ORIGINAL ARTICLE

Fat embolism syndrome in long bone fractures: Our experience at Lady Reading Hospital Peshawar

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Received:
7th February 2017

Accepted:
5th March 2017

Abstract

Objective: To find out the frequency, demography, clinical features and outcome of fat embolism syndrome in post traumatic long bones(femur, tibia) fractures.

Study design: Descriptive case series

Place and duration: Orthopaedic "A" Unit Medical Teaching Institution (MTI) Lady Reading Hospital (LRH) Peshawar from January 2014 to January 2017.

Material and methods: Patients both gender and age 16 years and above with closed fractures of femur and tibia fulfilling the inclusion criteria were included in the study. Patients were labelled to have fat embolism syndrome when Gurd and Wilson's at least two major or one major and four minor clinical features or laboratory investigations were positive. Demographic data of all the patients, their clinical presentation, time of onset of symptoms and outcome was documented.

Results: A total of 473 closed fractures of long bones, 283 patients of Tibia (59.8%), 190 cases of femur fracture (40.1%), in 458 trauma patients were assessed for fat embolism syndrome. Male patients were 410(86.6%) while female patients were 63(13.3%). Fat embolism syndrome was diagnosed in 13(2.8%) patients (mean age 28 years) using Gurd and Wilson's criteria. Bilateral femur shaft fractures were found in 6(1.26%) while isolated femur shaft fracture was found in 3(0.63%) patients. Tachycardia was present in all 13(100%) patients while hypoxemia in 10(76.9%) patients. Hemoglobin drop (average 6.2g/dl) was the most common in 5 patients (38.4%) laboratory findings. Mortality was found in 2 patients (15.3%).

Conclusion: Younger male patients with bilateral closed femur fractures were more prone to develop lethal fat embolism syndrome than isolated femur or tibia fracture. Tachycardia and hypoxemia were the most frequent and early clinical features while sudden drop in hemoglobin was the commonest laboratory finding of fat embolism syndrome.

Keywords: Fat embolism syndrome, long bone fractures, Gurd and Wilson's criteria

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Introduction:

The sub-clinical and asymptomatic manifestation of long bone fractures result in fat embolism which is the presence of fat globules in the parenchyma of lungs and circulation present in over 90 percent of traumatic events while fat embolism syndrome is a serious complication of fat emboli causing specific clinical signs and symptoms involving lungs, skin and brain and reported in only 3 to 4 percent of such patients.[1,2] It usually occurs 24 to 72 hours after traumatic long bone fractures but cases have been reported as early as 12 hours or as late as 2 weeks after initial trauma.[3] Fat embolism syndrome have been reported in 1 to 3 percent of patients with a single long bone fracture but with bilateral femoral fractures the risk increases up to 33 percent.[4]

Closed fractures have a higher frequency of developing fat embolism syndrome than open fractures.[5] In 1861 Zenker[6] mentioned for the first time fat globules in the pulmonary capillaries of an autopsy patient who sustained a crush injury

while Ernst von Bergmann[7] was the first person who did the clinical diagnosis of fat embolism syndrome in 1873. The exact pathophysiology of fat embolism syndrome is not yet known. Gauss,[8] 1n 1924 proposed the mechanical theory according to which long bone fracture causes release of fat globules and subsequent passage of these globules into the veins in the zone of injury and to the right side of heart into the capillaries of lungs causing mismatch of pulmonary ventilation perfusion leading to fat embolism syndrome. Lehman[9] proposed the biochemical theory of fat embolism in 1927 which states that release of excessive triglycerides by plasma mediators causing incomplete albumin binding and excessive triglyceride release into peripheral circulation and initiation of inflammatory changes and capillary occlusion in lungs and brain leading to fat embolism syndrome. Fat embolism syndrome had a mortality rate of about 10 to 20 percent or higher in 1960 but reduced to less than 10 percent due to early fracture fixation and more efficient modern supportive and lung therapy.[10] Fat embolism syndrome is diagnosed on the basis of a triad of clinical findings which include pulmonary, cerebral and skin manifestations.[11,12]

Hypoxemia, dyspnea and tachypnea is reported in 75 percent of patients of fat embolism syndrome while anemia, thrombocytopenia, tachycardia, pyrexia, oliguria and hematuria were also reported in some cases.[13] In order to reduce the mortality and complications of fat embolism syndrome it must be diagnosed early and prompt aggressive supportive therapy initiated as soon as possible.[14] Fat embolism syndrome can be prevented or its severity can be reduced if long bone fractures are fixed early thus reducing marrow fat release from the fracture site.[15] To reduce intramedullary pressure and risk of extravasation of medullary fat causing fat embolism syndrome during fracture fixation flexible narrow reamers, small size hollow nails, slow insertion of nails, distal venting and reamer irrigator aspirator systems are advised.[16-18]

In literature long bone fractures especially femur is a risk factor for developing fat embolism syndrome but unfortunately no data is available regarding its frequency, clinical features and outcome in our institution. We therefore conduct this study to find out the frequency, clinical features and outcome of fat embolism syndrome in our institution. Moreover we hope that this study will also improve the knowledge of doctors serving in Accidents & Emergency (A&E) Department to quickly diagnose fat embolism syndrome in patients admitted with a long bone fracture and respiratory signs and symptoms.

Material and Methods:

This descriptive study was conducted in Orthopaedics & Traumatology unit "A" Medical Teaching Institution (MTI) Lady Reading Hospital (LRH) Peshawar from January 2014 to January 2017. Patients of both gender and age above 16 years with closed fracture femur and tibia presented within 48 hours to accident and emergency orthopaedic department with suspicion of fat embolism syndrome having symptoms of breathlessness, cough and skin rashes were included in our study. Patients with open fractures, compartment syndrome, pathological fractures, head injury, spinal cord injury, chest trauma, vascular injury, abdominal injury and post operative patients were excluded from the study. Patients were labelled to have fat embolism syndrome when Gurd and Wilson's[11] at least two major or one major and four minor clinical features or laboratory investigations were positive. (Table 1) This criteria was applied to all the patients on admission and within 48 hours of admission and only those patients were included in the study who fulfilled the criteria. The study protocols were approved by the Ethical Review Board and informed written consent was obtained from all the participants. Complete history and physical examination was done in all cases. X-rays of the fracture extremity and chest was done. Hemoglobin, ESR, platelet count, urea and creatinine was done. Fundoscopy was done for retinal emboli. Oxygen saturation was monitored with continuous pulse oximetry. All the patients were initially managed conservatively in orthopaedic ward or intensive care unit on ventilator and definitive fixation of

Table 1:Gurd and Wilson's major and minor criteria for fat embolism syndrome

Major Criteria
1. Axillary or subconjunctival petechial rash
2. Respiratory symptoms with hypoxaemia PaO2<60 mmg Hg, FIO2=0.4
3. Cerebral depression unrelated to head injury
Minor Criteria
1. Tachycardia (110 or more)
2. Fever (38.5C or more)
3. Retinal emboli(on fundoscopy)
4. Fat globule in sputum or urine
5. Sudden drop in hemoglobin
6. Sudden drop in platelet count
7. High ESR

Table 2:Incidence and mortality of fat embolism syndrome in various studies

Study Name	Year	Incidence%(n)	Mortality%(n)
Schonfeld et al[12]	1983	15%(9)	None
Lindeque[19]	1987	23%(7)	None
Fabian[20]	1990	11%(10)	10%(1)
Robert[21]	1993	0.26%(20)	20%(4)
Bulger[22]	1997	0.9%(27)	7%(2)
Tsai[23]	2010	0.93(4926)	7.7%
Gupta et al[24]	2011	0.7%(12)	8.3%(1)
Koul et al[25]	2013	34%(935)	8.5%(3)
Our study	2017	2.8%(13)	15.3%(2)

fractures were done in the same admission in all those patients who survived. Methyl-prednisolone and anticoagulants were administered to all the patients of fat embolism syndrome. Blood and platelet were transfused where needed.

Demographic data of all the patients, their clinical presentation, time of onset of symptoms and outcome was documented. Data obtained was analyzed with SPSS(version 18). Frequency and percentages were calculated.

Results:

Over a period of three years, 473 closed fractures of long bones in 458 trauma patients were assessed for fat embolism syndrome in our unit. Male patients were 410(86.6%) while female patients were 63(13.3%). Mean age was 32 years (range 16 to 62 years). Majority 390(85.1%) of the patients were received to our unit within 6 hours of sustaining the fractures while remaining 68(14.8%) were received within 24 hours. Tibia fractures constituted 283 patients (59.8%) while femur fracture accounted for 190 (40.1%) fractures. Right side was involved in 415 patients (90.6%) while left in 43 patients (9.3%). Road traffic accidents was the causative event in majority 415 patients (90.6%) of patients while fall and physical assault was responsible for 28 patients (6.1%) and 15 patients (3.2%) respectively. Fat embolism syndrome was diagnosed in 13(2.8%) patients using Gurd and Wilson's criteria with least two major or one major and four minor clinical features or laboratory investigations present. All patients were male with road traffic accidents having the mean age of 28 years. About 9(69.2%) patients had the symptoms and signs of fat embolism within 6 to 12 hours of their fractures while the remaining 4(30.7%) patients had fat embolism syndrome in 12 to 48 hours after sustaining the fracture. Isolated femur shaft fracture was found in 3(0.63%) patients, bilateral femur shaft fractures in 6(1.26%), isolated tibial fracture in 2(0.4%) and multiple fractures (one floating knee and other contra-lateral femur and tibia fracture) were present in 2(0.42%) patients with fat embolism syndrome. Right side was involved in 8 patient (61.5%) while left in 5 patients (38.4%). Transvers or oblique femur fracture pattern was noted in 6(46.1%) patients, communited in 3(23%) and segmental in 2(15.3%) patients. The location of femur fracture was midshaft 8 patients(61.5%), proximal 3 patients(23%) and distal 2 patients (15.3%).

Tibial fractures were transverse in 3(23%) and communited in 1(7.6%) patient. All (100%, 4) tibial fractures were in mid shaft. Tachycardia was present in all (13, 100%) patients while hypoxemia in 10(76.9%) patients, fever in 6(46.1%), cerebral depression in 5(38.4%), hemoglobin drop(average 6.2g/dl) in 5(38.4%), low platelet (average 130000/dL) in 4(30.7%), high ESR (above 50 mm average) in 3(23%), rash in 2(15.3%) and retinal emboli in 1(7.9%) patient. Sputum or urine was not tested for fat globule in our patients. Nine(69.2) patients were managed in ICU while 4(30.7%) were managed in Orthopaedic ward. The mean hospital stay was 9 days(range 1 to 22 days). Mean ICU stay was 7 days(range 1 to 9 days). Ventilatory support was given to 8(61.5%) patients

while oxygen with nasal cannula was given to 5(38.4%) patients. Methylprednisolone and heparin was given in all cases. Blood and platelet transfusion was given to 9(69.2%) patients. The mortality was present in 2 patients (15.3%) and was due to respiratory depression and both patients had bilateral femur fractures. Interlocking nail femur and tibia was done in the same admission in all cases who survived. All the patients were regularly followed and no long term complications of fat embolism syndrome was found.

Discussion:

The incidence of post traumatic fat embolism syndrome in our study was in 13 patients (2.8%) while the mortality was in 2 patients (15.3%). In literature the incidence is reported to be as low as 0.2% to as high as 34% and mortality none to 20% (Table 2) One factor for this higher incidence in our study might be due to the fact that we were not able to fix most of these long bones fractures in the first 6 hours because studies[15] have shown that early fixation reduces the frequency or severity of fat symbolism syndrome. The cause of death in our study was mainly respiratory failure as in other studies. In our study all the victims of fat embolism syndrome were of male gender and younger age (less than 35 years). Other studies[25,26] reported the same observations but two studies[23,24] reported fat embolism syndrome in female patients and in elderly patients as well (70 years).

Patients with bilateral femur shaft fractures were more frequently affected than isolated fracture femur or tibia (1.26% versus 0.63% versus 0.4%) in our study. This has been confirmed by other studies.[23,26,27] Regarding the onset of symptoms we observed fat embolism syndrome as early as 6 hours after sustaining a fracture to a delay of 48 hours. Gupta[24] reported the onset of symptoms within 3.5 days and Tsai[23] reported in 11.37 to 97.5 hours. Tachycardia was present in 13 cases(100%) and hypoxiamia in 10 cases(76.9%) were the commonest early presenting symptoms in our patients. Koul et al[25] documented tachycardia in 94% and hypoxemia in 80% while Hysa[28] reported tachycardia in 100% and hypoxiemia in 84% cases. Hemglobin drop (average 6.2g/dl) was the most common in 5 cases (38.4%) laboratory findings in our patients of fat embolism syndrome. Tsai[23] reported a drop in hemoglobin in 84.6% (aazaverage 4.2 g/dL) while Koul et al[25] reported a drop in hemoglobin in 94% of their patients. We administered methylprednisolone and heparin to all the patients of fat embolism syndrome though their use is controversial but studies[1,12,19,23,28] favored their use to decrease the incidence and severity of the syndrome.

Our study had a few limitations. We did not check sputum or urine for fat globules in patients of pulmonary embolism. We were not able to perform ventilation perfusion scan or spiral CT or chest angiography to confirm the final diagnosis of fat empolism syndrome due to financial constraints.

Conclusion:

Younger male patients with bilateral closed femur fractures were more prone to develop lethal fat embolism syndrome than isolated femur or tibia fracture. Tachycardia and hypoxemia were the most frequent and early clinical features while sudden drop in hemoglobin was the commonest laboratory finding of fat embolism syndrome. Since fat embolism syndrome is diagnosed mainly on clinical features as there is no specific or confirmatory laboratory test. We therefore recommend a high index of suspicion in traumatic long bone fractures and monitoring of hypoxemia with continuous pulse oximeter in high risk patients. Furthermore early fixation of long bone fractures with small diameter unreamed nails or plating may reduce the incidence or severity of fat embolism syndrome.

Conflict of interest: None

Funding source: None

Role and contribution of authors:

Dr. Faaiz Ali Shah, MBBS, FCPS(Orthopaedics) Assistant Professor Orthopaedics & Traumatology Unit "A", Medical Teaching Institution (MTI), Lady Reading Hospital (LRH) Peshawar, conception/design of the study, data col-

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