Review Article

Fat Embolism and Fat Embolism Syndrome

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Abstract

Fat embolism (FE) occurs frequently after trauma and during orthopaedic procedures involving manipulation of intramedullary contents. Classically characterized as a triad of pulmonary distress, neurologic symptoms, and petechial rash, the clinical entity of FE syndrome is much less common. Both mechanical and biochemical pathophysiologic theories have been proposed with contributions of vascular obstruction and the inflammatory response to embolized fat and trauma. Recent studies have described the relationship of embolized marrow fat with deep venous thrombosis and postsurgical cognitive decline, but without clear treatment strategies. Because treatment is primarily supportive, our focus must be on prevention. In trauma, early fracture stabilization decreases the rate of FE syndrome; however, questions remain regarding the effect of reaming and management of bilateral femur fractures. In arthroplasty, computer navigation and alternative cementation techniques decrease fat embolization, although the clinical implications of these techniques are currently unclear, illustrating the need for ongoing education and research with an aim toward prevention.

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Fat embolism (FE) is defined as the presence of fat globules in the pulmonary or peripheral circulation, and FE syndrome (FES) refers to the clinical symptoms that follow an identifiable insult; it can result in the triad of respiratory distress, neurologic symptoms, and petechial rash. Frequently, FE occurs after trauma and during orthopaedic procedures.[1,2] Although typically considered benign, recent studies have identified possible links to neurocognitive impairment and deep venous thrombosis (DVT) formation.[3-5] Although FES exhibits potentially life-threatening effects, it is much less common. Despite its original description in the 17th century, FES remains incompletely understood. Both mechanical and biochemical mechanisms have been proposed to explain the clinical picture of FES. However, likely, it is a result of vascular obstruction, the body's response to embolized fat, and the trauma-induced inflammatory response. Diagnosis can be challenging, relying on a combination of clinical symptoms, laboratory results, and imaging findings. Further research directions include improving our understanding of predicting those at the risk of developing FES, accurately diagnosing the condition, and recognizing the more subtle effects of FE.

History

The clinical study of FES began in 1861 with Zenker, who reported the presence of fat droplets in lung capillaries of a railroad worker who died from a crush injury.[6] This report

remained an isolated case until 1865 when Wagner described the correlation of FE with fractures and attributed the origin of fat in the lungs to bone marrow. Von Bergmann was the first to describe the symptoms of FES, and in 1873, he reported a patient who fell from a roof and sustained a comminuted distal femur fracture. Sixty hours after the injury, he developed confusion, dyspnea, and petechiae and died after 19 hours. A massive pulmonary FE was found at autopsy. Czerny further examined the neurologic symptoms of FES and described them in 1875.[7] Although these observations were key in establishing a clinical pattern, it was not until the 1920s that the two main pathophysiologic theories of FES were first proposed. In 1924 Gauss[8] described the mechanical theory, and in 1927, Lehman and Moore[9] theorized about a biochemical explanation. Finally, in 1970, Gurd[10] presented the first set of diagnostic criteria (Table 1) based on his experience with a series of 100 patients with long bone fractures and coined the term "fat embolism syndrome."

Epidemiology

Fat in the peripheral circulation (FE) occurs fairly frequently. At autopsy, pulmonary FE has been found in 68% to 82% of blunt trauma patients.[1,2] During orthopaedic procedures, fat globules have been observed regularly passing through the heart and pulmonary circulation on ultrasonography (Figure 1). In 1995, Christie et al[11] performed transesophageal echocardiography (TEE) in 111 orthopaedic surgeries, including reaming of tibia and femur fractures, as well as cemented and uncemented hemiarthroplasty. Echogenic material was found traveling through the heart in 87% of procedures, and this material was confirmed as FE in a subset of 12 patients with blood sampling from the right atrium. Major emboli greater than 1 cm were found in 43% of patients.

The clinical entity of FES is much less common. In the older literature, it has been reported in up to 30% of orthopaedic trauma patients; however, recent studies show a much lower incidence.[6,7,12] In one of the largest clinical studies, a group from the Harborview reviewed 10 years of patients from their trauma database.[6] Using Gurd criteria,[10] 27 cases of FES were identified from 3,000 patients with long bone fractures with an incidence of 0.9%. The average age of patients was 31 years, and the onset was typically 24 to 48 hours after an injury. Ninety-five percent of these patients had fractures of the lower extremities, and it was more common in closed fractures. A more recent study in 2008 examined the *International Classification of Diseases, Ninth Revision* codes from the National Hospital Discharge Survey over a 26-year period including one billion patients.[13] Among all patients with fractures, the incidence was 0.17%. Of isolated fractures, femur fractures were the most common with a rate of 0.54%. This investigation excluded fractures of the femoral neck, which had only 0.09% incidence. Multiple fractures that included the femur had the highest incidence of 1.29%. The incidence was more common in male subjects, with a relative risk of 5.7, and it was more common in those aged 10 to 40 years.

Pediatric patients have a much lower incidence of FES. Although FE have been identified in 30% of pediatric cadavers at autopsy,[14] Stein et al[13] found no cases of FES among 1,178,000 children in their discharge database study. This discrepancy may be the result of the lower fat content in pediatric patients, where hematopoietic cells occupy nearly 100% of the volume at birth and decrease by 10% in each decade of life.[14] In addition, marrow fat composition in children may play a role. Children have a greater proportion of palmitin and stearin, which are less likely to cause an inflammatory response in comparison to olein found in adults.[13] However, patients with Duchenne muscular dystrophy warrant special consideration because they develop FES at a relatively high rate of 1% to 20% after minor trauma and fractures.[15]

Although FES is most commonly associated with trauma, it has also been reported rarely in nontrauma patients. Case reports document the occurrence of FES during bone marrow harvest, lung transplant, cesarean section, liposuction, and cosmetic procedures.[7,12,16,17]

Clinical Presentation

The classic triad of symptoms of FES is respiratory distress, neurologic changes, and a petechial rash.[10] Pulmonary symptoms occur first, typically 24 to 72 hours after trauma; but symptoms have been reported as early as 12 hours. A large embolus can cause sudden cardiopulmonary collapse; but more often, FES has an insidious onset with dyspnea, tachypnea, and hypoxemia. About half of all patients with FES develop respiratory failure that necessitates mechanical ventilation.[18] In a patient under anesthesia, findings include respiratory deterioration with hypoxemia, pulmonary edema, and decrease in lung compliance.[7]

Neurologic symptoms are present in up to 80% of patients, and usually, although not always, this symptoms occur after pulmonary symptoms. They begin with confusion and agitation similar to delirium, and it can progress to focal deficits, such as hemiplegia and aphasia, as well as seizures and coma. Commonly, upper motor neuron signs are also present.[7,12]

Table 1

Criteria for the Diagnosis of Fat Embolism Syndrome

Criteria	Findings	Points
Gurd criteria[a]	Major	
	Respiratory insufficiency	—
	Cerebral involvement	—
	Petechial rash	—
	Minor	—
	Fever	—
	Tachycardia	—
	Retinal changes	—
	Jaundice	—
	Renal changes	—
	Anemia	—
	Thrombocytopenia	—
	Elevated ESR	—
	Fat macroglobulinemia	—
Schonfeld criteria[b]	Petechia	5
	Chest radiograph changes	4
	Hypoxemia (PaO2 < 9.3 kPA)	3
	Fever (>38C)	1
	Tachycardia (>120 bpm)	1
	Tachypnea (>30 bpm)	1
Lindeque criteria[c]	Sustained Pao_2 < 8 kPa	—
	Sustained PCo_2 > 7.3 kPa or pH < 7.3	—
	Sustained respiratory rate > 35 bpm	—
	Increased work of breathing (dyspnea, accessory muscles, tachycardia, anxiety)	—

ESR = erythrocyte sedimentation rate, FES = fat embolism syndrome
[a] At least 1 major feature and 4 minor features needed for diagnosis.
[b] Cumulative score >5 required for diagnosis.
[c] One of the criteria indicates a diagnosis of FES.

Although part of the "classic triad" of symptoms, a petechial rash occurs in only 20% to 50% of patients. Its typical distribution is over the head, neck, thorax, axillae, subconjunctival space, and oral mucous membranes[12] (Figure 2). A similar rash can be found in sepsis and disseminated intravascular coagulation; however, the rash of FES is only found anteriorly on the body in nondependent areas, and it has never been reported on the back. Theoretically, this pattern of rash occurs because in a supine patient, the fat droplets (which float like oil on water) accumulate in the aortic arch and are then distributed through the carotid and subclavian vessels to nondependent areas.[12]

Although found to be nonspecific, other frequently reported signs and symptoms include tachycardia, hypotension, right heart strain, fever, retinopathy, renal changes, and coagulopathy. Overall mortality rates are 5% to 20%, usually because of respiratory failure or right heart failure.[7,12]

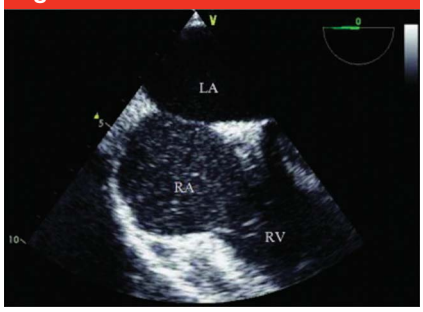
Pathophysiology

Two pathophysiologic mechanisms were proposed for the clinical manifestations of FES: the mechanical and biochemical theories. The mechanical theory was first presented by Gauss[8] in 1924. He proposed that trauma and fractures of long bones disrupts fat in the marrow and also tears intraosseous blood vessels. Typically, veins are characterized as having weak, flexible walls; however, in bone, they are contained within calcified tubules with rigid perivascular sheaths. This allows ruptured vein ends to remain open, and the negative venous pressure can draw free fat globules into the circulation. In cases of arthroplasty and during intramedullary instrumentation, intramedullary pressure increases forcing fat into the veins.[19] Once fat enters the circulation, it can create mechanical emboli and focal ischemia.

It was originally thought that to pass from the venous to the arterial circulation, the fat material (because of its large size) would have to pass through a foramen ovale from the right to the left atrium (present in 20% to 25% of adults),[20] either one that remained open from birth or one that reopened because of elevated pulmonary artery pressures. However, neurologic symptoms and skin lesions occur in patients without these anatomic variants. Other explanations are that fat is able to deform to travel through capillaries or that it passes through arteriovenous shunts present around the lungs.[20] Although logical, this theory does not explain the 24- to 72-hour delay in presentation, and just having fat in the circulation, even a large quantity of fat, does not in itself lead to the development of FES.[11]

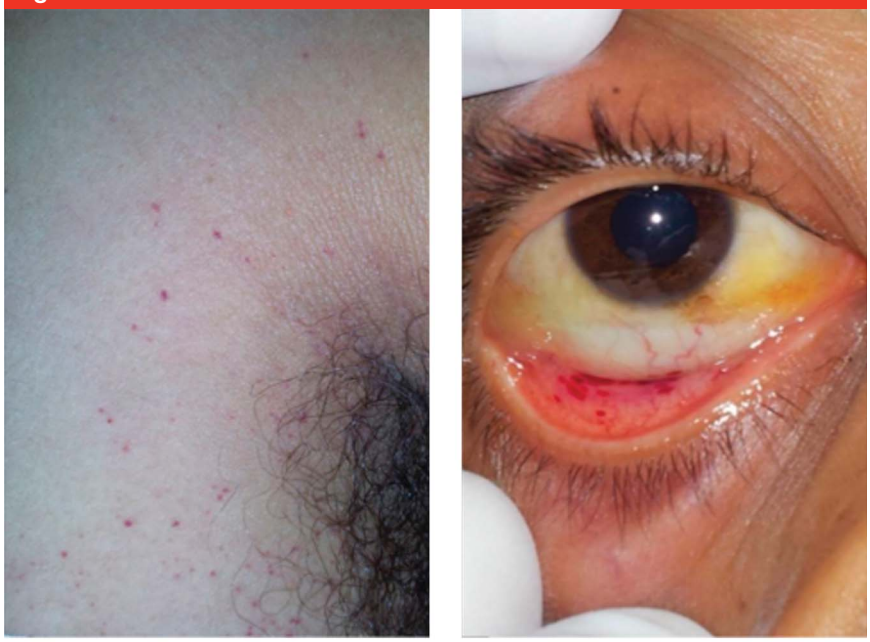
To explain cases of atruamatic FES, Lehman and Moore[9] described a biochemical theory in 1927. They proposed that after an insult or trauma, fat was mobilized from body stores and embolized into the tissues, initiating an inflammatory response. Since that time, we have learned that

Figure 1



Transesophageal echocardiography images showing microemboli in the right atrium during total knee arthroplasty. LA = left atrium, LV = left ventricle, RA = right atrium, RV = right ventricle. (Reproduced with permission from Zhao J, Zhang J, Ji X, et al: Does intramedullary canal irrigation reduce fat emboli? A randomized clinical trial with transesophageal echocardiography. *J Arthroplasty* 2015;30[3]:451-455.)

Figure 2



A **B**

Clinical images of axillary petechiae (**A**) and subconjunctival hemorrhage (**B**). (Reproduced with permission from Maghrebi S, Cheikhrouhou H, Triki Z, Karoui A: Transthoracic Echocardiography in Fat Embolism: A Real-Time Diagnostic Tool. J *Cardiothorac Vasc Anesth* 2017;31[3]:e47-e48.)

bone marrow fat embolized to the lungs causes the local releases of lipase, which breaks fat down into free fatty acids and glycerol. Free fatty acids are toxic to endothelial cells and cause vasogenic edema and hemorrhage.[20] These conditions release proinflammatory cytokines, such as tumor necrosis factor-alpha, interleukin (IL)-1 and IL-6, which can cause acute respiratory distress syndrome (ARDS). Elevated acute-phase reactants, such as C-reactive protein, have also been observed, and these reactants can cause lipids in the blood to agglutinate into larger molecules, which can occlude vessels. In addition, bone marrow fat is prothrombotic, and in the circulation, it is quickly covered in platelets and fibrin setting off the coagulation cascade, leading to thrombocytopenia and, in extreme cases, disseminated intravascular coagulation.[20]

In reality, the clinical symptoms of FES are likely a combination of mechanical vascular obstruction and the body's inflammatory response to trauma and embolized fat (Figure 3).

Diagnosis

Diagnostic Criteria

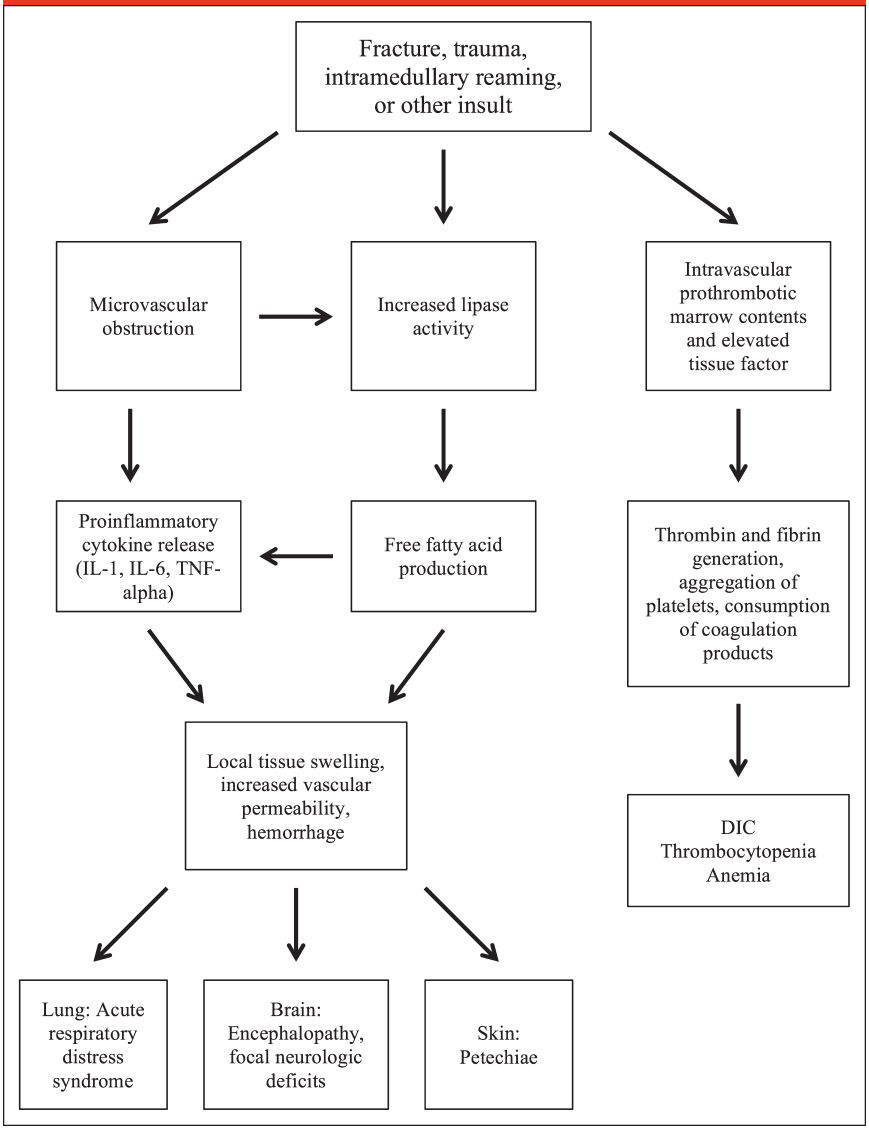
One of the challenges in the study and identification of FES is that there is no benchmark test. Gurd[10] was the first to identify diagnostic criteria based on a series of 100 patients. He divided his observations into major and minor criteria and suggested needing at least one major and four minor findings to make the diagnosis (Table 1). Lindeque focused on respiratory symptoms and created criteria based on a small series of 55 patients with long bone fractures and decreased oxygen saturation (Table 1).[7,12] Schonfeld described another set of criteria based on his opinion of the most important features[7,12] (Table 1). He weighted findings based on their observed specificity and chose scores of 5 or greater to define FES. Although these criteria have tried to standardize the diagnosis, they are all based on small series, and none have been prospectively validated.

Laboratory Studies

There are no laboratory tests specific to FES (Table 2); however, common findings include anemia, thrombocytopenia, and elevated inflammatory markers.[7] Elevated serum lipase can cause hypocalcemia, and albumin binds to free fatty acids leading to a decrease in free albumin. The presence of fat globules in the blood and urine has also been observed, but this is not specific to FES.[12,20]

Inflammatory cytokines have also been investigated as a predictor of FES. Based on the relationship of FES with systemic inflammation, Prakash et al[21] examined IL-6 levels in trauma patients. They found that at 12 hours after injury, IL-6 was significantly elevated in patients who went on to develop FES, diagnosed using Gurd criteria.

Figure 3



Flowchart showing proposed pathophysiologic mechanisms for the clinical manifestations of fat embolism syndrome. DIC = disseminated intravascular coagulation. (Reproduced with permission from KosovaE, Bergmark B, Piazza G: Fat embolism syndrome. *Circulation* 2015 January 20;131 [3]:317-320.)

Although fat in the lungs is nonspecific and can be seen in multiorgan failure and sepsis, bronchoalveolar lavage may also aid in diagnosis. Studies have attempted to determine specific characteristics of the amount and composition of this fat in FES. In FES, it has been shown that bronchial lavages had >30% of alveolar macrophages filled with lipid inclusions as compared with 13% to 15% in ARDS and 2% in normal control subjects. In addition, there are significantly elevated total cholesterol (10.2 μg/μg phospholipid in FES as compared with 3.7 to 4.2 in ARDS and 2.0 in control subjects) and lipid esters.[22]

Imaging

Imaging studies can be a useful adjunct to provide additional diagnostic information (Table 2). Typically, chest radiographs show bilateral diffuse or patchy ill-defined opacities (Figure 4), but this can also be seen in ARDS, pulmonary edema, aspiration, or infection.[23] High-resolution CT has more specific findings. It shows patchy ground glass opacities and consolidation with interlobular thickening called the "crazy paving" pattern[23] (Figure 5). The extent of these findings has correlated with disease severity.[24]

Although in some cases, brain CT may show diffuse edema with scattered hemorrhage, usually, it shows a negative result. However, MRI is more sensitive, and T2-weighted images typically demonstrate a "starfield pattern" with multiple, small, nonconfluent, hyperintense lesions.[25] These lesions are also bright on diffusion-weighted imaging and appear dark on susceptibility-weighted sequences (Figure 6). Brain MRI of these lesions is also very consistent in where they appear anatomically and correlate with autopsy findings. The lesions occur in the periventricular, subcortical, and deep white matter. This finding is in contrast to diffuse axonal injury, which has a similar appearance but with lesions at the gray-white matter junction.[25]

Treatment and Prevention

Treatment is primarily supportive care with goals being to maintain oxygenation and ventilation, support hemodynamics, and resuscitate with fluids and blood products. Beginning in the 1950s, some targeted therapies were attempted, including heparin, hypertonic glucose, increased fluid intake, aspirin, and corticosteroids, all without conclusive benefit.[7,12]

Recently, experimental studies have attempted to alter the renin-angiotensin pathway to prevent FES. In addition to acting as a vasoconstrictor, angiotensin II is also proinflammatory and profibrotic. This may contribute to the pathogenesis of FES

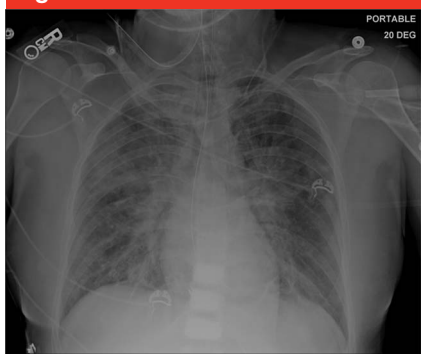
Table 2

Laboratory and Imaging Findings of Fat Embolism Syndrome

Lab/Imaging Study	Potential Findings
Laboratory studies	Anemia, thrombocytopenia, elevated ESR, CRP, and inflammatory cytokines, hypoalbuminemia, fat macroglobulinemia
Bronchoalveolar lavage	>30% alveolar macrophages with lipid inclusions
	Elevated total cholesterol and lipid esters
Chest radiograph	Bilateral diffuse or patchy opacities
Chest CT	Consolidation with interlobar thickening ("crazy paving" pattern)
Brain CT	Diffuse edema with scattered hemorrhage
Brain MRI	Multiple small nonconfluent hyperintense lesions on T2 ("starfield" pattern)
	Bright on diffusion, dark on susceptibility-weighted sequences

CRP = C-reactive protein, ESR = erythrocyte sedimentation rate

Figure 4



Chest radiograph of a patient with fat embolism syndrome showing bilateral diffuse patchy infiltrates.

in that fat in the lungs is taken up by macrophages, leading to the local release of renin followed by increased levels of angiotensin I and II. Fletcher et al[26] attempted to alter this pathway in a rat model by treating with the renin inhibitor aliskiren. In rats with FES induced by triolein injection, groups with aliskiren administration 1 hour after the injection showed significantly greater vessel diameter, decreased fibrosis, and lower fat content in vessels as compared with the control rats. Although experimental, this may represent a way to treat or provide prophylaxis against FES.

Given the lack of direct treatment options, an important goal is prevention. Based on the proposed role of the inflammatory process in FES, many randomized trials have examined the use of prophylactic corticosteroids. These are outlined in a 2009 meta-analysis that included seven randomized trials. From the pooled data of 389 patients, corticosteroids reduced the risk of FES by 78%, with no difference in the rates of infection and mortality.[27] A 2012 systematic review of randomized clinical trials found similar results.[28] Of a total of 223 patients receiving corticosteroids and 260 control subjects, 9 patients in the steroid group developed FES as compared with 60 patients in the control group ($P < 0.05$). It is difficult to interpret the results of these articles because they are based on small studies that used markedly different dosing regimens and had different duration of treatment. Because of the lack of high quality evidence, as well as the low incidence of FES and the potential risks of corticosteroid treatment, routine prophylaxis is not recommended.

Applications in Trauma

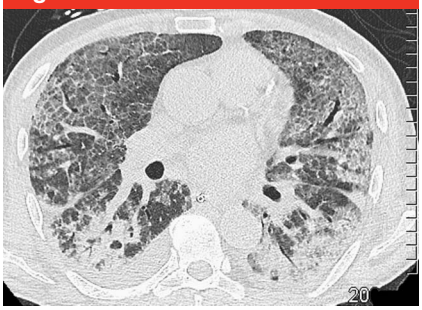
Concerns in orthopaedic trauma affecting FES that have been debated include timing to surgery, fracture fixation method, and the management of bilateral femur fractures.

Timing to Surgery

One of the earliest studies to examine the effect of timing was in 1976 by Riska et al.[29] He reported on a series of trauma patients seen with pelvic or long bone fractures or both from 1967 to 1974. During this time, at their institution, a transition from nonsurgical treatment to surgical treatment of fractures took place. They noted that while the number of fractures treated with early surgical fixation increased, the number of cases of FES decreased. When grouped by treatment type, they found FES rates of 22% in the nonsurgical group versus 4.5% in the surgical group. This was not randomized, and over this same period, supportive measures and resuscitation have also been changed; however, it was the first to give insight into the issue of fracture fixation timing. This was followed by a prospective randomized trial in 1989 that compared patients with isolated femur fractures with those with multiple injuries.[30] Within each group, patients were randomized to fixation either before 24 hours or after 48 hours. Significantly more pulmonary complications were found with late stabilization, both in the cases with isolated femur fractures and the multiply injured group. These studies were the basis for the principle of early total care.

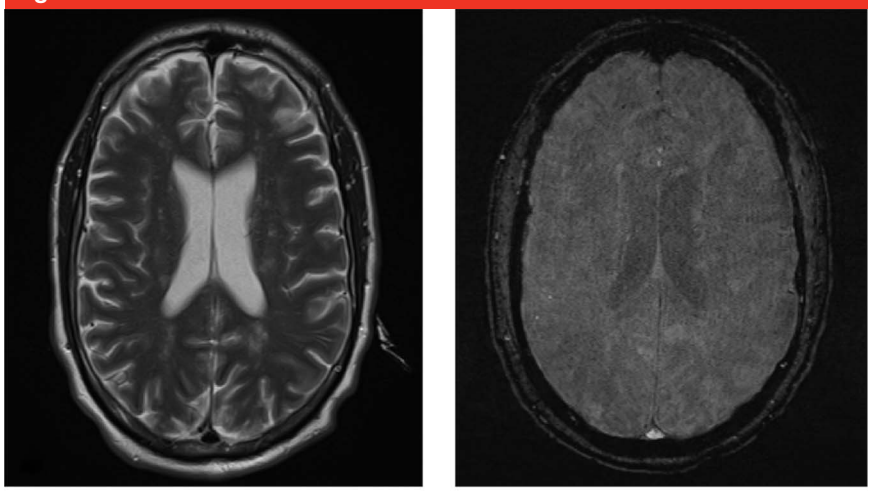
However, FES and pulmonary complications are just one of a multitude of factors to consider in the optimal timing of fracture stabilization in multitrauma patients. Although early definitive surgery is advantageous for most, there is a subset of patients for whom damage control orthopaedics followed by

Figure 5



Axial section of a chest CT scan showing interlobular septal thickening of the anterior bilateral lungs, described as the "crazy paving" pattern. (Reproduced with permission from Newbigin K, Souza CA, Torres C, Marchiori E, Gupta A, Inacio J: Fat embolism syndrome: State-of-the-art review focused on pulmonary imaging findings. *Respir Med* 2016;113:93-100.)

Figure 6



A **B**

Brain MRI of a patient with fat embolism syndrome showing multiple small nonconfluent lesions in the periventricuar and subcortical white matter that are bright on T2 sequences (**A**) and dark on susceptibility-weighted sequences (**B**).

definitive treatment at a later date may increase the chance of survival. Pape et al[31] recommend using findings of shock, coagulopathy, temperature, and soft tissue injury to risk stratify patients and guide treatment with early total care versus damage control. This concept has since been supported in multiple clinical studies, showing improved outcomes following similar algorithms.[32] Following these recommendations helps to balance the desire to prevent FES with the patient's physiologic ability to undergo surgical fixation.

Fixation Method

The mainstay of definitive treatment of femoral shaft fractures is a reamed intramedullary nail. It is known from animal and human studies that intramedullary reaming increases canal pressures and stimulates an inflammatory response.[33] Theoretically, this situation leads to an increased the incidence of FES compared with other fixation methods; however, this has not been conclusively shown in clinical studies. In reviewing pulmonary complications, Bosse et al[16] compared reamed intramedullary nails with plating in 453 patients. No significant differences in pulmonary complications or mortality were observed. This study was conducted at two different hospitals, one where nailing was more common and one where plating was routinely performed, which likely introduced confounding variables. Pape et al[34] randomized multiply injured patients in the borderline stable group to intramedullary nail versus external fixation. Six times greater incidence in acute lung injury was found in the intramedullary nail group, but no difference was found in ARDS or mortality. Reamed versus unreamed nails have also been investigated. The suggested benefits of reamed nails are higher union rates (particularly in distal fractures); the ability to place a biomechanically advantaged, larger diameter implant; and depending on fracture pattern, allow for early weightbearing.[6] The potential for increased pulmonary complications in reamed nailing has not been bourn out in the literature;[35] however, given small sample sizes, studies examining this topic may be underpowered.

Decreasing Embolic Load: Trauma

Several strategies for decreasing embolic load involve alterations to intramedullary reaming technique. In a sheep model, Mousavi et al[36] tested different reaming speeds, both advancement of the reamer and revolution of the reamer head. They created a midshaft osteotomy, induced hemorrhagic shock, and then resuscitated the sheep before reaming. The amount of embolized fat on TEE and intramedullary pressure was significantly lower with slower advancement and faster revolutions.

The use of a reamer irrigator aspirator (RIA) has also been proposed to decrease embolic load. In a canine model, Miller et al[37] compared three nailing scenarios, including unreamed, standard reaming, and RIA. A lower embolic load at the carotid artery was found on ultrasonography in the RIA group. On brain histologic assessment, the levels of heat shock protein (representing brain

stress) and hypoxia-inducible factor (representing ischemia) were significantly lower in the RIA and unreamed groups as compared to the standard reamer. Although seemingly promising, these techniques have yet to show clinical benefit in pulmonary or neurologic outcomes.

Bilateral Femur Fractures

Another situation that deserves special attention is the management of bilateral femur fractures. This is an uncommon injury pattern, and the literature consists of small case series. These series have shown that bilateral femur fractures treated with intramedullary nail have up to a 7.5% incidence of FES, have higher injury severity scores, resuscitation requirements, and rates of ARDS, as well as have longer hospital stays and higher mortality as compared with unilateral fractures with mortality rates of 5% to 6%, about 5 to 6× higher than isolated femoral shaft fractures.[38] No studies to date have compared different treatment modalities and surgical timing in bilateral femur fractures.

Applications in Arthroplasty

Joint replacement procedures also cause intramedullary pressurization, particularly with cementing, leading to embolization of fat into the circulation.[11] Fulminant FES with cardiopulmonary collapse is rare after arthroplasty, and descriptions are found only in case reports. However, the recent literature has examined other effects of FE in the setting of arthroplasty.

Pitto et al[5] evaluated the role of marrow embolization on the incidence of postsurgical DVT. They randomized 65 patients each to standard cementing technique versus cementing with the use of an intramedullary bone vacuum. The standard cementing group had significantly more severe and prolonged embolic events on TEE. Also, DVTs were detected in 18% of the patients in the standard group compared with 3% of patients in the bone vacuum group.

In addition, subtle cerebral findings have been attributed to FE. Several review articles have discussed this finding in the setting of arthroplasty and proposed that it could explain postsurgical delirium and cognitive decline.[3,4] This was originally studied in cardiac surgery requiring bypass, where increased fat embolic load correlated with a decline in postsurgical cognitive function. In the orthopaedic literature, despite being discussed in those recent review articles, just four original research articles have studied the relationship of FE on presurgical and postsurgical cognitive testing, three in the setting of arthroplasty and one in trauma.[3,4] None of these found a significant correlation, but interestingly, three out of four showed a trend that with increasing fat embolization, there was a decrease in postsurgical neurocognitive testing. The lack of significance may indicate that no relationship exists; however, the numbers in these studies are very small, and as such, they are likely underpowered.

The use of an intramedullary bone vacuum during cementation was shown to significantly decrease embolization of marrow contents.[5] Secondary cementation through a specially designed stem has also been described. In theory, this is a more gentle cementing technique because the stem is placed first and cement injected around it. Schmidutz et al[39] found a significantly lower embolic load on TEE with secondary cementation than with standard cementation.

In another approach, Zhao et al[40] attempted to alter the amount of embolized fat during total knee arthroplasty with an additional irrigation and suction step. Using implants that required tibial reaming, 30 patients were randomized to standard technique versus an additional irrigation and suction step to remove medullary contents before reaming. A significantly lower amount of fat embolization was noted on TEE in the modified technique group.

Computer navigation may also lead to decreased embolization of medullary contents. Typically, conventional knee arthroplasty uses intramedullary instrumentation, particularly in the femur, to set component alignment. This can cause an increase in intramedullary pressure leading to embolic showers. Computer-assisted surgery can potentially decrease the embolic load because it uses extra medullary guides. Several prospective randomized trials have compared computer-assisted procedures with standard total knee arthroplasty. Malhotra et al[19] evaluated embolic load after tourniquet release using TEE, transcranial doppler, and blood sampling from the right atrium. Decreased pulmonary and cranial emboli were found in the computer navigation group as compared with standard total knee arthroplasty group using an intramedullary femur guide.

These techniques may not be necessary or cost-effective in all cases, but they may be useful for certain high-risk patient groups.

Summary

Generally, FES is more common in male subjects, those aged 10 to 40 years, and those with closed long bone fractures, particularly of the femoral diaphysis, and multiple fractures. The clinical presentation includes respiratory distress, neurologic symptoms, and petechial rash 24 to 72 hours after injury. In trauma, strategies to limit FE include early fracture stabilization in stable patients, slow advancement, and fast revolutions of the reamer in reamed

nails, and RIA. Techniques in arthroplasty include the use of a bone suction device, secondary cementation technique with specialized stems, and computer navigation. Routine prophylaxis with steroids is not recommended. Possible future directions include the use of inflammatory markers, such as IL-6, to predict patients at the risk for FES, novel treatments such as altering the renin-angiotensin pathway, and examining the effect of embolic load on cognitive decline and delirium.

References

Evidenced based medicine: Levels of evidence are described in the table of contents. In this article, references 5, 19, 27, 28, 30, 35, 40 are level I studies. References 1, 11, 14, 21, 22, 34, 39 are level II studies. References 2, 10, 15, 16, 24, 25, 38 are level III studies. References 6, 13, 29, 32 are level IV studies. References 7, 12, 18 are level V expert opinion.

References printed in **bold type** are those published within the last 5 years.

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