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Author/s:

Sutherland, TR;Lynch, MJ;O'Donnell, C

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Authors:

Sutherland Tom R 1,2,3

Lynch Matthew 1,4

O'Donnell Chris 1

Affiliations

1. Victorian Institute of Forensic Medicine
2. St Vincent's Hospital Melbourne, Medical Imaging Department
3. University of Melbourne, faculty of Medicine, Dentistry and Health Science
4. Monash University, Department of Forensic Medicine

Author contact details:

Tom.sutherland@svha.org.au

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Post-mortem CT features of Fulminant Fatal Fat Embolization associated with Prosthetic Femoral Neck Replacement

Fat or fatty bone marrow embolism is a well-recognised, albeit uncommon complication of osseous fractures and is defined as the “presence of fat globules in the pulmonary microcirculation regardless of clinical significance”(1). The incidence of fat embolization relates to the bones involved and the complexity of the fracture (2) with the greatest incidence in long bone fractures, particular the femur due to the larger volume of the medullary cavity containing bone marrow. The reported incidence of fat embolism varies widely (3), in part relating to varied diagnostic criteria and the fact that many cases may be asymptomatic, not clinically suspected or have the symptoms masked by other disease processes (4). When trans-oesophageal echocardiography is used intra-operatively, evidence of fat embolization is noted in around 23% of cases of femoral hip prosthesis procedures (5), and most frequently occurs at times of increased intramedullary pressures such as during reaming and cement introduction, with mechanical venous injury occurring simultaneously.

A previously well 93-year-old female was undergoing an elective right total hip replacement under general anaesthesia and suffered cardiac arrest as the hip prosthesis was positioned in the osseous cement. Resuscitation was not attempted as the patient had an active “not for resuscitation” plan. Non-contrast post-mortem CT (PMCT) demonstrated a well-positioned right total hip replacement and a large volume of intramedullary cement, extending to the distal femoral metaphysis. A large cord of hypodense fat with mean density of -64 HU was evident in the mid inferior vena cava (fig 1) with further low density layering in the anti-dependent pulmonary outflow tract having a mean density of -93 HU(fig 3). Both of these intravascular deposits were of similar density to subcutaneous and retroperitoneal fat. Internal examination was not undertaken.

There are two main consequences of fat embolism, one being mechanical vascular obstruction and the second a biochemical response; both processes occurring simultaneously. The biochemical response involves platelet aggregation and the production of glycerol and free fatty acids that in turn result in toxic injury to adjacent cells (1). This biochemical response results in systemic symptoms that develop within 12-72 hours following the embolism producing the classic clinical triad of hypoxia, petechial rash and non-specific neurologic depression. In the elderly femoral fracture and joint replacement populations, this is frequently not recognised as symptoms are similar to delirium and post-operative pneumonia. Central fat is uncommonly encountered at imaging but is a marker of more peripheral microscopic emboli that cause a clotting cascade, pulmonary arterial hypertension and reduced oxygen transfer.

In patients with an acute post-operative deterioration undergoing a CTPA, a careful search for subtle fat deposits should be performed, and CT of the inferior vena cava should also be considered in addition, as it provides another opportunity to identify intravascular fat and initiation of appropriate therapy.

Figures

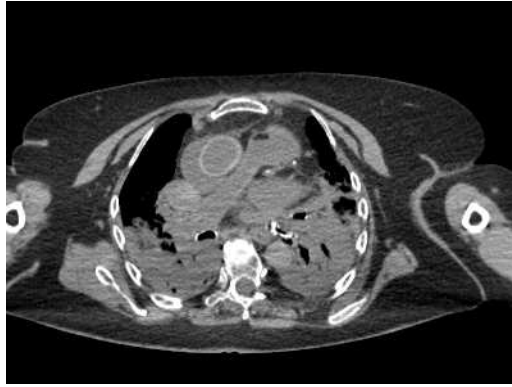
Figure 1 Coronal CT demonstrates a right hip prosthesis with extensive cement within the medullary cavity extending to the distal femoral metaphysis.

Figure 1 Sagittal CT shows a large cord of fat (arrow) within the sub-hepatic inferior vena cava,

Figure 2 Axial CT with a globule of fat within the ante-dependent aspect of the pulmonary outflow tract. Note that the degree of pulmonary consolidation is exaggerated as a post-mortem artefact.



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