DOWNLOAD PDF MANAGEMENT OF FAT EMBOLISM SYNDROME

Chapter 1 : Fat embolism - Wikipedia

The fat embolism syndrome (FES) is a rare clinical condition in which circulating fat emboli or fat macroglobules lead to multisystem dysfunction. In , Zenker first described this syndrome at autopsy.

Abstract Fat emboli occur in all patients with long-bone fractures, but only few patients develop systemic dysfunction, particularly the triad of skin, brain, and lung dysfunction known as the fat embolism syndrome FES. Here we review the FES literature under different subheadings. The etiology may be traumatic or, rarely, nontraumatic. Various factors increase the incidence of FES. Mechanical and biochemical theories have been proposed for the pathophysiology of FES. The clinical manifestations include respiratory and cerebral dysfunction and a petechial rash. Diagnosis of FES is difficult. The other causes for the above-mentioned organ dysfunction have to be excluded. The clinical criteria along with imaging studies help in diagnosis. FES can be detected early by continuous pulse oximetry in high-risk patients. Treatment of FES is essentially supportive. Medications, including steroids, heparin, alcohol, and dextran, have been found to be ineffective. Brain, clinical criteria, fat emboli, imaging studies, lung How to cite this article: Emergency management of fat embolism syndrome. J Emerg Trauma Shock ;2: In , Zenker first described this syndrome at autopsy. It is usually asymptomatic, but a few patients will develop signs and symptoms of multiorgan dysfunction, particularly involving the triad of lungs, brain, and skin. It varies considerably according to the cause. The actual incidence of FES is not known, as mild cases often go unnoticed. The other forms of trauma that may be rarely responsible for FES include massive soft tissue injury, severe burn, bone marrow biopsy, bone marrow transplant, cardiopulmonary resuscitation, liposuction, and median sternotomy. The non-traumatic conditions are very uncommon causes of FES; they are acute pancreatitis, fatty liver, corticosteroid therapy, lymphography, fat emulsion infusion and haemoglobinopathies. Pathophysiology Two theories are postulated for the occurrence of FES. First, there is the mechanical theory by Gassling et al. Microvascular lodging of the droplets produces local ischemia and inflammation, with concomitant release of inflammatory mediators and vasoactive amines and platelet aggregation. Acute-phase reactants, such as C-reactive proteins, cause the chylomicrons to coalesce and create the physiologic reactions described above. In an experimental study it is found that the intramedullary pressure increased up to mm of Hg during reaming of the cavity. The clinical manifestations may develop h after trauma and especially after fractures when fat droplets act as emboli, becoming impacted in the pulmonary microvasculature and other microvascular beds such as in the brain. Embolism begins rather slowly and attains a maximum in about 48 h. The initial symptoms are probably caused by mechanical occlusion of multiple blood vessels with fat globules that are too large to pass through the capillaries. Unlike other embolic events, the vascular occlusion in fat embolism is often temporary or incomplete since the fat globules do not completely obstruct capillary blood flow because of their fluidity and deformability. The late presentation is thought to be a result of hydrolysis of the fat into the more irritating free fatty acids, which then migrate to other organs via the systemic circulation. It has also been suggested that paradoxical embolism occurs due to shunting. The manifestations include tachypnea, dyspnea, and cyanosis; hypoxemia may be detected hours before the onset of respiratory complaints. These changes are nonspecific, ranging from acute confusion to drowsiness, rigidity, convulsions, or coma. Cerebral edema contributes to the neurological deterioration. The particular distribution of the rash is related to the fact that the fat particles float in the aortic arch like oil in water and thus get embolized to the nondependent areas of the body. Renal changes may include lipuria, oliguria, or anuria and hepatic damage may manifest as jaundice. The retina may show exudates, edema, hemorrhage, or intravascular fat globules. Differential Diagnosis Dyspnea and hypoxia can also occur with pulmonary embolis and pneumonia. Cerebral dysfunction will occur with hypoxia or meningitis, but the rash of meningococcal septicemia spreads rapidly all over the body. Diagnosis FES is commonly diagnosed on the basis of the clinical features and by excluding other causes. Blood gases will show hypoxia, with a paO 2 of less than 60 mmHg along with the, and presence of hypocapnia. Thrombocytopenia, anemia, hypofibrinogenemia, and increased erythrocyte sedimentation rate ESR are seen in FES, but are nonspecific findings. A decrease in hematocrit occurs within h and is attributed to

intra-alveolar hemorrhage. Cytological examination of urine, blood, and sputum may detect fat globules that are either free or within macrophages. This test is not sensitive and its absence does not rule out fat embolism. Fat globules in the urine are common after trauma. Preliminary investigations of the cytology of pulmonary capillary blood obtained from a wedged pulmonary artery catheter revealed fat globules in patients with FES and showed that this method may be beneficial in early detection of patients at risk. CT computerized tomography head: Findings may be normal or may reveal diffuse white-matter petechial hemorrhages consistent with microvascular injury. CT will also rule out other causes for deterioration in consciousness level. Performed for suspicion of pulmonary embolus, the findings from this scan may be normal or may demonstrate subsegmental perfusion defects. Spiral chest CT for pulmonary embolism: As the embolic particles are lodged in the capillary beds, findings of spiral chest CT may be normal. Parenchymal changes consistent with lung contusion, acute lung injury, or adult respiratory distress syndrome ARDS may be evident. Scanty data exist regarding MRI findings in patients with this syndrome; however, in one small patient group, multiple, nonconfluent, hyperdense lesions were seen on proton-density and T2-weighted images. It will show typical white matter changes along the boundary zones of major vascular territories [Figure 2]. In a small case study, five patients with trauma were monitored with intracranial Doppler sonography during intraoperative nailing of long-bone fractures. Cerebral microembolic signals were detected as late as 4 days after injury. This procedure may be of use in evaluating intraoperative release of marrow contents into the bloodstream during intramedullary reaming and nailing. The density of the echogenic material passing through the right side of the heart correlates with the degree of reduction in arterial oxygen saturation. Repeated showers of emboli have been noted to increase right heart and pulmonary artery pressures. Embolization of marrow contents through a patent foramen ovale has also been noted. They may also be seen in patients on lipid infusions. High flow rate oxygen is given to maintain the arterial oxygen tension in the normal range. Additionally, maintenance of intravascular volume is important, because shock can exacerbate the lung injury caused by FES. Albumin has been recommended for volume resuscitation in addition to balanced electrolyte solution, because it not only restores blood volume but also binds with the fatty acids and may thus decrease the extent of lung injury. Mechanical ventilation and PEEP may be required to maintain arterial oxygenation. Increased alveolar-to-arterial oxygen gradient and neurology deficits, including altered consciousness, may last days or weeks. As in ARDS, the pulmonary sequelae usually resolve almost completely within a year. Residual subclinical diffusion capacity deficits may persist. Residual neurological deficits may range from subtle personality changes to memory loss, cognitive dysfunction and long term focal deficits. FES alone has not yet been reported to cause global anoxic injury, but it may play a contributory role, acting along with other cerebral insults. Even severe respiratory failure associated with fat embolism seldom leads to death. Conclusion A high index of suspicion is needed to diagnose FES.

Chapter 2 : Fat embolism | BJA Education | Oxford Academic

Fat embolism syndrome (FES) is a rare clinical syndrome that can complicate a wide variety of clinical conditions, particularly those where fat is manipulated. Almost all cases of FES are due to long bone and pelvic fractures (bone marrow contains a high content of fat).

View Large Clinical presentation Fat embolism syndrome typically presents 24â€”72 h after the initial injury. Rarely, cases occur as early as 12 h or as much as 2 weeks later. Respiratory changes are often the first clinical feature to present. Dyspnoea, tachypnoea, and hypoxaemia are the most frequent early findings. The severity of these symptoms varies but a number of cases may progress to respiratory failure and a syndrome indistinguishable from acute respiratory distress syndrome ARDS may develop. Approximately one-half of the patients with fat embolism syndrome caused by long bone fractures develop severe hypoxaemia and respiratory insufficiency and require mechanical ventilation. Neurological features resulting from cerebral embolism frequently present in the early stages. The changes range across a wide spectrum from mild confusion and drowsiness through to severe seizures. The more common presentation is with an acute confusional state but focal neurological signs, including hemiplegia, aphasia, apraxia, visual field disturbances, and anisocoria, have been described. Seizures and decorticate posturing have also been seen. Fortunately, almost all neurological deficits are transient and fully reversible. The characteristic petechial rash may be the last component of the triad to develop. This produces a petechial rash in the conjunctiva, oral mucous membrane, and skin folds of the upper body, especially the neck and axilla. The rash appears within the first 36 h and is self-limiting, disappearing completely within 7 days. A number of minor features of fat embolism syndrome may be present and these appear to result from the release of toxic mediators secondary either to the initial injury or to dysfunctional lipid metabolism. The presence of any these features may be of help in diagnosis discussed later. Pathogenesis The mechanism producing fat embolism syndrome is not clearly understood; mechanical and biochemical causes have been proposed. Fat emboli may occur either by direct entry of depot fat globules from disrupted adipose tissue or bone marrow into the bloodstream in areas of trauma mechanical or via production of toxic intermediaries of fat present in the plasma biochemical. It is feasible that both mechanisms are involved, with embolized fat from traumatized tissues undergoing a subsequent biochemical degradation. Fractures of marrow-containing bone have the highest incidence of fat embolism syndrome and cause the largest volume fat emboli. This may be because the disrupted venules in the marrow remain tethered open by their osseous attachments. Therefore, the marrow contents may enter the venous circulation with relatively little difficulty. Further emboli will produce an increase in pulmonary artery and right heart pressures, and material can pass through a patent foramen ovale into the systemic circulation, resulting in paradoxical embolism. Some studies have demonstrated the appearance of embolic material in the systemic circulation in the absence of a patent foramen ovale, potentially explaining neurological disease and petechiae on the basis of obstructive microembolism. However, this theory does not sufficiently explain the 24â€”72 h delay in development after the acute injury. Production of toxic intermediaries biochemical theory There are a number of biochemical mechanisms potentially involved in the development of fat embolism syndrome. The most widely held is that the embolized fat is degraded in plasma to free fatty acids. Although neutral fat, such as is found in bone marrow, does not cause an acute lung injury, it is hydrolysed over the course of hours to several products, including free fatty acids, which have been shown to cause ARDS in animal models. Free fatty acids have also been associated with cardiac contractile dysfunction, which can be a feature of fat embolism syndrome. The plasma lipase concentration is increased in some patients. Serum from acutely ill patients has been shown to have the capacity to agglutinate chylomicrons, low-density lipoproteins, and liposomes of nutritional fat emulsions. C-reactive protein, which is elevated in these patients, appears to be responsible for lipid agglutination and may also participate in the mechanism of non-traumatic fat embolism syndrome. The delay in development of symptoms could be explained by the timescale required to produce these toxic metabolites. The onset of symptoms may coincide with the agglutination and degradation of fat emboli. Levels of circulating free fatty acids are moderately elevated in fracture patients compared with

controls. Nevertheless, evidence for these mechanisms of injury remains largely circumstantial. Diagnosis Diagnosis is usually made on the basis of clinical findings but biochemical changes may be of value. The major criteria are based on the classic triad and clinical diagnosis is made by the presence of respiratory insufficiency, neurological impairment, and a petechial rash.

Chapter 3 : Fat embolism - The Clinical Advisor

Fat embolism syndrome is a clinical diagnosis with a classic triad of presenting symptoms and signs consisting of hypoxemia, neurologic abnormalities, and a petechial rash. It occurs most commonly in patients with single or multiple long-bone fractures, though it can occur in a variety of clinical.

The majority of the morbidity and mortality associated with this condition is due to the development of ARDS. Meta-analysis of patients with long bone fractures and FES reported only two deaths, for a mortality rate of 1. The etiology of ARDS in a patient may be difficult to determine, and this is the most likely reason for the discrepant incidence and mortality rates reported in the literature. Worst outcomes are seen in patients with severe neurologic injury. Survivors have an excellent prognosis with minimal long-term sequelae of FES. This article describes the two basic mechanisms that cause fat to embolize: Extremely well-written, thorough review of fat embolism defining the disease, epidemiology, etiology, pathophysiology, clinical presentation, diagnosis, management and prognosis of FES. If you have time for only one reference, this is it. Review article of classic literature on FES including cause, pathophysiology, presentation, diagnosis and treatment. This article specifies that clinical suspicion and diagnosis is the key. Treatment modalities are summarized well for the learner. Often-cited article that summarizes content, theory and practical treatment measures for patients with fat embolism. Dermatologic review of manifestations, features of fat embolism syndrome. J Bone Joint Surg Br. This article describes that early fixation of major orthopedic injuries has significantly reduced pulmonary complications from these injuries. The best treatment option is prevention. A prospective, randomized clinical trial". Arch Orthop Trauma Surg. This article prospectively randomized 40 patients to drainage via a venting hole or not in patients undergoing total hip arthroplasty. TEE was used to observe as well as hemodynamic and blood gases. Prospective, randomized trial in Level 1 trauma center using two reamer systems for placement of IM nail for femur fracture by monitoring right heart fat via TEE. The reamer-irrigator-aspirator showed a nearly significant decrease in volume of fat as a conventional reamer. This study used lungs from 36 rats and randomized them to receive saline solution, FE or FE with N-acetylcysteine treatment. The corn oil micelle model induces acute lung injury with associated biochemical changes. Treatment with N-acetylcysteine alleviates the pathologic and biochemical changes from FE. Chin J Physiol Nov. These authors used a corn oil model to induce FES in fourteen rats to examine the biochemical markers produced. They found release of cytokines, nitric oxide and others and demonstrated that antioxidants and NOS inhibitors abrogated fat embolism changes. Rats were given triolein intravenously to mimic FES. One hour later they were given captopril Ang I converting enzyme inhibitor or losartan Ang II type 1 receptor blocker. Histology 48 hours later revealed decreased inflammatory, vasoconstrictor and profibrotic effects in lungs of rats treated with either captopril or losartan. This article reviewed randomly assigned groups to corticosteroids or standard treatment of FES from to Primary outcome was development of FES. Of the studies identified, 7 met criteria. Steroids reduced development of FES and risk of hypoxia with no difference in mortality of infection. This article, retrospective and prospective, reviews nine patients with FES using high-resolution CT of chest. Patterns of CT were recorded and analyzed. CT findings document ground-glass opacities in seven patients with thickened interlobular septa in five patients, patchy distribution in four patients and nodular pattern in two patients. Abnormalities resolved on average within J Emerg Trauma Shock. This case study discusses a patient with probable FES whose diagnosis was confirmed by transcranial Doppler embolic signals and magnetic resonance imaging changes. This prospective study looked at forty-two patients with femoral shaft fracture and evaluated for right-to-left shunt. They used a transcranial Doppler to see if there was evidence of emboli and if it was more pronounced with patients who had a shunt. Presence of shunt had more embolic brain signals and predicted neurologic symptoms. They found higher levels of total cholesterol, lipid esters, monoglycerides, and phospholipase A2 in FES group. This prospective study looked at relationship between embolic events during cemented total hip arthroplasty techniques and TEE. There were no clinical signs of fat embolism. This case report describes a posttraumatic death from diffuse fat embolization to brain and lungs without a right-to-left shunt. Mellor, A, Soni, N. Review article that looks at

incidence, etiology, pathophysiology, diagnosis and treatment of fat embolism. Supportive treatment results in good outcomes. This article was a ten-year review of patients diagnosed with FES at a Level 1 trauma center from to Twenty-seven patients were identified with incidence of 0. This remains a diagnosis of exclusion. Am J Med Sci. They demonstrated that the incidence of FES depends on bone involved, number of fractures, age and gender of patient. They conclude that FES rarely is the result of a medical condition. No sponsor or advertiser has participated in, approved or paid for the content provided by Decision Support in Medicine LLC.

Chapter 4 : Learning Radiology - Fat Embolism

Fat embolism syndrome (FES) is a serious clinical disorder occurring after trauma, orthopedic procedures and rarely in non-traumatic patients. Fat emboli develop in nearly all patients with bone.

Dixit Find articles by R. Gupta Find articles by R. Gupta Find articles by N. This article has been corrected. This article has been cited by other articles in PMC. Abstract Fat embolism syndrome is an often overlooked cause of breathlessness in trauma wards. Presenting in a wide range of clinical signs of varying severity, fat embolism is usually diagnosed by a physician who keeps a high degree of suspicion. The clinical background, chronology of symptoms and corroborative laboratory findings are instrumental in a diagnosis of fat embolism syndrome. There are a few diagnostic criteria which are helpful in making a diagnosis of fat embolism syndrome. Management is mainly prevention of fat embolism syndrome, and organ supportive care. Except in fulminant fat embolism syndrome, the prognosis is usually good. Diagnosis, diagnostic criteria, fat embolism, fat embolism syndrome, management INTRODUCTION Dyspnoea in the trauma wards is not a rare occurrence, and it is a usual practice for the respiratory physician to get a call for a patient in acute distress in the trauma wards. The causes of dyspnoea can vary in origin and in the gravity of situation it may lead to. The various causes of dyspnoea, secondary to trauma are pulmonary contusions, fat embolism syndrome, shock lung, thromboembolism etc. These conditions are to be differentiated from other causes of dyspnoea like metabolic causes and cardiovascular causes. Differentiating each clinical condition from the others, in optimistic terms is perplexing and in pessimist terms, almost impossible. Fat embolism syndrome is by far the most common cause, and also the most overlooked cause of dyspnoea. Affecting a diverse range of organs and organ system, fat embolism has always enjoyed a prominent picture in many controversies in autopsy meetings. Two terms of interest are fat embolism and fat embolism syndrome. Often used in place of the other, these are not interchangeable. Whereas fat embolism refers to the presence of circulating fat globules in circulation and pulmonary parenchyma, fat embolism syndrome FES is the clinical manifestation of fat embolism. It is associated with a complex alteration of hemostasis, usually presenting as a triad of respiratory insufficiency, altered sensorium, and petechiae. Aetiology FES is commonly seen in trauma wards and is usually associated with fractures of long bones or multiple fractures. Rarely, intra-osseous fluid administration, lipid soluble radio contrast, intravenous hyper alimentation, long term steroid administrations, liposuction, bone marrow harvesting and transplant are also implicated as iatrogenic causes for FES. Fat embolism and milder forms of FES may go undetected clinically, and in obvious clinical situation, the diagnosis is overlooked. A level I trauma center in India,[10] after a retrospective analysis of case records of 1, patients reported an incidence of 0. Pathophysiology Fat embolism syndrome is a disease affecting mainly capillaries and mainly on the venous side. Hence, the lung is the most common organ affected in FES. However, the fat globules and chylomicrons gain access to the systemic circulation and can also affect heart, brain, skin and retina. The various explanations offered for the systemic manifestations of FES are: It is not exactly understood why some patients develop fat embolism while others do not. The symptoms usually occur within 12 h to 72 h, but can occur over a wide range of time of 6 h to 10 days. Pulmonary embolism by fat globules lead to an increase in the perfusion pressure, engorgement of the pulmonary vessels rendering the lung more stiff, which in turn results in an increase in the workload of the right heart. As the work of breathing increases, the right side of the heart attempts to increase its output by dilatation, which requires an increased venous return. Electrocardiograms at this time will indicate right heart strain. The heart is most susceptible to the effects of hypovolemic shock with a decreased central venous return. Death at this stage is due to acute right heart failure. Chemical theory The lung responds to the presence of fat emboli by secreting lipase, which hydrolyses the fat into free fatty acids and glycerol. The free fatty acids acting locally to produce an increase in the permeability of the capillary bed, a destruction of the alveolar architecture, and damage to lung surfactant. The delay in onset of symptoms may be due to the time required for the production of toxic metabolites. Nevertheless the evidence for these mechanisms of injury is yet to be proven. The other hypothesis proposed are combination of both mechanical and biochemical theories- initiation of symptoms by

fat globules followed by the biochemical cascade responsible for the rest of the symptoms. Risk factors With the complexities of symptoms and intricacies in management, FES has always enjoyed the attention of physicians. It has not been possible to explain why only some people develop FES while others do not. The incidence is higher among trauma patients and with long bone fractures. Young children are more immune to this syndrome due to the relative preponderance of hematopoietic tissue. Closed fractures, multiple fractures, and conservative therapy for long bone fractures have a higher association with FES. They found that an injury severity score more than 16, the presence of femoral fracture, a combined abdominal and extremity injury, or abnormal vital signs at admission all were independently predictive of the later development of ARDS due to FES. Usually presenting with a delay of h, the classical triad consists of respiratory distress, cerebral signs and petachiae. Fat embolism syndrome can go unnoticed clinically or may present as an acute fatal event within hours of the inciting injury. A non-fulminant fat embolism, which is clinically apparent and milder than the fulminant form has also been described by Sevitt. It is often confused with postoperative symptoms such as pain, discomfort, or postoperative inflammatory process. Associated tachypnoea, tachycardia, and fever may be seen. Respiratory symptoms are usually benign and lung parenchyma tends to improve from third day. They may vary from minimal tachypnoea to development of ARDS. Seen within hours of injury, it results in severe physiologic impairment including respiratory failure, and altered mental status. Cause of death, if occurs, is usually due to acute right heart failure. Clinical symptoms Respiratory symptoms These are usually the first presenting features. Hypoxemia, tachypnoea, and dyspnoea are the initial findings. In some cases, the patients may progress to respiratory failure, requiring mechanical ventilation. Neurological symptoms The symptoms may appear within h and are highly varying. The symptoms may include irritability, anxiety, agitation, confusion, delirium, and coma, which are described as progressive changes and as single manifestation in individual cases. These symptoms are usually non-lateralizing, transient and fully reversible. Localizing signs, such as aphasia, anisocoria, apraxia in a patient with poly-trauma warrants a computerized tomography of brain to rule out hematoma, although in FES, a CT brain will be usually non-rewarding. Cutaneous manifestations Petechial rash may be the last component to develop in FES. Appearing within 36 h, this is believed to be pathognomic feature of FES. It can be easily missed in dark skinned persons, and is to be actively sought on the upper portions of the chest and axillae. Other common sites of involvement are conjunctiva, oral cavity and shoulders. Hepatic involvement may manifest as jaundice. Hypocalcemia and hypoalbuminemia also have been reported to occur in patients with FES. The elevated serum lipase or FFA may cause a drop in calcium while it is postulated that the FFA binding to albumin is responsible for hypoalbuminemia. An increased alveolar- arterial shunt fraction, in the absence of ARDS, within h of a potentially causative event is strongly suggestive of FES. Depending on ABG analysis for diagnosing hypoxia is a costly affair, which also causes discomfort to the patient owing to its invasive nature. Pulse oximetry is an equally reliable non invasive testing method to diagnose hypoxic patients who are at risk for developing FES. It also helps in diagnosing sub clinical cases more easily, where the only sign might be hypoxia. The abnormalities observed are tachycardia with non-specific ST â€”T changes, right axis deviation and RV strain. Though helpful in excluding traumatic causes of altered behavior, its use in FES is limited. Fat embolism syndrome lesions are characteristically located deep in the white matter, brain stem and cerebellum ganglia. However, this has been challenged by Aoki et al,[40] who concluded it was a non-specific finding. The absence of fat globules in BAL macrophages has a high negative predictive value and may help in ruling out fat embolism. Fat droplets in BAL have also been reported in patients with sepsis, hyperlipidemia, and patients on lipid infusions. This technique demonstrated a correlation between a reduction in oxygen saturation and fat emboli passing through right heart. It also demonstrated systemic embolization through patent foramen ovale. There is no pathognomonic and specific clinical sign, laboratory investigation or imaging modality that can accurately point out an early diagnosis of FES. Therefore, the diagnosis of FES depends largely on clinical features and ruling out other differential diagnoses. An early diagnosis can be made by close monitoring of pulse oximetry and ABG analysis in high risk patients. Diagnostic criteria for FES Gurd and Wilson[42 , 43] proposed a diagnostic criteria for fat embolism in suspected patients. One of the three major manifestations i. This criteria lacked lung pathology descriptions and arterial blood gas

findings. Murry and Racz[44] proposed the presence of tachycardia, tachypnoea, pyrexia, and cerebral involvement with a decreased arterial oxygenation as a diagnostic criterion for FES. More recently a quantitative means of diagnosis of FES was proposed by Schonfeld. The clinical symptoms are vague and non-specific and hence require detailed evaluation for ruling out each. However, the time of presentation and associated laboratory findings may be helpful in clinching the diagnosis. Apart from intra vascular volume loss, respiratory distress may be due to pulmonary contusion, thromboembolism, pneumonia, ARDS. The presence of clinico-radiologic and lab findings of pneumonic consolidation is usually not missed. Pulmonary contusion presents with radiologic findings within 6 h of injury and are not confined to lobar anatomy. Rapid evolution of cavity and its resolution suggests a diagnosis of post traumatic pulmonary pseudo cyst. The radiologic findings in contusion will always correspond anatomically to the external area of trauma. The appearance of petechial rash in patients of poly-trauma is considered by many to be diagnostic of FES.

Chapter 5 : Emergency management of fat embolism syndrome

Fat Embolism Syndrome (FES) Fat embolism syndrome (FES) is a collection of symptoms and complications that result from the presence of a fat emboli in the blood. FES most often develops as a result of a long bone fracture, such as a fracture of your femur (leg) bone.

This is an open access article distributed under the Creative Commons Attribution License , which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. Abstract Fat embolism syndrome FES is a life-threatening complication in patients with orthopedic trauma, especially long bone fractures. The diagnosis of fat embolism is made by clinical features alone with no specific laboratory findings. FES has no specific treatment and requires supportive care, although it can be prevented by early fixation of bone fractures. The patient received supportive management and a short course of intravenous methylprednisolone. Introduction Fat embolism was first described by Zenker [1] in in a railroad worker with a thoracolumbar crush injury. In , Ernst von Bergmann was first to make a clinical diagnosis of fat embolism in a patient who fell off a roof and sustained a comminuted fracture of the distal femur. FES usually occurs within 48 hours after trauma or during surgical procedures in most patients; however, most patients are usually asymptomatic. A small number of patients may exhibit signs and symptoms involving the lungs, brain, and skin. Case Presentation A year-old man with a history of hypertension and old left basal ganglia hemorrhage was sent to Thammasat University Hospital for right hip surgery. The patient had been well until 10 days before this evaluation, when he slipped and fell. He immediately felt a right hip pain. He was diagnosed with a fracture of the right femoral neck Figure 1 by an orthopedist in a private hospital who subsequently referred him for an elective surgery at Thammasat University Hospital. The orthopedic team at Thammasat University Hospital planned a total hip arthroplasty for the patient. Carotid duplex ultrasound and transcranial Doppler ultrasound were evaluated before surgery without significant abnormality. Unfortunately, during surgery, the patient developed sudden hypotension and was resuscitated with ml of normal saline load. Within five minutes, his blood pressure was stable. Radiograph of the hip before surgery left and after surgery right. One hour after surgery, the patient became unresponsive after weaning off anesthetic drugs. Medical consultation was requested for persistent unresponsiveness. On evaluation by the consultant physician, the patient was unable to answer any question or follow command; he had no spontaneous eye opening. The temperature was The pupils were equal, round, and reactive to light with constriction from 3 mm to 2 mm. The vestibulo-ocular and gag reflexes were normal. There was no nuchal rigidity. Deep tendon reflexes were normal. Examination of the lung revealed tachypnea and fine crepitation at both lower lung areas. Examination of the heart revealed persistent tachycardia and was otherwise normal. Full laboratory test results before and after surgery are shown in Table 1. The CT scans of the brain, performed without administration of intravenous contrast, revealed no evidence of acute intracranial hemorrhage, territorial infarction, or intracranial mass lesion. The chest radiograph revealed consolidation at both lungs, more prominent at both lower lung fields Figure 3 , which is consistent with acute respiratory distress syndrome. The patient was intubated and transferred to intensive care units for additional evaluation and treatment. The arterial blood gas after intubation is shown in Table 1. The CT angiogram of the chest was normal. Chest radiograph at 1 hour after surgery left and 24 hours after surgery right. At 24 hours after the conclusion of surgery, the temperature was The patient was withdrawing from painful stimulation of hands and feet. The remainder of the examination was unchanged. The chest radiograph was improved Figure 3. Transcranial Doppler ultrasound was monitored to detect microembolic signals MES for 30 minutes on days 1, 3, 7, and 10 after surgery. Right to left shunt was evaluated by transcranial Doppler ultrasound and echocardiogram after intravenous injection of agitated saline on days 1 and 7 after surgery. Both tests showed no abnormality. Six days after surgery, the temperature was He was somnolent but could open his eyes in response to loud verbal stimuli. Other examinations were unremarkable. He was extubated with stable oxygen saturation while breathing in ambient air. Heparin of units was administered subcutaneously for every 12 hours to prevent deep vein thrombosis. A physical therapist team was consulted

to start early rehabilitation. Fourteen days after surgery, he started to obey commands although with mild aphasia. He had stable vital signs. He was subsequently discharged from the hospital after 7 days. Discussion FES is most commonly associated with orthopedic trauma, with highest incidence among closed, long bone fractures of the lower extremities, particularly the femur and pelvis [2]. Risk factors are male, ages 10â€”40 years, multiple fractures, and movement of unstable bone fractures. Possible causes of FES are shown in Table 2. Although the pathophysiology of FES remains poorly understood, two main theories were proposed to explain pathology. Mechanical theory, described by Gossling et al. These fat droplets then travel to the lungs and occlude pulmonary capillaries and systemic vasculatures. They can also enter the arterial circulation via a patent foramen ovale or directly through the pulmonary capillary bed, causing the characteristic neurological and dermatologic findings of FES. Biochemical theory, described by Baker et al. Local hydrolysis of triglyceride emboli by tissue lipase produces glycerols and toxic-free fatty acids. These intermediate products lead to an injury to pneumocytes and pulmonary endothelial cells causing vasogenic and cytotoxic edema leading to a development of acute lung injury or respiratory distress syndrome. The biochemical theory helps explain the nonorthopedic forms of FES. FES usually has an asymptomatic interval for about 12 to 72 hours after initial insult and is then followed by a classical triad of findingsâ€”respiratory insufficiency, petechial rash, and neurologic manifestation. Symptoms vary from dyspnea, tachypnea, and hypoxemia to ARDS. Symptoms are usually nonspecific, for example, headache, acute confusion, convulsion, or as severe as coma. Dermatologic manifestation is usually seen within 24 to 36 hours and usually distributed in nondependent regions of the body such as conjunctivae, head, neck, anterior thorax, or axillary areas. Most rashes typically disappear within a week. Other nonspecific symptoms include fever, thrombocytopenia, jaundice, lipuria, haematuria, and retinopathy [7]. In severe cases, FES can be complicated by disseminated intravascular coagulation, right ventricular dysfunction, shock, and death. There are no universal criteria for diagnosis of FES. Diagnosis is made by clinical suspicion and characteristic findings on imaging methods. However, there have been three previously proposed criteria by different authors: Gurd, Schonfeld, and Lindeque Tables 3 â€” 5. Laboratory findings in FES are usually nonspecific. Some patients may develop thrombocytopenia, anemia, or even hypofibrinogenemia. Cytological examination of the urine and sputum may show fat globules, but their diagnostic role still remains controversial. He discovered that oil red O positively stained macrophages are frequently observed in traumatic patients irrespective of the presence of FES [11]. Many imaging modalities can facilitate the diagnosis of FES, but none is specific. Chest radiographic findings may show diffuse bilateral patchy infiltrates, consistent with acute respiratory distress syndrome, although this must be differentiated from pulmonary hemorrhage or pulmonary edema. Chest films of some patients were normal. Noncontrast CT scans of the brain may be normal or reveal diffuse white matter petechial hemorrhage. These lesions gradually disappear within a few weeks to a few months [12]. However, T2-weighted MRI scans are of limited help in the hyperacute phase as these abnormalities may take several days to develop, and the findings remain highly nonspecific. MES was detected as early as 36 hours after a long bone fracture and could persist for as long as 10 days [14]. The mainstay treatment of FES is preventive and supportive. Supportive correction of hypoxemia with supplement oxygen or mechanical ventilation is needed while the patient recovers. Additionally, monitoring of hemodynamic status and maintenance of blood volume is important in FES because shock can exacerbate the lung injury caused by FES. If patients exhibit neurologic involvement, frequent neurological examination and monitoring of intracranial pressure should be considered. Adequate anesthesia to limit sympathetic response to injury is also beneficial. No specific drug is recommended or has strong evidence for treatment of FES. Systemic corticosteroids, which may reduce inflammation, perivascular hemorrhage, and edema, may have benefit in patients with deterioration of lung functions, but there is no systematic evidence to back up such benefit. Heparin has been proposed as a treatment of FES, due to its stimulatory effect on lipase activity and clearance of lipid from the circulation. Nevertheless, an increase in free fatty acids in the circulation could exacerbate the underlying proinflammatory physiology. Systemic anticoagulation has also been considered as a potential for FES therapy, but in the setting of trauma and pre-existing hematologic abnormalities, anticoagulants may be harmful. Early stabilization of long bone fracture is recommended to minimize bone marrow embolization into

the venous system. Prevention is the most important aspect in patients with long bone fractures. Current evidence suggests that the use of corticosteroid for prevention of FES may be considered for initial prophylaxis. A recent meta-analysis of six small randomized, controlled trials found that prophylactic corticosteroid administration reduced risk of FES development RR 0. Surgical timing and techniques have important roles for preventing FES.

Chapter 6 : Fat Embolism Syndrome. Information on fat embolism | Patient

Fat is prothrombotic and pro-inflammatory Commonly associated with orthopedic fractures, especially long bone fractures of lower extremities (eg. femur) Occurance in Men > women, highest rates: ages y/o.

Box , Doha-Qatar Address for correspondence: This article has been cited by other articles in PMC. Abstract Fat emboli occur in all patients with long-bone fractures, but only few patients develop systemic dysfunction, particularly the triad of skin, brain, and lung dysfunction known as the fat embolism syndrome FES. Here we review the FES literature under different subheadings. The etiology may be traumatic or, rarely, nontraumatic. Various factors increase the incidence of FES. Mechanical and biochemical theories have been proposed for the pathophysiology of FES. The clinical manifestations include respiratory and cerebral dysfunction and a petechial rash. Diagnosis of FES is difficult. The other causes for the above-mentioned organ dysfunction have to be excluded. The clinical criteria along with imaging studies help in diagnosis. FES can be detected early by continuous pulse oximetry in high-risk patients. Treatment of FES is essentially supportive. Medications, including steroids, heparin, alcohol, and dextran, have been found to be ineffective. In , Zenker first described this syndrome at autopsy. It is usually asymptomatic, but a few patients will develop signs and symptoms of multiorgan dysfunction, particularly involving the triad of lungs, brain, and skin. It varies considerably according to the cause. The actual incidence of FES is not known, as mild cases often go unnoticed. The other forms of trauma that may be rarely responsible for FES include massive soft tissue injury, severe burn, bone marrow biopsy, bone marrow transplant, cardiopulmonary resuscitation, liposuction, and median sternotomy. The non-traumatic conditions are very uncommon causes of FES; they are acute pancreatitis, fatty liver, corticosteroid therapy, lymphography, fat emulsion infusion and haemoglobinopathies. First, there is the mechanical theory by Gassling et al. Microvascular lodging of the droplets produces local ischemia and inflammation, with concomitant release of inflammatory mediators and vasoactive amines and platelet aggregation. Acute-phase reactants, such as C-reactive proteins, cause the chylomicrons to coalesce and create the physiologic reactions described above. In an experimental study it is found that the intramedullary pressure increased up to mm of Hg during reaming of the cavity. The clinical manifestations may develop 24â€”72 h after trauma and especially after fractures when fat droplets act as emboli, becoming impacted in the pulmonary microvasculature and other microvascular beds such as in the brain. Embolism begins rather slowly and attains a maximum in about 48 h. The initial symptoms are probably caused by mechanical occlusion of multiple blood vessels with fat globules that are too large to pass through the capillaries. Unlike other embolic events, the vascular occlusion in fat embolism is often temporary or incomplete since the fat globules do not completely obstruct capillary blood flow because of their fluidity and deformability. The late presentation is thought to be a result of hydrolysis of the fat into the more irritating free fatty acids, which then migrate to other organs via the systemic circulation. It has also been suggested that paradoxical embolism occurs due to shunting. The manifestations include tachypnea, dyspnea, and cyanosis; hypoxemia may be detected hours before the onset of respiratory complaints. These changes are nonspecific, ranging from acute confusion to drowsiness, rigidity, convulsions, or coma. Cerebral edema contributes to the neurological deterioration. The particular distribution of the rash is related to the fact that the fat particles float in the aortic arch like oil in water and thus get embolized to the nondependent areas of the body. Renal changes may include lipuria, oliguria, or anuria and hepatic damage may manifest as jaundice. The retina may show exudates, edema, hemorrhage, or intravascular fat globules. Cerebral dysfunction will occur with hypoxia or meningitis, but the rash of meningococcal septicemia spreads rapidly all over the body.

Chapter 7 : Fat embolism syndrome - WikEM

A fat embolism (which via major trauma may progress to fat embolism syndrome) is a type of embolism in which the embolus consists of fatty material. They are often caused by physical trauma such as fracture, soft tissue trauma, or burns.

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Fat embolism is usually asymptomatic, but in the minority of the patients symptoms and signs develop as a result of dysfunction of several organs, notably of the lungs, brain, and skin, in which case the term fat embolism syndrome (FES) is reserved.

Chapter 9 : Assessment and management of fat embolism - Oxford Medicine

Fat embolism syndrome (FES) occurs when embolic fat macroglobules pass into the small vessels of the lung and other sites, producing endothelial damage and resulting respiratory failure (acute respiratory distress syndrome (ARDS-like) picture), cerebral dysfunction and a petechial rash.