The Trauma Professional's Blog

Trauma MedEd #63

Topic: Fat Embolism Syndrome Subscribe: www.TraumaMedEd.com February 2019

Fat embolism syndrome (FES) is one of those clinical problems that trauma professionals read about during their training, then rarely ever see. Although the clinical manifestations are frequently mild, they can progress rapidly and become life-threatening. This newsletter will help you better understand this condition, and provide details on diagnosis and treatment.

Fat Embolism vs Fat Embolism Syndrome

Fat embolism syndrome (FES) is a constellation of findings that arise from a single, unified cause: the escape of fat globules into the circulation (fat embolism). The ultimate resting places of those globules determine the specific manifestations of FES seen in clinical practice. When it occurs, it typically **becomes apparent 24 to 72 hours after injury.**

Simple fat embolism **occurs to some degree any time tissues containing fat are manipulated or injured**. It has been demonstrated during plastic surgical injections for cosmetic purposes and lipid infusions. It is **more frequently seen with orthopedic injuries, especially those involving the femurs and pelvis**. And it makes sense that the more fractures that are present, the more likely fat embolism will occur. **Embolism is also known to occur when performing orthopedic procedures**, particularly those involving the marrow cavity (intramedullary nailing), but has also been reported in total knee and hip procedures.

INSIDE THIS ISSUE

SPEAKING ENGAGEMENTS

29TH ANNUAL DUKE TRAUMA SYMPOSIUM

LOCATION: HILTON DURHAM, DURHAM NC

MARCH 28, 2019

"HOW TO IMPRESS THE TRAUMA SURGEON"

TEXAS HEALTH 21ST ANNUAL TRAUMA SYMPOSIUM

LOCATION: OMNI FORT WORTH HOTEL, FORT WORTH, TX

APRIL 4, 2019

"MASSIVE TRANSFUSION FOR TRAUMA: WHERE IS THE BLEEDING EDGE?

Fat embolism syndrome has a generally reported incidence of 1 - 10%, although I believe that is on the high side. I see a case every 3 – 4 years in a predominantly blunt, fracture-laden practice. Fat embolism without symptoms occurs much more frequently. A study from 1995 using transesophageal echo found evidence of emboli in 90% of patients with long bone fractures.

But how do these fat globules get into the circulation and produce such chaos? We know that they can be mechanically pushed into small venules when tissues containing fat cells or bone marrow are injured. In bone, there are numerous small venules located throughout that are anchored to it. When the bone is fractured, these venules tear and are held open so yellow (fatty) marrow can be pushed into them.

If enough emboli enter the blood stream, they may accumulate in the end vessels of tissues and block flow. Although this is a simple and appealing explanation, it may not be the full story. If the emboli primarily occur during and after injury, why does it take several days for the full-blown syndrome to develop?

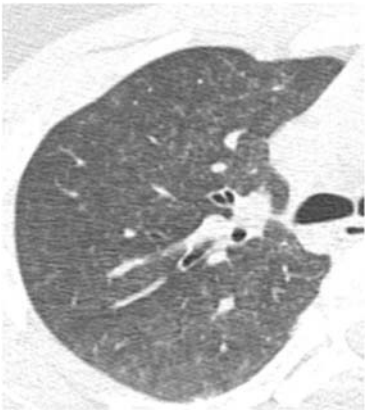
A likely explanation is that the **fat globules begin to degrade while in the circulatory system**. Breakdown into free fatty acids results in the release of a cascade of cytokines and other mediators. The inflammatory response around the end vessels create the gross pathology that we associate with fat embolism syndrome.

Clinical Manifestations Of FES

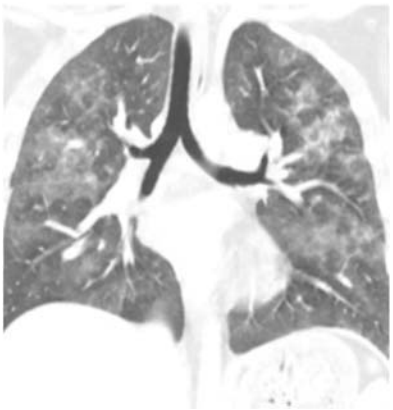
There are three organ systems that are classically involved in FES: **pulmonary, CNS, and skin**. Manifestations generally begin between 24 and 72 hours after injury. In rare cases, symptoms can begin within 12 hours. In my experience, these tend to be the ones that become the most severe and are frequently life-threatening.

Pulmonary (95% of cases): This is the most common manifestation of FES, and may occur without other signs and symptoms. Nearly all patients develop some degree of hypoxia. Progressive tachypnea and mild tachycardia may provide the first clinical clue if oxygen saturation is not being monitored.

Chest x-ray is usually unremarkable early on. And once the syndrome has developed, it is generally not helpful. CT scan is useful for defining the extent of pulmonary injury, but lags the clinical picture by several days. Findings are non-specific, usually consisting of small, ground-glass opacities in the periphery.



In this example, the opacities are very small and difficult to see.

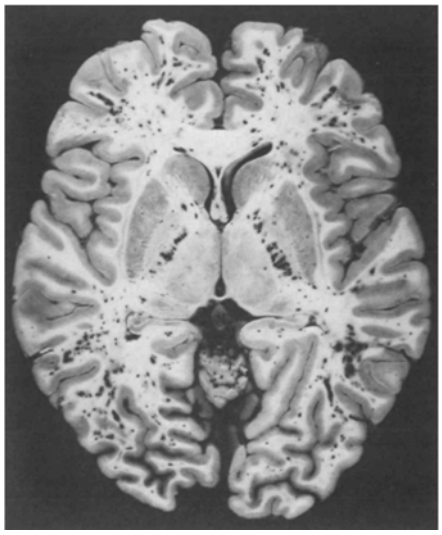


They're a little more obvious here!

Other CT findings include **small pulmonary nodules** in the upper lobes or along peripheral pulmonary vessels. These are thought to be areas of obstruction caused by the emboli. **Nonspecific pleural effusions** may be seen, and bronchial thickening has also been described. Rarely, fat globules may be seen in the lower extremity veins or IVC, and should immediately raise suspicion for developing FES even before symptoms develop.

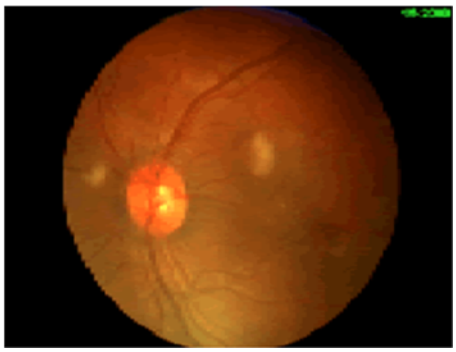
CNS (60% of cases): If they occur, CNS changes generally crop up **after the pulmonary manifestations** begin. Generally, they start as mild confusion, but can progress to decreasing level of consciousness and even coma. Focal neurologic deficits are occasionally seen, and seizures can occur.

The actual mechanism behind this appears to be very similar to the skin changes which will be described in the next section. Emboli occur in vessels predominantly in the white matter of the brain. This leads to petechial hemorrhages, which are likely due to the inflammatory mechanisms previously described.



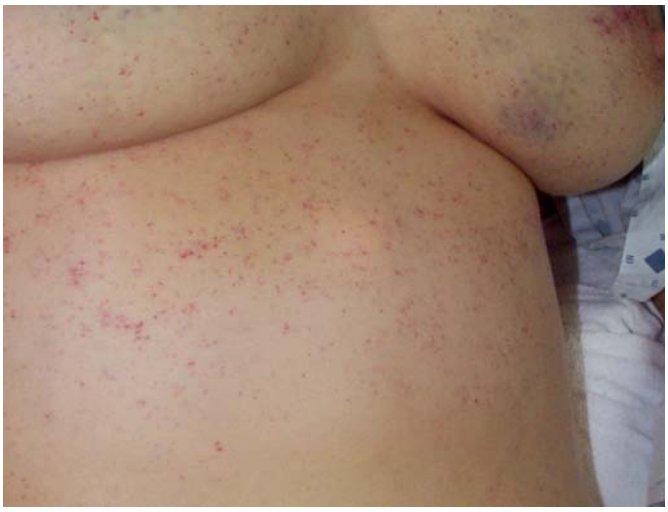
Note the numerous dark petechiae visible in the white matter in this specimen.

Retinal exam can also show evidence of fat embolism. Fat globules may actually be seen in the retinal vessels early.



Note the fat globules at the 9:30 and 2:00 positions to the optic nerve in the image above.

Skin (33% of cases): The most recognizable sign of FES is the petechial skin rash. This rash usually involves the torso, and axillary petechiae are very common. It can spread to involve the head and neck, and occasionally the extremities. Subconjunctival hemorrhages are sometimes seen. The rash tends to be transient and usually lasts only a few days. Here is an example of the classic petechial rash.



Other findings: Fat globules may be found in the urine in patients with FES. However, they are commonly present in patients with long bone fractures, so their presence is not helpful or predictive. Nonspecific findings such as fever, leukocytosis, anemia, and thrombocytosis are also relatively common. In severe cases, cardiac dysfunction, hypotension, and peripheral hypoperfusion can occur. I have personally seen necrosis of fingers and toes from a very severe case.

Unfortunately, the "classic" triad of mental status changes, skin rash, and pulmonary insufficiency are seen in only a small minority of patients. Typically, only one or two signs and symptoms appear at the same time, making diagnosis a bit challenging.

Diagnosis Of Fat Embolism Syndrome

A number of scoring systems have been developed to identify FES (Gurd's and Wilson's criteria, Schonfeld's criteria, Lindeque's criteria to name a few). Unfortunately, none of these are helpful. They were developed in the 1980s as part of the authors' studies on the use of steroids for treatment, and no one else has taken the time to study their sensitivity and specificity.

Diagnosis of FES is primarily clinical. It relies upon recognition of the principal findings on physical exam, and exclusion of more common conditions that may mimic it.

Here is a template for diagnosing FES:

Is your patient at risk? The vast majority of these patients will have fractures. One, or especially two or more long bone fractures (mostly the femur) are usually present. Other fractures that add risk are those involving the pelvis or bones that contain marrow, such as the ribs and sternum. Patients who have just undergone fracture repair are also at risk and will be discussed in the next section. Finally, patients who have had intraosseous lines placed are also at risk, regardless of the type of infusate.

What signs or symptoms have developed? Skin changes are very suggestive of FES if your patient is at risk. However, rashes are common manifestations of contact allergies, drug reactions, infectious diseases, and many other conditions. If those are ruled out, then the presence of risk factors plus a rash is sufficient to make the diagnosis.

Mental status changes are more difficult to pin on FES, even though it is a more common initial presentation than the rash. Since this is a trauma patient, you must rule out delayed manifestations of head trauma. Urgent CT of the head is required to do so. And typically, there will be no specific findings that point to FES. It is always a diagnosis of exclusion.

Pulmonary dysfunction requires a search for the usual suspects. A good physical examination of the chest coupled with a chest x-ray will help identify pneumothorax, hemothorax, or pneumonia. A chest CT may be indicated if pulmonary embolism is suspected.

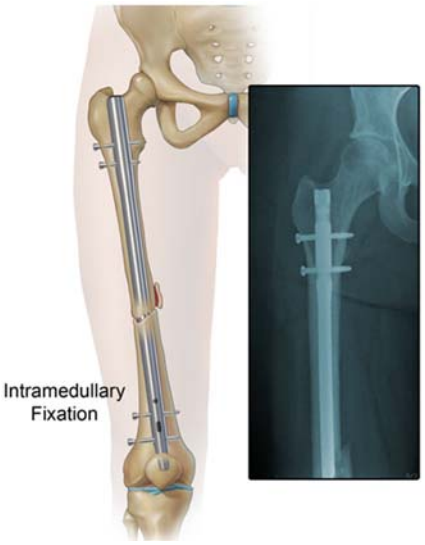
Once other more common clinical problems have been eliminated, you are left with the diagnosis of FES. There are no specific lab tests to draw, and more invasive studies are neither helpful nor indicated. **Fat embolism syndrome is a diagnosis of exclusion.**

FES And Orthopedic Surgery

Regardless of the exact mechanism for the development of fat embolism syndrome, in trauma it most commonly occurs when the medullary (bone marrow) cavity of a long bone is violated. This occurs first when the bone is fractured, and again when it is instrumented for fixation. The initial shower of emboli cannot

be prevented. However, ongoing emboli can be reduced with early fixation. This can be in the form of a good splint, or surgical external or internal fixation.

One type of internal fixation, intramedullary (IM) nailing, has been associated with embolism and FES for some time. This technique was introduced 80 years ago and has been refined significantly since. Here is a picture of a femur with an IM nail.



The nail is inserted proximally near the greater trochanter. The marrow cavity is first reamed to make insertion of the nail easier. This causes a number of changes in the physiology of and pressures within the marrow cavity. Pressure increases during the initial reaming, and hits a peak when the reamer enters the distal fragment. Once complete, there are no further increases as the nail is inserted. However, these pressure changes alter medullary blood flow and allow emboli to enter the venous system.

Reaming is actually beneficial in several ways. It simplifies and shortens the surgical procedure. And in animal models there is evidence that bone debris from the reaming process collects at the fracture site, creating an autograft that may improve healing.

A surgical group in Ireland has been using a novel technique for lavaging the marrow cavity during fixation for several years. Once the bone is entered proximally, a cut piece of suction tubing is inserted into the end of the bone. Suction is then applied for 2-3 minutes. The procedure continues, including reaming, then the suction procedure is repeated. Unfortunately, FES is uncommon, so it is difficult to judge whether their technique really works. The authors believe it is safe, but recommend formal studies to prove efficacy.

Use of an additional venting hole between the trochanters has also been studied in a small randomized trial. This allows for drainage of marrow during the reaming process, reducing any pressure rise. The number of embolic events detected using transesophageal echo was significantly reduced in the vented group (20% vs 85% of patients).

References:

1. *A Simple and Easy Intramedullary Lavage Method to Prevent Embolism During and After Reamed Long Bone Nailing. Cureus 9(8):e1609, Aug 2017.*
2. *Relevance of the drainage along the linea aspera for the reduction of fat embolism during cemented total hip arthroplasty. A prospective, randomized clinical trial. Arch Ortho Trauma Surg 119:146, 1999*

Prevention And Treatment Of Fat Embolism Syndrome

FES is uncommon, yet can be highly morbid or even fatal. Treatment is generally supportive, but there are a number of measures that can be taken to reduce the incidence.

Splint all long bone fractures early and well. Repeated movement at the fracture site increases the occurrence of fat embolism, which may in turn lead to FES. This tip applies to prehospital and emergency department providers alike. Do not let your patient leave the scene or ED without proper splinting.

Watch your fracture repair timing. Severely injured patients are not only anatomically damaged, but physiologically as well. They are as healthy as they are going to be when they roll in the door, so early definitive fixation (within 24 hours) is best done before they become “too sick.”

Consider using the “venting hole” technique described above to try to reduce embolism.

Use prophylactic steroids rarely, if ever. All of the studies on steroid use are small and mostly flawed in one way or another. FES is too rare to expose a large number of patients to the risk of receiving steroids. They should only be considered after seriously weighing all risks vs benefits.