

Early, reliable, utilitarian predictive factors for fat embolism syndrome in polytrauma patients

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Abstract

Background: Fat embolism is one of the apocalyptic pulmonary complications following high energy trauma situations. Since delay in diagnosis may have devastating consequences, early, easily accessible and relatively inexpensive investigations for risk stratification may prove useful, especially in developing nations. **Materials and Methods:** This prospective trial included a total of 67 young polytrauma patients, in whom the role of nine easily available, rapidly performable clinical or laboratory investigations (or observations noted at admission) in predicting the later occurrence of fat embolism syndrome were assessed. All the patients also underwent continuous monitoring of oxygen saturation with pulse oximetry. **Results:** The correlation between initial serum lactate (within 12 hours of injury) and hypoxia was statistically significant. There was a trend towards correlation with FES (by Gurd's criteria) ($P=0.07$), Sensitivity of 24-hour monitoring of oxygen saturation in predicting later pulmonary deterioration approached 100%. **Conclusions:** The combination of three factors including polytrauma (with NISS > 17), serum lactate > 22 mmol/l at admission (within 12 hours of injury) fall in oxygen saturation (SaO_2 below 90% in the initial 24 hours) predict the development of post-traumatic pulmonary complications, especially the fat embolism syndrome.

Key words: Fat embolism syndrome, polytrauma patients, pulse oximetry, serum lactate

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Introduction

Following a colossal rise in the vehicular traffic and a resultant increase in the incidence of road traffic accidents, polytrauma has become a cause of great concern in the developing nations. With a huge possibility of occurrence of diverse, grave, systemic complications ensuing such multiple injuries, the medical fraternity has begun to consider the vantage of a prophylactic approach prior to the onset of such untoward adversities in these circumstances.^[1-4] Pulmonary complications, which include direct blunt and penetrating injuries to the thoracic contents and other indirect pulmonary

consequences like systemic inflammatory response syndrome (SIRS), TRALI, fat embolism syndrome, pulmonary embolism etc., are unanimously considered to be the major issues that demand early attention and intervention in such multiple injured patients.^[5] Early diagnosis of these conditions requires identification of appropriate risk factors and clinical and laboratory indicators, that may guide management. This article proposes a cost-effective, screening protocol that may be employed for risk-stratification of polytrauma patients. Such a protocol may help in improving trauma care delivery, especially in the developing nations, where the demands for the emergency health-care facilities hugely overwhelm availability.

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Materials and Methods

The present study included a total of 67 patients in the age group of 16-40 years of both sexes, who were brought to our emergency orthopedic services following multiple skeletal injuries within 12 hours, over a period

of 6 years (between July 2004 and December 2010). Polytrauma was defined based on the New Injury Severity Score,^[6,7] and only patients whose scores exceeded 17 were considered. Patients with significant head, chest, and abdominal trauma, or those with history of pre-existing cardiac or pulmonary disease or chronic smoking were excluded.

All the patients were immediately resuscitated in accordance with the ATLS protocol. Relevant history of all details regarding the nature of accident, any prior treatment other extra-skeletal injuries, and pre-existing co-morbid illnesses was obtained.

After initial resuscitation and splintage, appropriate imaging for all the skeletal injuries was done. Clinical parameters such as pulse rate, respiratory rate, temperature, blood pressure, urine output, and central venous pressure were recorded at admission and 4 hourly. Routine investigations at admission included hematological biochemical and arterial blood gas results (using Rapidlab 855 (BAYER) blood analyzer). The necessary fracture stabilization procedures were carried out within 24 hours of admission.

The following parameters related to fat embolism syndrome were recorded regularly for 72 hours following admission:

- 4 hourly records of temperature, pulse rate, blood pressure, respiratory rate, and urine output
- Fundus and urine examination for fat globules, at least once a day for 3 days
- Posterior-anterior radiograph of the chest at least once-a-day
- Arterial blood gas analysis, 6 hourly on the first day and 12 hourly for next 2 days
- Complete hemogram and biochemical investigations including serum lactate at 24-hourly interval till third day of injury.

All these patients also underwent continuous pulse oximetry monitoring (CPOM) for capillary oxygen saturation. The diagnosis of fat embolism syndrome was made using the Gurd's criteria.^[8] Serum lactate estimation was done by the calorimetric method based on the highly sensitive color reaction of Egriwe. The clinical and laboratory parameters obtained at admission were analyzed and followed up for a later development of fat embolism syndrome or other pulmonary complications, to assess the predictive role of these parametric values.

Results

Out of the 67 polytrauma patients studied, 12 (17.9%) developed clinical fat embolism syndrome (as per Gurd's criteria) and 18 (26.9%) patients developed transient hypoxemia ($\text{PaO}_2 < 60$ mmHg). The general clinical and laboratory findings (as per the Gurd's criteria)^[8] in our patients during the initial 72 hours of monitoring are shown below [Table 1].

The clinical and laboratory observations at the time of admissions have also been noted and tabulated below [Table 2].

Discussion

Fat embolism syndrome, an entity first described as early as 1862, has remained a mystery to the orthopedicians till date, and the abstruseness on diverse aspects of its pathophysiology and management has been well-acknowledged.^[9,10] According to the current concepts the pathophysiology of this condition involves 2 mechanisms that have been put forth as mechanical and biochemical theories.^[11-14] The protagonists of the mechanical theory hypothesize that a mechanical block of the pulmonary vasculature by embolized fat globules is the culprit while the believers in the latter theory advocate a hormonal or an enzymatic (lipoprotein lipase) background.

This syndrome typically affects the young, muscular victims of high-energy trauma, following multiple long bone fractures.^[15] The multiple injuries not only initiate the influx of marrow fat into the systemic and pulmonary vasculature, but also induce a systemic inflammatory response that produces cytokines capable of causing pulmonary damage. The number of clinically evident cases of clamant respiratory distress in such a scenario only represents the tip of the iceberg, with a large number of lung injury remaining clinically inapparent.^[16]

The role of avoiding additional damage by surgical

Table 1: Clinical and laboratory parameters monitored in our patients

Clinical/Laboratory observation (initial 72 hrs)	No of patients
Petechiae	7
Hypoxia ($\text{PaO}_2 < 60$ mmHg; $\text{FiO}_2 \leq 0.4$)	30
Central nervous system depression	17
Tachycardia (> 110 beats/min)	19
Pyrexia (temperature > 38.5 degrees)	14
Fat globules in urine/sputum/retina	9/6/11
Sudden unexplainable drop in hematocrit	9
Sudden unexplainable drop in platelet values	7
Increasing ESR	8

ESR: Erythrocyte sedimentation rate

Table 2: Parameters at admission and their relation to FES

Parameter	FES	Specificity	Sensitivity	Statistical significance	Isolated hypoxia	Specificity	Sensitivity	Statistical significance	Total
Shock	6	67	50	0.69	7	54	39	0.62	30
Decreased hemoglobin	7	58	58	0.64	8	46	44	0.5	35
Raised ESR	6	76	50	0.25	7	68	39	0.64	25
Altered CRP	7	62	58	0.49	6	46	30	0.15	33
Hyperglycemia	7	71	58	0.20	9	68	50	0.21	28
Reduced platelet	5	76	42	0.49	8	68	44	0.38	25
Altered coagulogram	7	73	58	0.14	7	65	39	0.80	27
Raised lactate	7	88	58	0.07	11	78	61	0.003	24
Altered base deficit	6	86	50	0.18	9	76	50	0.12	23
Total	12				18				

FES: Fat embolism syndrome; ESR: Erythrocyte sedimentation rate; CRP: C-reactive protein

interventions in “border-line” and “unstable” patients is well recognized.^[16,17] Various mortality indicators have been postulated in polytrauma, including serum glucose, C-reactive protein, albumin, transferrin, base deficit, lactate, coagulation factors, platelets, hemoglobin, cholesterol, trauma scores, pro-inflammatory cytokines etc.^[15] The present study is designed to identify relatively reliable, accessible and rapidly assessable clinical or laboratory parameters that may help us in triaging polytrauma patients.

We had assessed the correlation of 9 clinical and radiological parameters observed at admission and the development of fat embolism syndrome and hypoxia (PaO₂ <60 mm Hg using CPOM or ABGA). All patients were monitored by continuous pulse oximetry for any transient hypoxia during the initial 72 hours. All factors included have previously been studied in post-traumatic scenarios and variously linked to mortality.^[15,17,18] Among the factors that were assessed, barring serum lactate ($P = 0.003$ for hypoxia and $P = 0.07$ for FES), the rest failed to show a significant association with either fat embolism syndrome or hypoxia. The sensitivity and specificity of the parameters in predicting the occurrence of FES or hypoxia were also determined. [Table 2]. This study demonstrated the superiority of serum lactate over other factors assessed. The literature^[19-30] also, has, for long, suggested serum lactate to be a reliable indicator for “occult” shock, tissue hypoxia, morbidity, and mortality in critically-ill patients in general and the adequacy of resuscitation. The situations leading to hyperlactatemia post-traumatically have been discussed,^[19] and the foremost mechanism popularly adverted involves a systemic imbalance between oxygen delivery and demand. Apart from the anaerobic lactate generation, various aerobic processes have also been discussed in post-traumatic and post-septic lactatogenesis:

- Increased aerobic glycolysis by cytokine-mediated cellular uptake of glucose or by

catecholamine-stimulated Na-K pump hyperactivity → increased pyruvate production exceeding capacity of pyruvate dehydrogenase complex

- Pyruvate dehydrogenase dysfunction
- Direct lactate production by lung
- Reduced clearance of lactate

These postulates have lately cast doubts upon the genuineness of considering lactate as a specific marker for tissue oxygen debt. This study shows significant correlation of lactate levels at admission and subsequent development of hypoxia.

Another marker that has been extensively studied^[22,23,31-33] in critically-ill patients is the base deficit, which is defined as the amount of alkali buffer required to titrate one liter of blood to a pH of 7.40. It exhibits similarity to lactate in being a validated measure of metabolic acidosis and shares qualities of ease and rapidity of acquisition as well as consistency of results. We, however, did not observe any correlation between altered base deficit and subsequent development of FES or hypoxia.

Literature also supports our observation that altered base deficit is a poorer marker (than lactate) of inadequate resuscitation or hypoperfusion or mortality in post-traumatic and ICU patients. This may be explained by the fact that base deficit is affected by a plethora of factors causing metabolic acidosis above and beyond anaerobic metabolism (like renal dysfunction, saline fluid resuscitation, gastrointestinal bicarbonate losses and diabetic ketoacidosis).

Another useful tool^[34] in such situations may be the non-invasive assessment of hypoxia using the continuous pulse oximeter. It is considered to be a superior indicator of hypoxia than arterial blood gas analysis, since that an uninterrupted, non-invasive monitoring enables the detection of even transient episodes of hypoxia.

We observed CPOM to be a very sensitive indicator for occurrence of any pulmonary or systemic deterioration. All the patients who developed fat embolism syndrome had at least a single episode of dip in oxygen saturation during the initial 24 hours following trauma (sensitivity 100%).

We, hereby, wish to highlight the role of early shock and intravascular hypotension in the pathogenesis of FES,^[35-39] which result in the development of sudden, negative pressure within the venous system, causing suction of the marrow adipose tissue into circulation. It would follow that restoration of circulation and tissue perfusion in the golden hour would play a key role in preventing post traumatic respiratory complications especially FES.

The findings in this study point to the importance of two specific investigations in the immediate post-admission period in polytrauma patients (NISS >17): serum lactate estimation (within 12 hours of injury) and continuous SaO₂ monitoring using pulse oximetry (CPOM) for at least the initial 72 hours. A serum lactate of more than 22 mmol/l or even a transient episode of hypoxia on CPOM may indicate a higher risk of pulmonary complications. These patients may need to be triaged higher than other polytrauma patients during their management and require a more watchful approach. Such risk stratification is essential to ensure appropriate emergency health care delivery, especially in the developing countries, where the hospital inflow of polytrauma cases far outstrips the infrastructure. Estimation of pro-inflammatory cytokines, albeit useful, may not be practical in the developing nations owing to restricted availability and cost.

We, thus, suggest this simple protocol in polytrauma patients that may be invaluable in developing nations. We recommend that these patients presenting with a triad of polytrauma (NISS >17), raised initial serum lactate and even a transient episode of hypoxia are at a higher risk for developing FES/post-traumatic hypoxia ($P < 0.02$, Chi square).

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