

Epipericardial Fat Necrosis

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Epipericardial fat necrosis, also known as mediastinal fat necrosis, is a benign self-limited disease and is considered a rare cause for acute chest pain. There are fewer than 40 case reports of this condition. The largest case series describes 11 patients [1]. Histopathological reports suggest a local inflammatory process within the epipericardial fat [2] with no established etiology to date.

We report on the case of a 68 year old patient who presented with acute pleuritic chest pain. Workup for acute coronary syndrome was negative. Her chest X-ray demonstrated a small left pleural effusion. Subsequent chest computed tomography (CT) scans revealed epipericardial fat necrosis.

PATIENT DESCRIPTION

A 68 year old female patient was admitted with complaints of acute chest pain lasting

for four days, chiefly in the left mid-axillary line and lower ribs. The pain first appeared while resting, and was exaggerated by inspiration and postural changes without any relation to physical exercise. There was no dyspnea, perspiration, or syncope event, and no preceding febrile illness or upper respiratory tract infection symptoms. Her past medical history included obesity, uncontrolled hypertension, and mild hypercalcemia with a suspected parathyroid adenoma.

Her vital signs at hospital admission were unremarkable except for elevated blood pressure. Physical examination revealed a right carotid bruit and a small hematoma on her left lateral chest of 1.5 cm. Heart and lung auscultations were normal. There was no pedal edema or venous congestion. Laboratory workup was negative for cardiac biomarkers. D-dimer test was slightly above the normal range. The electrocardiogram demonstrated sinus rhythm, without any ST changes or other findings associated with acute ischemia, pulmonary embolism, or pericarditis. The chest X-ray revealed a small left pleural effusion, and no signs of congestion or cardiomegaly.

A transthoracic echocardiogram demonstrated good global function, 60% left

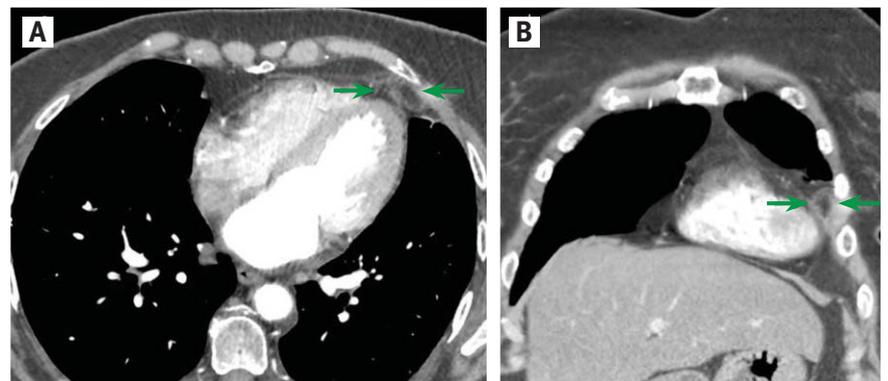
ventricular ejection fraction, without any valvular dysfunction, no regional wall abnormality, and no signs of pulmonary hypertension or right ventricular strain. There was a slight relaxation disturbance. A trial of pleurocentesis was unsuccessful. Direct ultrasound of the pleural space showed a small pleural effusion too small for pleurocentesis.

Assuming a non-cardiac origin for her complaints, a chest CT scan was performed. No signs of pulmonary embolism were found; however, a well circumscribed ovoid lesion was seen in the mediastinal fat, abutting the pericard in the left cardiophrenic angle [Figure 1]. The lesion had typical CT findings of epipericardial fat necrosis, which most likely was the source of the patient's chest pain.

COMMENT

Epipericardial fat necrosis is a rare and often overlooked etiology for chest pain. The incidence of this condition is still unknown, although one study documented epipericardial fat necrosis in as many as 2.5% within a cohort of patient admitted with acute chest pain who underwent chest CT during their evaluation [1].

Figure 1. Axial [A] and coronal [B] computed tomography images show a well-circumscribed ovoid fatty lesion (arrows) in the left cardiophrenic angle, abutting the pericard within the mediastinal fat



The common presentation is acute pleuritic chest pain, more frequently left-sided, with a negative workup for cardiac ischemia, pulmonary embolism, and pericarditis [3]. Physical examination is usually unremarkable. There are no elevations of both inflammatory and cardiac markers. Chest X-ray may reveal an ill-defined opacity near the cardiophrenic angle, ipsilateral to the side of the pain, occasionally with an associated pleural effusion. There are no validated data on the characteristics of the pleural effusion.

Chest CT is the modality of choice for diagnosis, demonstrating an encapsulated fatty lesion with inflammatory changes such as strands and thickening of the pericardium [3]. Epipericardial fat necrosis is more frequent in the left cardiophrenic angle, where anatomically there is more mediastinal fat.

The etiology of this disease remains unknown. One theory postulates acute torsion of a vascular pedicle as the cause of local ischemia and necrosis [2]. Another theory suggests increased intrathoracic pressure and beat-to-beat sheer force leading to hemorrhagic necrosis, especially where pre-existing fat abnormalities are present, such as hamartoma or lipomatosis [4]. The inflammatory histopathologic

appearance resembles conditions such as epiploic appendagitis and fat necrosis in the omentum or breast [2]. Baig and colleagues [5] hypothesized that thrombosis of abnormal arterioles that irrigate the epipericardial fat caused necrosis of this tissue.

There is no specific gender association of this condition. Obesity was previously considered a risk factor [1], but this condition may occur in patients with normal body mass index [5]. There are no other established risk factors.

Until recently, many patients had been treated with surgical excision of the lesion since the fatty mass seen on CT was suspected to represent neoplastic process such as liposarcoma. Since patient symptoms resolved after the surgery, resection was accepted to be the appropriate management. However, with increasing recognition that epipericardial fat necrosis is a benign self-limited process, it has become clear that surgical excision is not indicated [5]. Current management is conservative, and symptoms are managed with a short course of non-steroidal anti-inflammatory drugs. Follow-up CT can be performed to document resolution [1].

Epipericardial fat necrosis is probably an underdiagnosed condition that should be included in the differential diagnosis of

acute chest pain with a negative cardiopulmonary workup. A small pleural effusion without a clear explanation or nonspecific opacities near the cardiac silhouette on the chest X-ray may prompt further evaluation with chest CT. As symptoms might last for several weeks to months, a definite diagnosis based on typical CT findings prevents unnecessary or invasive investigation, with potential complications.

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Capsule

Distinct myeloid cell subsets promote meningeal remodeling and vascular repair after mild traumatic brain injury

Mild traumatic brain injury (mTBI) can cause meningeal vascular injury and cell death that spreads into the brain parenchyma and triggers local inflammation and recruitment of peripheral immune cells. The factors that dictate meningeal recovery after mTBI are unknown at present. Russo and co-authors demonstrated that most patients who had experienced mTBI resolved meningeal vascular damage within 2–3 weeks, although injury persisted for months in a subset of patients. To understand the recovery process, the authors studied a mouse model of mTBI and found extensive meningeal remodeling that was temporally reliant on infiltrating myeloid cells with divergent functions. Inflammatory myelomonocytic

cells scavenged dead cells in the lesion core, whereas wound-healing macrophages proliferated along the lesion perimeter and promoted angiogenesis through the clearance of fibrin and production of the matrix metalloproteinase MMP-2. Notably, a secondary injury experienced during the acute inflammatory phase aborted this repair program and enhanced inflammation, but a secondary injury experienced during the wound-healing phase did not. These findings demonstrate that meningeal vasculature can undergo regeneration after mTBI that is dependent on distinct myeloid cell subsets.

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Eitan Israeli

“A prince who is not wise himself will never take good advice”

Niccolo Machiavelli, (1469–1527), Italian diplomat, politician, historian, philosopher, humanist, and writer of the Renaissance period