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PULMONARY AND CEREBRAL FAT EMBOLISM AFTER BILATERAL TOTAL KNEE REPLACEMENT ARTHROPLASTY

Anaesthesiology

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ABSTRACT

Cerebral and pulmonary fat embolism syndrome is a rare and potentially lethal complication commonly seen in long bone fractures and intramedullary manipulation. The clinical triad of fat embolism syndrome consists of mental confusion, respiratory distress, and petechiae.1 This paper reports a case of cerebral and pulmonary fat embolism syndrome following uneventful elective bilateral total knee replacement. After initial recovery, the patient developed neurologic and respiratory symptoms on postoperative day one.

KEYWORDS

Arthroplasty, Fat embolism, Total knee replacement.

Introduction

Fat embolism is a common complication after pelvic and long bone fracture, and is commonly seen after procedures or conditions such as orthopaedic surgery, severe burns, liver injury, closed-chest cardiac massage and liposuction. In general, fat embolism is asymptomatic, but in 1-5% of patients, fat embolism results in the fat embolism syndrome associated with multiorgan dysfunction involving sites such as the lung, brain and skin due to direct entry of fat globules into the systemic circulation.[1]

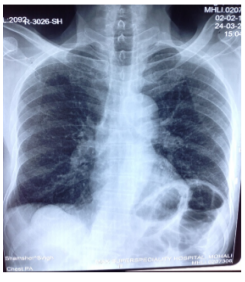
Fat embolism syndrome typically develops 24 to 72 hours after the initial injury, with either pulmonary or cerebral involvement confirmed by radiologic modalities. Risk factors include male sex,young adults, obesity, multiple fractures, and long bone fractures.2,3 The FES is usually prevalent in 1-5% of patients following trauma to the pelvis and long bones and rarely described in elective orthopaedic knee procedures.4So, we present an interesting case of fat embolism syndrome involving both brain and lung after total knee replacement, with a review of the literature including typical imaging findings.

Case Report

A 73 year-old male patient was admitted to max superspeciality hospital Mohali for bilateral total knee replacement (TKR). He had a 7-year history of progressive bilateral knee pain and limitation of walking due to severe osteoarthritis that did not improve with medicines and physical therapy.

The patient was relatively healthy without any previous medical or surgical history except for hypertension. The patient had a limitation of motion in both knee joints.His physical and neurological examination was normal .

Preoperative examinations such as routine laboratory tests(cbc,renal profile, liver functions, coagulation profile) and imaging studies such as chest X-rays, ecg ,echo pulmonary function tests were within normal limits. Before surgical intervention, his vital signs were: sinus rhythm pulse of 78/min, blood pressure of 128/80, respiratory rate of 16/min, a temperature of 37°C, and oxygen saturation on room air was 97%. The surgery was performed under combined spinal and epidural anaesthesia, and vital signs were stable throughout the operation, which lasted for 2.3 hours. He was transferred to orthopaedic post operative care unit with stable vitals alert in a good condition and postoperative knee x-rays was satisfactory. He remained stable throughout post operative day 0 and day 1 with normal vitals and epidural analgesia as per our protocol. However, on second postoperative day, he complained of chest tightness and difficulty breathing and became restless with a Glasgow Coma Score (GCS) of 8. His vital signs revealed sinus tachycardia (90/min) and tachypnoea (26/min), a blood pressure of 106/74, and an oxygen saturation of 92% on facemask with oxygen at 5 L/min. Arterial blood gas analysis showed ph 7.46 decreased PO2 as 63 mmHg pco2 27 hco3 23. With further deterioration in his neurological status he was immediately transferred to the Intensive Care Unit (ICU) where he required endotracheal intubation and mechanical ventilation due to desaturation and deteriorated level of consciousness. Neurological examination revealedthe patient to be stuporous with no focal signs and bilateral extensor plantar responses. A chest radiograph showed bilateral infiltrates **(Figure 1).** A postoperative laboratory test was unremarkable apart from a drop in hemoglobin from 15 to 8.6g/dl , despite an estimated blood loss was around 300 ml in drains for which he received 2 units of packed red blood cells. His platelets have dropped from 189×10^3 to 108×10^3.Immediate ECG revealed sinus tachycardia with no ischemic changes; cardiac enzymes were not elevated, and echocardiogram revealed no evidence of cardiac ischemia, thrombus, or atrial septal defect. Carotid doppler study revealed no atherosclerotic stenosis, and lower extremities duplex study was negative for deep venous thrombosis. No retinal fat or petechiae were seen. A cerebral computed tomogram (CT) was normal. Repeat ABG on 50% oxygen shows pH 7.35, pO2 80 mmHg, pCO2 36mmHg, saturations 95%.A lumbar puncture revealed no abnormality of cerebrospinal fluid. No other sources of sepsis were identified. At 48 hr



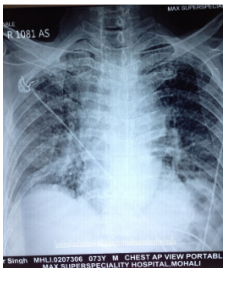
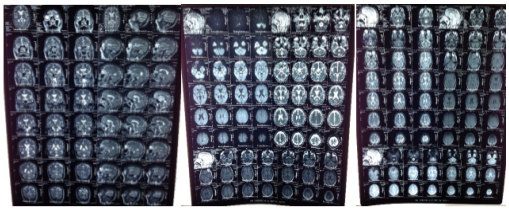


Figure 1 Preop chest xray Figure 2 chest xray showing infilterates

postoperatively, fat embolism was suspected, supported by the development of petechiae on the trunck , subconjunctiva and thrombocytopenia. Magnetic resonance imaging (MRI) was carried out five days postoperatively in which serial sections of brain were obtained in saggital, coronal and axial plains using T1, T2 and flair images .multiple tiny DWI hyperintense foci with low ADC values were seen diffusely scattered in bilateral cerebral parenchyma in bilateral frontal ,parietal ,occipital, and temporal lobes ,bilateral thalami, right basal ganglia and right cerebellum s/o multiple acute infarcts .Multiple T2 fluid-attenuated inversion recovery(FLAIR) hyperintense foci without restricted diffusion noted in bilateral periventricular and frontoparietal white matter s/o of chronic microangiopathic changes .rest of brain parenchyma shows normal MR morphology .Multiple tiny acute infarcts seen scattered in bilateral cerebral parenchyma were likely embolic shower.

Figure 3 MRI



Electroencephalography showed an abnormal diffuse theta recording with no focal abnormalities.

The patient was treated supportively with mechanical ventilation, supplemental oxygen and heparinization. The patient showed gradual improvement of the respiratory and neurological status and no further complications were noted. He remained in the hospital for 8 weeks with significant residual cognitive and motor impairment that required a special assistance for feeding and ambulation.

Discussion

Fat embolism occurs in almost all lower extremity trauma and intramedullary surgery. FES, however, is a severe multisystem manifestation of embolization that occurs much less commonly. Between 3%-5% of cases develop FES, which consists of damage and dysfunction of certain organs due to FE, usually occurring within 12-72 hours after the intervention or injury.5 FES is usually observed in cases of long bone fractures (0.9%-2.2%) 6, but it has also been described in certain orthopaedic procedures, with simultaneous bilateral knee arthroplasty being one of the most frequent (0.17%).[4]

The pathophysiology of fat embolism and its subsequent clinical manifestations is shrouded in controversy. However, three major theories have been described in the literature.7First, the Mechanical theory which states that traumatic injury to long bones results in the release of microscopic fat droplets into circulation by the disruption of the fat cell in the traumatized bone or the surrounding adipose tissues. The fat droplets are thought to enter into circulation due to pre-existing pathological arterial-venous communication, like a patent foramen ovale. Alternatively, fat droplets entry into the torn veins in the vicinity of the trauma due to an inherent pressure differential that exists between the venous pressure and intramedullary pressure. The droplets in the order of 7-10-micron escape the pulmonary filter and reach the systemic circulation. Second, the Biochemical theory which postulated where there is hydrolyzation of the fat, over a few hours, in plasma to free fatty acids and other mediators are considered the most plausible, as shown in animal models. Agglutination of chylomicrons and low-density lipoproteins facilitated by C-reactive proteins was found and reviewed also to be responsible for FES in non-traumatic models as well. Last, the coagulation theory proposed that the release of tissue thromboplastin, following traumatic long bone injury, result in activation of the complement system and extrinsic coagulation pathways and factor VII activation that leads to intravascular coagulation and subsequently lead to an increase in pulmonary permeability of the fat globules.

The triggering etiology of FES in TKR surgery was initially proposed to be due to cementing techniques and increased systemic monomer.8 This was demonstrated in animal models after simulated bilateral cemented arthroplasty. On the contrary, the development of FES was described after placement of the intramedullary femoral alignment guide during TKA, which released the bone marrow fat globules into the circulation before cement application, and advised canal marrow aspiration before alignment guide pin placement. The modification of surgical techniques to cementless TKR, and staging (unilateral versus bilateral) TKR procedures or the use of computer-assisted navigation has not been associated with a lower incidence of FES.8 However, irrigation of the femoral canal and aspiration of the bone marrow contents have shown satisfactory results, decreasing the incidence of FES9 The probability of facing FES after a revision arthroplasty is actually lower because the fatty tissue of the medullary canal was removed in the first surgery. It is believed that the multiple impacts and attempts to remove the prosthesis may be the cause of this complication in revision scenarios.[10]

The time onset of the clinical development of FES following knee arthroplasty was variable from an intraoperative cardiorespiratory collapse, before or after deflating the tourniquet, to the development of respiratory, cardiovascular and cerebral dysfunction minutes to hours in the postoperative period.[7]

The diagnosis of fat embolism is usually a clinical challenge and requires a vigilant high index of suspicion. It is a diagnosis of exclusion based on a myriad of clinical symptoms. Several organs may be affected; however, the 3 major clinical findings include respiratory dysfunction, neurological alterations, and skin rash of variable extension. Diagnosis is made by clinical criteria proposed by Gurd and Wilson11, requiring 2 major criteria or 1 major criterion plus 4 minor criteria to make the diagnosis (Table 1). The clinical symptoms and signs of FES are variable ranging from an asymptomatic patient, or the presence mild chest tightness and transient alteration of mental states, to coma and death.

Table 1

Gurd and Wilson criteria for diagnosis of FES.

Major criteria	Minor criteria
Respiratory distress	Tachycardia (>110 bpm)
Cerebral symptoms in non-head	Fever (>38.5_C)
injury patients	Jaundice
Petechial rash	Renal changes
	Retinal changes
	Drop in hemoglobin
	New onset thrombocytopenia
	Elevated erythrocyte sedimentation rate
	Fat macroglobulinemia

Two major criteria or 1 major criterion and 4 minor criteria suggest a diagnosis of FES

The use of advanced imaging and laboratory tests is complementary since they can help guide our differential diagnosis and rule out other entities. In addition, they may aid in patient prognosis, by describing the extent of ischemic damage and its reversal.1,12,13 Brain MRI is very useful when faced with diagnostic uncertainty due to its greater sensitivity, especially after a negative head CT, as we observed in our case.14,15 Chest radiography is a simple and significant initial investigation in patients with pulmonary symptoms possibly due to FES: it reveals an increase in bilateral pulmonary markings suggestive of interstitial pulmonary oedema, which is a consistent finding as in our case. A computed tomography (CT) scan would be useful to exclude intracranial haemorrhage. An MRI of the brain most accurately depicts the cerebral change in FES. Diffusion-weighted and susceptibility weighted imaging sequences are the mainstay of imaging sequences, with DWI picking up acute infarcts as early as 30 min after an onset of ischemia is documented.16Multiple tiny non-confluent T2-weighted MRI demonstrates hyperintense lesions with similar diffusion restriction in the cerebral deep white matters, typically in the region of the centrum semiovale and corona radiata. These lesions are micro-infarction due to microscopic fat embolisms. This shows a characteristic 'starfield' pattern, an association has been seen between the degree of the neurological defect and the size and distribution of the lesions. In our patient, the characteristic appearance of T2 and DWI were seen. These MRI findings along with the clinical scenario were associated with high accuracy in making the diagnosis of cerebral FES. Regarding the treatment, mainly supportive measures are required. Maintenance of arterial oxygenation within normal ranges and the adequate control of fluid resuscitation and avoiding fluid overload are fundamental for the prevention of shock, which can exacerbate lung and brain damage caused by FES .[14,17]

There are no comparative studies supporting the use of corticosteroids, while some studies note its ineffectiveness.18Finally, the use of heparin is not only considered ineffective, but it can worsen the clinical picture in the setting of FES by increasing the risk of haemorrhages and fatty acids in the circulatory system.19,20 Fortunately, the severe neurological symptoms of cerebral FES frequently resolve in most patients, but fatality and poor cognitive outcome, as in our unfortunate patient, may occur.

In summary FES is a significant cause of mortality and morbidity not only in trauma patients but increasingly recognized in association with elective orthopaedic surgery. Early diagnosis needs a high degree of clinical suspicion and early radiological investigations, such as MRI, for confirmation and prognostication.

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