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Case report

Trauma Related Fat Embolism Syndrome in Forensic Practice

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A B S T R A C T

The fat embolism syndrome (FES) in forensic practice is observed usually in cases of polytrauma related deaths. FES is rare, but serious complication after trauma. The most cases of post traumatic fat embolism are not fatal and it's very likely that many cases of mild fat embolism are overlooked. We describe a case of fat embolism syndrome in a young man after high energy injury. Upon concrete ceiling fell on his shoulders he had open fractures of both tibias and massive haematoma of the left hip joint. Transport immobilization of both legs was performed with admitting of analgesia during transportation to the hospital. Immediately after admission to hospital he underwent surgery. Several hours after osteosynthesis of both tibias, in ICU patient became hemodinamically and respiratory unstable. He developed global cyanosis and metabolic acidosis with significant decrease of oxygen pressure in the blood. Control chest radiograph showed typical »snow-storm« like pulmonary infiltrations. Deep bradicardy occurred followed by cardiopulmonary arrest. CPR was unsuccessful and the patient suddenly died within 12 hours from the incident. Histologic confirmation of fatty droplets the most commonly observed in the lung capillaries, brain capillaries or disseminated throughout the body remains diagnostic standard. We present one case of FES to emphasize the arising need of a quantitative analysis of both the size and localization of the fat emboli in order to grade the severity of FES and its relative contribution in pathophysiology of death. The postmortem diagnosis of fat embolism syndrome (FES), traditionally based on the histological demonstration of fat globules seems not to be enough, nowadays. A quantitative analysis of both the size and localization of the fat emboli has been discussed as reliable method of grading the pulmonary fat embolism in order to determine its relative functional contribution in death pathogenesis.

Key words: *fat embolism syndrome, forensic medicine, trauma*

Introduction

The fat embolism syndrome (FES) develops mostly in cases of long bone fractures. The prevalence of FES among all mechanical trauma (fracture mainly) patients has been reported to be between 0.25% and 1.25%[1]. Among patients with multiple bone fractures, the prevalence can reach up to 5%–10%[2].

Other causes of FES are associated with multiple traumas, decompression sickness, renal transplantation, blood transfusion, burns. The presence of fat droplets in the bloodstream is also associated with surgery procedures (liposuction) or orthopedic surgery, and infrequently in some non-traumatic conditions (fatty alteration of the liver, diabetes, acute pancreatitis).

A fracture of the ribs and sternum following the cardiopulmonary resuscitation (CPR) is probably the most common cause of pulmonary fat embolism seen at autopsy[3]. The postmortem diagnosis of FES is still traditionally based on the histological demonstration of fatty droplets. But as it has been observed by some authors there is an emerging need for a quantitative analysis of both the size and localization of the fat emboli in order to determine its relative functional contribution in death pathogenesis. This can be achieved primarily by grading the pulmonary fat embolism.

Case Report

We describe a case of FES in a young man after high energy injury. A male worker, 21 years old, had an accident on the tunnel construction site after the concrete

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ceiling felt on his shoulders and back. Open fractures of both tibias and hematoma without fracture of the left hip joint were detected (Figure 1). According to the Gustilo Classification the injury was classified as Grade III A and by AO classification type of fracture was 42C[4,5].

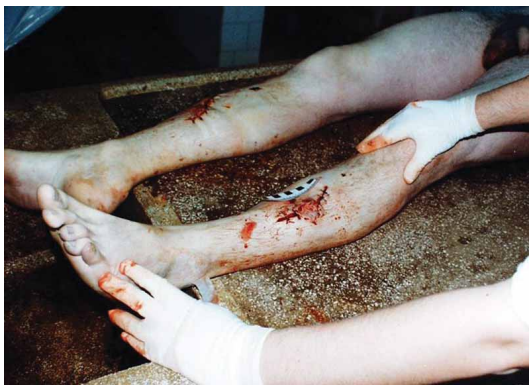

Fig. 1. Open fractures of both tibias and osteosynthesis point.

The transport immobilization of both legs and analgesia during transportation to the hospital were applied. Since the left sided thoracic pain was present the chest radiograph was performed with no signs of pneumothorax. Immediately after admission to the hospital he underwent surgery. Several hours after osteosynthesis of both tibias, in Intensive Care Unit (ICU) the patient became hemodinamically and respiratory unstable. Development of global cyanosis and metabolic acidosis with significant decrease of oxygen pressure in the blood was observed. The control chest radiograph showed typical »snow-storm« like pulmonary infiltrations (Figure 2). The symptoms of dyspnea become apparent followed with the neurological disturbances and Glasgow coma scale (GCS) estimated less than 5. The morning after, deep bradicardy occurred followed by cardiopulmonary arrest. CPR was unsuccessful and the patient suddenly died within 12 hours from the accident.

The autopsy was performed. The FES was confirmed. Macroscopically, on external examination petechiae were

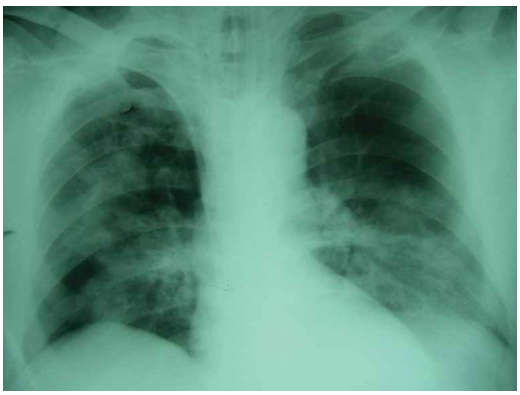

Fig. 2. Control chest radiograph with »snow-storm« like pulmonary perichilar infiltrations.

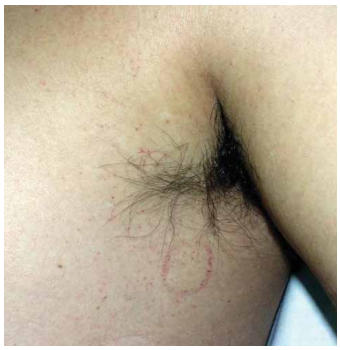

Fig. 3. Petechiae visible on the skin across the thorax.

visible on the skin across the chest and the back, the osteosynthesis on both tibias was also evidenced (Figure 3).

The internal examination showed macroscopically non-specific findings acute edema and lung congestion. Microscopically, the findings of fatty droplets in the lung alveolar capillaries (Figure 4) and in the renal glomeruli confirmed the diagnosis of FES.

Fig. 4. Histochemically (Oil-red O staining, x400), fatty droplets in lung alveolar capillaries.

A spectrum of pathophysiologic changes occurs with the phenomenon of fat embolism. At one end of this spectrum we have only sub clinical changes, and on the other end major pathophysiologic changes resulting serious disorders primary in respiratory and neurological system.

Discussion

Certain features of FES allow early clinical recognition. Symptoms are shortness of breath, followed by restlessness and confusion. Another striking feature or clinical sign of FES is that of the changing neurological symptom: the onset of restlessness, disorientation followed by marked confusion, stupor or coma[6].

One or two days after an injury, systemic fat embolism usually becomes apparent with characteristic clinical picture. FES can occur in immediate conjunction with

a precipitating factor or it can be delayed for up to 3 days, although 85% of cases are apparent within 48 hours[7].

Gurd and Wilson proposed the most widely accepted guidelines for the diagnosis of fat embolism, which require at least one sign from the major and at least four signs from the minor criteria[8]. Major criteria are: petechiae in a vest distribution, hypoxemia with PaO_2<60 mmHg, CNS depression and pulmonary edema. The minor criteria are: tachycardia>110 beats *per* minute, pyrexia>38.5°C, emboli visible in retina, fat in urine and sputum, unexplained drop in hematocrit or platelet count and increasing erythrocyte sedimentation rate. Other diagnostic systems have been also proposed namely, Schonfeld et al. proposed a fat embolism index that gives points for different diagnostic criteria[9,10].

Fat embolism is an important cause of acute respiratory distress syndrome (ARDS). The diagnostic workup of a patient suspected of having fat embolism syndrome include serial arterial blood gas measurements, as hypoxemia is one of the cardinal features. The clinicians should be vigilant in considering FES as a causative agent of postoperative respiratory distress. Serial chest radiographs as in our case can be used to observe the progression of ARDS infiltrates in the lungs, although it should be noted that chest radiographic changes are not often apparent in the initial stages of the syndrome[11].

Systemic fat embolism results in vascular obstruction and inflammation with petechiae seen within the brain and skin and demonstrable fat on microscopic examination of the brain and kidney using special stains and processing techniques. Pulmonary fat embolism detected microscopically is usually not associated with respiratory failure and the degree of fat embolism must be interpreted in the context of other factors such as the presence of other injuries and underlying natural disease processes[12]. The histological diagnosis of FES relies on observing fat globules in vascular spaces. Globules of fat within the pulmonary circulation may result in vascular obstruction, local vasoconstriction and pulmonary edema. The evaluation of functional contribution of FES to the cause of death in concrete case should be made. In attempt to do so different authors developed morphometric techniques of grading the pulmonary fat embolism. To accomplish that the total embolized tissue area should be investigated and compared to the total number of emboli; their mean area and the mean percentage of the embolized tissue area as suggested by Turillazzi et al.[13]. When mutually compared with the total tissue area of each sample and the total percentage of the embolized tissue area compared with the total tissue area of all slides. After that the surface area impact factor can be determine which can give us presumption of wound-producing capacity and the impact of the size of the surface area to which the force is applied.

The treatment of FES is primarily supportive. Early fracture fixation has decreased the incidence of pulmonary complications and fat embolism syndrome related to long bone fractures[14,15].

So the early surgical treatment with minimal movements[16] and combine with negative pressure during surgery are the goals of primary treatment. Because many patients suffer fat embolism syndrome in conjunction with multiple trauma, general supportive measures, including hemodynamic stabilization, maintained of normal electrolyte values, and prompt attention to orthopedic and soft tissue injury should be maintained[7,8].

Continuous follow-up of the patients with long bone fractures within first 72 hours, usage of fast-acting corticosteroids and good immobilization on the field are the first line in prevention of FES occurrence.

FES is rare, but serious complication of trauma. The most cases of post-traumatic fat embolism fortunately are not fatal[3]. This could explain why is likely that many cases of mild fat embolism are overlooked in ICU daily practice[6]. In forensic practice uncontrary the fat embolism is rarely observed but it should be suspected in trauma related deaths.

Lehman and Moore were among the first researchers to suggest that the phenomenon of posttraumatic fat embolism was the result of a change in droplet size of the endogenous plasma lipids[17]. This thesis has been supported by work of LeQuire et al. who found that the cholesterol content of the embolic fat was much higher than that of adipose tissue[18]. More recent investigations have demonstrated posttraumatic alterations in lipid metabolism and mobilization. Namely, elevated plasma levels of free fatty acids (FFA) and macroglobules and prolonged shifts in lipoprotein composition distinguish those patients at risk for developing fat embolism[19,20].

Conclusion

Forensic pathologist must consider FES as a potential cause of death in patients with long bone fractures. To establish fat embolism as a significant contributing factor to the cause of death one must identify systemic embolism. The postmortem diagnosis of FES, traditionally based on the histological demonstration of fat globules seems not to be enough, nowadays. Additional quantitative analysis of both the size and localization of the fat emboli has been discussed as reliable method of grading the pulmonary fat embolism in order to determine its relative functional contribution in death pathogenesis. In order to achieve this further analysis must be considered in cases of FES.

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SINDROM MASNE EMBOLIJE IZAZVAN TRAUMOM U SUDSKOMEDICINSKOJ PRAKSI

SAŽETAK

Sindrom masne embolije (SME) u sudskomedicinskoj praksi obično se razmatra u slučajevima smrti izazvanih politraumom. SME je rijetka, ali ozbiljna komplikacija traume. Većina slučajeva posttraumatske masne embolije nije fatalna pa su mnogi blaži slučajevi masne embolije vjerojatno previđeni. Mi opisujemo slučaj SME koji se razvio nakon mehaničke traume u mlađeg muškarca. Nakon što mu je betonski strop pao na leđa zadobio je otvoreni prijelom obje potkoljenice i opsežno krvarenje u području lijevog kuka. Učinjena je standardna imobilizacija prijeloma i analgezija tijekom transporta do bolnice. Odmah po primitku u bolnicu podvrgnut je kirurškom zahvatu. Nekoliko sati nakon osteosinteze prijeloma u jedinici intenzivnog liječenja pacijent je postao hemodinamski i respiratorno nestabilan. Razvila se globalna cijanoza i metabolička acidoza sa značajnim padom parcijalnog tlaka kisika. Kontrolna RTG snimka pluća pokazala je tipičnu infiltraciju pluća nalik »snježnoj oluji«. Nakon nastupa duboke bradikardije došlo je do kardiorespiratornog zastoja. Unatoč poduzetim mjerama oživljavanja, nagli smrtni ishod nastupio je unutar 12 sati od nezgode. Histološki dokaz masnih kapljica najčešće prisutnih u plućnim kapilarama, moždanim kapilarama i rasprostranjenih širom organizma ostaje dijagnostički standard. Prikazali smo slučaj SME da bismo istakli nužnost kvantitativne analize veličine i lokalizacije masnih embola s ciljem gradiranja SME i njegova razmjernog utjecaja u patofiziologiji smrti. Postmortalno postavljanje dijagnoze sindroma masne embolije, uvriježeno zasnovane na histološkom dokazivanju masnih kuglica, čini se, nije dovoljno u današnje doba. Kvantitativnu analizu veličine i lokalizacije masnih embola preporučujemo kao pouzdanu metodu za gradiranje masne embolije pluća s namjerom da se odredi značaj funkcionalnog doprinosa u patogenezi smrti.