CHAPTER 2

FAT EMBOLISM IN PATIENTS WITH
AN ISOLATED FRACTURE OF THE FEMORAL SHAFT

H.J. ten Duis, M.W.N. Nijsten, H.J. Klasen, B. Binnendijk

J Trauma 1988; 28: 383-390
ABSTRACT

Analysis of basic pathophysiologic variables in fat embolism patients is often restricted by the complexity of the different injuries present in each individual patient. To avoid this problem we investigated the presence of the fat embolism syndrome in patients with an 'isolated' fracture of the femoral shaft. Two groups were distinguished: those who had an open fracture or a closed fracture treated operatively within 24 hours after the accident (decompression group), and those who were treated initially conservatively (non-decompressed group). Clinical fat embolism occurred only in patients in the non-decompressed group (3.5%). They showed significantly higher temperatures, lower pulse rates, a progressive hemoglobin decrease, and a fracture localization more proximal ($p<0.025$) than the other patients in the non-decompressed group; they also showed pathophysiologic patterns significantly different from the patients in the decompressed group. Although the pathophysiologic mechanism of the onset of clinical fat embolism remains unclear, initial temperature elevations in combination with 'typical' fracture localization and fracture type appear to have a predictive value.

INTRODUCTION

The fat embolism syndrome is considered to be a symptom complex of acute respiratory distress, cerebral disturbances, and petechiae after one or more long bone fractures [1]. Many researchers have investigated the pathophysiology of this syndrome, though essential etiologic items remain unclear. Neither quantitative nor qualitative studies on fat embolization have been able to indicate consistent risk factors for the fat embolism syndrome. The clinical picture of full-blown fat embolism may develop after a single long bone fracture, although the incidence increases with the number of fractures. The investigation of a category of patients with single injuries enabled us to perform an analysis of possible etiologic factors without being influenced by accompanying injuries.

We studied a group of patients with an isolated fracture of the femoral shaft to determine which factors might predispose to the development of the fat embolism syndrome. Especially fracture site, fracture type and degree of comminution were analyzed and also minor fat embolism features like pyrexia, tachycardia, and thrombocytopenia.
MATERIALS AND METHODS

In the period 1967 through 1985, 172 consecutive patients, varying from 16 and 65 years of age, with an 'isolated' fracture of the femoral shaft, were studied from the moment they arrived at the Department of Traumatology at the University Hospital Groningen. Individuals with moderate and severe accompanying injuries were excluded, while patients with one minor additional injury (patella fracture, fracture of the clavicle or less) were included in the study. The investigation was performed on patients with a closed as well as an open fracture of the femoral shaft. Patients with a pathological fracture of the femoral shaft were excluded from this investigation.

Each patient was examined daily for the symptoms of fat embolism. Records of temperature, pulse and blood pressure were maintained as well as water and salt balance. The following investigations were performed daily for 6 days: supine chest X-ray, arterial blood gas analysis, hemoglobin, hematocrit, platelet- and white blood count, coagulograms, fibrinogen concentration levels, and serum electrolyte and triglyceride determinations. The X-rays of the femur, taken in the emergency room, were used to determine fracture localization in the femoral shaft, degree of comminution and fracture type. For fracture localization the shaft was divided into seven equal segments according to Kootstra [2] (Fig. 2.1).

The following fracture types were distinguished (we made use of the fracture quotient $Q=x/y$; $x$: length of fracture; $y$: width of femoral shaft at place of fracture): transverse $Q=1.0-1.1$; short oblique $Q=1.2-1.3$; oblique $Q=1.4-1.7$; longitudinal $Q>1.7$; spiral. The degree of comminution was

Table 2.1

| Accident cause in isolated fractures of the femoral shaft |
|--------------|-------|-------|
| Cause        | N    | %     |
| Fall         | 18   | 11    |
| Pedestrian   | 3    | 2     |
| Driver/passenger of Bicycle | 16   | 9     |
| Autobike     | 72   | 42    |
| Motorcycle   | 9    | 5     |
| Automobile   | 30   | 17    |
| Hit by heavy object | 7   | 4     |
| Miscellaneous | 17  | 10    |
noted: none, slight comminution with two or more fragments of less than 7 mm, moderate comminution with fragments from 7 mm to 2 cm, and serious comminution with multiple fragments greater than 2 cm with or without fragments of smaller size. Open fractures were treated, preferably with open reduction and internal fixation, on the day of the accident. In patients with a closed fracture, the fracture was reduced and immobilized in skeletal traction within 6 hours after admission. During the period 1967-1972 most closed fractures were treated conservatively; in the period 1972-1985 open reduction and internal fixation from day 10 onwards was usually the first choice of treatment. The diagnosis of fat embolism was made when at least two major symptoms as described by Gurd [3] were observed: 1) petechial rash, 2) respiratory distress with bilateral clinical and/or radiologic signs of pulmonary involvement, and 3) evidence of cerebral involvement unrelated to head injuries (primarily excluded). Two main groups of patients were distinguished: those who had a closed fracture and were initially (10 days) treated conservatively (non-decompressed group) and patients who had an open fracture, or a closed fracture but were treated initially (within 24 hours) operatively (decompression group).

No corticosteroids, antipyretics, or antiphlogistic medicaments were given in the initial phase of treatment. Student's t-test for unpaired values was used in the statistical evaluation of the results.

![Figure 2.2. Ages of the patients at the time of the accident.](image)

**RESULTS**

Of the 172 patients who satisfied the conditions of this study, 133 were males (77%) and 39 (23%) were females. The accident causes are given in Table 2.1. The average delay between accident and time of admission was 45 ± 35 minutes. The distribution of ages is given in Fig. 2.2. None of the patients showed any sign of shock at admission or during the initial phase of treatment.
There were 21 open and 151 closed fractures. Twenty-two patients with closed fractures were treated operatively on the day of the accident. Six patients (3.5%) were diagnosed as having fat embolism (Table 2.2), of whom four were given supplementary oxygen, and two patients had to be treated with mechanical ventilation. These patients all belonged to the primarily conservatively treated group with a closed fracture (129 patients). Neither the patients with an open fracture nor the patients with a closed fracture, initially treated operatively, developed two or more major signs of clinical fat embolism.

The fat embolism patients showed a fracture localization in segment two or three of the femoral shaft (mean 2.3). The average localization in the other patients was significantly more distal (mean 4.2; \( p < 0.025 \)). Fracture localization and degree of comminution are given in Table 2.3.

Temperature recordings revealed significantly higher temperatures on the day of the accident and day 1 in the fat embolism patients compared to the other patients in the non-decompressed group (\( p < 0.01 \), day 0; \( p < 0.005 \), day 1). Low initial temperatures were found in the decompressed group compared to the non-decompressed group (\( p < 0.01 \)) (Fig. 2.3).

The fat embolism group the initial pulse rate was as low as in the remaining patients of the non-decompressed group, but increased to significantly higher values during the rest of the observation period (\( p < 0.025 \)). Pulse rate values in the decompressed group generally followed the temperature pattern (Fig. 2.4).

All patients showed comparable decreases in hemoglobin and hematocrit values in the first 24 hours after the accident. Significantly lower values were observed in the fat embolism group from day 2 onwards (\( p < 0.025 \)) compared to the remaining patients in the non-decompressed group. This decrease was comparable to those of the open and/or primarily operatively treated patients (\( p < 0.001 \)) (Fig. 2.5).

---

**Table 2.2.** Major fat embolism symptoms present in patients of non-decompressed and decompression group.

<table>
<thead>
<tr>
<th></th>
<th>Non-decompressed</th>
<th>Decompressed</th>
</tr>
</thead>
<tbody>
<tr>
<td>FES (6)</td>
<td>No FES (123)</td>
<td>(43)</td>
</tr>
<tr>
<td>Petechial rash</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Pulmonary dysfunction</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>Mental dysfunction</td>
<td>4</td>
<td>1</td>
</tr>
</tbody>
</table>
All groups showed a temporary decrease in platelet count during the observation period. Platelet counts were slightly lower in the fat embolism group ($p<0.05$, day 1) (Fig. 2.6) and in the primarily decompressed group.

Arterial blood gas analysis revealed about equal values directly after the trauma in all groups. The fat embolism patients showed a significant decrease in PaO$_2$ and oxygen saturation levels on day 1 after the trauma ($p<0.001$) (Fig. 2.7). The fat embolism group had a PaO$_2$ of 8.0 kPa and an oxygen saturation of 90%, compared to 11.2 kPa and 96% for the non-decompressed group and 14.1 kPa and 97% in the decompressed group. After (symptomatic) treatment of the pulmonary dysfunction in the fat embolism patients, this group reached similar PaO$_2$ and oxygen saturation levels to the other groups around day 2 after the trauma. No important differences between the groups were observed in leukocyte counts, electrolytes, or BUN and creatinine levels, plasma protein, and albumin, as well as coagulograms. All patients survived and left the hospital in good health.

**DISCUSSION**

Fat embolism is a syndrome which includes the symptoms of respiratory failure, sensorium disturbances, and petechiae. The onset of the syndrome is unpredictable and there are no hard indications known which invariably
Figure 2.3. Mean with standard error of the mean of temperature values in patients of the fat embolism group (open squares), non-decompressed group (open circles) and decompressed group (filled circles) during the 6 day period following trauma. Statistically significant differences are marked with a star.

Figure 2.4. Mean with standard error of the mean of heart rate values in patients of the fat embolism group (open squares), non-decompressed group (open circles) and decompressed group (filled circles) during the 6 day period following trauma. Statistically significant differences are marked with a star.

lead to its development. The incidence - relatively rare in patients with single fractures - increases with the number of fractures or injuries [4].
In this study six cases of clinical fat embolism (3.5%) were observed. This percentage is comparable to other authors [5], considering the fact that this study only included solitary fractures.
Clinical fat embolism developed in patients who had a closed fracture and had not been operated on within 24 hours (non-decompressed group). In contrast, patients with an open fracture or those who had been operated upon early (within 24 hours; decompression group) using open reduction and internal fixation showed no clear evidence of pulmonary or cerebral disturbances or petechiae. This discrepancy between decompressed and non-decompressed patients, especially in the patients with closed fractures who were operated upon within 24 hours, suggests that induction phenomena
Fat embolism in patients with an isolated fracture of the femoral shaft

for the onset of fat embolism occurs in the first 24 hours. In this regard a well known factor is pulmonary embolization of marrow fat caused by fat globules, which are invariably found in the venous circulation after injury [5]. Although authors have tried to correlate the amount of fat globules with the incidence of fat embolism, it is unlikely that the syndrome is caused by this mechanism only. None of the patients with an open fracture or with a closed fracture who had been operated upon within 24 hours showed any evidence of fat embolism symptoms. Therefore, bone marrow embolization alone is unlikely to provoke fat embolism but it may compound the effect of other stresses in an injured patient to induce clinically apparent pulmonary problems. Especially local tissue ischemia by fracture hematoma may play an important role. Opening of the fracture evacuates the fracture hematoma and reduces tissue pressure around the fracture. In support of this (decompression) hypothesis is the investigation by Kallio in 1941 [6,7], who described a lower incidence of fat embolism in patients with open fractures. Concomitantly, this might be the reason why cases of postoperative clinical fat embolism were observed after acute 'closed' intramedullary nailing of the femoral shaft fractures [8].

Is the fat embolism syndrome related to fracture type or localization? Until now less attention has been paid to this criterion. All fat embolism patients in this series had a fracture localization in segment two or three of the femoral shaft, just cranial of the isthmus. At this place there several venous sinusoids within the marrow with large capillaries which do not have any wall musculature.

**Figure 2.7.** Mean with standard error of the mean of arterial $\text{pO}_2$ values in patients of the fat embolism group (open squares), non-decompressed group (open circles) and decompressed group (filled circles) during the 6 day period following trauma. Statistically significant differences are marked with a star.
The walls of these veins are very fragile here and are only embedded in depots of marrow fat [4].

This might explain why marrow fat just in this place might be released into the venous circulation.

The fat embolism patients were shown to have a short oblique or oblique fracture with a moderate degree of comminution (except the eldest patient with a spiral fracture) especially of fragments up to 2 cm. These fracture characteristics are often related to a certain degree of energy impact and are designated as high-energy injuries. Consequently, this moderate degree of bone marrow destruction will invariably be accompanied by fracture hematoma formation, soft-tissue injury, and consequently, in closed fractures, by local tissue hypoxia.

Interesting in this regard was that differences in blood loss reflected in hemoglobin and hematocrit decrease could not be found during the first 24 hours between the fat embolism group and the non-decompressed group. Significant differences were detected from day 2 onwards and seem therefore not related to the initial amount of hematoma formation.

The temperature pattern in fat embolism patients generally showed significantly elevated values within 24 hours after the accident, before any sign of pulmonary insufficiency was noticed. Here there was a significant difference to the main (non-decompressed) group with a highest mean of 37.9°C and 37.6°C in the group with any form of decompression. This pyrexia is a well known feature in patients with clinically apparent fat embolism, and it is probably induced through the release of interleukin-1 (endogenous pyrogen) by activated macrophages [9]. Fat globuli or toxic products released from the local ischemic tissues could serve as activators. The low initial pulse rate values (day of accident) in the fat embolism patients are of special interest because a rise in temperature is normally accompanied by an increase in pulse rate.

During the entire 6-day observation period the decompressed group was the least compromised concerning respiratory dysfunction. As expected, arterial PaO₂ and oxygen saturation were low at the onset of the fat embolism syndrome, although on admission differences between the groups could not be detected. As only two patients had to be treated by mechanical ventilation, the degree of respiratory distress was limited. Following the assumption that the pulmonary distress is an integral part of complement-induced PMN and platelet activation, the moderate degree of respiratory distress is in accordance with the only slight decrease in platelet numbers and the absence of severe disturbances in coagulograms.

The form of fat embolism described in this article is considered the nonfulminant subacute or classical form classified by Sevitt [11]. The fulminant fat embolism syndrome, which is often misdiagnosed and develops suddenly within a few hours after the accident, with an often fatal outcome was not observed in our selective series. It is unlikely that there will be any predictable sign for this form of fat embolism.

To our knowledge the patients described represent the first large series of patients investigated for the occurrence of fat embolism in a 'standardized' injury.

In this series a temperature increase to levels of 38.5°C or higher within 24 hours combined with a fracture of the femoral shaft just proximal to the isthmus seems to have some predictive value. If a patient shows these symptoms within 24 hours after the accident, one should be aware of the danger of a fat embolism syndrome during the following few days.
REFERENCES