CASE REPORT

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Case Report: Nonfracture-Associated Fatal Fat Embolism in a Case of Child Abuse

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ABSTRACT: Fatal fat embolism is usually thought of as a sequel to long-bone fracture, although cases secondary to soft tissue injury and atraumatic conditions have been infrequently reported. In this case of a two-year-old child-abuse victim who sustained multiple blunt traumatic injuries without skeletal fractures, pulmonary and systemic (brain and kidney) fat emboli were identified. At autopsy, all thoracic and abdominal viscera were intact; cranial contents exhibited only diffuse symmetrical petechial hemorrhages of the white matter. Because of the severe and widespread nature of soft tissue hemorrhage, and the absence of a grossly discernible cause of death, fat embolism was suspected. Using a combination of frozen section with oil red O staining and formalin-fixed osmium stained tissues, the immediate cause of death was determined to be diffuse fat embolism.

Review of the literature reveals a pathophysiologic basis for fat embolism in the absence of fracture, both as a consequence of an acute increase in local pressure at the site of trauma and an alteration of the emulsification of blood lipids during shock. In light of these findings, we present this case to remind the forensic science community to consider fat embolism as the cause of death in cases of blunt-force injury without fracture.

KEYWORDS: pathology and biology, injuries, child abuse, embolisms

Case Report

An autopsy was performed on a two-year-old black male at the request of local city police and the county coroner's office. The body of the victim was discovered at his home after the victim's mother telephoned local emergency services from another site.

Mechanism of Injury

Injuries were sustained by repetitive maternal beating with a belt over a six-month interval. The rate-limiting factor determining the length of each beating was "tiredness of the mother's right arm."

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Radiology

Before postmortem examination, total body radiographs revealed absence of osseous fractures (recent or remote) or periosteal reaction.

Exterior of the Body

The body had been bathed and clothed in clean garments. The body was that of a well-developed, well-nourished two-year-old boy. The body had a measured height of 35 in. (89 cm) and weight of 30 lbs (14 kg) (75th percentile). Presence of petechial rash could not be assessed because of body pigmentation. Bilateral periorbital ecchymoses surrounded diffuse scleral and conjunctival edema. Conjunctival petechiae were absent. The dorsal surface of the body demonstrated multiple diffuse abrasions and contusions over all surfaces.

Two parallel linear incisions were placed through the skin of the dorsum of the body extending from the upper borders of the scapulae to the posterior surfaces of the ankles. Diffuse subcutaneous hemorrhage was present on the back from the scapulae to the ilia bilaterally, in the proximal and distal thirds of each thigh, and in the distal two thirds of each leg (Fig. 1).

Anteriorly, diffuse contusions and abrasions were present over all surfaces. Several parallel linear contusions present on the thorax concorded in dimensions with a leather belt provided by city police. A "Y" incision over the anterior thorax and abdomen revealed a 1-cm hemorrhagic midline panniculus and diffuse fat necrosis from the superior aspect of the thorax to the symphysis pubis.

Examination of the extremities revealed ligature furrows about the wrists and ankles bilaterally and multiple contusions and abrasions.

Interior of the Body

The peritoneal cavity was without hemoperitoneum; all abdominal and retroperitoneal viscera were intact and without evidence of injury. Examination of the lungs and heart revealed no gross abnormalities. The head displayed focal frontal scalp hemorrhage bilaterally. The calvarium was intact. After formalin fixation, examination of the brain

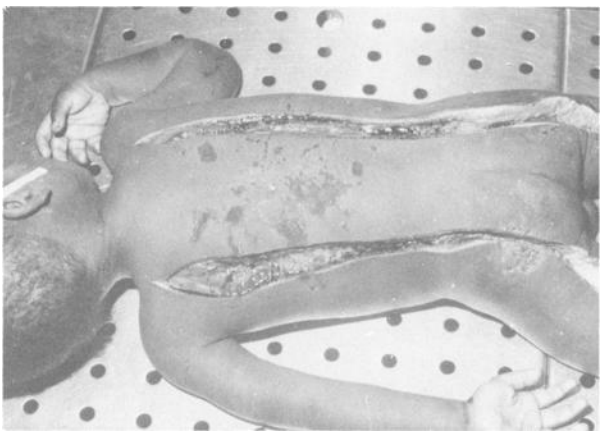

FIG. 1—*Dorsal view demonstrating diffuse subcutaneous hemorrhage exposed by parasagittal incisions.*

revealed diffuse symmetrical petechial hemorrhages of the white matter of the cerebrum and cerebellum (Fig. 2).

Microscopy

Routine hematoxylin and eosin preparations of lung tissue revealed focal chronic inflammation and atelectasis surrounding areas of probable fat embolization (Fig. 3). Oil red O stains on frozen sections of fresh lung tissue were positive for the presence of fat (Fig. 4). Routine hematoxylin and eosin staining of the cerebrum revealed multiple fibrin thrombi in the arterioles; concentric zones of clearing, containing material resembling fat, surrounded subcortical veins (Fig. 5). Presence of fat globules in the cerebrum was confirmed by osmium staining of fixed tissue (Fig. 6). Osmium staining of renal tissue also demonstrated the presence of fat globules (Fig. 7). Similar microscopic lesions were not present in tissues from an age-matched control.

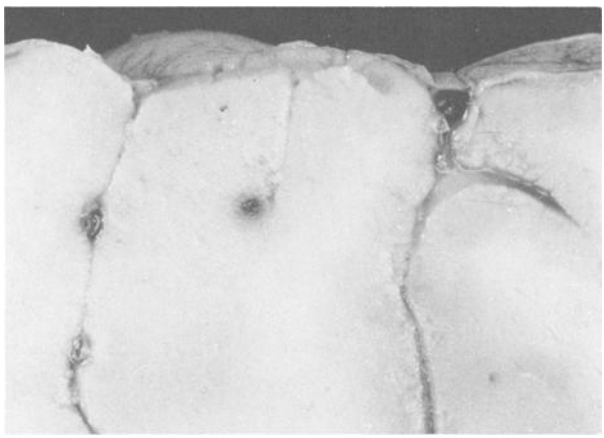

FIG. 2—*View of cerebrum demonstrating grossly visible petechial hemorrhage.*

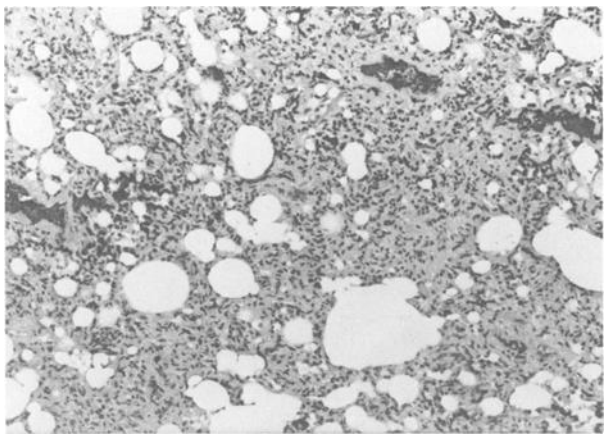

FIG. 3—*Lung, H&E staining, demonstrating atelectasis and chronic inflammation (×250).*

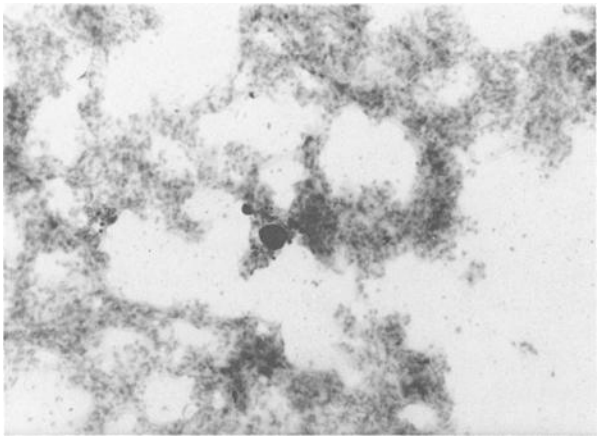

FIG. 4—*Lung, frozen section with oil red O stain, exhibiting fat globules within a capillary* (center) *and microdroplets within the interstitium* (left) *(×400).*

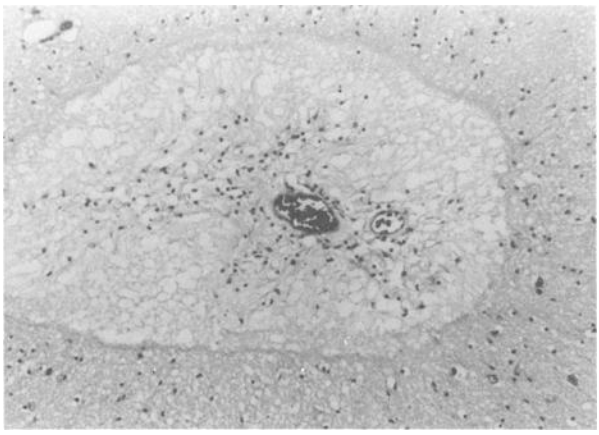

FIG. 5—*Brain, H&E staining. Concentric clearing and inflammation surrounding central vein (×250).*

Laboratory

Routine serum toxicology was negative. Urinalysis revealed a large amount of blood by chemistry strip; however, no red blood cells were seen by microscopy. Granular casts were seen, and the specific gravity was greater than 1.030.

Through history from the mother, it was ascertained that this child sustained severe, repeated soft-tissue injury over a period of months. Hypovolemia was present at the time of final insult, as indicated by the specific gravity of the urine (greater than 1.030). The immediate cause of death in this case was attributed to hypovolemia and pulmonary and systemic fat embolization.

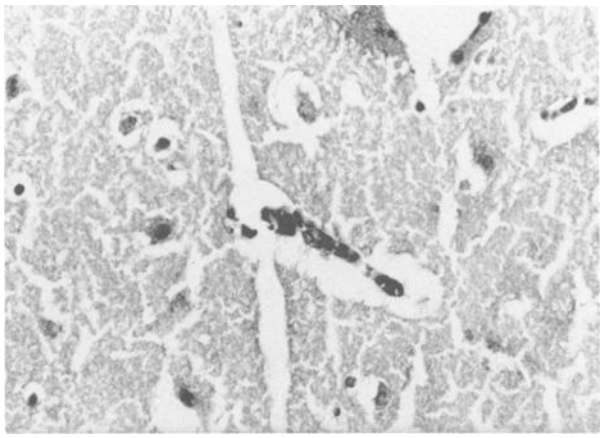

FIG. 6—*Brain, osmium tetroxide staining, demonstrating fat globules within an arteriole (×400).*

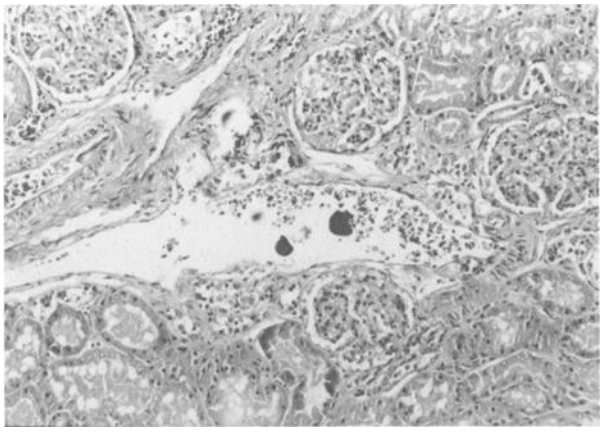

FIG. 7—*Kidney, osmium tetroxide staining, exhibiting fat globules within an arcuate artery (×100).*

Discussion

Fat embolism was first described by Muller in 1860, when he visualized fat globules in a patient's retinal vessels [*1*]. Since that time, fat embolism has become a well-known sequela of major trauma. The prototypical patient is a young adult, age 25 to 45 years, who has suffered severe impact trauma to the body as a whole, with resultant long-bone fractures [*2,3*].

Two theories have emerged to explain the pathogenesis of fat embolism. The mechanical theory proposes that physical disruption of fat cells liberates free fat, which, because of a local increase in pressure, is forced in torn veins in the vicinity [*2,3*]. The

biochemical (or physiochemical) theory states that tissue injury (regardless of etiology) initiates changes in the emulsion stability of blood lipids resulting in aggregation of intravascular fat [*1,4*]. Multiple experimental models have produced evidence supporting each theory [*1–3,5*]. Some researchers propose a combination of the two theories: a small mechanical embolization causes a change in the emulsion stability of blood lipids, thus producing a cascade of fat embolization [*1,5*].

It was found that emboli could originate from the marrow of fractured bone [*6–8*], and thus the observation that fat embolism rarely occurred in children, whose marrow contains little fat, was not surprising [*9*].

Fat embolism has now been identified in a variety of traumatic and atraumatic conditions. Injury of long bones remains the leading cause, and direct injury of subcutaneous fat is the second most common etiology [*10*]. Other conditions leading to fat embolism include carbon tetrachloride intoxication, extracorporeal circulation, nephritis, and shock [*1,4,9,11*].

Mild degrees of pulmonary fat emboli have been found in routine postmortem examinations [*1*]; this clinically insignificant finding has been termed "fat macroglobulinemia" [*10*]. True fat embolism is distinguished from fat macroglobulinemia by the presence of red blood cell aggregates, microthrombi, edema, inflammation, and tissue necrosis in association with fat globules [*10*].

Microscopic confirmation of the presence of fat globules requires special staining procedures. Oil red O staining is performed on frozen sections of fresh tissue. Osmium tetroxide can be used on formalin-fixed tissue and then embedded in paraffin. Some researchers feel that osmium tetroxide is superior to oil red O in localization of minute amounts of lipid [*12*]; osmium has the added advantage of being used on formalin fixed tissue.

In the case presented, fat embolization was suspected because of the extensive, severe subcutaneous tissue injury and the absence of apparent trauma to the viscera. Oil red O preparations of fresh lung tissue at the time of autopsy confirmed the diagnosis of pulmonary fat embolization. Later, osmium tetroxide staining of formalin fixed tissue revealed systemic fat embolization as well. It is suspected that the hypovolemic state of the victim at the time of final insult contributed to the pathogenesis of fatal fat embolism.

Conclusion

The fat embolism syndrome is a well-known sequel of severe trauma with long-bone fractures in young adults. It is reported less often in trauma without fractures and in other pathological conditions. Fat embolism is rarely discovered in children. Review of the literature failed to reveal any previously reported cases of fat embolism in child-abuse victims.

Diagnosis of fat embolism requires special staining procedures (oil red O or osmium tetroxide) to identify the presence of fat globules. In addition, surrounding tissue changes must be observed.

This case report is presented to remind the forensic science community to consider fat embolism as an immediate cause of death in cases of diffuse subcutaneous tissue injury without osseous fracture, regardless of the age of the victim.

Acknowledgments

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