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Case Report

Epi-Pericardial Fat Necrosis: A Case Report

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Abstract

Epipericardial fat necrosis (EFN) is a rare and benign cause of acute chest pain that may clinically mimic more serious cardiopulmonary conditions such as acute coronary syndrome or pulmonary embolism. It is characterized by inflammation and necrosis of the epipericardial fat tissue and is increasingly recognized due to the widespread use of computed tomography (CT). We report the case of a 42-year-old patient who presented with sudden acute left-sided pleuritic chest pain without significant past medical history. Physical examination and laboratory findings were nonspecific. Contrast-enhanced chest CT demonstrated a well-circumscribed encapsulated fatty lesion adjacent to the pericardium, surrounded by inflammatory fat stranding, consistent with epipericardial fat necrosis. Conservative treatment with analgesics and anti-inflammatory medication resulted in favorable clinical evolution with symptom resolution. Recognition of the characteristic imaging findings of EFN is essential to establish an accurate diagnosis, avoid unnecessary invasive investigations, and reassure patients regarding the benign and self-limited nature of this entity.

Keywords: *Pericardial Fat Necrosis; Mediastinal Fat Necrosis; Cardiophrenic Angle Mass*

Introduction

Fat necrosis involving systemic adipose tissue may arise in multiple anatomical locations. It is commonly observed in the breast and subcutaneous tissues following trauma, in the peripancreatic fat in the setting of pancreatitis, and within the epiploic appendages, where it manifests as epiploic appendagitis [1]. In rare instances, fat necrosis may involve the epipericardial adipose tissue. Epipericardial fat necrosis (EFN) is an inflammatory condition affecting this compartment, resulting in localized, encapsulated necrosis of adipose tissue [2]. Although rare, it represents a benign, self-limiting cause of acute chest pain that can clinically mimic more serious conditions, including acute coronary syndrome and pulmonary embolism [3].

Case Report

A 42-year-old woman with no significant past medical history presented to the emergency department with acute left-sided pleuritic chest pain evolving over 24 hours. The pain was sudden in onset, sharp in character, non-radiating, and exacerbated by deep inspiration and positional changes. She denied fever, cough, dyspnea, recent trauma, or cardiovascular risk factors. Physical examination revealed stable vital signs and localized tenderness over the left anterior hemithorax without signs of respiratory distress. Laboratory investigations

demonstrated normal cardiac enzymes, inflammatory markers, and complete blood count. Electrocardiography showed no ischemic abnormalities.

A chest radiograph revealed a subtle ill-defined paracardiac opacity adjacent to the left cardiophrenic angle. Subsequently, contrast-enhanced thoracic computed tomography (CT) demonstrated an encapsulated ovoid fatty lesion measuring approximately 37 x 10 x 24 mm TxAPxh within the left pericardial fat, surrounded by inflammatory fat stranding and associated with homolateral mild pleural effusion and very subtle atelectatic changes involving the ipsilateral lower basal lobe, as illustrated in the figures below (Figure 1-5). No pulmonary embolism, thickened pericardium, pericardial effusion, or mediastinal lymphadenopathy was identified. The radiological findings were highly suggestive of epipericardial fat necrosis.

Figure 1: *Axial contrast-enhanced chest CT scan demonstrating epipericardial fat necrosis. The image shows an ovoid, encapsulated fatty lesion located external to the left pericardium, surrounded by a thin soft-tissue rim (Blue arrow) and associated with adjacent inflammatory fat stranding (Green arrow), consistent with typical imaging features of epipericardial fat necrosis.*

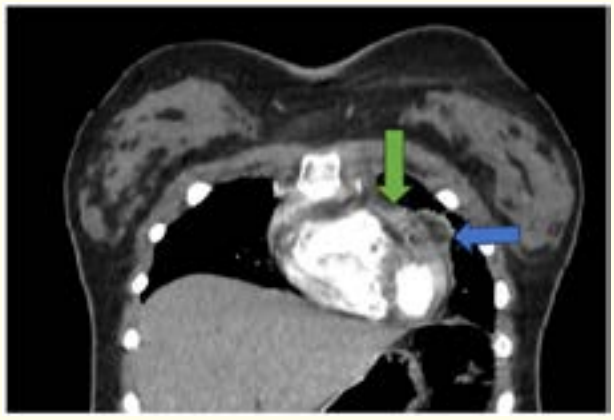

Figure 2: *Coronal contrast-enhanced chest CT scan demonstrating an ovoid encapsulated fatty lesion located external to the pericardia, surrounded by a thin soft-tissue rim (Blue arrow) and adjacent inflammatory fat stranding (Green arrow), findings suggestive of epipericardial fat necrosis.*

Figure 3: *Axial contrast-enhanced chest CT scan demonstrating minimal pleural effusion adjacent to the lower hemithorax (Blue arrow).*

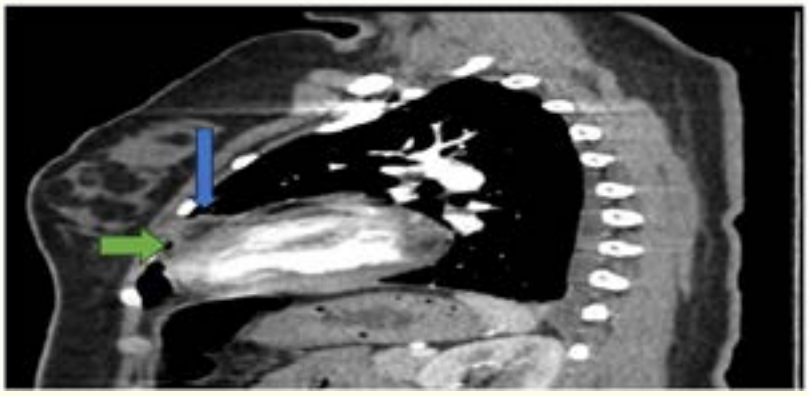

Figure 4: *Sagittal contrast-enhanced chest CT scan demonstrating an ovoid encapsulated fatty lesion located external to the pericardium, surrounded by a thin soft-tissue rim (Blue arrow), and adjacent inflammatory fat stranding (Green arrow), compatible with epipericardial fat necrosis.*

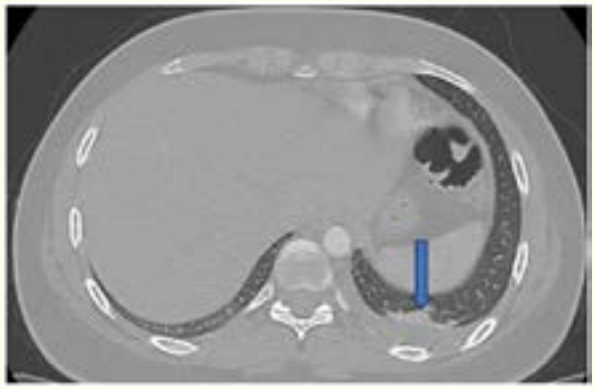

Figure 5: *Axial chest CT scan on the lung parenchymal window demonstrating mild lower lobe atelectatic changes (Blue arrow).*

The patient was managed conservatively with nonsteroidal anti-inflammatory drugs and analgesics, resulting in rapid symptomatic improvement within several days. Follow-up CT performed six weeks later demonstrated marked regression of the inflammatory changes and decrease in lesion size, confirming the diagnosis of pericardial fat necrosis. No recurrence was observed during subsequent clinical follow-up.

This case illustrates the characteristic clinical and radiological presentation of epipericardial fat necrosis, a rare and benign cause of acute chest pain that may mimic more serious cardiopulmonary conditions. Recognition of its typical CT appearance is essential to avoid unnecessary invasive investigations and to ensure appropriate conservative management.

Discussion

Epipericardial fat necrosis (EFN) is an uncommon benign inflammatory condition involving the adipose tissue adjacent to the pericardium. Although rare, it represents a potential cause of acute chest pain and may clinically simulate more severe cardiothoracic emergencies, including acute coronary syndrome, aortic dissection, and pulmonary embolism [4].

Fat necrosis may affect different adipose tissue compartments throughout the body, including the breast and subcutaneous tissues following trauma, the peripancreatic fat during pancreatitis, and the epiploic appendages in cases of epiploic appendagitis, in rare instances, this process can also involve the epipericardial adipose tissue [5,6].

A comprehensive review of the English-language medical literature published up to 2016 identified only 57 reported cases of epipericardial fat necrosis since its initial description in 1957 [1]. To date, the only available prevalence estimates originate from two retrospective Brazilian studies, which reported an approximate prevalence of 2% among patients presenting to the emergency department with acute atypical chest pain [7,8].

Epipericardial fat necrosis affects both males and females without a clear sex or age predilection, and no definitive predisposing factors have been identified in the literature. In our case, the patient was female [9].

The pathophysiology of epipericardial fat necrosis remains incompletely understood, and two principal mechanisms have been proposed to explain its development. One hypothesis suggests that torsion of a pedunculated fatty appendage vascularized by a feeding pedicle may result in ischemia and subsequent fat necrosis [10,11].

An alternative theory implicates repetitive mechanical stress and shearing forces generated during the cardiac cycle, particularly in the presence of pre-existing structural abnormalities of the adipose tissue such as lipoma or hamartoma [12,13].

To date, no definitive risk factors for EFN have been identified. Although obesity was initially considered a potential predisposing factor, subsequent reports failed to demonstrate a consistent association [14].

Patients with epipericardial fat necrosis typically present with acute pleuritic chest pain, which may clinically mimic pulmonary embolism or acute pericarditis. The pain is generally ipsilateral to the lesion, most frequently involving the left hemithorax, and may persist for several weeks. Additional manifestations such as syncope, tachycardia, and dyspnea have occasionally been reported [15].

Physical examination may demonstrate pericardial tenderness on palpation as well as a pericardial friction rub [16] whereas laboratory investigations, including inflammatory markers, as well as electrocardiographic findings are usually unremarkable, in some cases, elevated C-reactive protein levels have been observed, likely reflecting an inflammatory response associated with necrosis of the epipericardial fat [17].

These findings are globally concordant with our case, in which the patient presented with acute pleuritic chest pain associated with normal ECG and laboratory results, including CRP and cardiac marker [15].

The diagnostic evaluation of epipericardial fat necrosis generally begins with chest radiography. Although chest X-ray findings may suggest the diagnosis, they are not sufficiently specific to establish it with certainty. The most commonly described radiographic feature is an ill-defined paracardiac opacity adjacent to the cardiomediastinal silhouette, which may raise suspicion for an underlying mediastinal mass lesion [18].

Chest computed tomography (CT) represents the second-line and most decisive imaging modality in the diagnostic evaluation of epipericardial fat necrosis, as it enables accurate characterization of the lesion and precise assessment of its anatomical location. The typical CT appearance consists of an ovoid paracardiac fatty lesion surrounded by a thin capsule, associated with a central area of increased attenuation and adjacent inflammatory fat stranding; these findings are considered highly suggestive of EFN [19]. These imaging features were concordant with those observed in our patient [19].

Other CT findings associated with this pathology include pleural effusion, pericardial thickening, and moderate atelectasis [20]. In our case, only a mild ipsilateral pleural effusion and minimal atelectatic changes were identified.

These imaging features sus mentioned, when recognized, are highly suggestive of the diagnosis and may obviate the need for further invasive evaluation [21].

However, due to limited awareness of this entity, it has historically been misinterpreted as a mediastinal neoplasm, leading in the past to unnecessary surgical excision of the affected fat tissue. Until the early 2000s, surgical resection was often considered the definitive management approach to exclude malignancy. Subsequent recognition that the CT appearance is analogous to fat necrosis in other anatomical sites, together with documented spontaneous regression on follow-up imaging, has established epipericardial fat necrosis as a benign and self-limiting condition that can be confidently diagnosed based on its typical radiological pattern [21].

No invasion of adjacent structures, including the chest wall or myocardium, is typically observed in epipericardial fat necrosis. Nevertheless, the inflammatory process may extend toward the pleural fissures due to the anatomical continuity of the epipericardial fat with the pleural adipose tissue [22]. The surrounding inflammatory fat stranding observed on CT is comparable to that described in other forms of fat necrosis, such as omental torsion and epiploic appendagitis [23]. In certain cases, peripheral or central calcifications may develop within the lesion over time [24]. The presence of inflammatory stranding associated with encapsulated fatty tissue and preservation of diaphragmatic integrity are considered highly suggestive radiological features of EFN [25].

Historically, epipericardial fat necrosis was predominantly managed by thoracotomy with surgical excision of the lesion. Surgical treatment generally resulted in symptom resolution without significant postoperative complications or short-term mortality. Between 1957 and 2005, approximately 20 surgically treated cases of EFN were reported in the literature. Although surgery was initially recommended to confirm the diagnosis and exclude thoracic neoplasms, subsequent studies demonstrated that epipericardial fat necrosis can be reliably diagnosed on CT and managed conservatively, with spontaneous regression on follow-up imaging [4].

Conclusion

In conclusion, epipericardial fat necrosis is a rare and underrecognized cause of acute chest pain that may mimic more serious cardiopulmonary conditions, leading to diagnostic uncertainty and unnecessary invasive investigations. The nonspecific clinical presentation represents a major diagnostic challenge, making imaging particularly computed tomography, essential for establishing an accurate diagnosis. Recognition of its characteristic radiological features is crucial to avoid misdiagnosis and inappropriate management, as the condition is benign and usually resolves with conservative treatment.

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