Fat embolism syndrome

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Abstract

The authors review the fat embolism syndrome. After a brief introduction about pathogenesis, they describe the symptoms, diagnosis, therapy and prognosis of the fat embolism syndrome.

Key words: fat embolism syndrome.

Introduction

Fat embolism syndrome is a severe and life-threatening condition occurring usually as a complication of long bone fractures (in 0.9% – 2.2% of cases), particularly when the femur is involved or bone marrow handling during orthopaedic surgeries (0.5% – 0.8% of cases).[1]

The knowledge of such clinical entity goes back to 1862, time in which Zencker observed for the first time fat droplets in the pulmonary capillary bed in autopsied of multi-traumatized individuals.[1,2,3]

Fat embolism as a clinical entity is known since 1873, when von Bergman describes the first clinical case, relating it with the observations made previously by Zencker.[1,2]

Pathophysiology

Until now, the pathophysiology of such entity has been incompletely known, with several theories explaining several aspects of fat embolism syndrome emerging.[4]

One of such theories – Gauss theory – is based on the fact that the bone marrow is rich in the fatty tissue, especially in the lower limbs and pelvis. Blood vessels existing there have a fragile wall without a muscle layer. When the bone marrow suffers aggression (fractures, orthopaedic surgery), it is easy to admit that lipid droplets pass to the venous system causing embolism in the pulmonary capillary.[4]

Symptoms relating to the CNS would be explained by an embolism of the cerebral circulation by lipids droplets passing from the right atrium to the left atrium through the foramen ovale that according to some authors remains permeable 20 – 34% of the population.[4]

Another theory gives an important role to the C reactive protein, CRP, as cause of such syndrome. [1] CRP is synthetized in the liver responding to several infectious, inflammatory and neoplastic agents. Chylomicrons (CM) and very low density lipoprotein (VLDL) agglutination is seen in the presence of CRP high levels and in calcium dependency. [1] Lipids aggregated in such manner will cause embolism in the microcirculation of organs – target, where they are hydrolysed and shown to be toxic to the endothelial cells basis membrane, triggering the emergence of fat embolism syndrome.[1] Clinical cases where only the central nervous system is affected would be therefore explained.[5] The presence of lipids in the pulmonary capillary damages its cells, increasing its permeability as lysosome membranes break and inflammatory process mediators are called to the place, causing interstitial oedema which is the anatomical clinical basis for the adult respiratory distress syndrome.[6]

The fat embolism syndrome frequency changes in the literature reviewed, whether or not the lightest cases not in need of therapy are considered.

Around 90% of cases correspond to long bone fractures (pelvis and lower limbs), but it has also been seen in other circumstances where there is an aggression on the subcutaneous adipose tissue (lipoaspiration) or the one composing the long bones bone marrow (Orthopaedics surgery), as well as clinical situations following high levels of C reactive protein (multi-trauma without fractures, acute pancreatitis, diabetes mellitus, burns, drepanocytosis).[7]

Gauss mechanical theory explains the current observation of how rare the development of fat em-

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Received for publication on the 4th February 1997

bolism syndrome really is, in paediatric individuals where the bone marrow of long bones is not abundant in adipose tissue.[4]

Symptomatology

The fat embolism syndrome develops from 12 – 72 hours after the originating event in 90% of cases. Its clinical expression is most often made by dyspnoea; [1] petechiae in the upper half trunk and conjunctiva but it can reach the whole body;[3] CNS dysfunction causing seizures, confusion, drowsiness, stupor and coma;[1,8] retinopathy and febrile syndrome.[3]

As several fat embolism syndrome conditions are associated to cranial trauma, it might be difficult to allocate symptoms referring to the CNS exclusively to microembolism by lipids particles. It is possible cerebral structures are affected in the absence of pulmonary lesions.

Fever, possibly reaching 39°C, is a common and early sign albeit non-specific. Retinopathy due to fat embolism is translated by small retinian infarctions causing vision changes. Tachycardia is also an early sign. In a multi-traumatic patient this can be due to numerous factors and therefore it is only useful when combined with other diagnostic criteria.[1] The rare occurrence of fat embolism in cardiac tissue leads to disseminated myocardial necrosis, which is translated electrocardiographically with an inner depression of the ST segment, a low voltage of the ST segment and several degrees of atrioventricular block.[1]

Diagnosis

In laboratorial tests hypoxemia is found at an early stage[9] (64% of multi-traumatised), low hematocrit and leucocytosis. The last two endpoints are not specific and can raise the problem of differential diagnosis with other conditions.

Thorax teleradiography can be normal or containing bilateral infiltrate, indicating in such case the likely evolution and to adult respiratory distress syndrome.

Cranial encephalic CT scan can show oedema and small ischaemic areas[10] or it can be entirely normal.

Cranial encephalic NMR can restrict areas of ischemia and demielinisation of the white matter in cases where the CNS has been affected.[10]

Histologically, there are areas of haemorrhage in the brain without preferential location. It is only possible to show a lipid embolism within a few minutes after the trauma.[4]

The research for fat droplets can be made in the expectoration, bronchoalveolar lavage, clotted blood (cryostat test) and urine. Lipids in the urine have been found in 6% and in the expectoration in 40% of the fat embolism syndrome. These last two laboratory tests have not shown enough sensitivity and specificity to prove the usefulness to the diagnosis.[11]

The research of lipids in the clotted blood (cryostat test) has been shown to be a sensitive method for the clinical and subclinical diagnosis of fat embolism in syndrome, being positive in 52% of patients affected by such pathology and in 85% of multi-traumatised with multiple fractures carriers of such syndrome.

Bronchoalveolar lavage for the research of fat droplets is a method with high specificity (63%), quick and that is within our reach for the diagnosis of fat embolism syndrome, enabling in some cases to predict its progression.[12]

As most signs and symptoms are not specific, the diagnosis for the fat embolism syndrome is presumptive, based in a suggestive clinical condition within the context of a pathological situation with a triggering potential.

Therapy

The foreseen therapy for a long time is support. It should be used mechanical ventilation with or without introducing positive pressure at the end of the expiration (peep) and that should be made whenever necessary, by developed hypoxemia.[4]

The commitment by the documented CNS by clinical or image methods should be treated with hyperventilation and osmotic diuresis, with the purpose of solving the edema following such situations.[10] Corticoid administration, heparin, alcohol or dextran does not have a proven efficacy. [12] In the literature reviewed we found a study dated from 1983 showing that corticoids can have a prophylactic efficacy in the fat embolism syndrome.[4]

The pathophysiological basis for the beneficial effect of cortical therapy is based on its property of stabilising lysosome membranes, preventing them from rupture and a call to the local for mediators of the inflammatory process.

Heparin beneficial effect should be due to its capacity of preventing and improving thromboembolic phenomena which can occur concomitantly to fat embolism syndrome. It is not recommended the ad-

ministration of high doses of heparin in these cases as it can lead to important haemorrhages. Low-dose heparin is not usually followed by importance accessory effects and it is useful preventing thromboembolic phenomena, but it is not proven that it has any influence in the fat embolism syndrome.[4]

Situations of stress where a multi- traumatised patient is an example, are followed by adrenaline release and subsequent lipids mobilisation. Administering glucose and insulin is against the induced lipolysis by the hypercatabolic condition, reducing the lipids substrate to form the fat embolism syndrome. Its beneficial role is not totally demonstrated.[4]

Concluding, fat embolism syndrome is a self--limited disease which lesions are totally reversible if the patient is treated during the critical phase.[10]

Prognosis

The mortality rate found is variable according to researchers and it is widely dependent on the remaining pathological conditions associated to fat embolism syndrome.[4] The most effective methods for preventing it is the early fracture immobilization. ■

References

1. Müller C, Rahan BA, Ulrich Pfister Richard P. Meinig. The incidence, pathogenesis, diagnosis and treatment of fat embolism. Orthopaedic Review 1994; 23(2): 107-117.

2. Vance BM. The significance of fat embolism. Arch Surg 1931; 23: 426-465.

3. Peltier LF. The Diagnosis and Treatment of fat embolism. Journal of Trauma 1971; 11(8):661-667.

4. Fabian TC. Unravelling the fat embolism syndrome. N Engl J Med 1993; 329(13): 961-963.

5. Hulman G. Pathogenesis of non traumatic fat embolism. Lancet 1988; 18: 1366-1367.

6. Gossling HR, Doohue TA. The fat embolism syndrome. JAMA 1979; 241(25): 2740-2742.

7. Vichinsky E, Williams R, Maya Das, Earls AN, Lewis N. Pulmonary fat embolism: A distinct cause of severe acute chest Syndrome in sickle cell anemia. Blood 1994; 83 (11): 3107-3112.

8. Shulz PG, Weiner SP, Harber L, Armstrong D. Neurological Complications from fat emulsion therapy. Ann of Neurol 1994; 35(5): 628-630.

9. Fabian TC, Horts A, Stanford DS, Patterson R. Fat embolism syndrome: Prospective evaluation in 92 fracture patients. Critical Care Med 1990; 18(1): 42-46.

10. Shonfeld SA, Ploysongsans K, Ralph Dilisio, Crissman JD, Miller E. Fat embolism prophylaxis with corticosteroids. A prospective study in high risk patients. Ann Inter Med 1983; 99:438-443.

11. Scopa M, Magatti M, Rossitto P. Neurologic symptoms in fat embolism: care report. Journal of Trauma 1994; 36(6): 906-907.

12. Chan KM, Tham KT, Path MRC, Chiu HS. Post-traumatic fat embolism. Its clinical and subclinical presentations. Journal of Trauma 1984; 24(1): 45-49.

13. Chastre J, Fagon JY, Soler P, Fichelle A. Bonchoalveolar lavage for rapid diagnosis of the fat embolism syndrome in trauma patients. Ann Intern Med 1990; 113:583-588.