Acute Fatal Fat Embolism Syndrome In Bilateral Total Knee Arthroplasty – A Review Of The Fat Embolism Syndrome

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Citation

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Abstract

Background:Although fat embolism syndrome is known to be relatively common in cases of multiple traumatic fractures, it is rare in cases of total knee arthroplasty. We describe a case of a 75 year old male who underwent bilateral total knee arthroplasty. After release of the first tourniquet, he developed a drop in oxygen saturation. He subsequently deteriorated, requiring intubation. The clinical diagnosis of fat embolism syndrome was made. We review diagnosis, investigation and prevention of the syndrome. Conclusion: Fat embolism syndrome can occur unexpectedly in elective reconstructive orthopedic procedures. One should have a high degree of clinical suspicion of fat embolism syndrome when a patient deteriorates perioperatively. The increased risk of fat embolism with bilateral total knee arthroplasty compared to unilateral total knee arthroplasty should also be considered.

INTRODUCTION

Embolism of fat during bone injury or manipulation is a common, usually benignoccurrence. However, fat embolism syndrome (FES) is a serious condition consisting of neurological, pulmonary, cutaneous and hemodynamic changes, associated with a high mortality that occurs in a minority of these cases. Clinical events leading to FES are well known. The incidence is more common in high velocity long bone fractures, however, it has been reported to be related to other events such as endomedullary bone manipulation, liposuction, total parenteral nutrition, bone marrow harvest and transplant, burns, acute pancreatitis and others [1] . Although fat emboli are common with both unilateral (TKA) and bilateral (BTKA) total knee arthroplasty, FES as a complication of BTKA is rare. Here we describe a case of fulminant FES after BTKA.

CASE PRESENTATION

A 75-year-old male presented for BTKA for osteoarthritis. His past medical
history included type II diabetes mellitus and treated hypothyroidism. He had a recent
normal adenosine thallium stress test and echocardiogram. Other pre-operative
evaluations were within normal limits. Continuous spinal anesthesia was selected.

Two mg of midazolam was used for sedation; supplemental oxygen was provided with
2 liters oxygen via nasal cannula with an oxygen saturation of 100%. The left knee was completed first.

An extramedullary tibial cutting guide was used for the tibial preparation and an intramedullary guide was used for the femoral preparation. The tourniquet for the left knee was deflated 15 minutes after surgery began on the right side after closure was completed on the left. Immediately after deflation it was noted that the oxygen saturation decreased to 90-94%. The blood pressure was 140/80 mmHg and the pulse was 60 beats per minute. The FIO2 was increased to 8 liters, and oxygen saturation
improved to 98% with deep inspiration. There were no complaints of chest pain or
shortness of breath. The patient was fully alert and orientated. The tourniquet from the
right knee was released after completion of surgery 104 minutes after the release of the
left sided tourniquet. Post-operative oxygen saturation was 92% on 8 liters nasal cannula
(NC) prior to transfer to the PACU. Estimated blood loss was minimal. Total fluids given
included 2 liters of Lactated Ringers and 500 cc of Hextend®.

On arrival to the PACU the initial oxygen saturation was 94% on a simple face
mask. The lungs were clear to auscultation bilaterally. The heart rate was 60 beats per
minute with a blood pressure of 110/60 mmHg. Three hours after arrival to the PACU,
there was a rapid deterioration in mental status. The patient was orientated to person only
but not to place or time and was somnolent. Breathing became increasingly labored with
an oxygen saturation of 75-80% on 10-L face mask and the respiratory rate was 30
breaths per minute. Crackles in both lungs were noted. The decision was made to control
the airway due to worsening respiratory distress. Copious secretions were suctioned from
the endotracheal tube. Post intubation arterial blood gas showed a pH of 7.22, PaO2 of 64, PaCO2 of 56 on 100% FIO2. Empirical treatment was started with furosemide and morphine, with no improvement. A chest x-ray taken 4 hours post-op showed bilateral
pulmonary infiltrates. Serial EKG's showed no acute changes. Cardiac enzymes were
normal. A transthoracic echocardiogram showed no acute left ventricular dysfunction, a
normal right ventricle, and no valvular lesions. Urine, blood, and sputum were sent for fat
staining. The urine and blood were negative; however, the sputum stained positive for
copious extracellular fat. The patient became comatose and a computerized axial
tomogram (CAT) scan of the brain which showed no acute changes.
He was transferred to the Surgical Intensive Care Unit. The clinical course was
marked by hemodynamic instability requiring norepinephrine and vasopressin for
hypotension. A MRI of his brain was performed on post-op day 2 (figure 1). This showed foci of acute ischemia suggestive of embolic phenomena consistent with fat embolism
syndrome. A repeat transthoracic echocardiogram remained normal with no evidence of a
patent foramen ovale (PFO) on contrast study. An EEG on post-op day 4 showed severe
diffuse encephalopathy. There was no petechial skin rash. Other laboratory studies
showed thrombocytopenia with a platelet count of 53,000/µl on post-op day 3. The ICU
stay was complicated by GI bleeding and renal failure. The coma never improved. A
repeat cranial MRI showed evolving watershed infarction throughout the cortices
bilaterally (figure 2). The neurological consult service diagnosed severe encephalopathy with a very poor prognosis .The decision was made to withdraw care. He was placed on the palliative care service, and expired on post-op day 22.

MRI showing foci of ischemia suggestive of fat embolism syndrome:
Figure 1 is from post operative day 2 showing multiple hyperintense areas consistent
with multiple emboli.

Figure 2 is from post operative day 14 and shows evolving cortical infarctions

Figure 1

Figure 1

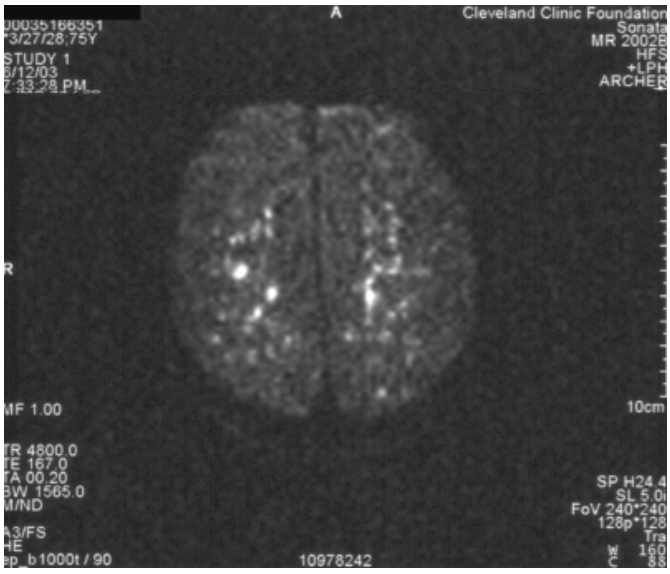
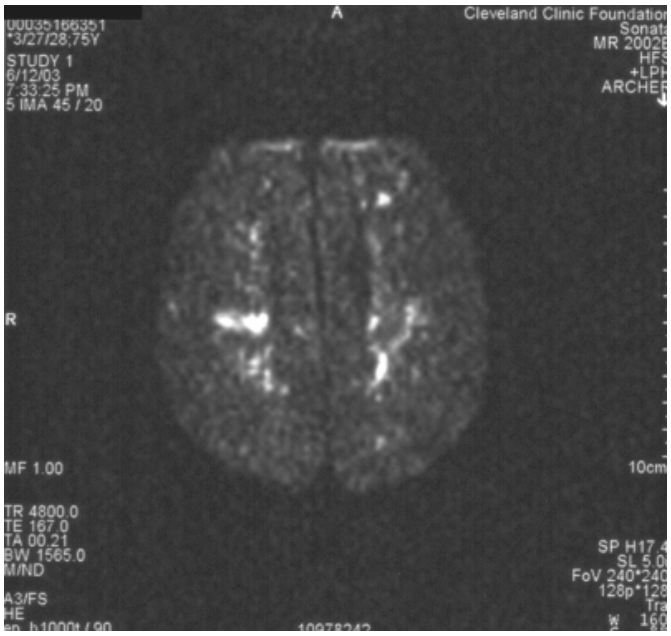


Figure 2

Figure 2



DISCUSSION

INTRODUCTION

Our patient suffered a clinically significant embolic event after release of the first
pneumatic tourniquet during BTKA. He went on to develop a rapidly deteriorating
clinical course of respiratory failure, mental status changes, evidence of CNS emboli and
progressive deterioration leading to his death. We would like to discuss the incidence, diagnosis and treatment of the fat embolism syndrome.

Fat embolism syndrome (FES) is a serious condition consisting of neurological, pulmonary, cutenuous and hemodynamic changes, often associated with a high mortality. Zenker first described fat embolism in 1862. [2] Von Bergman made the first clinical description of the FES in 1873. [3] Fat emboli are fat globules within the circulation, which may or may not produce clinical sequelae. The FES, however, is a more rare condition consisting of a constellation of neurological, pulmonary, cutaneous and hemodynamic changes, thought to be due to the presence of fat globules within the circulatory system.

INCIDENCE

The reported incidence of FES varies. The incidence is more common in traumatic fractures and lower limb fractures; however it has been reported to be related to other events such as liposuction, total parenteral nutrition, bone marrow harvest and transplant, burns, acute pancreatitis and others. [1] In a 25 year review of patients with high-risk fractures, the incidence was reported to be as little as 0.26% with a mortality of 20%. [4] Another review of femoral and tibial fractures showed the incidence to be as high as 29% with a mortality rate of 0%. [5] Gurd et al reported the incidence to be 19% in trauma patients. [6]

Many studies suffer from criticism that they focused on multiply traumatized patients whose concomitant injuries may have made it difficult to clearly define the contribution of FES towards the overall morbidity and mortality of these patients.

Ganong studied tibial and femoral fractures in young skiers with no other medical problems or confounding injuries. He found the incidence of FES to be 23% with a mortality of 0% in these patients. [7] Another criticism is that the variable defining criteria of what the FES consists of, contributes to the differences in the incidences reported. The occurrence of fat embolism syndrome after total knee replacement in more rare. The incidence has not yet been reported.

PRESENTATION

The presentation of the FES can be subclinical, clinical or fulminating. The signs and symptoms involve the pulmonary, neurological, renal, cardiac and dermatological systems. FES is a diagnosis of exclusion and is based on clinical criteria. There is no specific sign, symptom nor test, which is pathognomonic to the FES. It may often be confused with other conditions such as SIRS or sepsis.

The subclinical form of fat embolism syndrome may often go unnoticed especially if there are other injuries, which may often confound the clinical picture. Microscopic embolisation of fat occurs during most long bone operative procedures and after most long bone fractures. Christie et al, documented embolic evidence of fat on Echocardiography in 97 out of 110 patients having reaming of a fractured femur or tibia. [8] Despite the regular occurrence of microscopic fat embolisation, FES with multiorgan involvement is rare. Gurd et al established clinical criteria for FES into major, minor and laboratory findings. Others have modified this criteria based on perceived insensitivity. Below are the various criteria.

GURDS CRITERIA:

Major Criteria (one necessary for diagnosis)
Respiratory insufficiency
Cerebral involvement
Petechial rash

Minor Criteria (four necessary for diagnosis)
Pyrexia > 39.4
Tachycardia >120 beats per minute
Retinal changes
Jaundice
Renal changes

Laboratory Findings (one necessary for diagnosis) Anemia Thrombocytopenia Elevated ESR Fat macroglobulaemia [6]

LINDEQUES CRITERIA

Sustained Pa O2 less than 60 mmHg Sustained Pa CO2 greater than 55 mmHg or pH less than 7.3 Sustained respiratory rate of greater than 35 breaths per minute even after adequate sedation Increased work of breathing. [5]

A patient developing one of the above criteria was judged to have developed FES.

Schonfeld in his study used the fat embolism index score to diagnose fat embolism syndrome, with a score greater than 5 indicating its likelihood.

SCHONFELDS FAT EMBOLISM INDEX SCORE:

Figure 3

Symptom	Score
Pethechiae	5
Alveolar infiltrated	4
Hypoxemia Pa O2 <70 mmHg	3
Confusion	1
Fever >38 degrees C	1
Heart rate >120 beats per min	1
Resp rate >30 breaths per min	1

9

Lindeques criteria may be criticized for focusing only on the pulmonary system. Those features may be present in patients with ARDS with a cause other than fat embolisation i.e. burns, septicemia aspiration or multiple transfusions.

Regardless of the reported criteria for the diagnosis of FES, one must have a high index of suspicion for its occurrence in orthopedic patients and patients with traumatic fractures. Most patients have onset of symptoms between 25 and 48 hours after injury, with those presenting at less than 12 hours usually having a more fulminant presentation. [10] . Gurd found the period between injury and onset of symptoms to be 4 hours to 15 days. An average being 46 hours. [6]

LABORATORY EVALUATION

There are no pathognomonic tests to diagnose the FES. However there are several important tests, which may aid in identifying the FES.

Arterial blood gas must be examined. A PaO2 of less than 60 mmHg with no other obvious lung pathology in the orthopedic setting is highly suspicious of FES. [5] An alveolar-arterial gradient of greater than 100 mmHg indicates a high likely hood of FES. [11]

Blood and urine may be examined for fat; however positive findings are unspecific in the diagnosis in FES. [11] Gurd et al found fat globules greater than 8 microns circulating in the serum of all his documented cases. He stated that even though the relationship of large fat globules to the pathogenesis of the clinical picture remains obscure, the demonstration of their presence can be helpful in the diagnosis. [6] As well as examining blood and urine for fat, bronchoalveolar lavage may also be examined for fat. The macrophages may be stained for fat using the Oil Red O stain. However this is non-specific marker. Fat stained macrophages are frequently observed in trauma patients. [12] Anemia, thrombocytopenia elevated lipase and ESR may be found with the FES. [6]

Chest X-ray may show bilateral infiltrated resembling ARDS. EKG may show ST and T wave changes and signs of right heart strain.

T2 weighted MRI images have been shown to be the superior imaging in the diagnosis of cerebral FES. CT scans are often negative. [1314] MRI images typically show multiple non-confluent areas of high intensity signals or bright spots on a dark background, known as a “starfield pattern”. This

"starfield pattern" has been suggested to be pathognonomic of acute cerebral microinfarcts. Diffusion weighted MRI images should be done if FES is suspected as they are helpful in aiding the diagnosis. [1415] As well as being quite sensitive to the presence of cerebral microinfarcts, the T2 signal MRI images have identified evidence of cerebral microinfarcts as early as 4 hours after the inset of neurological symptoms and correlate well with the clinical severity of brain injury. [14]

PATHOPHYSIOLOGY

Two theories exist as to the pathophysiology of FES.

MECHANICAL THEORY

This theory suggests that the fat arise from damaged bone, travels through torn veins and lodges in distal capillaries causing mechanical obstruction.

BIOCHEMICAL THEORY

This suggests that stress and trauma alters the stability of chylomicrons already present in the bloodstream, which coalesce to form fat droplets.

Initially the fat emboli aggregate in the pulmonary circulation causing a mechanical obstruction. This leads to pulmonary hypertension and increased right heart strain. The decreased right-sided output leads to decreased left-sided filling pressures, hypotension and further pulmonary hypertension. Lipase is secreted in response to the excess fat and causes hydrolysis of the fat. This leads to release of free fatty acids and vasoactive substances, which causes increased permeability of capillary beds. Damage to type 2 pneumocytes leads to an ARDS type picture in the lungs. Micro thrombi form involving lipids, platelets and fibrin. This heralds the onset of DIC. Fat globules may cross into the systemic circulation via a PFO, AV shunts or through the pulmonary capillaries. This results in CNS emboli and ischemia, which may be further worsened by hypoxia. [16]

However, neurological involvement and evidence of cerebral emboli may be present without a PFO. A case report describes a 77-year-old who arrested during femur fracture fixation. FES was confirmed at autopsy. No PFO was found on autopsy. However fat globules were present in post capillary pulmonary venules. This suggests fat had traversed the pulmonary circulation. [17] In dogs that had cemented arthroplasties, fat globules were found deposited in the brains of all the dogs. None of these dogs had PFO'S. [18]

TREATMENT

Treatment is supportive. Special effort should be made to preventing hypovolemia and hypoxia. Aggressive respiratory support is often needed with frequent use of mechanical ventilation. Intravascular volume must be supported. Inotropes and pulmonary vasodilators are required frequently. Invasive hemodynamic monitoring may aid in deciding on treatment options. Exacerbation of CNS ischemia from hypotension should be avoided. Coagulopathy may be treated with platelets and fresh frozen plasma. Transfusion of PRBCS should be administered as needed.

More specific therapies have been studies, including steroids. Lindeque et al looked at 55 patients with tibial, femoral fractures or both. They divided the patients into those receiving steroids or placebo. They found the use of steroids maintained O2 levels and decreased free fatty acids in the serum. They found a 35% reduction in the incidence of FES in the steroid group. [5] Schonfeld et al also looked at the efficacy of corticosteroid treatment in prophylaxis of FES in patients with long bone fractures. They found the incidence of FES to be 0 out of 21 patients receiving steroid versus 9 out of 41 in patients receiving placebo. [9]

PREVENTION

With regard to trauma, it has been suggested that splinting and early reduction and fixation of a fracture prevent complications such as fat embolisation. However, this is controversial. It has been debated that manipulating bone during fixation may actually increase the incidence of FES. Bone et al looked at 178 patients who had early versus delayed stabilization of acute femoral fracture. They found delayed stabilization greatly increased the incidence of fat embolisation, ARDS and pneumonia. [19] Bulger et al found no notable difference in the incidence of FES among those undergoing operative fixation within 24 hours compared to those undergoing fixation at more than 24 hours. [10]

With regard to knee arthroplasties, preventative measures in surgical technique have been described including the use of fluted instead of round intramedullary alignment rods. Venting of the long bones decreases the intramedullary pressure, but may weaken the bone. [20] A study on cadaver dog bones found a 90% reduction of pressures generated with distal venting during intramedullary reaming; suggesting again that the use of venting holes may decrease pressure and thus less embolisation of marrow particles. [21]

Newer computer guided systems that do not use intramedullary cutting guides may also reduce the risk of serious emboli and their consequences.

UNILATERAL VERSUS BILATERAL TOTAL KNEE ARTHROPLASTY

Fat emboli occur commonly during TKA, even though FES is rare. BTKA may increase the incidence of FES compared to unilateral TKA. Bilateral total knee arthroplasty (TKA) can be performed concurrently, sequentially or as a staged procedure at two different time periods. This patient's BTKA were performed concurrently. Kim studied the presence of fat in blood at various critical events during TKA. [22] He concluded that fat embolism occurred in 65% of BTKA and 46% of unilateral TKA. Berman et al.used transesophageal echocardiography to evaluate emboli after tourniquet release in TKA. They concluded that some degree of embolization occurred in all patients. [23] Lane et al looked at 100 patients receiving unilateral TKA and 100 patients receiving bilateral TKA. They used identical pre, intra and post op protocols. They found post op confusion to be 4 times greater in the bilateral group than the unilateral group, 29% versus 7%. This was thought to represent and increased incidence of fat embolisation secondary to a 2-fold increase in the load of fat showered. [24]

The total embolic load released into the pulmonary vasculature during concurrent bilateral TKA may be very large. The increased mass of embolized fat may lead to severe hemodynamic and neurologic decompensation. Once the osteotomies on the second leg have been started, it may not be optimal to abandon the procedure even in the face of physiologic decompensation. Other potential complications may emerge from bilateral TKA. The significant volume shifts and acid washout of tourniquet deflation are endured twice. The patient under discussion unfortunately suffered a significant embolic event after deflating the first tourniquet and the second side was already underway. While it is impossible to determine whether subsequent emboli from the second side contributed to his deterioration, it might have been a good choice to postpone the second side after a clinically significant embolic event occurred on the first side. Some have advocated using changes in pulmonary vascular resistance to decide whether to proceed with the second knee in a sequential manner. [252627] Kelly et al reviewed 163 patients who were scheduled for bilateral TKA under one anesthesia. In their protocol, they used a Swan Ganz catheter to monitor cardiac output, pulmonary vascular resistance at induction, before inflation of either tourniquet, after completion of the first TKA, once tourniquet had been deflated for 15 minutes and at the end of the case. The patients were admitted to the ICU for monitoring overnight. With this protocol they identified patients:

1- with an increased pulmonary vascular resistance (PVR) who would be poorly suited to withstand the insult of fat embolisation from bilateral TKA,

2- with hemodynamic instability after one knee who were at risk for the second procedure.

3- who suffered fat embolisation after one knee and who would be at risk for a fatal outcome if further fat embolisation were to occur after the second knee.

They cancelled the second TKA in patients whose PVR was greater than 200 dyn.s/cm5 either before any surgical procedure was initiated or after the first TKA was completed or in patients whose PVR doubled after the first procedure. A total of 10.4% of patients had the second procedure cancelled. Thus the use of monitoring enabled them to identify patients at greatest risk for complication and thus cancel the second procedure. [25] Dorr et al documented monitoring of pulmonary artery pressures (PA press) and subsequent cancellation criteria for sequential bilateral TKA. They cancelled 6.3% of the second knee procedure secondary to raised PVR. They had no postoperative evidence of FES. [27]

CONCLUSION

BTKA represents a significant physiologic insult to patients. All patients have
some degree of fat embolization with both pulmonary and systemic emboli being
commonly found. Most patients recover uneventfully but some suffer from a range of
FES related complications. The outcome of our patient demonstrates that even relatively
healthy patients for BTKR can have severe complications from fat embolism.

The FES is an unpredictable condition with a varied presentation. There are no obvious determining factors. Thus it is up to the clinician to be highly vigilant with regards to trauma and orthopedic patients. The first step is to be able to recognize the FES and then respond quickly and aggressively in the treatment.

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