

Epipericardial fat necrosis – a rare cause of pleuritic chest pain: case report and review of the literature

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Abstract

Epipericardial fat necrosis (EPFN) is an uncommon cause of chest pain. Typically manifesting as severe acute chest pain, EPFN can be mistaken for a serious disorder, such as pulmonary embolism or myocardial infarction. We report a case of EPFN, diagnosed tentatively based on clinical and radiographic findings, with documented resolution of the lesion on chest CT. According to our literature search, this is only the second case in which chest CT alone was used to both diagnose the lesion and track its resolution. It is the first documented case diagnosed and followed as such in North America.

Key words: fat necrosis, pleuritic, chest pain.

Introduction

Epipericardial fat necrosis (EPFN) is an uncommon cause of chest pain. Typically manifesting as severe acute chest pain, EPFN can be mistaken for a serious disorder, such as pulmonary embolism or myocardial infarction. We report a case of epipericardial fat necrosis, diagnosed tentatively based on clinical and radiographic findings, with documented resolution of the lesion on chest CT. According to our literature search, this is only the second case in which chest CT alone was used to both diagnose the lesion and track its resolution. It is the first documented case diagnosed and followed as such in North America.

Case report

A 70-year-old man with a history of borderline hypertension and mild obesity was admitted with a 2-day history of left-sided pleuritic chest pain, left shoulder pain, and mild dyspnea. On the night of admission he awakened with severe pleuritic pain in the left mid-axillary area. The pain was not positional and did not radiate, and was not reproducible by palpation. He had mild dyspnea which he attributed to splinting to control the severity of his pain. There was no associated diaphoresis, palpitation, leg tenderness/pain, syncope, or near syncope. He did not experience any substernal burning, nausea, emesis, anorexia, or other gastrointestinal symptoms. He had been on a long airplane flight two weeks prior to his presentation. His past medical history was notable for borderline

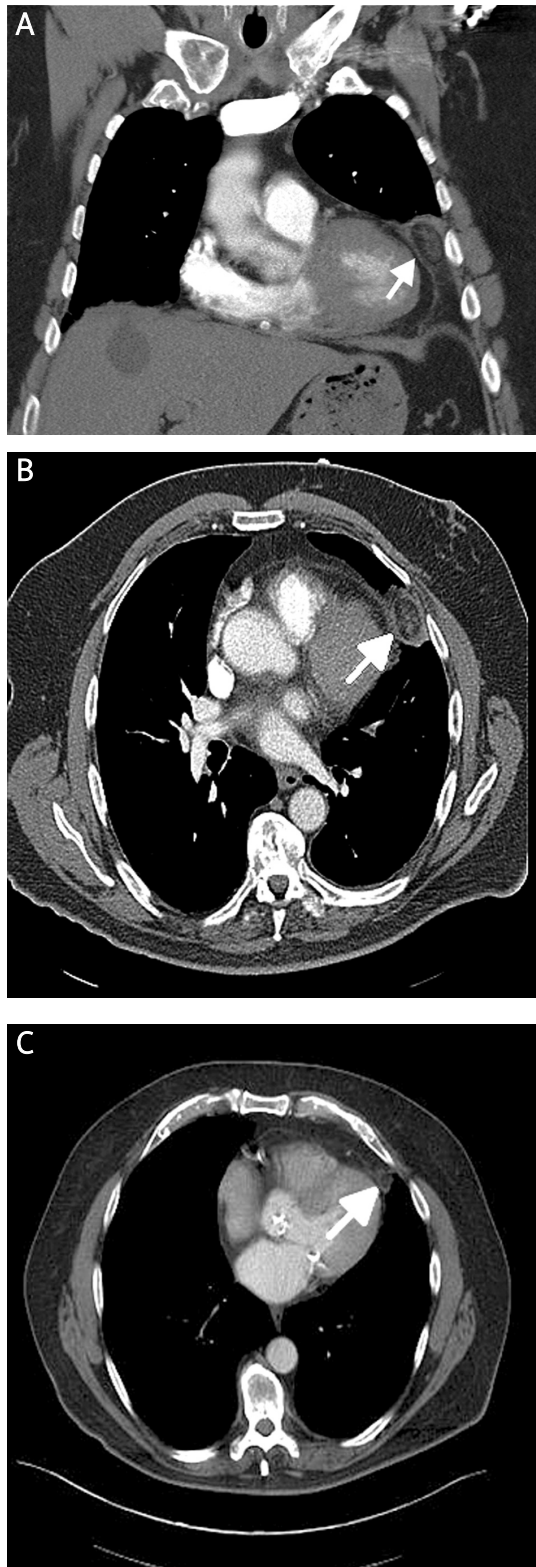


Figure 1. Chest CT: **A)** Coronal section shows low attenuation of the pericardial fat (arrow). An area of pathologic fat necrosis and inflammation is seen lateral to the pericardial fat, corresponding to the shadow seen on admission CXR. An axial section (**B**), shows the necrotic area (arrow). Six months later (**C**), the lesion measured 1.4 cm

hypertension, gout, remote peptic ulcer disease, and mild obesity. He was a nonsmoker and did not consume alcohol or use illicit drugs. He reported being active with a daily exercise program including daily treadmill exercise, with no noted changes in exercise tolerance or capacity. On evaluation, his blood pressure was 156/90 mmHg with a pulse of 81 and regular. He was afebrile, there were no palpable nodes, and cardiac rate and rhythm were normal, as were chest and lung exams. ECG showed normal sinus rhythm, Q waves in lead III, and non-specific ST and T-wave abnormalities, with no evidence of acute ischemia. Cardiac enzymes on admission were CK 87 U/L (nl: 70-185), CK-MB 3.7 ng/ml (nl: 0.0-6.0), and troponin T was undetectable (nl: < 0.029 ng/ml). Eight h later, CK and CK-MB had decreased to 83 U/L, and 3.1 ng/ml, respectively, and troponin T remained undetectable. A final troponin T level was measured and was undetectable. Given unequivocal cardiac enzyme levels and ECG findings, the clinical suspicion for myocardial ischemia was very low. Accordingly, no additional diagnostic tests for MI were ordered. Chest radiograph revealed a soft tissue density adjacent to the left heart border. Chest CT identified a 6.4 by 2.5 cm low-density soft-tissue shadow in the left lung base. A second, smaller paracardiac density (2.9 × 2 cm in diameter) was also noted. The presumptive diagnosis of epipericardial fat pad necrosis was made based on these findings (Figures 1A-B). The patient was admitted, observed overnight, and discharged the following morning, his pain having resolved.

Three months following discharge, the patient experienced recurrent left pleuritic chest pain. An ECG performed at this time was unchanged compared with the prior ECG, with no evidence of ischemia. Initial cardiac enzymes: CK 70 U/l, CK-MB 3.2 ng/ml, troponin T undetectable. At 8 h, enzymes were: CK 44 U/l, CK-MB 2.6 ng/ml, troponin T undetectable. Final cardiac enzymes: CK 34 U/l, CK-MB 2.6 ng/ml, troponin T undetectable. Imaging studies showed that the area of increased attenuation in the paracardiac fat had decreased in size, to 2.2 cm by 1.4 cm. The left-basilar density had resolved. Six months later, the paracardiac lesion had decreased in size to 1.4 cm in diameter (Figure 1C). The patient was monitored with serial CT imaging studies for 12 months after his acute episode, and his symptoms did not recur.

Discussion

Epipericardial fat necrosis is an uncommon cause of chest pain. Typically manifesting as severe acute chest pain, EPFN can be mistaken for a serious disorder, such as pulmonary embolism or myocardial infarction. Diagnosis has generally

Table I. Previous cases of epi-pericardial fat necrosis

Case	First author	Year*	Age [years]	Sex	BMI [kg/m ²]	Complaint	Size [cm]	Treatment
1	Jackson	1957	52	M	29.9	Right-sided chest pain	5 × 2.5 × 2	Exploratory thoracotomy
2	Jackson	1957	63	M	31.6	Left-sided chest pain	8 × 4.5 × 2.5	Exploratory thoracotomy
3	Jackson	1957	47	F	39.4	Left-sided chest pain	7.4 × 6 × 2.5	Exploratory thoracotomy
4	Kasserman	1958	23	M	21.9	Left-sided chest pain	7 × 4.5 × 2	Exploratory thoracotomy
5	Chester	1959	46	M	22	Left-sided chest pain	10 × 7.5 × 3.0	Exploratory thoracotomy
6	Perrin	1960	56	M	NR	Right-sided chest pain	9 × 7 × 1.5	Exploratory thoracotomy
7	Kyllonen	1961	42	F	NR	Left-sided chest pain	3 × 2.5 × 2.5	Exploratory thoracotomy
8	Chipman	1962	40	M	NR	Left-sided chest pain	7.5 × 5 × 4.5	Exploratory thoracotomy
9	Kyllonen	1964	51	M	NR	Left-sided chest pain	2.5 × 5	Exploratory thoracotomy
10	Behrendt	1968	23	F	Moderately obese	Left-, then right-sided chest pain	4.5 × 1.5 × 1	Exploratory thoracotomy
11	Takkunen	1970	50	M	NR	Left-sided chest pain	1.5	Exploratory thoracotomy
12	Wychulis	1971	52	M	Obese	Left-sided chest pain (3 cases)	5 × 2.5 × 2	Exploratory thoracotomy
13	Wychulis	1971	63	M	Obese	Right-sided chest pain (1 cases)	–	Exploratory thoracotomy
14	Wychulis	1971	64	M	Obese	Left-sided chest pain	8 × 4.5 × 1.5	Exploratory thoracotomy
15	Wychulis	1971	47	F	Obese	Left-sided chest pain	–	Exploratory thoracotomy
16	Webster	1974	58	F	28.2	Left-sided chest pain	3 × 1.5	Exploratory thoracotomy
17	Stephens	1988	37	F	19.7	Right-sided chest pain	4 × 1.5 × 1	Exploratory thoracotomy
18	Bensard	1990	67	F	Non-obese	Left-sided chest pain	3 × 3	Exploratory thoracotomy
19	Inoue	2000	55	M	25.9	Left-sided chest pain	5 × 3.5 × 2	Video-assisted thoracic surgery (VATS)
20	Takao	2004	45	M	NR	Hemoptysis	NR	Biopsy of pericardial fat
21	Pineda	2005	54	F	Non-obese	Chest pain	NR	Analgesics, documented radiologic follow-up
22	Pineda	2005	50	M	Non-obese	Left-sided chest pain	NR	Exploratory thoracotomy
23	Lee	2007	45	M	22.3	Intermittent dizziness, pitting edema	6 × 5	Exploratory thoracotomy
24	Van den Heuvel	2010	55	F	NR	Left-sided chest pain	NR	Analgesics
25	Present case	2011	70	M	34.8	Left-sided chest and shoulder pain	6,4 × 2.5	Analgesics, documented radiologic follow-up

*Year of publication

required surgical resection for diagnosis. Pathologically, associated lesions resemble necrotic tissue more commonly found in epiploic appendages of the colon or in the breast [1], with a predominance of lipid-filled macrophages, inflammatory changes, and extravasation of blood [2]. Only 24 previous cases (see Table I) have been described in the medical literature [1-16]. However, of these 24 cases, 22 required surgical excision and histologic examination for diagnosis. Only one prior case [7] involved a tentative diagnosis of EPFN, symptomatic treatment, and documented resolution of the lesion without surgery.

The pathogenesis of EPFN remains unclear. Potential causes include torsion of a pedicle, a pre-existing structural abnormality that may make tissue more vulnerable to necrosis, and heavy lifting, which could cause intravascular pressure changes significant enough to cause hemorrhage into adipose tissue. The associated pain of epipericardial fat necrosis is almost always pleuritic – sharp in quality, with intermittent peaks. In general, patients typically experience acute pleuritic chest pain. The pain has been described as left-sided in 16 patients [1-8, 11-13, 17], right-sided in 5 [1, 3, 9, 10, 18], and was without further clear description in 3 cases [7, 15, 16]. In most instances, the onset is acute, on the order of days to weeks, however in uncommon cases, the pain can persist for close to one year [1, 3]. EKGs are normal, with rare exception [2, 6, 9]. Cardiac enzymes, leukocyte counts, and other laboratory tests, when performed, have been non-contributory.

Diaphoresis and syncope can complicate the presentation. Cases have generally been unrelated to trauma or infection. Obesity has been suggested as a risk factor, but from our literature review, only 42% (10 out of 24 patients) were described as being at least moderately obese. Still, the volume of pericardial fat is typically increased in overweight individuals [19]. Narrowing the differential diagnosis can be difficult, given the generally nonspecific presentation of EPFN, and radiographic similarities between fat necrosis and other fat-containing masses, including liposarcoma and lipoma. Historically, this ambiguity made surgical management essential [1, 3, 4, 9]. With advanced CT, important EPFN findings can include an encapsulated fatty lesion, with or without adjacent pericardial thickening [7]. Fat stranding in the lesion may assist in diagnosis. Chest pain in tandem with these radiographic features is highly suggestive of EPFN [7]. Although rare, it is important to consider this benign entity, based on its usual clinical presentation, combined with negative markers for MI and other cardiopulmonary pathology. A paracardiac density or opacity with or without a cardiophrenic or adjacent pleural effusion on

routine chest film can suggest the diagnosis and prompt further evaluation with chest CT.

In conclusion, first described by Jackson, Clagett, and McDonald in 1957 [3] the definitive diagnosis of epipericardial fat necrosis has generally required surgical resection and pathologic examination. Of 24 previous cases, the case we presented represents only the second case of EPFN diagnosed and followed through resolution, using chest CT imaging studies alone. Given this prior report combined with the clinical course of our patient, we have presented additional evidence that strengthens the case for conservative management of this condition, and suggests that although EPFN is rare, it may be under-diagnosed. Both radiologists and practicing clinicians should be aware of the clinical presentation of this uncommon but benign condition and include it in their differential diagnosis of chest pain.

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