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Acute epicardial fat necrosis: Case report of a 61-year-old male with pleuritic chest pain

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Abstract

Background: Acute Epicardial Fat Necrosis (AEFN) is a rare but significant clinical entity characterized by inflammation and necrosis of the epicardial adipose tissue. Frequently mistaken for more common cardiac or pulmonary conditions, such as acute myocardial infarction or pleuritis, AEFN poses a diagnostic challenge due to its nonspecific symptoms.

Case: We present the case of a 61-year-old male with a history of dyslipidemia and hypothyroidism who presented with pleuritic chest pain affecting the entire left hemithorax. The initial clinical presentation raised suspicion for Acute Coronary Syndrome (ACS), but diagnostic imaging ultimately revealed AEFN, leading to conservative management and eventual resolution of symptoms.

Conclusion: This case highlights the importance of considering AEFN in the differential diagnosis of chest pain and emphasizes the role of imaging, particularly Computed Tomography (CT), in avoiding unnecessary invasive interventions.

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Keywords: Acute epicardial fat necrosis; Acute chest pain; CT scan; Diagnosis.

Introduction

Acute Epicardial Fat Necrosis (AEFN) [1,2], is an uncommon cause of acute chest pain that can mimic serious cardiac and pulmonary conditions, often leading to unnecessary interventions if not accurately diagnosed. The epicardial fat is located between the pericardium and myocardium, playing a significant role in the homeostasis of coronary vessels and myocardial function. However, under certain pathological conditions, this fat tissue can undergo inflammatory changes, leading to necrosis and presenting with acute chest pain.

Although the exact pathogenesis of AEFN remains unclear [3], several factors have been implicated, including localized ischemia, trauma, or metabolic conditions such as obesity and diabetes mellitus. Imaging, particularly CT scans, plays a crucial role in diagnosing AEFN, as it can reveal fat stranding, low-density lesions in the epicardial fat, and associated inflammatory changes.

In this article, we describe the case of a 61-year-old male with dyslipidemia and hypothyroidism who presented with acute chest pain, initially suggestive of ACS. This case underscores the importance of considering AEFN in the differential diagnosis of chest pain, particularly in patients with atypical presentations.

Case presentation

Patient presentation

A 61-year-old male presented to the emergency department with a 48-hour history of progressively worsening chest pain. The pain was sharp, pleuritic in nature, and radiated diffusely across the entire left hemithorax. It was exacerbated by deep breathing and positional changes and relieved to a small extent by sitting upright. The patient denied any recent trauma, cough, fever, or chills. He also denied a history of previous similar episodes, palpitations, dyspnea, or diaphoresis. His past medical history was notable for dyslipidemia and hypothyroidism, both managed with atorvastatin and levothyroxine, respectively. The patient reported adherence to his medication regimen.

On physical examination, the patient was afebrile with stable vital signs: blood pressure of 130/85 mmHg, heart rate of 78 beats per minute, respiratory rate of 16 breaths per minute, and oxygen saturation of 98% on room air. Cardiac auscultation revealed a regular rhythm without murmurs, rubs, or gallops. Lungs were clear to auscultation bilaterally, and there was no tenderness on palpation of the chest wall. The abdominal and extremity examinations were unremarkable.

Initial workup

Given the patient's age and the nature of his chest pain, an ACS was the primary concern. The initial workup included:

Electrocardiogram (ECG): Normal sinus rhythm without ischemic changes. No ST-segment elevation or depression.

Cardiac biomarkers: Troponin I levels were within the normal range at presentation and upon serial testing.

Chest X-ray: Unremarkable, with no evidence of pleural effusion, pneumothorax, or consolidation.

Despite the lack of objective evidence for ACS, the atypical presentation and persistent pain warranted further evaluation. A CT of the chest and abdomen was performed to rule out pulmonary embolism, aortic dissection, and other potential causes of pleuritic chest pain.

Imaging findings

The CT scan of the chest showed no evidence of pulmonary embolism, aortic dissection, or coronary artery pathology. However, it did reveal an area of fat stranding and low-density changes in the epicardial fat along the left anterior and lateral aspect of the pericardium. These findings were consistent with acute epicardial fat necrosis. No pericardial effusion or signs of myocarditis were observed.

Diagnosis

Based on the clinical presentation, laboratory findings, and imaging results (Figure 1), a diagnosis of acute epicardial fat necrosis was made. Given the benign and self-limited nature of AEFN, the patient was managed conservatively with Nonsteroidal Anti-Inflammatory Drugs (NSAIDs) for pain control and close outpatient follow-up.

Clinical course and follow-up

The patient experienced significant improvement in his symptoms within a few days of starting Nonsteroidal Anti-Inflammatory Drugs (NSAID) therapy. He was discharged from the hospital after 48 hours of observation, with instructions for continued NSAID use and outpatient follow-up. At his two-week follow-up visit, the patient reported complete resolution of his chest pain. A repeat chest CT performed one month after the initial presentation showed resolution of the fat stranding and inflammatory changes in the epicardial fat, confirming the diagnosis of AEFN.

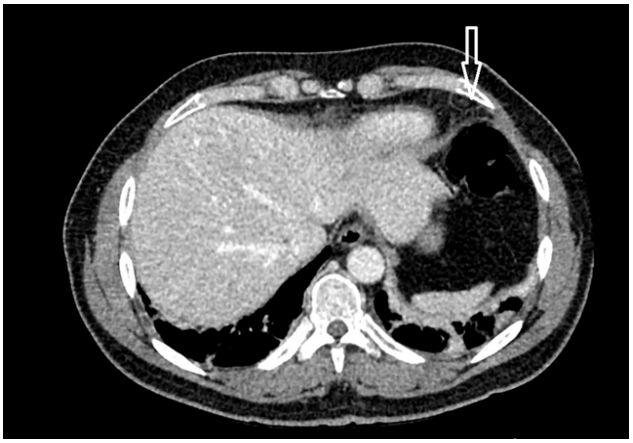

Figure 1: Clinical image.

Discussion

Epicardial fat is a visceral adipose tissue located between the myocardium and visceral pericardium. It plays an important role in protecting the coronary arteries by acting as a cushion and energy reservoir, and it is metabolically active, producing pro-inflammatory and anti-inflammatory cytokines. However, under certain pathological conditions, epicardial fat can become a site of inflammation and necrosis.

The pathogenesis of AEFN is not fully understood, but several mechanisms have been proposed. One hypothesis is that localized ischemia in the epicardial fat due to mechanical stress or vascular compromise leads to fat necrosis. Another theory suggests that systemic inflammation, particularly in the setting of metabolic disorders like obesity, dyslipidemia, and diabetes mellitus, contributes to the development of AEFN. In the present case, the patient had a history of dyslipidemia, which may have predisposed him to epicardial fat dysfunction.

Clinical presentation and diagnostic challenges

AEFN often presents as acute chest pain, which can be pleuritic in nature, as seen in this case. However, because the symptoms of AEFN are nonspecific, it is frequently misdiagnosed as more common conditions such as acute coronary syndrome, pulmonary embolism, or pericarditis. The pleuritic nature of the chest pain in AEFN is thought to result from inflammation of the adjacent pericardium or pleura due to the necrotic epicardial fat.

The differential diagnosis of pleuritic chest pain is broad and includes life-threatening conditions such as ACS, pulmonary embolism, aortic dissection, and pneumothorax. In the absence of specific clinical or laboratory findings, imaging plays a crucial role in distinguishing AEFN from these more serious conditions. In this case, the initial workup, including an ECG, cardiac biomarkers, and chest X-ray, was unremarkable, prompting further investigation with a CT scan, which ultimately revealed the pathognomonic findings of AEFN.

Imaging findings

CT imaging is the gold standard for diagnosing AEFN [4]. The

typical findings include areas of fat stranding and low-density lesions within the epicardial fat, often associated with inflammation or thickening of the adjacent pericardium or ipsilateral pleural effusion [5]. These findings are crucial in differentiating AEFN from other causes of chest pain, as the clinical presentation alone is insufficient for a definitive diagnosis. In this case, the CT scan revealed characteristic signs of AEFN, allowing for a non-invasive diagnosis and guiding appropriate management.

Management and prognosis

The management of AEFN is conservative, as the condition is typically self-limiting but the pain may persist for several weeks [6]. NSAIDs are the mainstay of treatment, providing relief from pain and inflammation [7]. In rare cases where symptoms are refractory or complications arise, corticosteroids or other anti-inflammatory agents may be considered. In our patient, NSAIDs provided effective symptom relief, and his symptoms resolved completely within two weeks.

The prognosis of AEFN is good [8], with most patients experiencing full recovery without long-term sequelae. Repeat imaging may be performed to confirm resolution of the inflammatory changes, but this is not always necessary if the clinical symptoms improve. In this case, a follow-up CT scan showed complete resolution of the fat necrosis and inflammation, confirming the diagnosis and successful treatment.

Conclusion

Acute epicardial fat necrosis is a rare and underrecognized cause of acute chest pain, often misdiagnosed as more serious conditions such as acute coronary syndrome or pulmonary embolism. This case highlights the importance of considering AEFN in the differential diagnosis of pleuritic chest pain, especially when initial workup is inconclusive. CT imaging plays a pivotal role in diagnosing AEFN and avoiding unnecessary invasive procedures. Conservative management with NSAIDs is generally effective, and the prognosis is excellent, with most patients recovering fully without complications.

Increased awareness of this condition among clinicians can lead to more accurate diagnoses and prevent overtreatment in patients presenting with acute chest pain. Collaboration between Primary Care and Emergency Department is important for the diagnosis and monitoring of the patient's evolutionary course.

Declarations

Contributions: EB & MAL participated in the diagnosis, treatment and follow-up of the patient. All authors participated in the writing and revision of the manuscript. All authors read and approved the final manuscript.

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