1918, the virus had become much deadlier. The second wave then hit Europe and most of the civilized countries including the United States. Records show that the young were now much more severely affected. There was a significant change in the population statistics of those infected. Also, the mortality rose sharply, with October 1918 being the deadliest month on record for the pandemic.

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POTTER RANDALL COUNTY MEDICAL SOCIETY (PRCMS) OFFERS HELP TO TROUBLED PHYSICIANS

If you, or a physician you know, are struggling with addiction, depression or burnout and are unsure what to do or whom to contact, the Potter-Randall County Medical Society is here to help. We offer face-to-face confidential sessions with the PRCMS Physician Health and Wellness Committee, made up of your physician peers who know and understand recovery. Please don't struggle alone when help is a phone call or an email away. Whether you are calling for yourself, your practice partner, or as a family member of a physician, contact Cindy Barnard, PRCMS Executive Director, at 806-355-6854 or prcms@suddenlinkmail.com. Membership in PRCMS is not required.

Unfortunately, records also show that the pattern of medical response may have made the problem worse. As overcrowding was known to be problematic, elderly patients with the flu were managed at home. It was reasoned that these patients would do poorly anyway. Younger patients with their stronger immune system were crowded into already overcrowded hospitals. No one thought that this would harm them. Using the common logic that the younger individuals would do better and therefore have to be less guarded, crowding the younger individuals was advocated. This grouping caused the number of cases to geometrically increase. The death rates soared. While the elderly recovered at home, the young died in the cities and in the hospitals.

More isolated countries like Denmark were fortunate in that their exposure to the first wave helped develop population immunity. This helped them survive the second (deadlier) wave. Denmark was exposed to both strains of the virus, one in early 1918, and the other in August of 1918. Initial death rates in Copenhagen were at 0.02%, in line with the initial flu exposure from the first wave. When the second wave hit Denmark, the population mortality, although significantly higher at 0.27%, was still far lower than many other European countries. Unfortunately, in some European countries, there was no first wave and hence no development of immunity. These countries initial exposure was to the second deadlier wave. Because of this, they suffered much heavier losses. In these countries, mortalities were as high as 5% of their entire population.

A third wave hit sometime in early 1919. By then, the virus had again started to mutate. This third strain was much less virulent. More importantly, most of the population around the world had by now been exposed. Records show minimal mortality from this third wave.

Lessons learned from the 1918 Pandemic

The Spanish Flu is thought to be the most devastating health catastrophe in modern history. Some of the fault lies in the initial response. It was treated lightly like the ordinary flu. The lack of awareness of its severity allowed it to get a strong foothold in Europe, the United States, and throughout the world. In the United States, the institution of the Sedition Act added to the spread of the disease. Under this legislation, the disease and its transmission were downplayed so as not to affect morale during the war effort, resulting in a false sense of security. This delay in understanding the gravity of the disease both in the United States and abroad cost thousands of additional lives.

The Spanish Flu was the first pandemic to spread across the entire globe using the new modalities of transportation. Previous pandemics did not spread so easily. As populations were infected and patients died, the diseases often ran aground. The epidemic was self-limited. This was not so with the Spanish Flu. The Spanish Flu was the first pandemic to spread without constraint to the four corners of the world. With the new modalities of transportation, the disease was not physically constrained. There were no impediments. It's spread across the seas, land and air showed that simple barriers no longer worked. It also showed that much more stringent rules of isolation and containment are necessary if there is any hope of saving lives with new pandemics, such as our current Covid-19 pandemic.

The Spanish Flu presented in three waves. The first wave behaved much like the seasonal flu. As the first wave ebbed, regulations were relaxed. Then came the more deadly second wave. There were few attempts at isolation, making the second wave impossible to control. The lack of foresight in planning for this second wave cost millions of lives. Also, the lack of attention to additional factors such as bacterial superinfections and tuberculosis, further added to the mortalities seen. Regrettably, overcrowding in urban areas, the exposure of the population without restriction, malnutrition in some of the population, and the lack of access to health care in rural areas led to far more deaths than would have been expected.

There are many lessons to be learned from the upheaval and the deaths caused by the Spanish Flu pandemic of 1918. This could not be truer than now. In 2020, we are faced with the pandemic of COVID 19. This virus has proved to be nearly as sinister and just as cataclysmic. We need to look at the Spanish Flu, its presentation, its transmission and its mutations for lessons on how to protect our patients and ourselves. We must learn from our past short-sightedness and ignorance, and use these lessons to chart a safe path towards the future.

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Massive Basal Cell Carcinoma with Associated Lymphadenopathy: Successful Treatment with Complete Surgical Excision

By Ganesh Maniam, Dr. Alan Sbar

BACKGROUND:

Basal cell carcinoma (BCC) is the most common skin malignancy, and is usually indolent (1). Metastasis can occur via blood vessels and lymphatics, but metastasis is exceedingly rare at only 0.003% - 0.55% of cases – far less than squamous cell carcinoma or melanoma (1). The presence of lymphadenopathy is also a rare finding with basal cell carcinoma, given the low incidence of metastasis. This paper presents a case of massive basal cell carcinoma with associated lymphadenopathy that was successfully treated with complete surgical excision.

CASE REPORT:

A 55-year-old man presented with left arm skin lesion (Figure 1) that had recently become painful. The lesion initially appeared 5 years ago as a flat red discoloration of approximately 1 – 2 millimeters in diameter. The lesion had progressively increased in size and pain. The patient did not have any occupational sun exposure, but did have a family history of basal cell carcinoma in his father. Physical exam revealed an ulcerated left lateral upper arm lesion that was raised, non-blanching, erythematous, and draining serous fluid. The patient also had painless left axillary lymphadenopathy, 2 cm, without any preauricular, postauricular, submandibular, submental, anterior cervical, posterior cervical, or supraclavicular lymphadenopathy. There were no other suspicious lesions on skin survey. Biopsy of the lesion revealed basal cell carcinoma. The patient underwent soft tissue ultrasound of the left axilla, which revealed a single benign-appearing left axillary lymph node. Despite the pathological diagnosis, the exam raised the question of metastasis. Surgical resection was performed via wide local excision with left sentinel lymphadenectomy, as well as lymphadenectomy of the palpable node. On excision, the lesion appeared to have invaded deep dermis, but not the subcutaneous tissue. Surgical pathology noted the peripheral and deep resection margins were free of carcinoma, and the left axillary nodes were negative for metastatic disease.

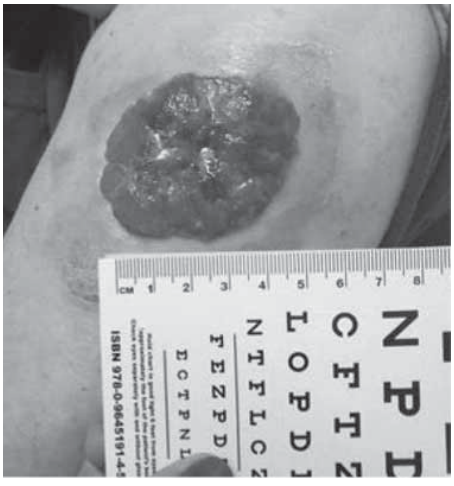

Figure 1. ***Photograph of lesion***

DISCUSSION:

Epidemiologically, BCC affects males more than females (2:1), and the majority of cases are in Caucasian patients. The median age for primary tumor onset is 45 years (2). Diagnosis of BCC requires a skin biopsy, while diagnosis of metastatic BCC as per Lattes & Kessler guidelines requires (a) primary BCC tumor originating from skin rather than mucous membrane, (b) metastasis occurring at a distant site, meaning that direct extension must be ruled out, and (c) similar histopathology between the primary and metastatic tumors (3). Treatment of BCC is surgical intervention, while metastatic BCC requires additional surgical treatment for regional metastasis and combination chemoradiation for distant metastasis, with cisplatin having been found to be the most effective chemotherapeutic agent (2). In this case, the patient did not have metastatic BCC but rather only localized BCC, albeit massive in size.

The presence of lymphadenopathy is certainly a unique feature in this case, as BCC has only rarely been associated with regional lymphadenopathy. Cases are quite sparse in the literature, but one case described a 59-year-old Caucasian woman with a 2-month history of painless left axillary lymphadenopathy who was found to have infiltrative BCC on histopathological examination following lymphadenectomy. The primary tumors on her chest and shin were approximately 5 mm in size, and were excised with clear margins (2). Another case discussed a 67-year-old man who was transferred to oncological surgery due to a 3 x 5 cm recurrence of BCC on his right scapular region, which had been excised 16 years ago. A subsequent thorax CT showed mediastinal, carinal, subcarinal, and aorticopulmonary lymphadenopathy. He was treated with wide margin surgical excision and scapulectomy, but the BCC had already invaded the scapular bone and bronchial wall (4). A third case presented a 68-year-old Caucasian woman involved with an 8 year history of gradually enlarging ulcers on the left lower leg and left shoulder that were draining and associated with both left axillary lymphadenopathy and left inguinal lymphadenopathy. This was diagnosed as BCC following biopsy specimen, and ultimately required multiple surgical excisions to treat (5). Finally, a case was reported of a 62-year-old Caucasian man with an extensive history of BCC who presented with right-sided cervical lymphadenopathy. Fine needle aspiration biopsy revealed BCC, requiring surgical excision and radiotherapy (6). Additionally, a 2015 study looked at selected case reports of BCC and

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included a presentation of BCC of the ear with cervical lymphadenopathy (7).

Prompt recognition and diagnosis of BCC, as well as early surgical excision, will be most effective in preventing metastasis and further complications. While metastasis of BCC is exceedingly rare, the presence of lymphadenopathy remains a concerning sign of lymphatic metastasis. The clinical presentation of the large, ulcerated lesion with palpable lymphadenopathy required appropriate diagnostic workup. Following lymphadenectomy, histopathological examination of the nodes in this case actually revealed benign findings rather than metastasis. In this case, the massive basal cell carcinoma and lymphadenopathy of this patient was successfully treated with wide margin surgical excision and lymphadenectomy.

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Severe Hemolytic Jaundice due to ABO Incompatibility

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Abstract

A 28-year-old G6P5015 delivered an early term average for gestational age female. The mother's blood type is O+ and the neonate's blood type is B+, giving her ABO incompatibility that required intense phototherapy and multiple doses of IVIg. Case reports demonstrating this level of severity and rapid onset of hyperbilirubinemia in a newborn with ABO incompatibility are extremely rare. We use this case to explore the debate between intravenous immunoglobulin (IVIg) and double volume exchange transfusion (DVET). More research is needed to understand if IVIg is effective in preventing the need for double volume exchange transfusion.

Introduction

We report a case of a mother who has O+ blood type giving birth to an early term female with B+ blood type. The patient was transferred to the NICU after intense phototherapy due to hemolytic disease caused by ABO incompatibility. It has been reported that hemolytic disease of the fetus and newborn (HDFN) is present in 3 to 80 out of 100,000 births per year (1). Elevated bilirubin levels are dangerous for neonates because unconjugated bilirubin can cross the blood-brain barrier when it is not bound to albumin because it is fat soluble. When albumin becomes saturated with unconjugated bilirubin or a pharmacological agent causes the bilirubin to unbind, then the bilirubin easily crosses the blood-brain barrier. In the brain, bilirubin has a tendency to cause cell death by apoptosis which leads to bilirubin induced neurologic dysfunction (BIND). This may manifest as disorders in vision, hearing, gait, speech, cognition, and language. Acute Bilirubin Encephalopathy (ABE) is either reversible by treatment or results is the permanent dysfunction called BIND (2). This is a serious complication that should be avoided.

The mortality in patients before any intervention was 50%, which then decreased to 25% after the implementation of exchange transfusion in 1945. Occasionally, neonates with hyperbilirubinemia will not respond sufficiently to phototherapy. In these cases, a decision of the next appropriate step to lower the bilirubin must be made. There are currently two available options for intensive therapies: intravenous immunoglobulin (IVIg) and double volume exchange transfusion (DVET). These therapies have different benefits and risks that must be carefully considered when treating a neonate.

Case description

A 28-year-old African American G6P5015 mom vaginally delivered an early term average for gestational age female born at 37 weeks. The mother is O+ blood type and the neonate is B+ blood type giving her an ABO incompatibility that was DAT positive. The mother had other children with jaundice, but none required intense therapies. Cord bilirubin and maternal IgG titers could not be measured in this case due to rapid onset of labor. However, the neonate's serum bilirubin at 6 hours of life was elevated at 8.33 mg/dL putting her at high risk for severe hemolytic disease of the newborn. Her total serum bilirubin (TSB) was 19.66 mg/dL on day of life 3 (DOL 3) requiring her transfer to the NICU for more intense therapies. Her reticulocyte count was 19% and her hematocrit was 42%. The newborn was placed on intense phototherapy including 2 sets of lights with two light bulbs on each one and a bili blanket underneath. She received intravenous immunoglobulin (IVIg) 1 g/kg in order to bind to the mothers anti-B antibodies and decrease the rate of hemolysis. Lastly, she received IV 10% dextrose at 60 mL/kg/day along with breast milk in the NICU which was discontinued 3 days later. TSB peaked at 20.63 mg/dL on DOL 3. The neonate's bilirubin started to decline the next day to 16.97 mg/dL. On DOL 5 the TSB increased by 0.4 mg/dL, so a second dose of 1 g/kg IVIg was given. It took four more days to wean the phototherapy to just 1 light. One worrisome feature was that her hematocrit dropped from 42% (DOL 3) to 29% (DOL 10) indicative of continued hemolysis by her mother's antibodies. This and the elevated reticulocyte count of 5-6% prompted a third and final dose of IVIG. DAT IgG was positive for anti-B at DOL 11.

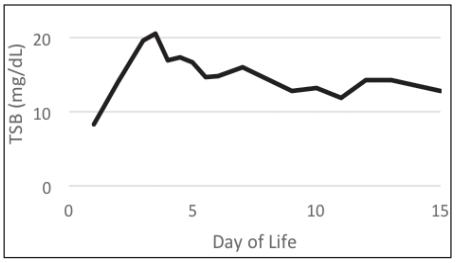

Figure 1. ***Total serum bilirubin (TSB) in mg/dL from birth (day of life 1) to post-hospitalization follow-up (day of life 15).***

Her total serum bilirubin slowly declined with manageable rebounds after being off phototherapy for 2 days, so the patient was discharged home on iron supplements. Her TSB was 12.78 mg/dL on follow up 2 days after discharge. Neonatal testing for other conditions such as glucose-6-phosphate dehydrogenase deficiency and hereditary spherocytosis was negative. Mother has no significant maternal medical history or gestational complications. All gestational screening and toxicology were negative.

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Discussion

This specific case was brought to our attention due to the severity and rapid onset of hyperbilirubinemia. While many ABO incompatibilities are either self-limiting or require phototherapy, this case met the protocols for intensive therapy.

A case report published in 2018 explained a similar case of severe ABO hemolytic disease of the newborn requiring double volume exchange transfusion (DVET). An O+ African American mother delivered a B+ neonate. DAT was positive for anti-B and anti- A,B. Maternal IgG titer 1024. She was similarly started on fluids and IVIg (1g/kg) at 21 hours of life. The newborn's TSB rose from 17.3 mg/dl (18 hours) to 17.6 mg/dl (28 hours) and did not respond to IVIg in contrast to our case. Since there was in fact a small rise in TSB, the neonate met AAP criteria for DVET. DVET occurred and TSB decreased to 10.5 mg/dL and hematocrit increased from 34% to 45% post procedure at 32 hours of life suggesting that the rate of hemolysis had decreased (3). When comparing this case to ours, it is notable that their patient was released on hospital day 4 after receiving DVET, in contrast to ours which was released on hospital day 13 after three doses of IVIg. The shorter hospital stay may have been due to the use of DVET rather than IVIg.

In a study done in 2002 on 136 healthy term newborns with ABO incompatibility, a serum bilirubin measurement of >4 mg/dL at the sixth hour of life was the most sensitive for detecting severe disease while the critical bilirubin levels of >6 mg/dL at the sixth hour of life was specific for neonates that will develop severe hemolytic disease of the newborn (4). In our case the TSB as the 6th hour of life was 8.33 mg/dl indicating a high likelihood of severe disease. Total serum bilirubin (TSB) should be measured if TcB exceeds the 75th percentile of normal on the TSB nomogram, or if it exceeds the 95th percentile of normal on the TcB nomogram (5). To decide whether treatment is needed, the Bhutani nomogram

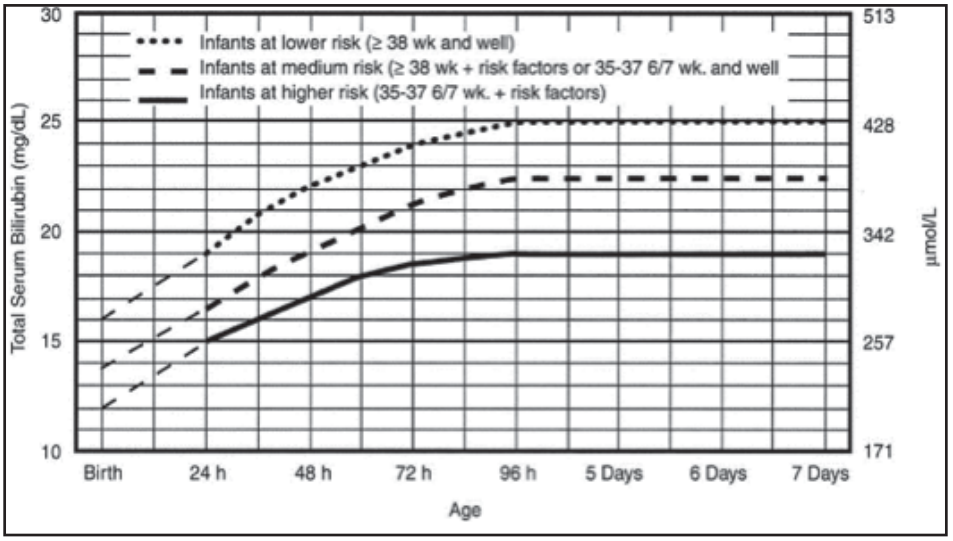

Figure 2. ***Exchange Transfusion Nomogram2***

is commonly used. In this patient, the neonate was in the high-risk zone of the nomogram from 12 hours until the 5th day of life, which means treatment was necessary.

While there is controversy about the use of IVIg due to lack of substantial studies, AAP recommends its use in proven hemolytic disease of the newborn (2). As with any therapy, there are side effects which must be considered. Some of the immediate reactions which can occur include flushing, urticaria, pain, transfusion related acute lung injury (TRALI), and transfusion-associated circulatory overload (TACO). Delayed reactions which can occur include acute kidney injury, hemolysis, neutropenia, and more. Additionally, there have been cases which report an increased risk of necrotizing enterocolitis in newborns following administration of IVIg (6). When administering IVIg, it is therefore important to take whatever steps possible to decrease the incidence of side effects via a slower infusion rate, and adequate hydration.

Another method of treating hyperbilirubinemia of the neonate is double volume exchange transfusion (DVET). Using this for hyperbilirubinemia is important as it both removes circulating bilirubin, as well as antibodies in hemolytic disease of the newborn. DVET takes about 1.5 hours. It is recommended to start by exchanging only a 5 ml aliquot of blood at a time so that the stability of the neonate can be monitored. While it is an effective procedure, the associated risks include infection, transfusion reactions, circulatory overload, changes in electrolytes, necrotizing enterocolitis, and others (8). In a study of 55 neonates that required 66 DVETs between 1992 and 2002, they reported thrombocytopenia in 44%, hypocalcemia in 29%, and metabolic acidosis in 24%. This study also demonstrated a decreased risk of adverse events from 88% to 58% when only an umbilical vein catheter was used compared to using both an umbilical vein and artery catheter (7). While severe complications are rare, this procedure is time-consuming and requires many resources to appropriately perform. Used in the correct setting, however, DVET can greatly aid in the management of hyperbilirubinemia. The AAP recommends use of a nomogram charting hours of life against total bilirubin to determine if a patient is a candidate for DVET therapy (Figure 2).

Deciding whether to administer IVIg before DVET is a controversial decision. While there have been various studies examining the role of IVIg in reducing the rates of DVET use, a recent meta-analysis of these studies demonstrated a high-risk of bias in the studies which advocates for IVIg use. The studies which the meta-analysis determined to have a low-risk of bias showed no association between IVIg use and decreased need for DVET (9).

However, the AAP still recommends the use of IVIg in instances where the bilirubin continues to rise despite intensive phototherapy, or if it is within 2-3 mg/dL of the threshold for DVET (2). With our patient, IVIg helped to decrease hemolysis following administration. Although studies have shown that both are effective, there have not been high-powered studies comparing the two treatments (9). Since we know that elevated bilirubin in the neonate can lead to long-term neurological consequences, we must continue to study safe, effective, and evidence-based treatments.

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Melanoma Cancer

By Ananya Bharadwaj, Steve Urban, MD

What is malignant melanoma?

Melanoma is the most dangerous kind of skin cancer. It arises in pigment cells called melanocytes, the cells that give color to the skin. Damage to melanocytes, usually caused by ultraviolet light exposure, can cause DNA mutation and uncontrolled cell growth. Spread of the malignant cells to distant parts of the body is much more common in melanoma than in other skin cancers. The incidence of melanoma is increasing at a greater rate than any serious cancer; it is estimated that 1 in 50 Caucasian patients will develop melanoma in their lifetime.

As per the American Cancer Society (ACS), about 100,350 individuals will be diagnosed with melanoma in 2020, with about 6850 deaths expected in the same period of time.

What are the risk factors for melanoma?

As mentioned above, ultraviolet (UV) light exposure is a major risk factor for developing melanoma. Fair-skinned patients are at risk; patients who suffered multiple sunburns in childhood and adolescence are at even higher risk. Melanoma is 5 times more common in Caucasians than Hispanics and 20 times more common in Caucasians than African Americans. Although most moles are not malignant, certain dysplastic (atypical) moles may pose a risk for melanoma. Patients with an inherited condition called dysplastic nevus syndrome are at very high risk for developing melanoma. Other risk factors include age, family history of melanoma and immunocompromising conditions.

When should I get a mole checked?

Melanoma can appear anywhere on the body, not only where sunlight hits. For this reason, it is crucial for individuals to keep a watch on their moles, lesions, or suspicious colored areas on the skin. Unusual moles or new or changing moles may be a sign of melanoma. About half of melanomas arise from normal skin (i.e. not from a changing mole); so a new spot on the skin or a spot that is changing in size, shape, or color should be checked by a specialist.

Worrisome features for melanoma can be remembered by the ABCDEs:

A - Asymmetry:

B - Border: An irregular, ragged or notched edge.

C – Color: Benign moles are usually uniform in color. Variegated shades of brown, black or blue may suggest melanoma.

D - Diameter: 6 mm or larger (larger than the diameter of a pencil eraser).

E - Evolving: Changes in size, shape or color. A bleeding or itching mole is of concern.

How is melanoma diagnosed in a suspicious or changing mole?

If melanoma is suspected, the lesion needs to be biopsied. Most commonly, excisional biopsies (where the entire lesion is removed) or saucerized shave biopsies are performed (see article in this issue by Dr. Summer Clark). The specimen is sent for histology and, in many cases, immunochemical and genetic testing. A crucial piece of information is depth of invasion. As a general rule, invasion of < 1 mm confers favorable prognosis, whereas invasion deeper than 2 mm is of concern. Ulceration of the lesion and high growth rate (as assessed by the number of mitoses) are prognostically important as well.

What kind of surgery is done for melanoma?

Surgical removal of the tumor is necessary to cure melanoma. Tumors with depth 1 mm or less are usually resected with a 1 cm margin of normal tissue. Most deeper or ulcerated tumors are managed with 2 cm resection margin and are considered for sentinel lymph node biopsy. If the sentinel node shows evidence of disease, a more extensive removal of the nodes in that regional lymph node area is performed (e.g. for a lesion on the arm, this would be lymph nodes in the armpit). Between 20 and 50% of patients with lymphatic spread of the tumor can be cured with removal of the lymph nodes (often combined with adjuvant therapy).

What is adjuvant therapy? Can it be useful in melanoma?

Adjuvant therapy is treatment given when there is no overt evidence of remaining disease; its purpose is no kill residual cancer cells and to prevent recurrence. Recurrent or metastatic disease in melanoma, as in most cancers, is a dire occurrence; so those at high risk can be treated expectantly to decrease this risk. Deciding how best to treat patients with metastases to regional nodes is the subject of much current research.

Melanoma responds poorly to chemotherapy; so adjuvant chemotherapy (as is often used in premenopausal breast cancer or colon cancer) is not used. Patients whose lymph node biopsies reveal metastatic cancer are treated either with adjuvant targeted therapy or with immunotherapy (see below). These agents produce few side effects but are very expensive.

Does anything help metastatic melanoma? Metastatic melanoma has traditionally been considered a very lethal cancer, but this outlook has been radically altered by novel treatments: targeted therapy and immunotherapy.

Targeted therapy (signal transduction therapy): Some patients with melanoma have activating mutations that cause uncontrolled cell growth. A patient's tumor can be tested for these mutations; if the cells are positive for this mutation, several drugs are available to suppress

the overactive enzymes. These medications block the mutated BRAF or MEK genes and have dramatic benefit in some patients who harbor these mutations.

Immunomodulating therapy: Cancer cells develop mechanisms to evade destruction from the body's immune cells (in particular, T-cells). T-cells can be stimulated to attack the previously "invisible" cancer cells by counteracting certain signals that check or inhibit the activity of the T-cells. These drugs are called checkpoint inhibitors. One kind of checkpoint inhibitor uses monoclonal antibodies to block the CTLA-4 molecule; other drugs block a protein called PD-1. Both kinds of checkpoint inhibitors can produce dramatic responses, even in patients with metastatic melanoma, as in demonstrated by the long remission of former president Jimmy Carter.

Preventive measures against melanoma:

Limit exposure to ultraviolet (UV) rays:

a. Avoid UV light. Risk of melanoma and other skin cancer can be reduced by wearing long-sleeved shirts, wearing hats and applying sunscreens. The daily use of sunscreens with a sun protection factor (spf) of 30 or more can lower the risk of melanoma by 50%.

b. Avoid tanning beds: Increased risk of melanoma has been found even with the occasional use of tanning beds. Exposure to radiation from tanning beds or lamps before the age of 35 increases the risk for melanoma by 75%.

c. Protect children from excessive sun exposure.

Watch for abnormal moles: Check your skin for any new or growing moles and, if any are suspicious, discuss this observations with your doctor.

Melanoma and other skin cancers are common in the Texas Panhandle area due to the increased exposure to sunlight throughout the year. Although advances in treatment have occurred, almost 7000 people still die in the U.S. every year from metastatic melanoma. Careful attention to prevention and early diagnosis can reduce the risk of melanoma and may save numerous lives.

History of Lyme Disease

By Rouzbeh K Kordestani, MD, MPH

Introduction

Lyme disease (Lyme borreliosis) is an infectious disease caused by the bacterium Borrelia, which is spread by ticks. The bacterium is a spirochete and is transmitted often along with other bacteria through seasonal spread in areas endemic with the presence of ticks. The disease process is prevalent in the northeastern United States but can be encountered elsewhere, even in Texas. However, several types of borreliosis are prevalent in different areas of the world. In North America, *Borrelia burgdorferi* and *mayonii* are common, while in Europe and Asia, *Borrelia afzelii* and *garinii* are the main culprits. In the United States alone, the disease process affects over 300,000 people a year.

Lyme disease most often presents first with a rash. This distinctive rash (erythema migrans) can be accompanied by fever, chills, headache, fatigue, neck stiffness, and body aches. It can present months after the initial infection with recurrent fever, myalgias, disseminated skin lesions, facial nerve palsy (sometimes bilateral), and heart block. There may be tingling in random patterns in the upper and lower extremities, aseptic meningitis, or other neurological signs such as confusion, abnormal gait, abnormal ocular movements, slurred or impaired speech and shaking. Months to years later, persistent and destructive joint inflammation (usually in one or a few large joints) predominates. The lack of a definitive pattern can make the disease hard to diagnose. Delay in diagnosis and treatment can result in permanent joint, neurological or cardiac injury. Permanent heart sequelae (Lyme carditis) can produce cardiomegaly, ventricular dysfunction and congestive heart failure in up to 15% of patients so affected.

The History of the Disease

As far back as 1720s and 1730s, explorers in the areas of the northeast (lands later to become parts of the United States) commented on the seasonal presence of ticks and tick-associated illnesses. Later, as the areas of the northeast were slowly developed and the local forests were harvested, the ticks and disease retreated to the remaining wooded areas of the northeast. European scientists from Ireland and from England had been exposed to ticks from wooded areas in their native Europe and had described a disease similar to that described in North America. In northern Europe, this pattern of presentation had been described as far back as 1749.

In Europe, specifically in Germany and France, multiple descriptions of the disease pattern and the neurological sequelae appeared in the 1800s and in the 1900s. Again, unfortunately, no diagnosis had been made. These are often descriptions of the diseases of the day and of the region.

In 1948, Carl Lennhoff from the Karolinska Institute in Sweden made some initial observations in regards to a skin condition that seemed to be linked to a tick disease. He studied the skin lesions and the causative organisms. He and his colleagues began the initial treatments of the tick disease and its neurological sequelae with penicillin. The initial responses were favorable. However, still there was no unifying or overarching diagnosis.

Lyme Disease in the United States

Around 1975, a large cluster of young patients in the northeast, in the townships of Lyme and Old Lyme Connecticut, presented with a seemingly abnormal pattern of aches and pains. These young patients were initially misdiagnosed as having juvenile rheumatoid arthritis (JRA). When the pattern of presentation and the symptoms were later examined and noted to be atypical for JRA, a secondary evaluation was begun by a group of scientists. Drs. Snydman and Steere from the Epidemic Intelligence Service and Dr. Malawista and his team from Yale University began to intensively study the patients and their disease process. They categorized the disease as "Lyme arthritis." They also noted that the pattern of presentation and the symptoms were similar to previously described syndromes. These included meningopolyneuritis, sheep tick fever, and Bannwarth Syndrome. They found that these entities were all the same. Unfortunately, they could still not isolate the causative organism.

While Dr. Malawista and his team at Yale were working on the diagnosis and treatment of the disease, Dr. Willy Burgdorfer, a renowned Swiss parasitologist/bacteriologist, was studying a similar disease presentation at the Hamilton Lab/ Rocky Mountain Biological Laboratory. Dr. Burgdorfer was in fact studying the tick-borne disease Rocky Mountain Spotted Fever. It was there that he was presented with data from the Yale group. He had been studying many arthropod-transmitted diseases when he saw the data from the Lyme disease population. He quickly deduced the presence of hitherto unrecognized bacterial strains in the tick population causing the Lyme neuritis. He found the offending bacterium to be a spirochete. It was reasoned that the ticks might carry multiple bacteria and could co- infect patients with multiple types of organisms. With this in mind, Burgdorfer and his team quickly isolated the spirochete organisms from the ticks from the northeast. They then showed that the patients in the northeastern United States who had presented with neurological signs and the Lyme arthritis were in fact victims of the tick attacks. The spirochete bacteria were confirmed to be the causative organism, while ticks were confirmed to be the vector for the infection.

Dr. Burgdorfer published his data and his conclusions in 1982 in Science. Antibiotic treatment soon followed, and the Lyme disease, especially when identified early, became treatable.The causative (spirochete) organism was given the name *Borrelia burgdorferi* which is the name it holds today.

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