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OF THE ARKANSAS MEDICAL SOCIETY
VOL. 117 • NO. 4
OCTOBER 2020

IN THIS ISSUE

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PRSA 2019 Prism Award Finalist Printed Periodicals

by BENJAMIN THARIAN, MD, MRCP, FACP, FRACP, FASGE

Director of General and Advanced Endoscopy, Assoc. Prof of Medicine, UAMS, Little Rock, Ark.

Curing Early Cancer: Role of Advanced Endoscopy

Gastrointestinal endoscopy involves a variety of procedures to diagnose diseases of alimentary canal (esophagus, stomach, duodenum, small and large bowel) with the help of camera attached to a tube (endoscope). Advanced endoscopy includes procedures which are beyond the scope of a regular endoscopy and requires high innovation, persistence, and ability to manage complication with controlled aggression.

According to Center for Disease Control (CDC), some of the alimentary canal tumors such as esophageal cancer, colorectal cancer have higher incidence and mortality in Arkansas compared to rest of the U.S. This could be due to lack of access to endoscopy centers and decreased awareness of cancer screening procedures. However, with the availability of more gastroenterologists in the state, ability for cancer screening (colon and rectal) has been increasing. Advanced endoscopy takes one more step to access various parts of the gastrointestinal tract.

Previously, surgery was the main modality to assess, diagnose, and formulate a plan of treatment for the bile duct, gallbladder, and small bowel. However, with the availability of advanced ultra-slim cameras, ultrasounds, and imaging, the ability to access these difficult areas of the gastrointestinal tract has become possible without surgery.

Recently, I took care of a 50-year-old software engineer, a technically savvy individual who came to my clinic with some sticking sensation in his food pipe. His initial workup showed that he had a small thickening in the middle part of the esophagus. He was diagnosed with esophageal cancer, early type, restricted to the first layer (mucosa). It was a superficial cancer of the esophagus. He performed an extensive research given his internet skills and was significantly concerned about his life ahead. The news of cancer shell-shocked him. His research on the internet showed esophageal cancer had worse outcomes. He was depressed. However, with the role of advanced endoscopic procedures, cancers restricted to superficial layers (mucosa or submucosa) could potentially be targeted with advanced techniques (Endoscopic Mucosal Resection [EMR], Endoscopic Submucosal Dissection [ESD]) in centers with high endoscopic expertise. His esophageal lesions were removed and his cancer cured. He was very grateful that his cancer was treated without major surgery or chemotherapy.

Similarly, I recently evaluated a physician from out-of-state who came to UAMS to get an evaluation concerning stomach pain. He had initial workup of his stomach with endoscopy and was diagnosed with a small fat tumor (lipoma), which was benign and not cancerous. However, his symptoms were still present. He requested that I have a second look at his symptoms. We performed a repeat procedure and found that the fat tumor was unusual in its appearance; hence, an ultrasound was attached to the endoscope (Endoscopic Ultrasound [EUS]) and revealed that the benign tumor was atypical with a suspicion for cancer. We took a sample of this tumor and analyzed it microscopically, revealing a rare cancer that was then removed completely by surgery. The technique of EUS helped us to diagnose and stage the cancer in its early part, which resulted in the removal and potential cure.

With advances in the development of minute ultrasounds, we can access the bile duct, target stones in the bile duct that are difficult to remove, and access gallbladder and small bowel. Special procedures like EUS are used to take samples for microscopic analysis from any organs (nodes, tumors) surrounding the gastrointestinal tract. They could potentially be used to relieve pain (by targeting nerve supply) and jaundice (by relieving blockage) in advanced pancreatic cancer. They could be used to target early cancerous lesions, potentially curing them.

The Future Awaits

These minimally invasive procedures are used to access internal gastrointestinal tract with no skin incisions. This results in faster recovery time, no scars, reduced blood loss, and – eventually – patients walking out of the hospital on the same day after the procedure. With the advent of these new techniques – and the ability to target the esophagus, stomach, and colon cancers – we anticipate a paradigm shift in detecting tumors at an early stage and removing them, resulting in increased cure.

In the future, robotic technology with virtual reality will have a significant role in enhancing and improving these techniques. Virtual techniques, using high-tech software and state-of-the-art stimulators, have resulted in significant advances in training with minimal risk to the patient. This should help trainees early in their career to develop and enhance their visual and technical knowledge in reaching parts of the gastrointestinal tract that were never thought to be a possibility before. With the use of robotic-assisted techniques, future gastroenterologists should be able to integrate and treat with minimum discomfort to the patient.

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by S.D. TAGGART, MD

The Deadliest Month: The Spanish Flu of 1918 in Rural Arkansas, A Historical Perspective

In 1917, a novel virus emerged in northern China and within a year, a pandemic engulfed the world. In 1918, the world population was 1.8 billion, between 20 to 50 million people died as a direct result of the Spanish Flu. Estimates vary widely as to the mortality rate associated with that round of the Flu, but it is generally agreed that 3-5% of those infected with the virus died.

On December 31, 2019, the World Health Organization was alerted to a novel virus in the coronavirus family similar to SARS, MERS, and the common cold. On March 11th, 2020, the head of the World Health Organization declared the Covid-19 outbreak a worldwide pandemic. The world population is now at 7.8 billion people. Since we really don't have any idea as to how widely it has spread it is far to early to even comment on mortality rates and potential impact.

This article is an attempt to put in perspective what the people of rural Arkansas faced 100 years ago, what tools that had to deal with the pandemic, and how they responded.

Benton Courier, Oct. 10th, 1918

Benton and Vicinity is in Grip of an Epidemic of Spanish Influenza. Public Places Closed. No epidemic has had its grip on this country and county as has the Spanish influenza, which, during the past week has placed itself in almost every home… it has been announced that many churches will have no services Sunday…The American Bauxite Company had to shut down its plant on Tuesday on account of so many men being sick, and several have died in that section of the county."

Gurdon Times, Oct. 19th, 1918

" A gloom of sadness has been casted over our town and community this week by the several deaths caused by pneumonia as result of the influenza so persistent here and throughout the whole country."

Stuttgart Daily Leader Oct 17th, 1918

" The present epidemic in its death dealing wake during the past week claimed several of our citizens and friends, in its sweep, neither the old or the young are spared…"

During the Yellow Fever outbreak of 1888, shot gun quarantine was a popular method of virus containment.

During the year of 1918, good news was hard to come by. In the spring, the Germans went on the offensive and the Allied forces were battered. Until the late summer of 1918, World War I was still in doubt. New recruits from across the country continued to be drafted. Daily papers in small towns across Arkansas carried headlined articles about the course of the war. The Red Cross aggressively campaigned for supplies and contributions. Letters to the editor and personal correspondence admonished people not to be a "slacker"; that is, not supporting the war effort.

The Spanish Flu struck the state in the last few days of September in 1918 and for four weeks held them in a literal death grip. Scientists speculate that the influenza began first attacking human beings with the advent of the Agricultural Revolution (circa 7000 BCE) and the domestication of pigs and chickens. The first documented occurrence of the flu was 2400 years ago recorded by Herodotus, a Greek historian. As human populations increased, and people began to congregate in larger numbers, crowd diseases like the flu began to occur in unpredictable cycles. When DeSoto and his fellow travelers made their way into the new world of the Western Hemisphere, they brought with them several of the crowd diseases such as smallpox, measles and flu. These illnesses found in the indigenous populations a virgin people who had no herd immunity. For several centuries, there were dramatic epidemics, killing off large parts of the resident populations of North, Middle, and South America.

The Italians coined the phrase Influenza in the 15th century and the French referred to the same disease as La Grippe. In the 19th century, local newspapers such as the Arkansas Gazette were replete with references to La Grippe. In 1889-1890, an influenza labeled the Russian Flu made its way through Europe and crossed the Atlantic. Even though Arkansas was 98% rural, the flu pandemic of that year killed 365 people in Arkansas before it receded.

Before we begin to discuss the flu of 1918, it is important to remember that this was not the first time the state of Arkansas had

been threatened by violent deadly diseases. Since 1804, the region had dealt with endemic malaria and typhoid fever. There had been recurring threats of smallpox, cholera, and yellow fever during the 19th century.These illnesses would appear unannounced and wreak havoc on parts of the populations. Prior to the 1860s, most of these illnesses were grouped together into fever illnesses; the population and their physicians had very little idea as to the cause or how to deal with them. With the advent of the germ theory in the 1860's, this began to change. Even before the germ theory, quarantine was known to be a useful tool. "Shot Gun" quarantine was an unofficial but useful tool to prevent the spread of these disease processes.[1] Armed groups of men would stand guard at the entrance to a town and you were not allowed to enter if they didn't know you.

By the late 1870s, state-wide temporary Boards of Health were created to help establish more widespread quarantine against yellow fever. In 1913, the state of Arkansas created a permanent Board of Health, that eventually morphed into the Arkansas Department of Health. Between 1913 and 1918, the head of the State Board of Health, Dr. Charles Garrison, began to establish the rules and regulations that would be used to protect the public's health. A list of reportable diseases was established; interestingly, influenza was not included on this list.

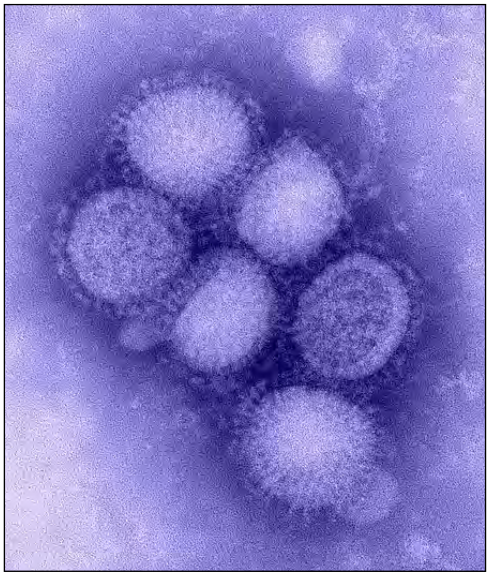
The new State Board of Health was active in remote counties before the epidemic of 1918, but most of their efforts were aimed at smallpox immunization. Dr. J. W. Melton of Solcomb and Dr. J.M. Phillips were the unofficial county health officers and as early as 1916-1917, they were instrumental in helping to carry out the State Board of Health mandated smallpox immunization policy for school attendance.

Camp Pike Hospital

Another change in rural Arkansas that impacted the spread of any epidemic disease was the advent of the railroads. In the post-Civil War era, the rail systems began to create a spiderweb of train tracks across the state making even the most remote places in rural Arkansas accessible. With an illness like the flu that may have an incubation period of two or three days, this modern convivence became a two-edged sword. An asymptomatic person could board a train in Little Rock and hours later disembark in Paris, Arkansas; soon, the flu had a foothold in a new town. Early in the epidemic, small towns as widespread as Carlisle, Newport, Wilmot, Stuttgart, Waldon, Subiaco, Paris, Hunter, and Dermott reported significant caseloads of flu.[2]

The Flu Arrives

The first area of the state to be affected by this wave of the flu was Camp Pike, located northwest of North Little Rock. Camp Pike was established in 1917, and by September of 1918 there were 54,000 soldiers being trained at the post; making it the second largest town in Arkansas.[3,4] Young soldiers crammed into over-crowded barracks provided a tinderbox for the rapid spread of the flu. By late-September, there were thousands of cases of flu on the post, overwhelming the base hospital.[5] Despite, the dramatic rates of infections among the troops and high death rates, the U.S. Surgeon-General, Rupert Blue, said: "There is no cause for alarm if precautions are observed."[6]


Electron microscope of H1N1 virus.

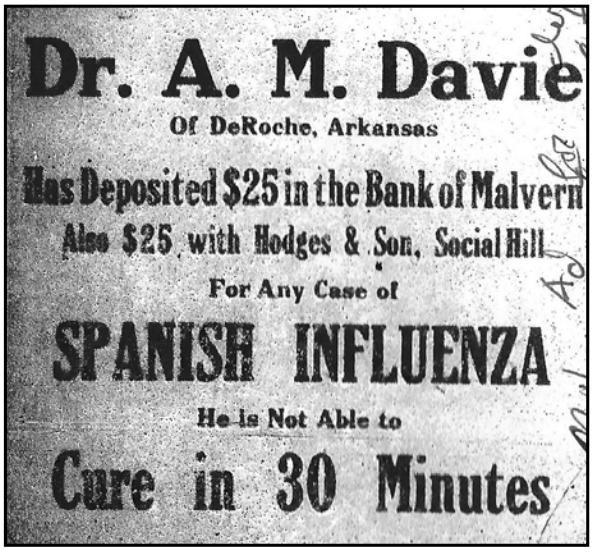
It is no surprise that in mid-September, Dr. James C. Geiger, the U.S. Public Health Officer in charge at Camp Pike in North Lit-

tle Rock echoed his boss's sentiments "it is just simple, plain old la grippe."[7] As late as October 4, when the civilian cases of flu began to mushroom in the central Arkansas area, both Dr. Geiger and Dr. Charles Garrison of the Board of Health continued to attempt to prevent panic in the general population with calming statements.

Drift vs Shift

The flu virus has a heightened ability to change the face it shows to the human population. On the surface of this virus are two common type of proteins (designated as N and H) that provide a fingerprint that the human immune system can recognize and produce antibodies. Once infected with the virus, the human system produces immunologic memory, protecting against repeat infection or at least moderating the effect of the virus. The proteins on the surface of the virus have the ability to undergo change so that the human host can't recognize it. The terms viral drift and shift are used to describe how this change occurs. In most cyclic epidemics of influenza, the virus undergoes a viral drift; meaning, that it changes, but not much. In the event of a viral shift, the change is enough that most humans can't recognize the virus and the epidemic is much worse. The Flu of 1918, represented a dramatic viral shift and the result was a worldwide pandemic. There is speculation that there were similarities between the Russian Flu in 1889 and the Spanish Flu of 1918. Most flu epidemics strike the very young and those over the age of 65. In 1918, the flu had a much larger impact on young adults than the older population. One explanation for this change would have been that those in the over 65 age group were exposed in 1889 and therefore had a degree of immunologic memory.

A second element that intensified the impact of this flu on the younger humans has to do with a vigorous immune system. Young adults have a stronger immune system, the virus attack is met with a strong overwhelming response; this is now known as a "cytokine" storm.[8] It is thought that this response may have been responsible for the dramatic increase in pneumonia, central system infections, and death observed in otherwise healthy young people during the 1918 pandemic.



Advertisement for a cure for the Spanish Flu from Dr. Davie that appeared in a 1918 edition of the Malvern Metore.

What tools did they have to fight the flu?

The Chinese have a saying; "when hope is gone, the ultimate sanity is grasping at straws." Often, families relied on folk medicine and family remedies. If home remedies failed, they called the doctor. The physician's armamentarium was quite limited. Three medicines served as the most commonly used: calomel (mercury), quinine, and laudanum (opium). These medicines had a long history in treating fevers of other origins, especially calomel and quinine. In hindsight, the mercury made the influenza worse. Quinine, commonly used, for malaria and other fever illnesses, may have modulated the fever and opium had an impact on the muscle aches and headaches associated with the flu.

Even in normal times, patent medicine advertising provided a large part of the revenues for newspapers; during epidemics this increased. Tanlac is a good example of a medicine that was advertised in obvious drug ads in local papers like the Benton Courier. In addition, articles were published that appeared to be legitimate articles, quoting U.S. Government authorities. Reading through the articles, it becomes clear that the purpose of the articles was to sell a drug like Tanlac.[9] An interesting aside, is the repetition of the same article in many small-town newspapers across the state.

After the epidemic in 1889-1890, Dr. Richard Pfeiffer identified

what he thought was the cause of flu. The bacteria he identified was incorrectly named Haemophilis influenza. He immediately began work on an anti-serum to combat the illness. The anti-sera had been available for at least a decade to Arkansas physicians. During the 1918 epidemic, physicians in Arkansas were encouraged to give the anti-sera to their patients. In Searcy (White County), Dr. John Jones publicly encouraged his fellow physicians to give the serum to their patients.[10] Despite some placebo effect, the anti-sera was of no value. It was not until the 1930's that the influenza virus was identified and the belief in the role of H. Flu in influenza completely disappeared.

As with any other catastrophe there are always those willing to take advantage of the gullible. On October 26, 1918, a Dr. A.M. Davie of DeRoche, Arkansas (Hot Spring county) posted an advertisement in the Malvern paper that read: "Have deposited $25 in the Bank of Malvern and $25 with Hodges and Son in Social Hill. For any case of Spanish Influenza, I am not able to cure in 30 minutes."[11]

In October of 1919, the Arkansas Medical Society Journal published a symposium detailing the experience of a number of physicians from across the state. Dr. H. N. Street of Lonoke (Prairie County) related: "this was the worst epidemic form of any disease I have encountered in thirty years of practice." Dr. A. G. Henderson of Imboden (Lawrence County) "I treated 510 cases –eight of those developed pneumonia; eight died." Dr. C.W. Dixon of Douglas (Lincoln County) "I live in the backwoods of Lincoln County. During the epidemic, I had quite a territory to cover. I could cover half in one day and the other half the next." The symposium detailed the various different approaches to treating the flu and, in the end, the conclusion was that bed rest, nourishment and isolation were the best tools in the doctor's bag.[12]

Quarantine and avoiding contact with sick people were the only effective tools available in 1918. Lip service was given to wearing flu masks, if you were coughing and sneezing, as well as hand washing but the most important tool was quarantine and avoidance. Luckily, most of the residents of rural Arkansas had a bit of a protective barrier in their normal isolation. In the case of the Spanish flu, this protective shield was only modestly successful and for the most part only delayed many of the smaller communities being affected.

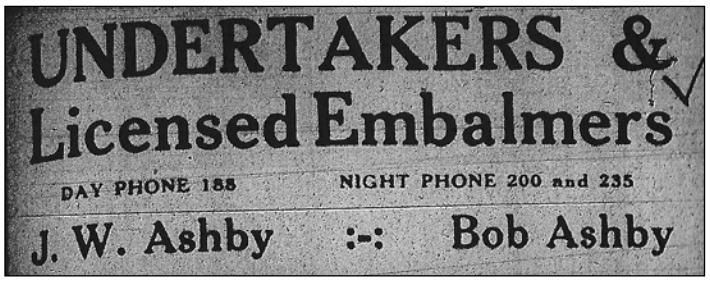
On October 4, 1918, Dr. Geiger from the Public Health Service and Dr. Charles Garrison from the Arkansas Board of Health, were pronouncing the situation well-in-hand but at the same time encouraging voluntary isolation of flu victims.[13] By October 5, there had been 800 new cases of flu in the Little Rock and North Little Rock.[14] Multiple small towns from Dermott to Paris were reporting significant cases loads. On October 7, any pretense of control over the lethal wildfire was abandoned and Dr. Garrison, with the blessings of the Governor, declared a mandatory statewide quarantine.[15]

A group of residents from Claradeon, Arkansas, wearing masks.

Many of the old records no longer exist as to how the quarantine was carried out. But in general, Dr. Garrison of the Board of Health would contact the County Health Doctor and the County Judge. They would then enlist the Sheriff who was charged with enforcing the quarantine. The quarantine prohibited public gatherings such as schools, churches and encouraged businesses to open on a limited basis. Children under the age of eighteen were not allowed on the street; it is clear the quarantine was honored in most counties but not all. In Scott, Arkansas, the local authorities failed to stop a circus from performing.[16] Dr. Garrison sent an officer and had the owner of the circus arrested. The Mayor of Booneville chose to ignore the quarantine. Dr. Garrison telegraphed the Mayor and threatened to seal of the town unless the he ordered the quarantine.[17]

Within days, the small towns of Arkansas became ghost towns. The streets were quiet, many stores were closed, and churches and school grounds stood emp-

ty.[18] In many cases, businesses and plants did not open because they didn't have enough healthy employees. The American Bauxite Mining Company in Saline County was closed for at least one day.[19] The Benton Courier had trouble producing a newspaper because of employees off sick with the flu.[20] Physicians and what nurses they could muster went about their calls. There is a report of at least three physicians dying of the flu in Crawford and Sebastian counties in northwest Arkansas.[21] There are numerous accounts of multiple members of households being infected and no one to care for them. Caskets were often makeshift affairs thrown together by able-bodied friends. There are multiple reports of areas in rural Arkansas where there was no one in the family well enough to attend funerals.[22] The car, still a novelty, on the dirt roads of Arkansas, was used to transport physicians on their calls. Dr. Christopher Columbus Gray in Independence County recruited his sons to drive from call to call so he could sleep in between visits.[23]



Ashby's Funeral Home ad that appeared in a 1918 edition of the Benton Courier.

By all appearances, the quarantine worked because by the third week in October the report of new cases had begun to subside, and talks began about lifting the quarantine. Remote areas like Pettigrew in Madison County had completely escaped the epidemic and thought they were in the clear. On October 25, 1918, they had a public gathering at the school and in the space of two days, everyone in town had the flu. Despite this exception, most areas of the state were showing a sharp drop in new cases of the flu.[24] The TB Sanitorium, south of Booneville, instituted a strict quarantine and had no flu at all.[25] On October 26, a partial lifting of the quarantine was beginning for areas of the state hit earlier.[26] By November 4, 1918, the statewide quarantine was lifted but it was left up to individual counties based on their situation.[27]

What were the results of the Spanish Flu Pandemic of 1918?

It should be noted that health statistics in Arkansas are notoriously inaccurate until the 1940s. There is no way to know for absolute certain how many died in Saline County but what data that is available make it clear that the rural counties suffered like the rest of the state. Rough death totals suggest that 20 to 50 million died around the world, 500,000 died in the United States and 7000 people in the state of Arkansas died in this wave and another wave that occurred in the winter of 1919.[28] The Funeral Registry at Ashby's Funeral Home in Benton is one of those sad snapshots that we do have access to. For the years between 1917 and 1919, the funeral home averaged seven to fifteen funerals per month with the exception of the month of October 1918 when they performed 71 funerals. Almost as quickly as the epidemic came, it was gone, and, in November of 1918, the funeral home was back to its normal rate of deaths and funerals.[29]

The short-term economic consequences of the Spanish Flu were obvious. Merchants in Little Rock recorded drops of 40-70% in their business. Clearly, this was equally true throughout the state. A study done in 2007 on the long-term economic impact suggests that those born during the time of the epidemic had reduced educational attainment, higher rates of physical disability, and lower incomes. Because of its propensity to strike young adults, the flu had a lingering effect on business and social life well into the 1920s.[30]

Why was the Spanish flu forgotten?

There are any number of theories as to why we seemed to have forgotten this dreadful month 100 years ago.

Even though psychiatry has moved on from Freudian theory, one of his ideas still holds sway and that is the idea of repression. Simply stated, human beings and society in general tend to banish painful memories from their consciousness. This was a painful time, and everyone was faced with this dreadful specter of a painful death not unlike the Plague of the 14th century.

The epidemic was paired with the ending of WWI, November 11, 1918. "All Clear on the Western Front" signaled the end of the "War to End All Wars" and for most people the epidemic. These two painful memoires concluded on almost the same day.

Could it happen again?

Epidemiologists seem in agreement that another deadly pandemic like that of 1918, is not a matter of IF but of WHEN. In 1918, there were 1.8 billion people worldwide and now there are 7.8 billion people on our lonely, little planet. There are more of us to get infected and spread the infection. Unlike 100 years ago, we tend to live in cities and towns. But still today in many parts of the world, sanitation is lacking, and these areas act as breeding grounds for new viral flu strains. Probably more important, we now travel at rapid rates from one part of the world to another in less than two days. Much like the train in the early 20th century, jets act as a two-edged sword, providing us with access to the rest of the world and transmitting disease just as quickly.[31]

This article is part of an ongoing series from Dr. Sam Taggart. For a complete set of referencess, email ams@arkmed.org.

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by LESLI BIRKHEAD

Memories of T.E.D. Birkhead

I never knew him, but I've heard about my great grandfather, T.E.D. Birkhead, for years from his son, my grandfather, James Birkhead Sr., and my dad, James Birkhead Jr.

T.E.D. was a country doctor in poor, rural areas of Arkansas. While it's not known exactly where he practiced medicine, he lived near Lonoke.

T.E.D. practiced medicine in the late 1800s and early 1900s. He rode in a horse and buggy or walked to patients' homes. Of course, he carried an old doctors bag. Most of his patients were not able to pay him. Some repaid him by giving him some of their livestock. Others paid what they could, and many did not pay at all. Payment was not as important to my great grandfather as treating the ill and injured.

During his practice years, the smallpox epidemic was in full swing. There was no cure or treatment. He would visit several homes to help his patients feel better. He treated all ages, including older adults and very young children. Some of the smallpox victims survived. The others, he could only try to make comfortable or get there in time to close their eyes as they had passed away prior to or during his arrival. Though T.E.D. had a wife and children of his own, he would still make house calls. One of his children often accompanied him on his house calls. In 1903, he lost some of his children and his wife from the smallpox virus. Most likely, he brought the virus home from visiting the sick and dying. However, four daughters survived and pledged their lives to the medical field. I believe three were nurses.

T.E.D remarried and, in 1911, my grandfather was born. Two years later, T.E.D. passed away. It is not known what happened to him. He was buried in Arkansas and a Woodsmen of the World monument is placed at his gravesite.

Due to the current pandemic of the coronavirus (Covid-19), I decided to write a tribute to my great grandfather, T.E.D. Birkhead. I've only written poetry for two years. My book, "Black Mountain, A Collection of Poems," was published in January 2020. The first poem I have attached here, "The Doctors Bag," is the tribute poem to my great grandfather. The second poem, "A Viral Story," was written for everyone affected by this coronavirus, most especially medical personnel, first responders, and those helping in any way.

A Viral Story

They are the front-line fighters
From physicians and nurses to aides
Everyday more and more Covid-19 patients
So many that in the halls they laid

In a bed if lucky
On a ventilator when critical
So many workers behind the scenes and curtains
They're the true heroes for one and all

Pushed beyond human limits
And driven to near insanity
So tired they cannot think
But they continue for all humanity

Their patients are the entire family
Holding a phone or tablet for them to see
Unable to touch their loved one
That is how it has to be

The hearts of those frontline workers
Continued to break every shift
However, when one patient survives
A party sendoff gave them a minute's lift

Behind the smiles and cheers
They cried inside yet tears appeared
Flatliners waiting as they turned around
They'd go back in and don their gear

"How long will this continue
I cannot take it anymore"
But they put on their PPEs
To see a new patient behind the doors

And all of those who helped
From bringing food to shouting, 'Thank you'
They're as much a part of the story
As well as those staying at home too

For all of those involved somehow
Let their anguish be released
Bring focus to their hearts and minds
So, one day again they can live in peace

There's no cure for this virus
So, there's no ending to this rhyme
"God keep them strong and keep them going,
Bless them all til a cure they find"

The Doctors Bag

He traveled to the homes of many
Who were injured or not well
On that old country dirt road
Through the snow as it fell

Beyond the hills and valleys
With his only shoes worn out
His doctor's bag in hand
That's what life was all about

Most had no way of paying
Many gave a grateful 'thank you'
Some would pay with a few nickels
Others paid with a chicken or two

It wasn't the money he wanted
He just wanted to cure their ills
It was all about their feeling better
Not about how they'd pay his bill

He'd pull from his doctor's bag
And listen to the heart and lungs
Sometimes he'd just close their eyes
He treated them all, old and young

Without knowing what he was walking into
And smallpox so contagious
There was no cure or treatment
His house calls so courageous

A disease so easily transmitted
It was taking many lives
Eventually it came home
And took his children and wife

Arriving with his horse and buggy
He'd walk in and take off his hat
It was all about helping them get well
That was the man with the doctor's bag

by RASHMI BALASUBRAMANYA[1]; ACHALA DONURU MBBS, FRCR[2]; VINAY KANDULA, MBBS, FRCR[3]; JOANNA CHAN MD[2]; MARUTI KUMARAN, MD[4]; MAANSI PAREKH, MBBS, DNB[1]

1. Department of Radiology, Thomas Jefferson University Hospitals, 132 S 10th St, 1079 Main Building, Philadelphia, PA
2. Department of Pathology, Thomas Jefferson University Hospitals, 132 S 10th St, Suite 285 Main Building, Philadelphia, PA
3. Department of Medical Imaging, 1600 Rockland Rd, Wilmington, DE
4. Department of Radiology, Temple University Hospitals, 3401 N Broad St, Philadelphia, PA

Disseminated Aspergillus of the Heart: A Case Report of Intracavitary, Myocardial, and Valvular Fungal Involvement

Introduction

Invasive aspergillosis is a life-threatening, opportunistic infection that occurs most often in immunocompromised patients. Myocardial abscess is a rare and potentially fatal condition. Metastatic myocardial abscess, in the setting of infective endocarditis, has been infrequently reported. Herein, we report a case of disseminated aspergillus infection with vertebral body osteomyelitis, renal aspergillosis, myocardial abscess, and endocarditis in a liver-transplant patient. A high index of clinical suspicion is required to make a prompt diagnosis. Final diagnosis may need a combination of serological tests and imaging, particularly in instances where biopsy is non-yielding or cannot be performed.

Case Description

A 64-year-old female underwent liver transplantation for fulminant Hepatitis A liver failure six months earlier. She presented to the hospital with pain radiating down her right lower extremity. She complained of chronic lumbosacral back pain that had persisted ever since her transplant, however progressively worsened. She denied any numbness, tingling, or loss of sensation. MRI of the spine was ordered, which revealed osteomyelitis and discitis of L2/3 with an epidural phlegmon (Figure 1). Incidentally, she was found to have a new hypodense right kidney lesion (Figure 2). Biopsy of the epidural phlegmon and right kidney lesion revealed aspergillus (Figure 3). Serum aspergillus galactomannan antigen was positive at that time. She was started on Amphotericin B and eventually transitioned to Isavuconazole as her QT prolongation on EKG did not allow for Voriconazole. Chest CT performed for new onset hypoxia revealed a filling defect in the left atrium (Figure 4) and an additional low density myocardial mass arising from the free wall of the left ventricle felt to represent a myocardi-

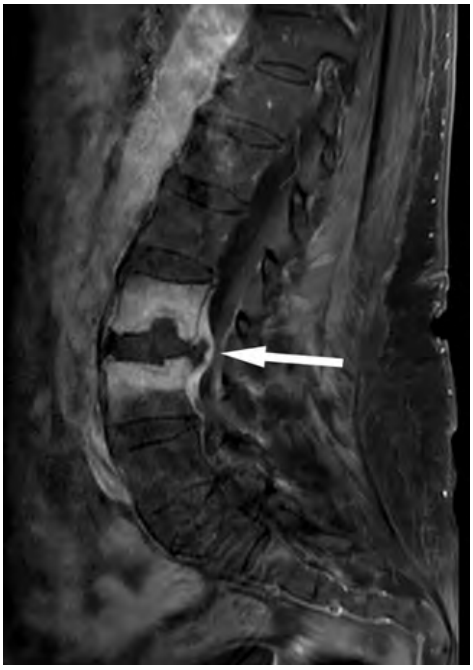

Figure 1: MRI lumbar spine post contrast sagittal image demonstrates avid enhancement involving the L3 and L4 vertebral bodies with associated endplate deformities

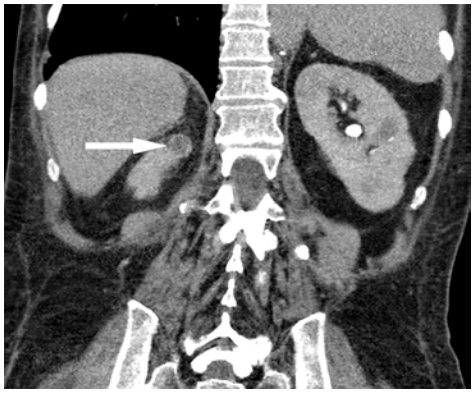

Figure 2: Rim-enhancing lesion in the right upper renal pole concerning for an abscess (arrow).

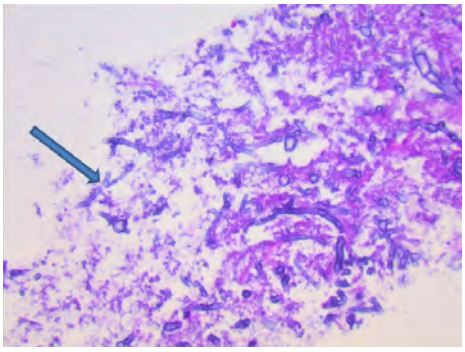

Figure 3: Biopsy of the right renal lesion. Fungal elements with 45-degree branching consistent with aspergillus species. H&E 400x

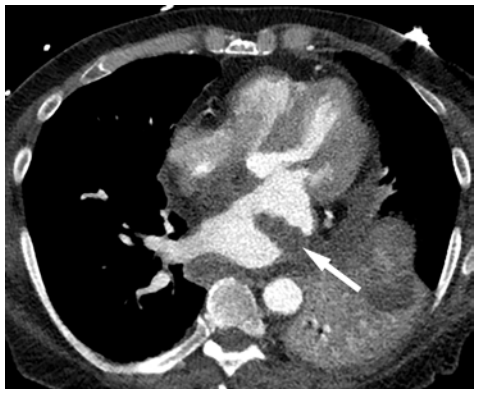

Figure 3: Contrast-enhanced image from a Chest CT demonstrates a lobulated filling defect in the left atrium, along the ligament of Marshall (warfarin ridge).

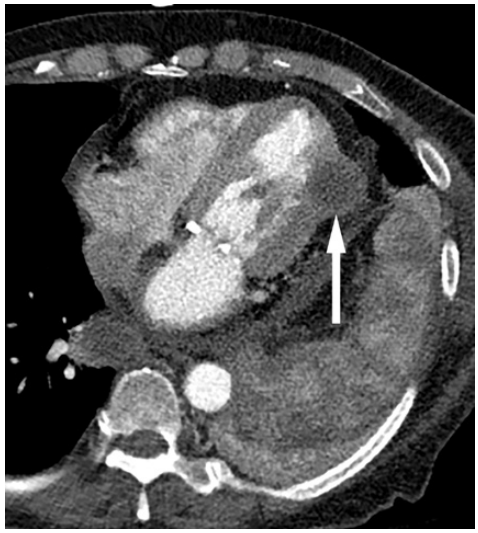

Figure 5: Contrast-enhanced image from a Chest CT demonstrates a hypodense lesion arising from the left ventricular lateral wall myocardium. Low attenuation in the center suggests necrosis.

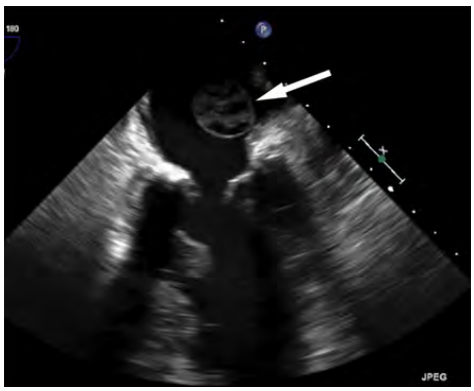

Figure 6: Round, mobile mass with septations attached to the lateral wall of the left atrium (arrow).

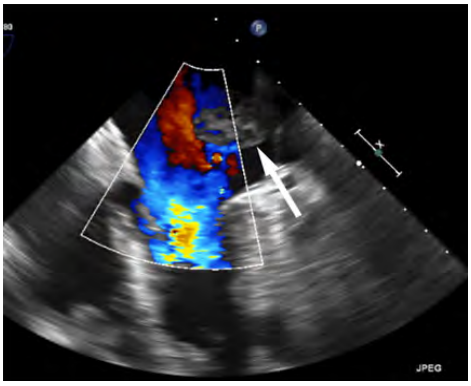

Figure 7: Round, mobile mass with septations attached to the lateral wall of the left atrium (arrow) without internal vascularity.

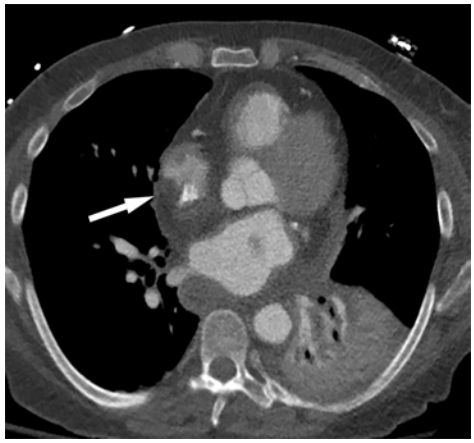

Figure 8: Filling defect in the right atrium (arrow).

al abscess invading into the epicardium, and possibly the pericardium (Figure 5). Transesophageal echocardiogram was performed for further characterization and revealed a mobile mass with septations in the left atrium, atypical for a thrombus (Figure 6, 7). Hence, the constellation of findings was felt to be most consistent with cardiac involvement of aspergillus. Repeat imaging showed an increase in size in these lesions and a new right atrial lesion (Figure 8) despite therapy with Isavuconazole. The cardiothoracic surgical team was consulted; however, given prognosis and extensive nature and high risk of surgical intervention, no biopsy or surgical intervention of cardiac lesions was performed.

Discussion

Invasive aspergillosis is a serious opportunistic infection that occurs in a variety of immunocompromised patients such as those receiving chemotherapy for hematological malignancies or organ-transplant patients, and it has high morbidity and mortality rates. Its incidence has been estimated at around 10% - 14%, and the mortality rates may be as high as 27% - 60%, despite antifungal therapy.[1,2]

Cardiac invasive fungal infection is difficult to prove premortem and has an especially high mortality rate. In the case of Aspergillus endocarditis, the mortality rate reaches 96% if the patient is treated by medication alone, and 68% even if surgical resection is performed.[3-6] Aspergillus accounts for 24–28% of all cases of fungal endocarditis.[7] There are four different types of manifestations of cardiac aspergillosis described in literature.[8-10] Three of these types are listed as intracavitary mass, myocardial abscess, and endocarditis; all of these were seen in our case. This is a very rare scenario and, to the best of our knowledge, so many different manifestations of cardiac aspergillosis in a single patient has only once been previously reported in literature.[11] The fourth variety includes embolization of aspergillus into the coronary arteries, which can lead to a myocardial infarction. The clinical diagnosis of Aspergillus endocarditis is usually challenging due to its non-specific presentations, and it is rarely isolated from blood cultures. A high level of clinical suspicion is needed for early diagnosis.[12]

References

1. P. Eggimann, J. C. Chevrolet, M. Starobinski, et al., "Primary Invasive Aspergillosis of the Digestive Tract: Report of Two Cases and Review of the Literature," Infection, Vol. 34, No. 6, 2006, pp. 333-338.
2. L. Pagano, M. Caira, A. Candoni, et al., "Invasive Aspergillosis in Patients with Acute Myeloid Leukemia: A SEIFEM-2008 Registry Study," Haematologica, Vol. 95, No. 4, 2010, pp. 644-650.
3. H. Kume, T. Yamazaki, M. Abe, H. Tanuma, M. Okudaira and I. Okayasu, "Increase in Aspergillosis and Severe Mycotic Infection in Patients with Leukemia and MDS: Comparison of the Data from the Annual of the Pathological Autopsy Cases in Japan in 1989, 1993 and 1997," Pathology International, Vol. 53, No. 11, 2003, pp. 744-750. http://dx.doi.org/10.1046/j.1440-1827.2003.01548.x
4. P. Nenoff, C. Kliem, M. Mittag, L. C. Horn, D. Niederwieser and U. F. Haustein, "Secondary Cutaneous Aspergillosis Due to Aspergillus Flavus in an Acute Myeloid Leukaemia Patient Following Stem Cell Transplantation," European Journal of Dermatology, Vol. 12, No. 1, 2002, pp. 93-98.
5. T. Yamazaki, H. Kume, S. Murase, E. Yamashita and M. Arisawa, "Epidemiology of Visceral Mycoses: Analysis of Data in Annual of the Pathological Autopsy Cases in Japan," Journal of Clinical Microbiology, Vol. 37, No. 6, 1999, pp. 1732-1738.
6. A. S. Kalokhe, N. Rouphael, M. F. El Chami, K. A. Workowski, G. Ganesh and J. T. Jacob, "Aspergillus Endocarditis: A Review of the Literature," International Journal of Infectious Diseases, Vol. 14, No. 12, 2010, pp. e1040-e1047.
7. Meshaal MS, Labib D, Said K, Hosny M, Hassan M, Abd Al Aziz S, et al. Aspergillus endocarditis: Diagnostic criteria and predictors of outcome, A retrospective cohort study. PLoS One 2018;13:e0201459.
8. GP Bodey , S Vartivarian. Aspergillosis. Eur J Clin Microbiol Infect Dis. 1989; 8:413–437.
9. P Vaideeswar. Aspergillus pancarditis manifesting as hospital-acquired infection: report of two cases and review of literature. Cardiovasc Pathol. 2010; 19:e253–e257.

For more references, email ams@arkmed.org.

by J. CORBIN NORTON; NICKOLAS D. SCHERZER, MD; JEFFREY T. ALLISTON, MD; DENNIS W. JACKS, MD; DOUGLAS E. CLAYBROOK,MD; DAVID A. LUPO, MD

Department of Urology, UAMS, Little Rock, Ark.

Villous Adenoma of the Prostatic Urethra: A Rare Tumor in a Rare Location

Introduction

While villous adenomas of the gastrointestinal tract are common place, their location within the urinary tract is exceedingly rare. To date, only a handful of case series and reports have been published. In addition, most reported cases involve either the urinary bladder or urachus, while villous adenoma of the urethra is even less common. As these lesions' malignant potential is still not fully understood, we present an additional case of villous adenoma of the prostatic urethra to draw further attention to this scarcely seen lesion.

Case Report

The patient that this report will describe is a 75-year-old caucasian male who presented with obstructive lower urinary tract symptoms that had progressively worsened over the course of a year. Urinalysis was negative for hematuria and positive for bacteria, white blood cells, and leukocyte esterase. He was being treated for a urinary tract infection with macrolide antibiotics. He was initially seen a few months earlier for similar symptoms. Cystoscopy at that time showed a papillary tumor in the prostatic urethra at the level of the verumontanum, with an exudative film extending to the bladder neck. Repeat cystoscopy revealed a papillary lesion that occluded the entire prostatic urethra. The patient underwent a transurethral resection of the prostatic urethral tumor extending from the bladder neck to the verumontanum, resulting in an open channel. Direct visualization of the bladder did not reveal any other co-pathologies.The prostatic curettings were evacuated and sent for pathology. Specimens consisted of tan-pink soft tissue measuring 9.5cm x 6.8cm in aggregate. Microscopic examination was consistent for villous adenoma with no evidence of high-grade dysplasia or invasive carcinoma. CT of the chest, abdomen, and pelvis following the procedure showed no evidence of malignancy or metastatic disease. The patient was diagnosed with villous adenoma of the prostatic urethra. At clinical follow-up, patient reports no complications and improved lower urinary tract symptoms. He will be followed with a cystoscopy three months postoperatively to monitor for recurrence.

Discussion

Villous adenomas are benign glandular neoplasms more commonly seen in the gastrointestinal tract. While rare, they can also occur anywhere along the urinary tract, with cases most commonly reported in the bladder, followed by the urachus, urethra, and ureter.[1-3] This case illustrates a 75-year-old white male with a villous adenoma of the prostatic urethra. This is a rare location with isolated reported cases and two case series reported within the literature. Since 1981, there have only been two other cases of villous adenoma of the urinary tract at our institution, with neither being recent enough for records to review.

Villous adenomas are more commonly found in men who range in ages from 53 to 93, with an average age of 69.6 years[2]. Patients typically present with hematuria, irritative voiding symptoms and sometimes mucosuria[1,2] Our patient did not present with hematuria but instead had obstructive urinary tract symptoms with urgency, increased frequency, and difficulty voiding.

There is still debate regarding the exact pathogenesis of villous adenomas within the urinary tract, but generally there are two proposed mechanisms. The first discussed by Atik et al., describes the shared embryologic origin of the rectum and urinary bladder. They both are derived from the cloaca, which is an endoderm lined portion of the terminal hindgut. During embryogenesis, the urorectal septum divides the cloaca dorsally into the rectum and anal canal, and ventrally into the bladder and urogenital tract. A potential defect in this process may occur, leaving behind cloacal remnants that could later develop into a glandular epithelial neoplasm[4]. The second proposed mechanism suggests a chronic irritation-metaplasia-dysplasia-carcinoma sequence. Irritation in the form of infection, chemical injury, or calculi can damage urothelial stem cells resulting in glandular metaplasia[5]. This theory is supported by the presence of neutral mucins, acidic sulphomucins, and sialomucins within the villous adenomas and in cystitis glandularis[6] as well as similar genetic compositions between villous adenoma and dysplastic regions of flat metaplastic mucosa.[5].

Villous adenomas of the urinary tract share the same histologic features as their colonic counterparts. They are characterized by papillary projections lined by pseudostratified columnar epithelium with mucin-producing goblet cells. Epithelial cells often display nuclear atypia and mild dysplastic changes.[3] Unsurprisingly, immunohistochemistry between villous adenomas of the colon and urinary tract share similarities in that they are strongly positive for cytokeratin 20 (CK20) and carcinoembryonic antigen (CEA) and are most often negative for epithelial membrane antigen (EMA).[1,3,4,7] Cytokeratin 7 (CK7) is positive in 50%-56% of villous adenomas of the urinary tract, which helps differentiate it from intestinal villous ade-

Table 1

Villous Adenoma of the Urinary Tract Features	
Average Age	69.6 years
Presenting Symptoms	Hematuria, mucosuria, irritative voiding symptoms, obstructive urinary symptoms
Pathogenesis	Two proposed mechanisms: 1. Shared embryologic origin of rectum and urogenital tract, with possible cloacal remnants left behind leading to a glandular neoplasm. 2. Chronic irritation-metaplasia-dysplasia-carcinoma sequence.
Distinguishing Histologic Features	Positive for CK20, CEA, and CK7 (differentiates urogenital from intestinal villous adenomas)
Treatment	Surgical resection

nomas.[1,8] Immunohistochemistry was not performed in this case.

Studies have shown that villous adenomas can coexist or potentially transform into malignant tumors such as adenocarcinoma in situ, invasive adenocarcinomas, and urothelial carcinomas.[1, 2, 9, 10] Cases of pure villous adenomas usually have good prognosis with surgical resection considered curative. Patients who present with co-existent invasive carcinomas may require more aggressive treatments and have a higher risk for recurrence.[1,2] A case series by Seibel et al. had 18 reported cases of villous adenoma, three (16.7%) of which involved the prostatic urethra. All three were associated with coexistent adenocarcinoma, and one of which developed local recurrence.[2] Another case series by Cheng et al. examined 23 cases of urinary tract villous adenoma, of which only two (8.7%) involved the urethra. One of these two cases developed multiple recurrence over a six-year follow-up period[1]. In addition, a case report by Zarineh et al., described an isolated villous adenoma of a female urethra that displayed features of high-grade dysplasia that reoccurred following transurethral resection.[7] These findings suggest that villous adenomas of the urethra may have higher rates of co-existing malignancies and subsequently higher rates of recurrence as compared to villous adenomas located elsewhere along the urinary tract. In our case, pathology did not show any coexistent carcinomas and the patient remains disease free at latest follow-up.

Conclusion

In conclusion, villous adenomas of the urinary tract are rare lesions that share many histological similarities with their colonic counterparts. They have been associated with coexistent malignancies and possible malignant transformation. For this reason, it is important for the clinician to identify this uncommon lesion, excise it completely, and thoroughly sample the urinary tract for a possible co-malignancy. Close follow-up of these patients is also imperative.

References

1. Cheng, L.M.D.M., Rodolfo M.D.; Bostwick, David G. M.D., Villous Adenoma of the Urinary Tract: A Report of 23 Cases, Including 8 with Coexistent Adenocarcinoma. The American Journal of Surgical Pathology, 1999. 23(7): p. 764.
2. Seibel, J.L., et al., Villous adenoma of the urinary tract: a lesion frequently associated with malignancy. Hum Pathol, 2002. 33(2): p. 236-41.
3. Sung, W., et al., Villous adenoma of the urinary bladder. Int J Urol, 2008. 15(6): p. 551-553.
4. Atik, E., et al., Villous adenoma of the urinary bladder: rare location. Contemp Oncol (Pozn), 2012. 16(3): p. 276-7.
5. Adegboyega, P. and A. Adesoken, Tubulovillous adenoma of the urinary bladder. Modern pathology : an official journal of the United States and Canadian Academy of Pathology, 1999. 12(7): p. 735-8.

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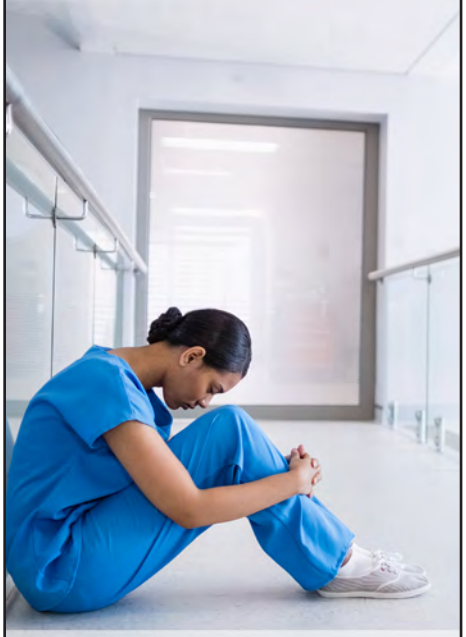

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A CLOSER LOOK AT QUALITY

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Primary Care Fellowship Training: Addressing barriers and finding new solutions to addressing difficult healthcare issues (Part one)

CHAD RODGERS, MD, FAAP AND HANNA WINDLEY, MPA

We were all well trained through medical school and residency of taking care and managing sick patients but also improving health for our patients through prevention, education, and coaching. Our best training most likely occurred during the first 3-5 years of practice. The experience of caring for patients in your practice setting (academic vs. private, rural vs. urban, large vs small practice) teaches us a lot and refines our skill of the "art of medicine". Continuing medical education through self-study, conferences, learning modules, and other modalities is the cornerstone of remaining well trained as doctors. Our career is one of lifelong learning and constantly adapting to new knowledge and ways of practice.

Many physicians experience frustration within a few years of practice not just with the healthcare system, but despite their best efforts and training, their patients don't seem to be getting better. We work on communication styles, motivational interviewing, care plans, close follow up, calls from our nursing staff, but still our patients struggle.

Arkansas has many challenges as a rural state. Economic hardship, poor access to primary and specialty care, low educational attainment and poor health literacy, multigenerational poverty, systemic racism, and many other social determinants of health. Good evidence continues to show that your zip code is a greater predictor of your health outcomes than your genetic code. Eighty percent of what occurs outside our clinic exam room or hospital doors impacts our patient's health.

Childhood trauma, referred to as Adverse Childhood Experiences (ACEs), impacts not only our children's current mental and physical health, but many adults' past experiences contribute to their current health and their ability to improve their health. The more ACEs you have experienced, the more likely you are to struggle with chronic physical health problems as well as mental health. Factors that foster resilience in children, homes, and communities are protective of the impact of ACEs. It's not what is "wrong" with our patient, but perhaps "what happened" to our patient. (For more info about ACEs visit afmc.org/aces

Arkansas also ranks highly in the areas of obesity, opioid and other drug use, as well as poor behavioral and mental health. These are all very complex issues which makes them very difficult to manage. The fact is these healthcare issues can not be solved by the Primary Care Physician alone within the clinic or hospital. It is more than a medication, a procedure, or a therapy.

Within a few years of practice, many physicians identify health issues they would like to confront and adopt new practices or new ways of addressing long-held troublesome diagnoses. But practice is challenging with limits of time, energy, knowledge, and especially the administrative burdens related to managing our patients. Many times, we find ourselves looking to the West or East coast or even urban centers for solutions. But the rural setting is different. Not that there are not solutions, but there are unique, potentially innovative strategies, to improve health care.

In 2018, AFMC sought and was awarded a Primary Care Training Enhancement (PCTE) grant which created a fellowship program

targeting primary care physicians in rural Arkansas to be trained and provided a stipend to offset clinical time or support out of practice time to learn and develop new strategies for addressing health issues related to obesity, opioid use, and mental health. During the life of this grant, an average of five fellows have and will be selected to participate in this fellowship program. The first cohort is nearing completion and currently finalizing their projects. A second Cohort has completed the curriculum phase and are developing their projects to implement over the next few months. Applications for the third cohort opened July 31st. By completion, 20 primary care providers will have completed training and projects to disseminate potentially best practices to address these and other complex health care issues.

Acknowledging the impact of ACEs and Social Determinants of Health, fellows develop new treatment strategies, care plans, community interventions, and resources through a Trauma Informed Care lens. (For more information about Trauma Informed Care: https://www.chcs.org/topics/trauma-informed-care/)

A twelve-week educational curriculum was developed by AFMC in conjunction with Arkansas College of Osteopathic Medicine (ARCOM) who has also incorporated elements into their graduate medical education program. The curriculum is broken down into six 2-week modules to be completed on the Primary Care Physician's own time with online discussion boards, technical assistance calls, and support from AFMC and ARcare staff.

The modules included subjects related to Social Determinants of Health, Adverse Childhood Experiences, Public Heath and Population Management, Practice Transformation, and Physician Leadership. The modules were developed using multiple formats including recorded lectures and power point presentations, articles and other related readings, case studies, followed by online discussion questions. During the second cohort, short calls at the end of each module were held to allow for interactive discussion between fellows and faculty as well as support staff for any technical assistance questions.

Arkansas Good Medicine℠

With completion of the educational course, fellows began to develop the plan for their project to address issues related to obesity, mental health, and opioid use. Brief abstracts of the projects were submitted and literature reviews started. Technical assistance is provided by AFMC, ARcare, and ARCOM staff and faculty. Throughout their project, fellows will collect pertinent data and will work with the AFMC Analytics team to analyze their findings. At the end of the program period, a report is completed, and presentations made that are shared with all the fellows and shared on AFMC's website.

All fellows from cohorts 1 and 2 have reported satisfaction with the program and have provided feedback to continue to improve the fellowship program to be carried forward into future cohorts. A member of cohort 1 stated, "I enjoyed learning more about trauma-informed care, I feel this is vital especially when practicing in a small rural community." They also have reported increased knowledge in these areas and have been able to teach their staff new ways of looking at things and doing things in their practice to improve care. Many anticipate continuing changes to their practice after the fellowship is over. A member of cohort 2 elaborated on this by saying, "I definitely feel more knowledgeable about this subject [ACEs and Trauma-informed care] and research. Having that knowledge is key to understanding the patient, their health conditions, and how to approach and treat the patient."

AFMC continues its relationship with ARCOM and ARcare as we move forward recruiting new fellows and expanding opportunities to other rural practices and learning institutions. Not only has this been a great learning experience for all, it will create new thoughts and practices about the delivery of health care in Arkansas and improve health for Arkansans. ▲

Dr. Chad Rodgers is Chief Medical Officer at AFMC and practices at Little Rock Pediatric Clinic.
Ms. Hanna Windley, MPA, is a program and policy analyst for AFMC and is the project director of the Arkansas Good Medicine fellowship program.

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OCTOBER 2020

by SPENCER MCCLURE; SANDRA MARCHESE JOHNSON, MD, FAAD

Drug-Induced Bullous Pemphigoid

We present a 76-year-old white man with a history of gastroesophageal reflux disease and type two diabetes (last A1C was 7.3 in August 2018) who presented with pruritic erythematous patches and plaques on his scalp, back, and arms. The patient had a tonsillectomy in 1950, an appendectomy in 1960, and two carpal tunnel operations, with the first in 2007 and the second in 2014. Other medical history of note is he received a tetanus and pneumococcal vaccination in 2014, a varicella vaccination in 2015, an influenza vaccination in 2017, and he suffered from a deep vein thrombosis in June of 2018. His medication regimen consists of Atorvastatin 10mg at night, Finasteride 0.5 mg in the morning, Glyburide/Metformin 10mg/1000mg bid, Losartan/HCTZ 50mg/12.5mg in the morning, Metanx bid, Tamsulosin HCL 0.4mg at night, and Warfarin 1mg at night.

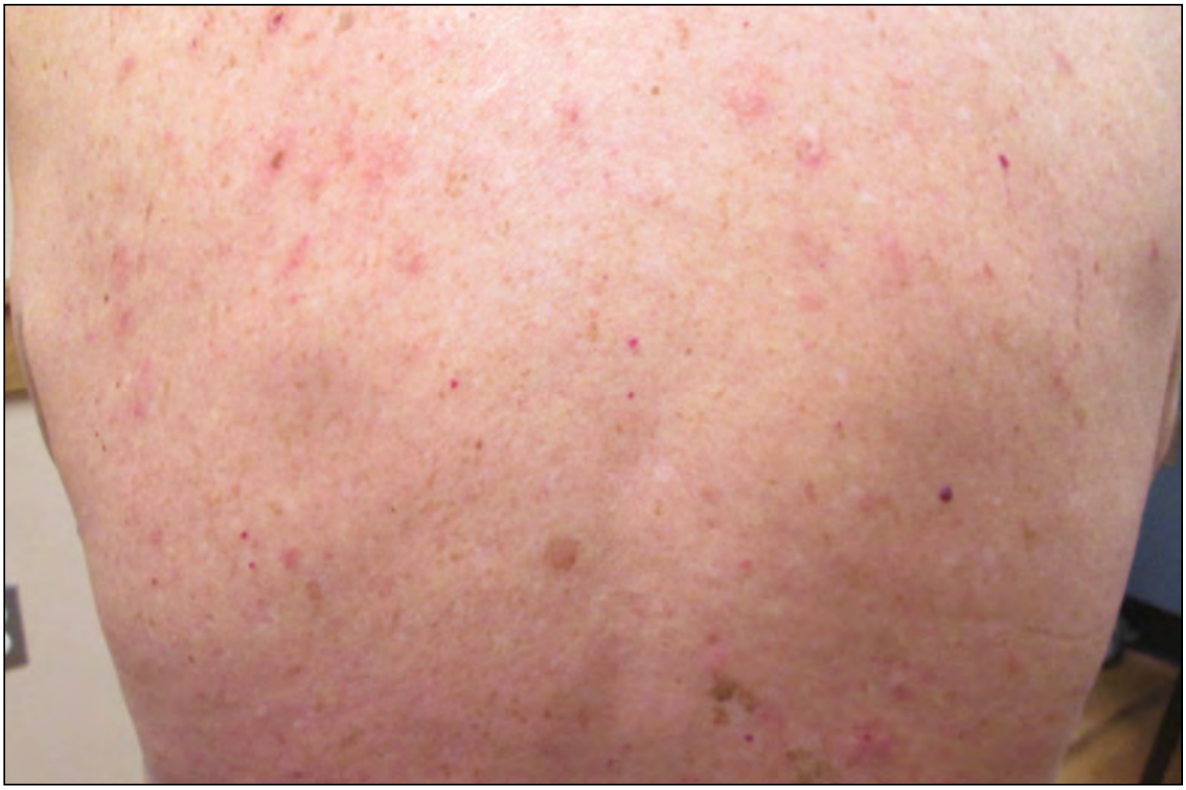

Photo 1: Erythematous papules, some of which are urticarial in nature scattered on the back.

On physical exam, patient had erythematous papules, some of which were urticarial in nature scattered on the back, scalp, and upper extremities bilaterally. (Photo 1). Skin biopsies were performed that revealed superficial perivascular infiltrate with eosinophils and sub epidermal detachment. Direct immunofluorescence of perilesional skin revealed linear IgG and C3 deposits at the basement membrane zone without IgA, IgG or fibrin. Serum tests revealed BP 180s to be 31 U (normal less than 9U) and BP 230s to be 16 U (normal is less than 9). The diagnosis of bullous pemphigoid was determined. The patient would get relief while taking oral prednisone with a minimum of 20 mg in the morning. Attempts at steroid-sparing agents were not successful including doxycycline, niacinamide, methotrexate, cyclosporine, ultraviolet light and mycophenolate mofetil. A second opinion was obtained from the UAMS Department of Dermatology. It was determined that the patient could be experiencing drug-induced bullous pemphigoid from his omeprazole for GERD or his underlying diabetes as well as diabetes treatments. The patient is still suffering with poorly controlled diabetes and poorly controlled bullous pemphigoid.

Bullous pemphigoid is the most common autoimmune blistering disorder, and it has become increasingly common over the past decade.[1] It is part of a group of "pemphigoid diseases" that are defined by autoantibodies against the dermal-epidermal junction. It is predominantly associated with a more elderly patient population with onset usually occurring in the eighth decade.[2] Clinical presentation most commonly is described by tense, fluid-filled blisters and erythema. Severe pruritus is observed in almost all patients. Mild oral lesions can be observed in 10-20% of patients but other mucosal surfaces are rarely effected.[3] Bullous pemphigoid has been commonly associated in literature with different conditions, but up to half of all patients with bullous pemphigoid also have a neurological disease with Parkinson's, cerebrovascular disease, epilepsy, and multiple sclerosis being among the most common.[4,5,6,7] Onset of bullous pemphigoid is often variable. Occasionally trauma, burns, UV radiation, and drugs

Chart 1: Drugs Commonly Associated with Bullous Pemphigoid

Drug Class	Examples
Dipeptidyl Peptidase-IV (DPP-IV) Inhibitors	Vildagliptin, Linagliptin
Other Antidiabetic Medications	Metformin, Gliclazide, Glimepiride
Aldosterone Antagonists	Spironolactone
Loop Diuretics	Furosemide
Antipsychotics	Phenothiazine Aliphatic Chain
Checkpoint Inhibitors Anti-PD1/PD-L1	Pembrolizumab, Nivolumab, Dirvalumab
Angiotensin-converting Enzyme Inhibitors	Quinapril, Fosinopril, Lisinopril
Anticoagulants	Acetylsalicylic Acid, Acenocoumarol

are triggers of symptoms.[8] The treatment for drug-induced bullous pemphigoid is unique in that it differs from the guideline-based approach of treatment for classic bullous pemphigoid and focuses on removing the suspected offending agent.[9] After drug removal, many patients experience rapid improvement in their symptoms with very little recurrences.[10] Some of the most commonly cited offending drugs are, DPP-4 inhibitors (a relatively new drug class commonly used in the treatment of type 2 diabetes), other antidiabetic medications (ex. Metformin), diuretics, antipsychotics, Anti PD-1/Anti PD-L1, ACE inhibitors, and anticoagulants, as shown in Chart 1.[11-12] Clinical presentation and histological findings may be indistinguishable from the classic form of bullous pemphigoid as there is no specific recognized biomarker of the drug-induced variety. Onset in younger patients, and within a mean of three months after beginning a new medication can be helpful variables in the recognition and subsequent treatment of drug-induced bullous pemphigoid.[10] More offending drugs are being recognized frequently and that is likely to keep occurring as new pharmaceuticals are produced.

References

1. Ujiie H, Nishie W, Shimizu H. Pathogenesis of bullous pemphigoid. Dermatol Clin. 2011;29:439–446.ix.
2. Schmidt E, Zillikens D. Pemphigoid diseases. Lancet. 2013;381:320–332.
3. E Schmidt, R della Torre, L Borradori Clinical features and practical diagnosis of bullous pemphigoid Dermatol Clin, 29 (2011), pp. 427-438
4. K Taghipour, CC Chi, A Vincent, RW Groves, V Venning, F WojnarowskaThe association of bullous pemphigoid with cerebrovascular disease and dementia: a case-control stud Arch Dermatol, 146 (2010), pp. 1251-1254
5. YJ Chen, CY Wu, MW Lin, et al. Comorbidity profiles among patients with bullous pemphigoid: a nationwide population-based study Br J Dermatol, 165 (2011), pp. 593-599
6. SM Langan, RW Groves, J West The relationship between neurological disease and bullous pemphigoid: a population-based case-control study J Invest Dermatol, 131 (2010), pp. 631-636
7. S Bastuji-Garin, P Joly, P Lemordant, et al. Risk factors for bullous pemphigoid in the elderly: a prospective case-control study J Invest Dermatol, 131 (2011), pp. 637-643
8. VA Venning, F Wojnarowska Induced bullous pemphigoid Br J Dermatol, 132 (1995), pp. 831-832
9. Venning VA, Taghipour K, Mohd Mustapa MF, Highet AS, Kirtschig G. British Association of Dermatologists' guidelines for the management of bullous pemphigoid 2012. Br J Dermatol 2012; 167: 1200–1214.
10. Stavropoulos PG, Soura E, Antoniou C. Drug-induced pemphigoid: a review of the literature. J Eur Acad Dermatol Venereol. 2014 Sep;28:1133–1140.
11. Patsatsi A, Vyzantiadis TA, Chrysomallis F, Devliotou-Panagiotidou D, Sotiriadis D. Medication history of a series of patients with bullous pemphigoid from northern Greece – observations and discussion. Int J Dermatol2009; 48: 132–135.
12. Denise Miyamoto, Claudia Giuli Santi, Valéria Aoki, Celina Wakisaka Maruta Bullous Pemphigoid An Bras Dermatol. 2019 Mar-Apr; 94(2): 133–146.

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Human Monocytotropic Ehrlichiosis in Immunocompromised Patient

Introduction

Human monocytic ehrlichiosis (HME) is a potentially life-threatening tick-borne rickettsial disease (TBRD) caused by the obligate intracellular gram-negative bacteria, Ehrlichia chaffeenis. Fatal HME presents with acute ailments of sepsis and toxic shock-like symptoms that can evolve to multi-organ failure and death. The most common clinical symptoms are fever, headache, myalgias, and arthralgias. Rash can also be seen more commonly in pediatric cases. Although most known HME cases are not fatal, approximately 3–5% of all Ehrlichia infections in the U.S. result in death despite patients receiving appropriate care.[5] The characteristic lab findings include significantly lowered total white blood cells, neutrophils, and platelets along with mildly elevated transaminases.[1] The first-line treatment for HME are tetracyclines and their derivatives, particularly doxycycline. Typically, patients treated with doxycycline are afebrile within one to three days after initiation.[2] Alternatively, rifampin has been shown to have rapid bactericidal effects in vitro and several reports have shown successful treatment with chloramphenicol.[2]

Case Report

Our patient is a 50-year-old female undergoing chemotherapy treatment consisting of fluorouracil (5-FU) and mitomycin for squamous cell carcinoma of the anal canal. She presented to the emergency department after experiencing two weeks of fatigue, shortness of breath, and cough, which was initially dry and subsequently became productive. The patient also reported diffuse body aches and reported a temperature of 39.4°C. The patient was also an active smoker, with an average of one pack per day for 30 years.

On arrival to the ED, physical exam was significant for tachycardia and crackles in the lower lobe of the left lung. She had a temperature of 37.3°C, heart rate of over 100 beats per minute, blood pressure of 130/90mmHg, and oxygen saturation of 97.5%. Lab values showed white cell count of 5.6K/ul with 76.5% neutrophils, ANC of 4.29K/ul, hemoglobin of 12g/dl, platelet count of 179K/µl, sodium of 131mmol/L, and potassium of 4.1mmol/L. Chest x-ray revealed left basilar atelectasis, and CT revealed interstitial pulmonary edema. The patient was given vancomycin and cefepime and admitted for treatment of presumed pneumonia. Upon admission, the patient was started on ceftriaxone and azithromycin.

Twenty-four hours after admission, the patient's shortness of breath persisted and she began spiking temperatures as high as 39.2°C. Blood pressure dropped to 103/42mmHg and heart rate was measured at 110 beats per minute. Repeat laboratory values showed that her potassium had dropped to 2.6mmol/L and her calcium had dropped to 6.1mg/dl. Liver function tests showed elevated alkaline phosphatase of 108 IU/L and aspartate aminotransferase of 51 IU/L along with lower albumin of 2.2g/dl. Blood labs with manual differential showed total white cell count of 2.73 k/ul with absolute neutrophil count of 0.9 k/ul. Blood cultures reported no growth at 24 hours. A more detailed history elicited that she had discovered a tick on her waistline while showering the week before. The patient was then switched to a regiment of vancomycin, cefepime, and doxycycline while awaiting the results of a tick panel that included PCR detection tests for Anaplasma and multiple Ehrlichia species.

Within 24 hours of starting the doxycycline, the patient's fever, cough, and shortness of breath had subsided, and lungs were clear to auscultation on exam. PCR tests returned positive for Ehrlichia chaffeensis. The patient's WBCs and platelets continued to drop. Forty-eight hours after initiation of doxycycline, her WBC was measured at 2.31K/ul and platelets were measured at 52K/ul. Due to the drastic clinical improvement, the patient was discharged four days after admission with instructions to continue doxycycline P.O. for a total 10-day course.

Discussion

Pathogenic rickettsiae and ehrlichiae are likely emerging diseases that cause under-recognized presentations, so clinicians must be aware of this disease in order to make a timely and appropriate diagnosis. The number of cases of Ehrlichia chaffeensis infections reported to the CDC has been on the rise. Between the years 2000 and 2017, the number of reported cases has increased eight-fold from 200 cases in 2000 to 1642 cases in 2017.3 Knowledge of the presentation and treatment of HME is especially useful for physicians practicing in Arkansas, as the disease is most commonly seen in the southern central states. Over 50% of reported cases come from only four states (Arkansas, Missouri, Virginia, and New York).[3] HME could be related to individuals engaging in outdoor occupations and/or living in close proximity to areas that favor tick habitats. It is very likely that endemic regions in the U.S. are affected by a combination of factors including climate, land use, and socio-economic conditions. Given the prevalence of Ehrlichiosis in Arkansas, physicians can mitigate risk of contraction by counseling their patients on preventative measures against tick

bites. Whenever the patient is in a situation where risk of tick exposure is high, they should be encouraged to apply permethrin spray to their clothing or even do something as simple as tucking their pant legs into their footwear as both of these have been shown to be protective against tick-borne illnesses.[4]

When evaluating a patient (especially one who is immunocompromised) with a febrile illness, flu like symptoms and the triad of anemia, thrombocytopenia, and elevated transaminases, one must always remember to include infections from Ehrlichia or other tick-borne pathogens in their differential diagnosis given their ability to act as opportunistic infections. Because a laboratory confirmation is required to meet the case definition, reporting may be influenced by a patient's access to health care.

It is likely that, given the prevalence of Ehrlichia chaffeensis in Arkansas and the relatively low socioeconomic status of many Arkansans, the actual incidence of infection is drastically underreported. Prospective seroepidemiology studies performed in Arkansas have demonstrated that the rate of asymptomatic infections could be as high as 2/3 total infections.[4] It stands to reason that an infection in an immunocompromised patient could result in severe pathology that otherwise would have been asymptomatic.

With patients in an immunocompromised state, early detection and treatment becomes all the more essential. Ideally, a patient with a clinical picture resembling HME should be started on empiric doxycycline within 24 hours of the onset of symptoms. Beginning treatment within 24 hours has been shown to have lower rates of being transferred to the ICU, decreased requirement for mechanical ventilation, and shorter hospital stay duration.[5]

Our case demonstrates the nonspecific but severe presentation of HME in the immunocompromised patient. It also highlights the importance of early clinical suspension of HME gained by the history of tick exposure and triad of anemia, thrombocytopenia, and elevated transaminases in a febrile patient. Our case also emphasizes the rapid response of the disease to tetracyclines and the importance of early empiric therapy in highly endemic areas such as Arkansas.

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References

1. Ismail,Naheed; McBride, Jerry. Tick-Borne Emerging Infections. Clinics in Laboratory Medicine 2017;37(2) 317-340
2. Paddock CD, Childs JE. Ehrlichia chaffeensis: a prototypical emerging pathogen. Clin Microbiol Rev. 2003;16(1):37–64. doi:10.1128/cmr.16.1.37-64.2003
3. Centers for Disease Control and Prevention, National Center for Emerging and Zoonotic Infectious Diseases (NCEZID), Division of Vector-Borne Diseases (DVBD) https://www.cdc.gov/ehrlichiosis/stats/index.html
4. Steven J. Yevich, José L. Sánchez, Robert F. DeFraites, Cornelia C. Rives, Jackie E. Dawson, Iyorlumun J. Uhaa, Barbara J. B. Johnson, Daniel B. Fishbein, Seroepidemiology of Infections Due to Spotted Fever Group Rickettsiae and Ehrlichia Species in Military Personnel Exposed in Areas of the United States where Such Infections Are Endemic, The Journal of Infectious Diseases, Volume 171, Issue 5, May 1995, Pages 1266–1273, https://doi.org/10.1093/infdis/171.5.1266 Pavelites JJ, Prahlow JA (2011) Fatal human monocytic ehrlichiosis: a case study. Forensic Science Medicine and Pathology 7: 287–293

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