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INFECTIOUS PATHOGENS

Ultrasound Imaging: Zika Virus

There has been a devastating disease that has only recently emerged from Brazil called Zika virus, which causes microcephaly in newborns. In this article, I will describe the history of the Zika virus, the way that it is spread, and how ultrasound is used to detect the cranial defects the Zika virus causes.

Zika virus was first discovered in 1947, and named after the Zika forest in Uganda (1). In 1952, the first human cases of Zika virus disease were detected. Outbreaks of Zika have been reported in Africa, Southeast Asia, and the Pacific Islands (1). Before 2007, 14 cases had been documented. In May, 2015 an alert was issued regarding the first confirmed Zika virus infection in Brazil (1). On February 1st, 2016 the World Health Organization (WHO) declared Zika virus a public health emergency of international concern (PHEIC).

Special points of interest:

- Zika virus was discovered in 1947
- WHO declared Zika virus a public health emergency on February 1st, 2016
- Zika virus is carried by mosquitos
- Increasing number of babies born with microcephaly since the outbreak of Zika virus

The mosquito that carries the virus is called "Aedes aegypti" and is not found in Colorado. This type of mosquito goes as far north as the Carolinas and the southern states, particularly Florida and Texas. The mosquito lives in tropical environments and feeds almost exclusively on human blood. Mosquitos become infected when they feed on a person already infected with the virus. Infected mosquitos can then spread the virus to other people through bites. (5)

The biggest challenge in Brazil since the outbreak of Zika virus is an increase in the number of babies born with microcephaly. Microcephaly is a condition where a baby's head is much smaller than expected. *Figure 1.* During pregnancy, a baby's head grows because the baby's brain grows. Microcephaly occurs because a baby's brain has not developed properly during pregnancy. In the United States, microcephaly occurs from 2-12 babies per 10,000 live births (1). Causes include infections such as rubella, toxoplasmosis, and cytomegalovirus. Other causes include severe malnutrition, alcohol, or interruption of blood supply. (2) In Brazil, babies with microcephaly have been reported among mothers who were infected with the Zika virus while pregnant.

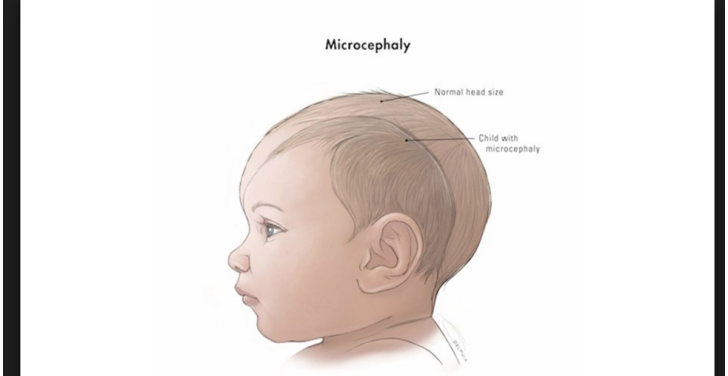

Figure 1: Diagram of Microcephaly

In the United States, detailed US fetal anatomic survey performed at the optimum time of 18 - 22 weeks gestation age will detect the majority of serious defects (2). Effective US screening for CNS anomalies can be performed by examination of 3 crucial axial planes through the fetal brain:

Transthalamic Plane: used to measure the biparietal diameter and head circumference *Figure 2.*

Transcerebellar Plane: landmarks are the thalami, third ventricle, and cerebellar hemisphere *Figure 3.*

US: Zika Virus Continued...

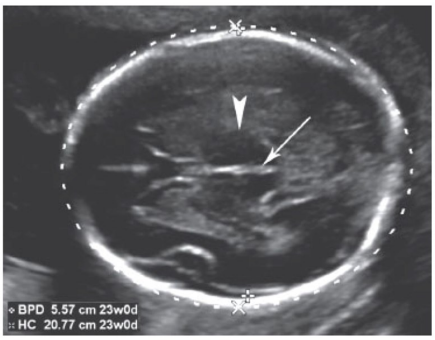

Figure 2: Transthalamic plane: paired thalami (arrowhead), midline of the third ventricle (arrow), biparietal measurement and head circumference measured in this plane.

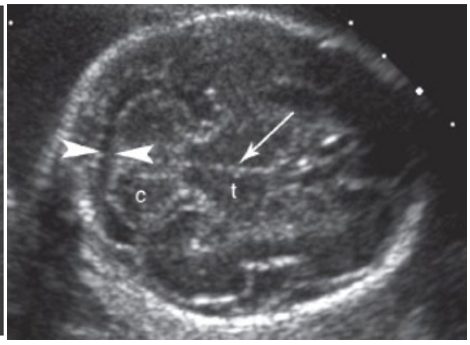

Figure 3: Transcerebellar Plane: Landmarks are the thalami (t), third ventricle (arrow), and cerebellar hemispheres. Cisterna Magna is measured (arrowheads).

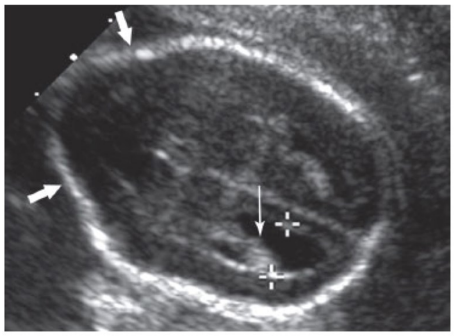

Figure 4: Transventricular Plane - Early Ventriculomegaly. The choroid plexus hangs dependently in the atrium of the downside lateral ventricle (skinny arrows). Note the bossing of the frontal bones (thick arrows) giving the outline of the cranium similar to a lemon shape.

Transventricular Plane: Level of the atria *Figure 4*.

Microcephaly in fetal imaging is usually defined as head measurement (e.g. circumference) falling under two standard deviations expected for age or under the 3rd percentile. Other authors advocate the use of three standard deviations which increases specificity. (3) Importantly, the head circumference is the only measurement used, as biparietal diameter can be altered by the shape of the head.

If a patient comes to the department of radiology and has a history of recent travel to place with ongoing Zika virus transmission, there is a recommended testing algorithm. (4). The patient will get a blood test for the Zika virus. If the patient test is positive or inconclusive, consider serial fetal ultrasounds every 2 weeks. If the patient is negative for Zika virus, perform a fetal US to detect microcephaly or intracranial calcifications. If microcephaly or intracranial calcifications are present, retest for the Zika virus and consider amniocentesis. If microcephaly or intracranial calcifications are not present, then patient should return to routine prenatal care. (4) *Figure 5*.

Hopefully this article helps the reader understand a little more about the Zika virus, and the history of the disease. Information about the Zika virus is evolving. The current Zika virus guidelines are to test for the disease in pregnant patients, and use ultrasound to measure the head circumference of the fetus. If the measurement is 2 or 3 standard deviations below the mean, then it is consistent with microcephaly.

Dr. Michael Rogan

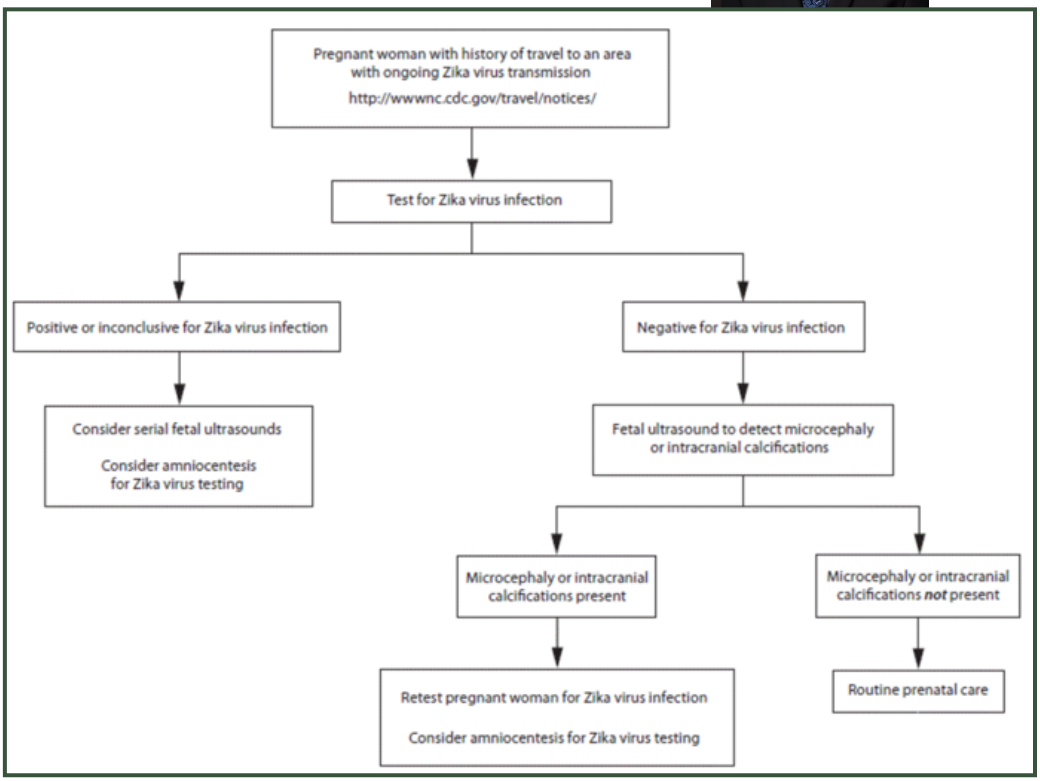

Figure 5: Imaging Guideline for Zika Virus

References

1. website: CDC.gov
2. Brant, William E., Helms, Clyde A. Fundamentals of Diagnostic Radiology. 4th Edition. 2012.
3. Di Muzirio, Bruno D., Microcephaly. raediopedia.org
4. Oduyebo, T. et al. Update: Interim Guidelines for Health Care Providers. CDC website.
5. denver.cbslocal.com

Computed Tomography: Retroperitoneal Fasciitis

Clearly, MR is more sensitive than CT for detection of infection due to its intrinsically higher contrast to noise and sensitivity to enhancement with gadolinium. It is the modality of choice for assessment of diskitis in the spine, encephalitis in the brain, and acute infection in bones and joints.

However, there are some obvious reasons and some not-so-obvious reasons that CT is the modality of choice in many situations where infection is suspected clinically.

In this article, I provide a brief summary of situations where CT is better suited for assessment of infection and then go into much more detail about a rare but rapidly fatal entity called Retroperitoneal Fasciitis in which CT plays a crucial role.

First, the obvious uses of CT over MR in infection:

- Contraindication to MR (incompatible pacemaker /metallic foreign body / implanted devices / etc)
- Patient 's general condition or peripheral devices preclude MR
- Severe claustrophobia in cases where sedation is not an option
- Availability of scanner

The not-so-obvious uses of CT over MR in infection:

- Pulmonary / pleural infections in general. CT provides much better detail of lung parenchyma / central cavitation / pleural calcification.
- Abdominal infection where it is critical to identify extra-luminal gas that might indicate bowel perforation or post -op leak. CT exquisitely defines gas against soft tissue. MR can be very difficult to interpret. Also, peristalsis during the MR acquisition makes the assessment even more difficult. Breath hold in a very sick patient is less critical for CT.
- Identifying calcification in the liver can provide a specific diagnosis of shistosomiasis ("tortoise shell" pattern).
- Sequestrum in chronic osteomyelitis, which can be a source of ongoing infection, can be difficult to identify by MR (however MR is better at determining whether the sequestrum is viable or dead).
- Necrotizing fasciitis of the extremities. Gas is the hallmark and much easier to identify and more reliably demonstrated with CT. Classically, affected tissue reveals absence of contrast enhancement (necrotic) and presence of gas (from gas producing organisms).
- Retroperitoneal fasciitis, not because MR can't show it, but because CT is always the first line in these very sick patients.

RETROPERITONEAL FASCIITIS

Recognition of any infection is critical for timely care of the patient and to avoid significant long-term consequences like joint destruction, spread to epidural space, etc.

Although rare, Retroperitoneal Fasciitis (RF) is a potentially lethal rapidly progressive infection involving the retroperitoneal tissues and fascial planes of the abdomen and pelvis.

The disease can range from a simple infection of the fascial planes to a fulminant necrotizing form (NRF), which is associated with thrombosed vessels and infarcted tissue. The distinction between the non -necrotizing and the necrotizing form of retroperitoneal fasciitis may not be possible on purely clinical or imaging findings, however, spread across fascial planes and, particularly, retroperitoneal gas suggests the more severe necrotizing form of RF.

It is important for both technologists and Radiologists to be aware of this condition as early treatment can be life saving. The first reported case was in 1991, and for the first few years of recognition it was considered uniformly fatal.

The patient will present with extraordinary pain and rapid clinical decline. Unlike necrotizing fasciitis of the legs, there are no external visible clues to the diagnosis.

A history of recent surgery (especially gynecological), recent child birth/C-section or a history of known lower extremity necrotizing fasciitis should increase suspicion.

Predisposing factors include diabetes, obesity, advanced age, alcoholism, cirrhosis, leukemia, HIV, renal failure, and septicemia. Iatrogenic causes include steroid therapy and chemotherapy.

Because there are no specific clinical means to detect it, NRF often presents late with the patient in septic shock and hypotension.

The disproportionate pain is thought to be due to intramuscular edema causing a compartment syndrome – often involving the psoas muscles.

Lab studies are nonspecific – elevated white count, sed rate, C-reactive protein.

Most cases have an identifiable source of infection on CT - appendicitis, diverticulitis, pyelonephritis, perianal abscess, colon cancer, perforation, postsurgical complication or spread

Retroperitoneal Fasciitis:

a potentially lethal, rapidly progressive infection involving the retroperitoneal tissues and fascial planes of the abdomen and pelvis.

The disease can range from a simple infection of the fascial planes to a fulminant necrotizing form (NRF), which is associated with thrombosed vessels and infarcted tissue.

CT: Retroperitoneal Fasciitis Continued…

from an infected thigh. Some cases have no clear source of infection.

The responsible bacteria depend on the source – usually polymicrobial when the source is abdominal/pelvic and usually a single organism when the source is the extremities (in particular Beta-hemolytic strep and MRSA).

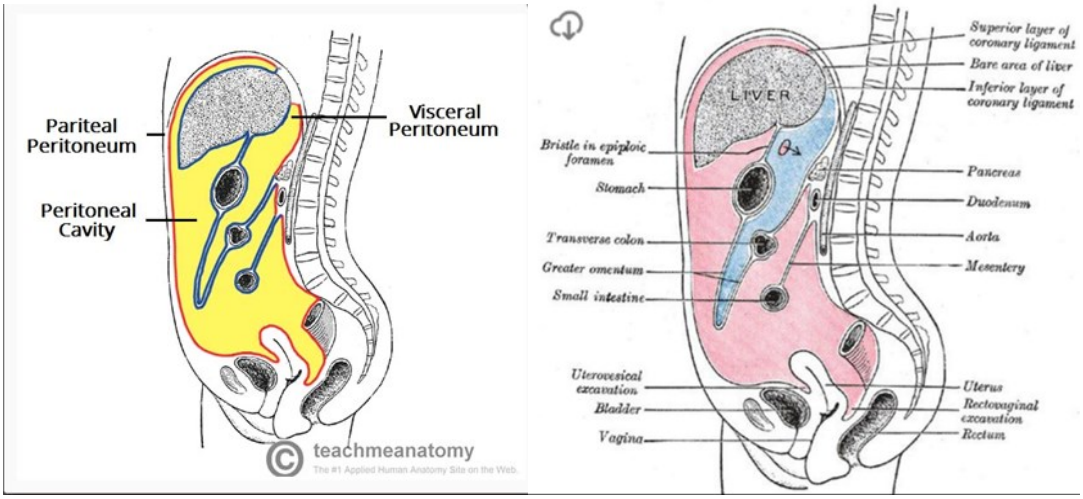

Figure 1 & 2: Case courtesy of Dr. Jeremy Jones, Radiopaedia.org rID: 36226

It is helpful to understand how the infection spreads in order to recognize it.

It spreads along retroperitoneal fascial planes. These fascial planes are potential spaces (ie – they only become a 'space' when distended by inflammation or infection).

Some descriptive and illustrative anatomy will help:

The peritoneal space can be thought of as a balloon with two surfaces – visceral and parietal. Imagine pushing your fist into the posterior surface of the balloon. Your arm is the vascular pedicle for the small bowel mesentery that feeds the small bowel that is now wrapped by a surface of the balloon (called the visceral layer of the peritoneum). Also imagine the liver, spleen, stomach, transverse colon and sigmoid colon all pushing into the balloon. All of these structures become covered by a visceral layer of peritoneum. The rest of the balloon layer is known as the parietal peritoneum. The peritoneal space is between the visceral and parietal layers. Disease of these organs usually results in peritoneal fluid / blood / infection.

By the way, in the figure above (Figures 1 & 2) – right, the pink is the "greater" peritoneal cavity and the blue is the "lesser" peritoneal cavity or sac that resides behind the stomach, anterior to the pancreas.

The retroperitoneal space (or better the "extra-peritoneal space") completely surrounds the peritoneal space - anteriorly, posteriorly and laterally and inferiorly. It is that tissue between the parietal peritoneum and the transversalis fascia (the fascia that lines the innermost of the 3 body wall muscle layers).

Within the retroperitoneal space are (Figures 3, 4, & 5):

- Anterior pararenal space (APS) contains the pancreas, anterior and descending colon, and portions of the duodenum
- Perirenal space (contains kidneys, adrenals and proximal ureters)
- Posterior pararenal space (PPS) contains mostly fat.

Gerota's fascia (aka Retromesenteric plane/fascia – 'RMP') separates the perirenal space from the anterior pararenal space

Zuckerkandl fascia (aka Retrorenal plane/fascia – 'RRP') separates the perirenal space from the posterior pararenal space.

The RMP and the RRP are potential spaces that can expand with inflammation / bleeding / infection.

Laterally, these 2 fascia (RMP and RRP) meet to form the Lateral Conal Plane/fascia (LCP).

Inferiorly, they fuse to form the 'Combined Interfascial

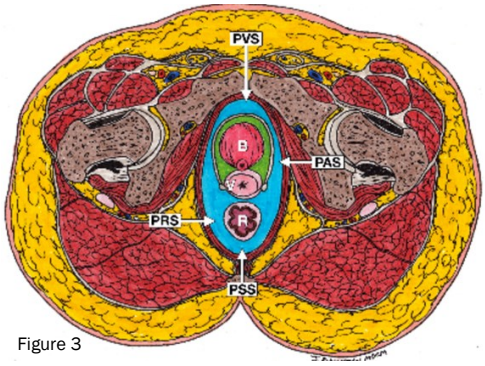

Figure 3

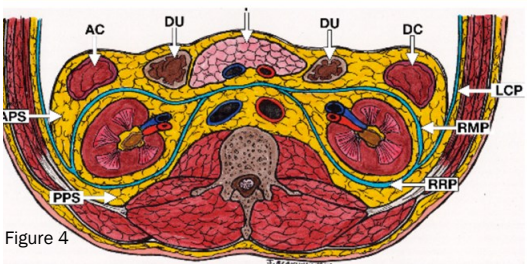

Figure 4

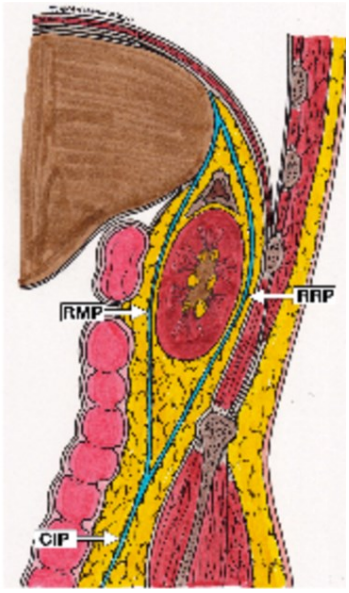

Figure 5

CT: Retroperitoneal Fasciitis Continued...

Plane' (CIP). The CIP is in continuity with the pelvic extra-peritoneal spaces (spaces around the bladder, rectum and anterior to the sacrum). The pelvic extra-peritoneal spaces are in continuity with each other and with the retroperitoneal spaces superiorly.

The point of all this detailed anatomy is that Retroperitoneal Fasciitis can originate within any retroperitoneal structure (kidneys, ureters, pancreas, duodenum, ascending or descending colon, pelvic organs) and can directly spread to anywhere else in the retroperitoneum along or through the planes described above (along fascial planes if less aggressive or non-necrotizing, through fascial planes if aggressive or necrotizing).

The following sets of images in Tables 1, 2, & 3 are examples:

TABLE 1—

Example 1: (Figures 6 & 7) Non-necrotizing retroperitoneal fasciitis from pyelonephritis.

The two axial CT images shows inflammatory changes involving the right lateral conal plane and the combined interfascial plane (white arrows) and in the presacral space (black arrows)

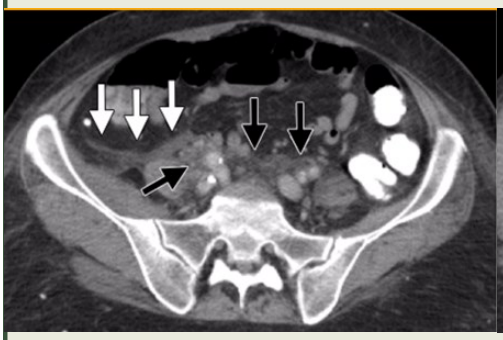

Figure 6

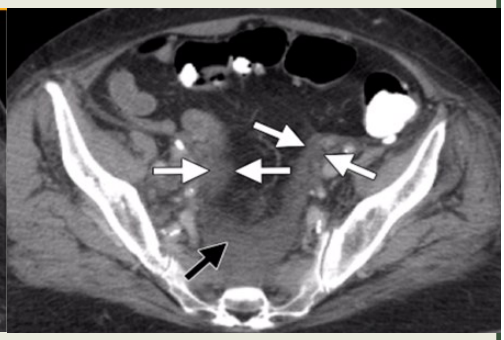

Figure 7

TABLE 2—

Example 2: (Figures 8 & 9) Necrotizing retroperitoneal fasciitis from pyelonephritis. Note the extravasated contrast from necrosis of the ureters (white arrows), lateral conal fasciitis (black arrows). Also note the collapsed IVC from septic shock.

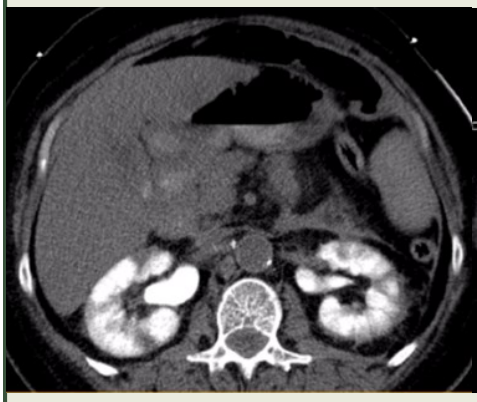

Figure 8

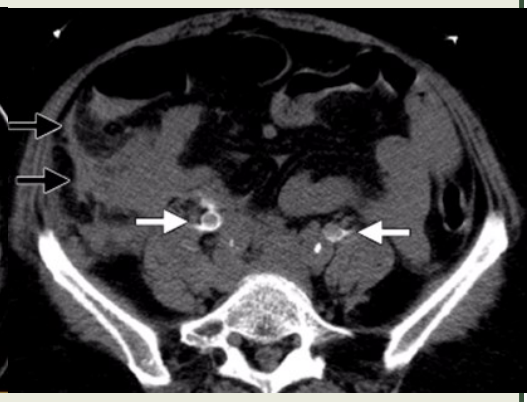

Figure 9

TABLE 3—

Three examples of NRF spreading from below:

In Figure 10 anterior thigh NF spreads cephalad via the inguinal canal.

In Figure 11, perianal NF spreads cephalad directly.

In Figure 12, medial thigh NF spreads cephalad via the obturator canals.

From the pelvic extra-peritoneal spaces, there is free access to the rest of the retroperitoneum.

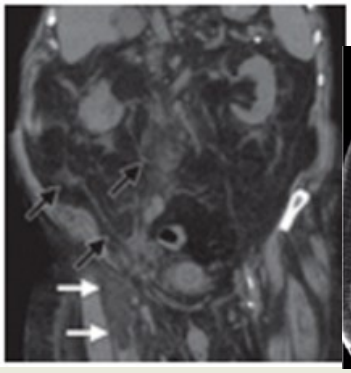

Figure 10: Anterior Thigh NF

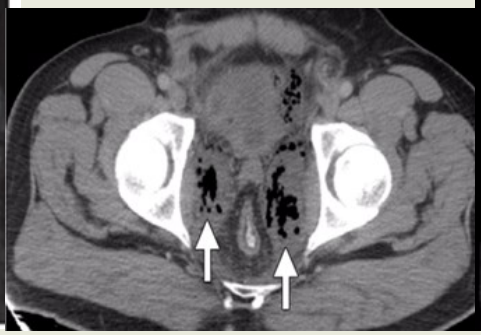

Figure 11: Perineal NF

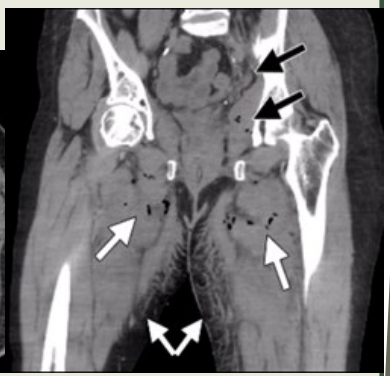

Figure 12: Medial Thigh NF

CT: Retroperitoneal Fasciitis Continued...

CT is usually the first to discover evidence of retroperitoneal fasciitis.. The patient is typically very ill and rapidly deteriorating and often post-op.

CT is 100% sensitive (findings are always present) and it is over 80% specific (the correct diagnosis can be made correctly 4 out of 5 times). A few hours can make a big difference in the patient's outcome – so - it is important to be aware of the findings and to alert the Radiologist in the appropriate clinical setting.

So what do you look for?

- Asymmetric retroperitoneal fascial thickening / fluid / enhancement
- Muscular edema (especially psoas /iliacus muscles) and adjacent fat stranding
- Enhancing retroperitoneal fluid collections (abscesses)
- Extravasated excreted contrast from an involved ureter
- Gas along fascial planes is highly concerning but not commonly seen
- A collapsed IVC indicating septic shock is an ominous sign.

CT often also finds the source of the infection as well (eg – diverticulitis, perforated viscus, perineal abscess, thigh cellulitis).

Both the clinical diagnosis and the imaging diagnosis of retroperitoneal fasciitis can be difficult as there are numerous non-infectious causes of inflammation in the retroperitoneum (pancreatitis / retrocecal appendicitis / pyelonephritis / diverticulitis). Also, gas in the retroperitoneum can be from non-infectious sources (for example recent surgery, benign perforation of a diverticulum or benign subcutaneous emphysema). Third spacing in chronically ill patients with extensive dependent edema can also mimic the CT signs of retroperitoneal fasciitis although usually very symmetric in appearance.

So if you see the typical signs of NRF on a CT, how do you know when to get concerned and call the Radiologist?

Get concerned when the CT findings described above are in the setting of:

- Inordinate pain
- Rapid clinical deterioration
- Immunosuppression
- Recent surgery (especially any gynecologic surgery) or delivery.

Treatment for the Necrotizing form of Retroperitoneal Fasciitis is repetitive surgical debridement (sometimes every 1 to 2 days) and aggressive antibiotic therapy. This once uniformly fatal disease a couple decades ago is now seeing up to 50% survival with this aggressive approach. Most important to survival is early diagnosis and that is where the CT technologist plays a critical part of the team that saves a life.

CONCLUSION:

Retroperitoneal Fasciitis is a disease spectrum ranging from simple fasciitis to the potentially life threatening necrotizing form. Absence of gas and absence of transgression of fascial planes does not exclude the necrotizing form. The diagnosis should be considered in post-surgical and post-partum patients presenting with sepsis and inordinate pain. Early recognition of the typical imaging appearance is crucial in directing timely life-saving treatment.

Dr. Richard Pacini

So what do you look for on CT images?

- Asymmetric retroperitoneal fascial thickening / fluid / enhancement
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- Enhancing retroperitoneal fluid collections (abscesses)
- Extravasated excreted contrast from an involved ureter
- Gas along fascial planes is highly concerning but not commonly seen
- A collapsed IVC indicating septic shock is an ominous sign.

Magnetic Resonance: Imaging of Spinal Infectious Processes:

Discitis/Osteomyelitis/Epidural Abscess

MR is frequently ordered to evaluate for infection in the spine, the 3 most common manifestations being discitis, osteomyelitis and epidural abscess (or epidural phlegmon). Osteomyelitis and discitis are usually seen at the same time as vertebral body osteomyelitis usually occurs first followed by extension into the adjacent disc space. Discitis/osteomyelitis and epidural abscesses are predominately bacterial in origin with the most common pathogen being Staphylococcus aureus. The most common symptom associated with these entities is worsening back pain.

In the early stage of the infectious process MR is the most sensitive modality to evaluate for these entities. Radiographs and CT can confidently diagnose discitis/osteomyelitis when the disease is more advanced. Both radiographs and CT are inferior to MR with regards to the diagnosis of an epidural abscess. A significant epidural abscess may be present which can be too subtle to visualize on CT.

The primary imaging findings of discitis/osteomyelitis are low vertebral body signal on T1 pre-contrast, high vertebral body signal on STIR, increased signal in the disc space on T2 and STIR, disc space height loss, irregularity and destruction of the vertebral body endplates, adjacent paraspinal/psoas muscle edema and abscesses, and vertebral body and disc space enhancement. **See figure 1.**

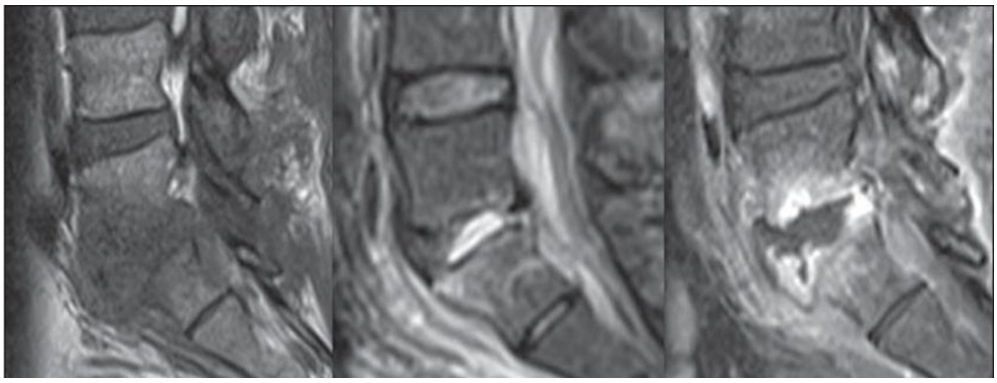


Figure 1 discitis/osteomyelitis: Pre contrast T1, pre-contrast T2 and post-contrast T1 acquisitions show low vertebral body marrow signal on pre-contrast T1, fluid signal within the disc on T2, irregularity of the vertebral body endplates on post-contrast T1 and vertebral body as well as posterior disc space enhancement on post-contrast T1 acquisition. There is also prevertebral soft tissue enhancement on the post-contrast sequence.

Epidural abscesses exhibit confluent high T2 and STIR signal within the anterior, lateral and posterior aspects of the spinal canal with variable degrees of canal narrowing depending on the thickness of the abscess. Epidural abscesses usually, but not always, will exhibit rim-enhancement on post-contrast sequences. **See figures 2 and 3.**

Contrast enhanced sequences are an essential component to evaluate for both discitis and epidural abscess. Contrast enhancement of the disc space, adjacent vertebral bodies and paraspinal soft tissues (most frequently the psoas muscles in the lumbar spine region) combine to increase the confidence in diagnosis of discitis/osteomyelitis. The contrast enhanced images aid in differentiating an epidural space phlegmon versus an epidural space abscess. An epidural space phlegmon is a localized infection without formation of a

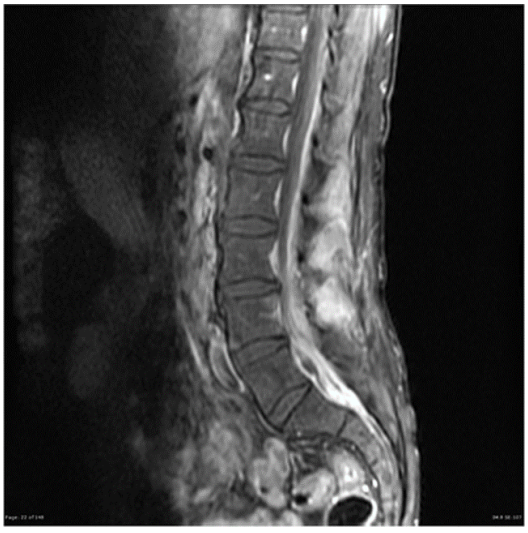


Figure 2 epidural abscess: Post-contrast fat saturated T1 sequence shows a rim enhancing abscess in the anterior aspect of the lumbar canal at the L4 and upper L5 levels. There is additional diffuse posterior epidural space enhancement in the posterior aspect of the canal in keeping with a phlegmon.

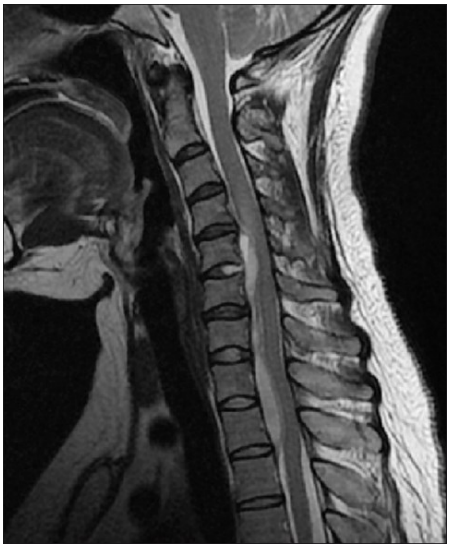


Figure 3 epidural abscess: Sagittal T2 sequence shows high signal loculated epidural space abscess in the anterior aspect of the canal extending from C5 through T2. This results in severe canal narrowing and cord compression at the C5 and C6 levels.

MR: Imaging of Spinal Infectious Processes Continued…

drainable fluid collection which will exhibit variable enhancement, from minimal to diffuse enhancement, and thickening of the epidural space. An epidural space abscess contains internal fluid which consequently will appear as a rim -enhancing fluid collection on the post-contrast acquisitions.

Patients with spine infections are typically in pain and may have many medical co-morbidities which increase the challenge of imaging with MR. A frequent occurrence with these patients is the need to acquire multiple acquisitions due to excessive patient motion. This can be especially challenging when the entire spine needs to be imaged which may require multiple sequences to be obtained and may be a major time sink. If at all possible trying to get the patient to understand that they may have a serious disease process present for which imaging is crucial for diagnosis may aid in getting the patient to comply if the early image acquisitions are limited by motion. If you, as the MR technologist, notice a potential abnormality and the patient is not complying with the exam the radiologist could be contacted to try to tailor the exam to obtain the highest yield sequences, which are typically the post contrast sagittal and axial T1 sequences as well as the pre-contrast sagittal and axial T2 sequences. Sequences such as the axial pre-contrast T1 acquisitions could be dropped from the exam in those instances of patient non-compliance. If the patient is tolerant all of the sequences in the protocol for the spine MR without and with contrast should be obtained.

Additionally if the patient is in significant pain it may be useful to have the ordering clinician attempt to help reduce the pain with medication prior to the exam (if the patient's medical condition allows this) to facilitate a less disruptive exam. This could however be a double edged sword as pain relief may cause the patient to be less inhibited/ forgetful and therefore less compliant with lying still.

If you see an abnormality that you feel is out of the ordinary and worrisome (such as an epidural fluid collection/abscess resulting in canal narrowing or cord compression) do not hesitate to send the early obtained sequences and contact the radiologist which may facilitate a more rapid diagnosis and may facilitate a quicker consultation with the neurosurgeon/spine surgeon who may be needed to decompress the patients spine with a surgery.

Additionally notify the radiologist if you notice a fluid collection within the spinal canal that appears to extend above or below the level of imaging. It is not uncommon for epidural abscesses to extend above or below the imaged area. For example in the lumbar spine an epidural abscess may extend into the lower thoracic spine. In these instances it would be beneficial to image the complete extent of the epidural abscess at the initial time of imaging for treatment as well as follow-up purposes.

Another point with regards to imaging of the thoracic spine in general (whether just imaging a routine outpatient for chronic pain or for imaging a more acutely ill patient with a potential spine infection) is that a scout localizer needs to be performed at the start of the exam so the vertebral body and disc space levels can be precisely defined. Every thoracic spine should have an adequate scout localizer which includes the cervical spine (and ideally the lumbar spine). The obtained axial images should be linkable, on the radiologist's PACS/viewing station, with the scout localizer. This scout localizer is used to number the thoracic vertebral bodies which is necessary to precisely determine the level of abnormality in case of the need for surgery.

Lastly, fat-saturated post-contrast T1 acquisitions are obtained to allow better visualization of epidural abscesses as well as surrounding paraspinal inflammation. Fat-saturated sequences may be significantly degraded by the presence of spinal hardware. In those cases where the fat saturated post contrast sequences are significantly degraded by hardware artifact, non-fat saturated post-contrast T1 acquisitions can be additionally obtained which may be less affected by the artifact.

Dr. Nicholas Statkus

Whether just imaging a routine outpatient for chronic pain or for imaging a more acutely ill patient with a potential spine infection, a scout localizer needs to be performed at the start of the exam so the vertebral body and disc space levels can be precisely defined.

References:

1. Figure 1 from Radiol Bras vol.46 no.3 São Paulo May/June 2013
2. Figure 2 from radiopaedia.org (epidural abscess and facet joint septic arthritis)
3. Figure 3 from Ohyagi M, Ohkubo T, Taniyama T, Tomizawa S, Okawa A, Yokota T, Mizusawa H. Spinal epidural abscess caused by *Bacteroides fragilis*group after dilation and curettage for incomplete abortion. J Global Infect Dis 2012;4:132-4

Diagnostic Imaging: Pediatric Lead Toxicity

Etiology, Pathophysiology, and Radiology

ETIOLOGY OF LEAD TOXICITY

Lead is a ubiquitous and versatile metal and has been used since ancient times and the history of public exposure to lead, in food and drink, is extensive. Lead poisoning was common in Roman times because of the use of lead in water pipes and wine containers.

As early as 1904, it was determined that lead paint in the home was responsible for poisoning children. Despite that, lead paint was not banned until 1978. Deteriorating paint in pre-1979 housing remains the most common source of lead exposure in children accounting for 70% of elevated levels. Lead in water pipes is also a source of lead exposure as evidenced by the water crisis in Flint, Michigan. Other sources of exposure include batteries, putty, cement, imported canned food, jewelry and cosmetics.

Exposure in children is generally from ingestion of products containing lead. Children are more susceptible than adults to the adverse effects of lead exposure. The physiologic uptake rates of lead in children are greater than in adults and are more susceptible to the affects of lead. Absorption of lead is inversely proportional to particle size. Large particles such as paint chips are poorly absorbed whereas fine dust particles licked from fingers or other objects are much more readily absorbed. Absorption is also inversely proportional to nutritional status. Lead absorption is augmented in the presence of iron, zinc, and calcium deficiency. High fat diet is also associated with increased absorption. Dietary components such as phytates found in green leafy vegetables bind lead particles increasing their elimination. Absorption is inversely proportional to chronological age. In general 30-50% of lead ingested by children is absorbed vs 10% of that ingested by adults.

PATHOPHYSIOLOGY OF LEAD TOXICITY

Lead exerts numerous adverse mechanisms of toxicity. Lead has a high affinity for sulphydryl groups and is particularly toxic to multiple enzyme systems. Many of lead's toxic effects also result from lead's ability to inhibit cellular function requiring calcium. Lead binds to calcium activated proteins with a much (105 times) greater affinity than calcium.

Encephalopathy caused by lead poisoning is considered the most detrimental health hazard. Microvasculature of a developing child's brain is uniquely susceptible to high level lead toxicity, characterized by cerebellar hemorrhage, increased blood-brain barrier permeability and vasogenic edema.

Lead toxicity can also cause neuropathy and involve the motor, sensory and autonomic systems. Lead also interferes with heme biosynthesis and cause anemia at high blood levels. At low levels, lead causes microcytosis (decreased volume and hemoglobin in a red blood cell) and a compensatory increase in the number of red blood cells. Lead can also cause acute or chronic nephropathy. Chronic nephropathy can lead to hypertension and gout.

Accumulation of lead in bone cells has toxic consequences altering bone cell function. Most retained lead in the human body is ultimately deposited in bones. A lead line refers to the metaphyseal line of increased radiodensity that occurs in lead poisoning. This line is due to impaired resorption of calcified metaphyseal cartilage.

RADIOLOGIC FINDINGS

- Dense Lines at Metaphyses—a "lead line" or "lead band". This appears at a blood lead level between 70-80 micrograms/dl (Figure 1)
- Punctate radiodensities in the GI tract of a child who ingested lead paint chips. (Figure 2)
- Chronic lead exposure-alternating bands of sclerosis indicating different periods of exposure (Figure 3)

Dr. Amy Hayes

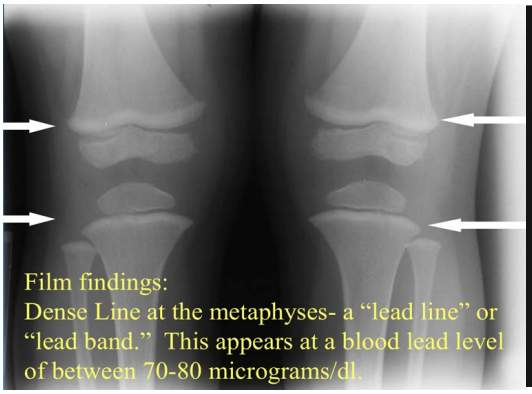


Figure 1

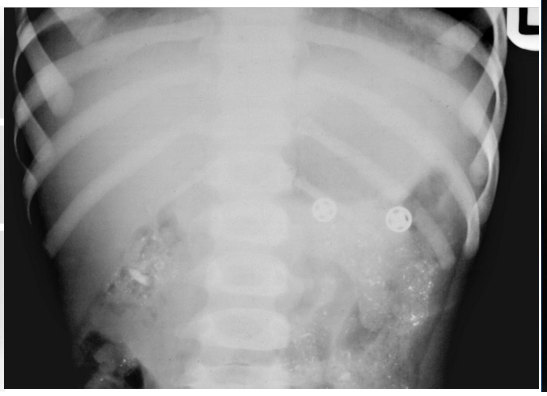


Figure 2

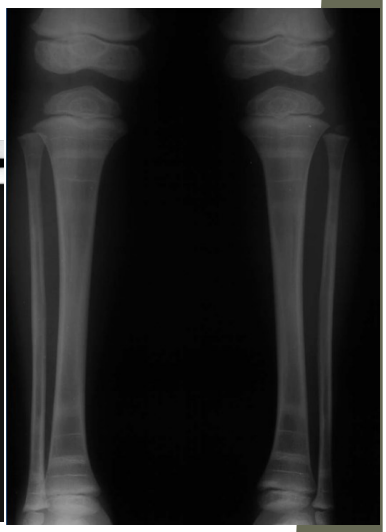


Figure 3

"On the Case"

55 year old male presents with massive hemoptysis.

Images from a CT pulmonary angiogram are shown below.

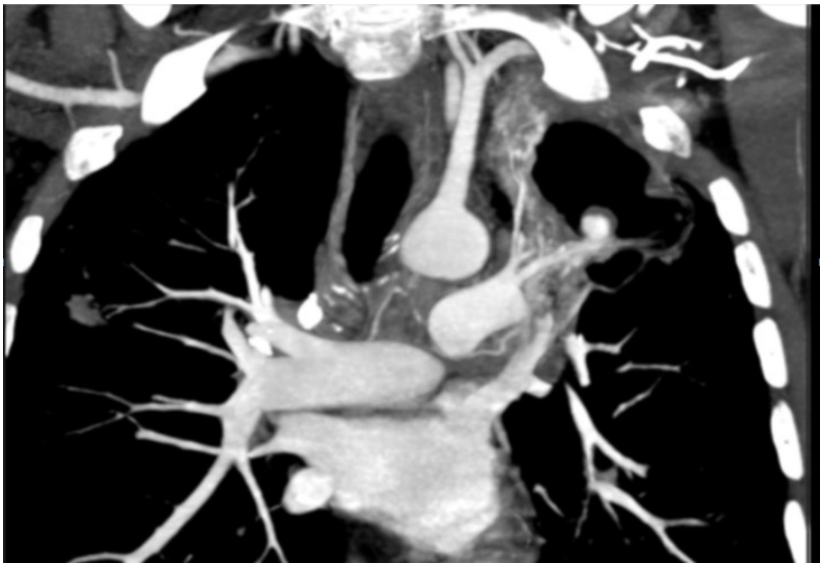
1. What are the findings?

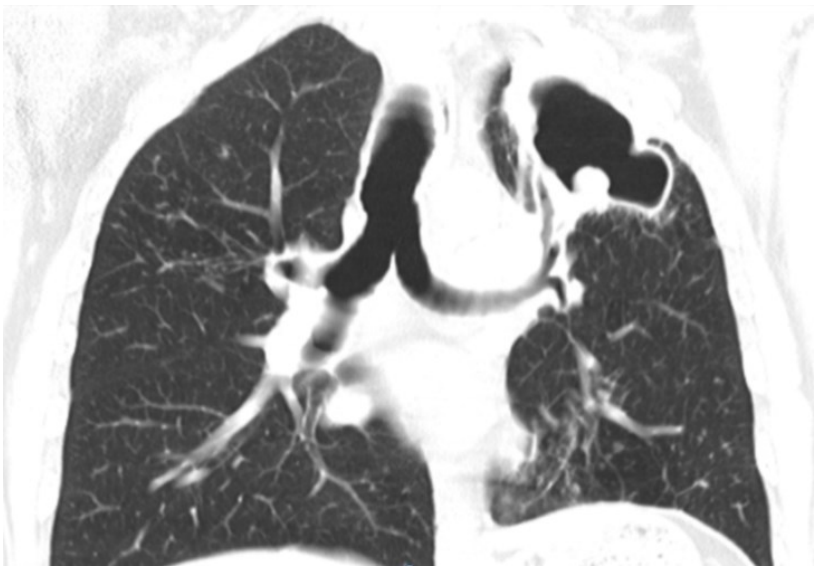
2. What is the diagnosis?

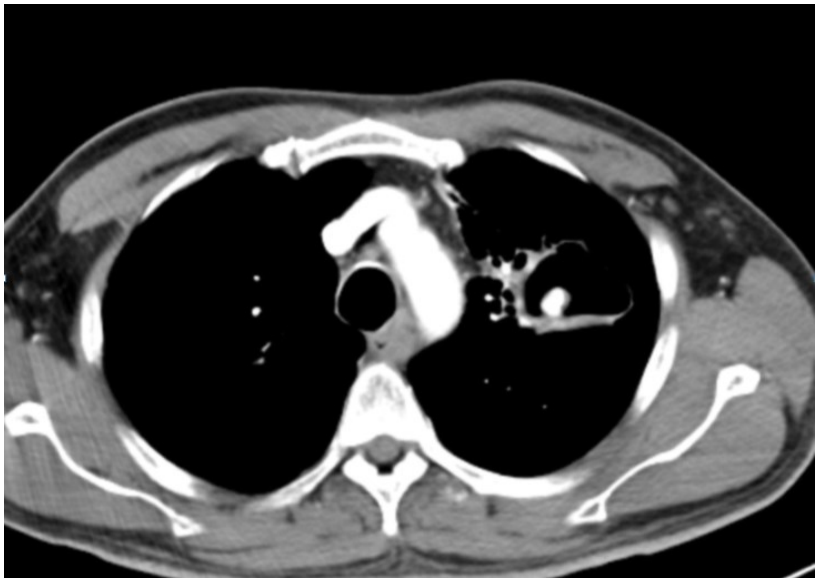
3. What interventional procedure would you do next?

Please Submit your answers to Andi, AMIC Client Services Manager, by April 30th to be eligible for a prize.

Andi Dresen
Client Service Manager
a.dresen@advmedimaging.com







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