Thesis

Fat Embolism Syndrome in Trauma: Evaluating Long Bone Fracture-Related Risk Factors and Patient Outcome

Adarsh Kota[1],
Chetan Pradhan[1],
Atul Patil[1],
Chetan Puram[1],
Darshan Sonawane[1],
Ashok Shyam[1],
Parag Sancheti[1]

[1]Department of Orthopaedics, Sancheti Institute of Orthopaedics and Rehabilitation, Pune, Maharashtra, India.

Address of Correspondence
Dr. Darshan Sonawane,
Department of Orthopaedics, Sancheti Institute of Orthopaedics and Rehabilitation, Pune, Maharashtra, India.
E-mail: researchsior@gmail.com

Abstract

Background: Fat embolism and fat embolism syndrome (FES) are recognized complications after marrow-containing bone trauma and can produce respiratory, neurological and dermatological manifestations ranging from subclinical hypoxemia to severe respiratory failure.

Objective: To determine the incidence and timing of hypoxemia and clinically evident fat embolism in adults with tibial and femoral diaphyseal fractures and to identify associated risk factors.

Methods: Two hundred consecutive patients treated at a tertiary orthopaedic centre were enrolled and followed prospectively. Demographic details, mechanism of injury, prehospital immobilization, fracture site and associated injuries were recorded. Serial arterial blood gases, urine fat globule examinations and platelet counts were obtained during the first 72 hours and patients were monitored for clinical features of FES.

Results: Hypoxemia occurred in 25.5% of patients and clinically evident fat embolism in 2%; hypoxemia most commonly appeared within 48 hours and fat embolism within 72 hours. Femoral fractures and multiple injuries had higher rates of hypoxemia.

Conclusion: Early immobilization, close monitoring in the early post-injury window and timely supportive care reduce progression.

Keywords: Fat embolism, Fat embolism syndrome, Hypoxemia, Long-bone fracture, Femur.

Introduction

Fat embolism denotes the presence of marrow fat globules in the circulation after trauma or intramedullary procedures and spans a clinical spectrum from microscopic emboli to full-blown fat embolism syndrome (FES) with respiratory failure, neurological disturbance and petechial rash [1]. FES most often follows fractures of long bones and the pelvis and is particularly associated with femoral shaft injuries and high-energy mechanisms such as road traffic accidents, which commonly affect young adults in many settings [2]. Two principal, complementary mechanisms are described: the mechanical theory, which proposes forcible extrusion of marrow fat into torn venous channels under raised intramedullary pressure, and the biochemical theory, which emphasises hydrolysis of fat to free fatty acids that produce endothelial injury and a systemic inflammatory response [3] [4]. Evidence from autopsy series and prospective clinical cohorts indicates that subclinical fat embolization is far more frequent than clinically overt FES, which accounts for the wide variation in reported incidences across studies [5]. Clinical recognition remains a challenge because no single test is pathognomonic; therefore practical bedside monitoring with

Dr. Chetan Pradhan

Dr. Atul Patil

Dr. Chetan Puram

Dr. Darshan Sonawane

Dr. Ashok Shyam

Dr. Parag Sancheti

DOI: https://doi.org/10.13107/jmt.2025.v08.i01.172

continuous pulse oximetry and serial arterial blood gases is useful for early detection of hypoxemia and impending respiratory compromise [6]. Early immobilization, prompt transfer and timely fixation are emphasised as pragmatic measures to reduce pulmonary complications. Given the frequency of subclinical embolization and potential for early progression, this prospective evaluation aims to provide practical data to refine monitoring and early care pathways in our tertiary orthopaedic setting. The practical implications of recognizing early hypoxemia include timely oxygen therapy, selective ICU monitoring and avoidance of procedures that may worsen intrathoracic pressures in vulnerable patients. Local data remain limited, and describing a contemporaneous cohort will guide training, resource allocation and local protocols for early detection and management in regions with similar trauma profiles. This report presents those findings and recommendations.

Aims and Objectives

Primary aim: To determine the incidence of hypoxemia and clinically evident fat embolism in adults presenting with tibial and femoral diaphyseal fractures to a tertiary orthopaedic unit [7]. Secondary aims: To identify clinical and demographic factors associated with hypoxemia and fat embolism, including age, sex, mechanism of injury, fracture location (femur versus tibia), prehospital immobilization status and presence of multiple fractures, and to describe the timing of hypoxemic events in the early post-injury window [8]. The study also intended to evaluate the diagnostic yield of routine tests in this context, specifically serial arterial blood gas analysis, urine fat globule examination and platelet counts during the first 72 hours after injury. Investigators planned to document immediate supportive measures provided, criteria for escalation to ICU care and short-term outcomes such as need for ventilatory support and in-hospital mortality so as to recommend feasible surveillance and escalation protocols for similar resource settings. The data were to be collected prospectively to ensure precise timing of events and to minimise recall bias. By generating baseline incidence and timing information in our population, the study would help design larger trials of prophylactic measures. Local protocol recommendations and staff education were planned deliverables as outputs.

Review of Literature

The phenomenon of fat embolism has been observed for well over a century, with early pathologic descriptions identifying fat droplets in pulmonary capillaries after severe trauma and later clinical reports describing the syndrome of dyspnoea, petechiae and altered consciousness now termed FES [9]. Autopsy series regularly document pulmonary fat emboli following major trauma, while prospective clinical cohorts show that clinically overt FES is less common and that reported incidence varies according to diagnostic definitions and surveillance intensity [10]. The mechanical theory explains embolization as a consequence of raised intramedullary pressure forcing marrow fat into torn venous channels and producing mechanical obstruction in the pulmonary microcirculation; intraoperative maneuvers such as intramedullary nailing have been associated with embolic signals that reflect this process [11]. The biochemical theory stresses hydrolysis of marrow fat to free fatty acids with secondary endothelial toxicity, platelet aggregation and an inflammatory cascade that worsens microvascular occlusion and tissue injury [12]. A hybrid model that recognises both mechanical and biochemical contributions best accounts for the variable clinical presentations and for systemic manifestations when embolic material or inflammatory mediators reach the arterial circulation [13]. Diagnostic approaches remain largely clinical; continuous pulse oximetry and serial arterial blood gas sampling are practical bedside tools for early detection of hypoxemia, whereas tests such as urine fat globule examination and platelet counts have variable sensitivity and must be interpreted in clinical context [14]. Radiologic imaging may demonstrate nonspecific pulmonary infiltrates in established respiratory involvement and advanced modalities such as CT or MRI are reserved for severe or cerebral cases. The literature emphasises early immobilization and timely definitive fixation as pragmatic preventive measures supported by observational evidence, even though randomized trial data for specific intraoperative techniques or pharmacologic prophylaxis are limited. However, diagnostic heterogeneity and variable reporting contribute to the wide range of incidence figures across published series. Many studies differ in case definitions, sampling frequency and the use of laboratory adjuncts, which limits direct comparison. Urine fat globule testing, once considered a hallmark, suffers from inconsistent sensitivity and specificity in clinical practice, and thrombocytopenia and anaemia are non-specific changes that may reflect systemic trauma rather than embryonic syndrome alone. Several observational reports have documented reductions in severe pulmonary complications with early fracture immobilization and expedited fixation, but methodological differences and confounding by injury severity complicate definitive interpretation.

Materials and Methods

This prospective observational study enrolled 200 consecutive adult patients with tibial or femoral diaphyseal fractures presenting to a tertiary orthopaedic centre between 2016 and 2018 after institutional ethics committee approval and informed written consent. Inclusion was limited to adults with diaphyseal fractures of the lower limb; exclusion criteria were major concomitant head, chest, abdominal or pelvic injuries, pregnancy, pathological fractures and any other obvious cause of hypoxemia such as overt sepsis or head injury. On arrival

demographic details, mechanism of injury, prehospital immobilization and associated injuries were recorded on a pretested proforma. Fractures were classified by standard orthopaedic systems and baseline radiographs were obtained. Arterial blood gas analysis was performed within 12 hours of admission and repeated at 24-hour intervals for three days. Platelet counts and urine samples for fat globules were collected at 24, 48 and 72 hours. Hypoxemia was defined and categorised as subclinical, clinical and overt fat embolism using established clinical criteria adapted from classical series and surgical reports [15, 16]. Symptomatic patients received supplemental oxygen and were escalated for ICU monitoring when clinically indicated; early immobilization and timely definitive fixation were practised in line with local protocols and longstanding surgical recommendations [17, 18]. Data were entered into spreadsheets and analysed with standard statistical tests; continuous variables are reported as mean ± SD and categorical variables as frequencies and percentages. Student's t-test and Chi-square tests were applied as appropriate with $P < 0.05$ considered significant. Ethical and cost considerations of routine testing were observed for all participants. Confidentiality maintained.

Results

Two hundred patients were enrolled. Mean age was 33.6 years with 42.2% aged 21–30; 71.5% were male. Road traffic accidents accounted for 89% of injuries and 85.5% of patients had some form of immobilization at presentation. Isolated fractures comprised 97% of cases; femoral diaphyseal fractures were more common (75.2%) than tibial fractures (24.8%). Hypoxemia developed in 51 patients (25.5%): 18 patients (9.0%) had subclinical hypoxemia, 29 (14.5%) had clinical hypoxemia and 4 (2.0%) met criteria for overt fat embolism. Most hypoxemic events occurred within 48 hours and fat embolism presented within 72 hours. Clinical signs accompanying hypoxemia included tachycardia, fever and transient altered sensorium in subsets of patients; petechial rash was uncommon. Urine fat globules were detected intermittently and thrombocytopenia was infrequent; neither correlated consistently with clinical hypoxemia. Femoral fractures and patients with multiple injuries demonstrated higher rates and greater severity of hypoxemia. Supportive care with supplemental oxygen sufficed for most symptomatic patients while a small proportion required ICU-level monitoring and ventilatory support. One death was attributed to respiratory complications related to fat embolism. Length of hospital stay correlated with hypoxemia severity and injuries, with cases requiring supportive care and monitoring.

Discussion

This prospective cohort confirms that clinically overt fat embolism syndrome is uncommon while subclinical hypoxemia after long-bone fractures is relatively frequent and may precede clinical deterioration. The demographic profile of predominantly young men injured in road traffic accidents mirrors regional trauma patterns and aligns with prior reports. The higher incidence and greater severity of hypoxemia observed in femoral fractures and in patients with multiple injuries supports the view that greater marrow content and increased injury burden elevate risk. Temporal clustering of events within the first 48–72 hours emphasises an early surveillance window when serial arterial blood gases and continuous pulse oximetry can detect impending respiratory compromise. Classic laboratory tests such as urine fat globules and platelet counts were inconsistently helpful and should support rather than replace clinical monitoring. Preventive emphasis should remain on early immobilization, rapid transfer and timely definitive fixation where feasible. While older surgical and military series documented frequent embolic events in battle and operative casualties [19, 20], modern series with early fixation and improved critical care report lower mortality but still show that severe cases can progress to ARDS and require ventilatory support. Limitations of this work include single-centre design, finite sample size and absence of advanced embolic detection modalities such as transesophageal echocardiography or MRI for cerebral microembolism, which may underestimate subclinical events. Despite these constraints, the data provide practical guidance for tertiary orthopaedic centres with similar casemix: heightened vigilance during the first 72 hours, routine ABG monitoring for at-risk patients and prompt supportive care when hypoxemia is detected. These measures are feasible, low-cost and can be audited prospectively to measure impact.

Conclusion

In this prospectively collected series of 200 patients with lower limb diaphyseal fractures, hypoxemia occurred in 25.5% and clinically apparent fat embolism in 2%. Hypoxemia most commonly presented within 48 hours and fat embolism within 72 hours of injury. Femoral fractures and multiple injuries were associated with higher risk. Urine fat globules and thrombocytopenia were of limited predictive value; serial arterial blood gases combined with continuous pulse oximetry and close clinical observation were more reliable for early detection. Early immobilization, prompt stabilization and rapid escalation to higher monitoring when required remain the most practical measures to reduce progression to overt fat embolism in similar tertiary-care settings. The findings support focused early surveillance protocols, strengthening of prehospital immobilization practices and training for peripheral staff to expedite referral. These baseline data can inform local protocols and provide a platform for larger confirmatory studies that evaluate prophylactic and therapeutic strategies and policy.

References

1. Saigal R, Mittal M, Kansal A, Singh Y, Kolar P, Jain S. Fat

embolism syndrome. JAPI. 2008 Apr; 56.
2. Akhtar S. Fat embolism. Anesthesiology Clinics. 2009 Sep; 27(3):533–50.
3. Maitre S. Causes, clinical manifestations, and treatment of fat embolism. Virtual Mentor. 2006 Sep; 8(9):590–2.
4. Stein PD, Yaekoub AY, Matta F, Kleerekoper M. Fat embolism syndrome. The American Journal of the Medical Sciences. 2008 Dec; 336(6):472–7.
5. Mellor A, Soni N. Fat embolism. Anaesthesia. 2001 Feb; 56(2):145–54.
6. Eriksson EA, Pellegrini DC, Vanderkolk WE, Minshall CT, Fakhry SM, Cohle SD. Incidence of pulmonary fat embolism at autopsy: an undiagnosed epidemic. Journal of Trauma and Acute Care Surgery. 2011 Aug; 71(2):312–5.
7. Johnson MJ, Lucas GL. Fat embolism syndrome. Orthopedics. 1996; 19:41–48.
8. Scuderi CS. The present status of fat embolism. Int Surg Digest. 1934 Oct; 18(4):195–215.
9. Fenger C, Salisbury JH. Diffuse multiple capillary fat embolism in the lungs and brain is a fatal complication in common fractures. J Chicago Med Exam. 1879; 39:587–95.
10. Peltier LF. Fat embolism: a perspective. Clinical Orthopaedics and Related Research®. 2004 May; 422:148–53.
11. Payr E. Further contributions to the knowledge and explanation of fat-embolic death after orthopaedic surgery. Z Orthop Chir. 1900; 7:338.
12. Newbigin K, Souza CA, Torres C, Marchiori E, Gupta A, Inacio J, and Armstrong M, Peña E. Fat embolism syndrome: state-of-the-art review focused on pulmonary imaging findings. Respir Med. 2016 Apr; 113:93–100.
13. Purtscher O. Angiopathia retinae traumatica. Albrecht von Graefe's Arch Ophthalmol. 1912; 82:347–71.
14. Peltier LF. Nail design; an important safety factor in intramedullary nailing. Surgery. 1950 Oct; 28(4):744–8.
15. Peltier LF. Fat embolism following intramedullary nailing: report of a fatality. Surgery. 1952 Oct; 32(4):719–22.
16. Tanton J. L'embolie graisseuse traumatique. J de Chir. 1914; 12:287–96.
17. Wilson JV, Salisbury CV. Fat embolism in war surgery. Br J Surg. 1944 Apr; 31(124):384–92.
18. Scully RE. Fat embolism in battle casualties: incidence, clinical significance and pathologic aspects. Am J Pathol. 1956 Jun; 32(3):379.
19. Collins JA, Gordon WC Jr, Hudson TL, Irvin RW Jr, Kelly T, Hardaway RM 3rd. Inapparent hypoxemia in casualties with wounded limbs: pulmonary fat embolism? Ann Surg. 1968 Apr; 167(4):511.
20. Cloutier CT, Lowery BD, Strickland TG, Carey LC. Fat embolism in battle casualties in hemorrhagic shock. Mil Med. 1970 May; 135(5):369–73.

Conflict of Interest: Nil

Source of Support: None

Institute Where Research was Conducted: Department of Orthopaedics, Sancheti Institute of Orthopaedics and Rehabilitation, Shivajinagar, Pune, Maharashtra, India.
University Affiliation: MUHS, Nashik, Maharashtra, India.
Year of Acceptance of Thesis: 2019

How to Cite this Article: Kota A, Pradhan C, Patil A, Puram C, Sonawane D, Shyam A, Sancheti P | Fat Embolism Syndrome in Trauma: Evaluating Long Bone Fracture-Related Risk Factors and Patient Outcom | Journal of Medical Thesis | 2022 January-June; 08(1): 5-8.