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Case Report

Epipericardial fat necrosis diagnosed by cardiac CT in a patient with apical hypertrophic cardiomyopathy ☆

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ABSTRACT

Epipericardial fat necrosis (EFN) is a rare benign cause of chest pain, that is frequently overlooked. EFN involves the necrosis of fat tissue in the mediastinum, and presents on computed tomography (CT) as an ovoid lesion of fat attenuation surrounded by a rim of soft tissue attenuation. This case report describes a case of a 50-year-old man diagnosed with EFN on cardiac CT, which was incidentally associated with apical hypertrophic myocardiopathy. Notably, the detection of EFN proved difficult on arterial phase images during coronary CT angiography, whereas it was much easier to detect on delayed phase images. EFN should be considered in the differential diagnosis of chest pain, and careful examination of mediastinal fat is crucial for accurate diagnosis.

Introduction

Epipericardial fat necrosis (EFN) is known as a relatively rare benign condition causing chest pain [1–7]. EFN was first reported in 1957 by Jackson et al. [8] and is thought to be a very rare condition, but reports of EFN have increased recently. Giassi et al. [5] reported that EFN was found in 2.15% of the patients undergoing CT for chest pain in the emergency department. In this case report, we present a case of EFN diagnosed by cardiac CT in a patient with apical hypertrophic cardiomyopathy (HCM).

Case report

A 50-year-old man presented to the emergency department with left anterior chest pain. The pain had been present for approximately 8 hours and worsened with changes in

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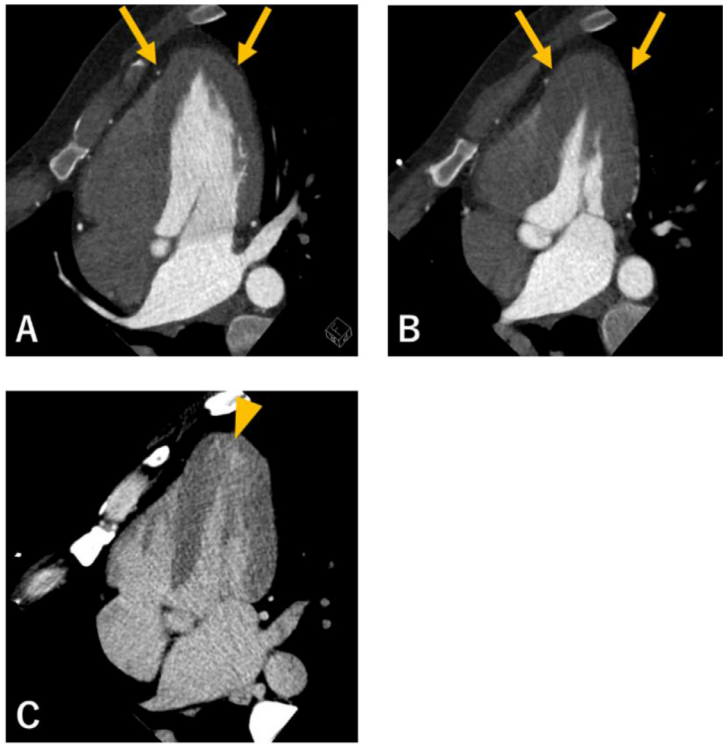

Fig. 1 – Cardiac CT 4-chamber images (A: diastole, B: systole, C: systole, delayed phase).
There is a focal hypertrophy of the left ventricular apex (arrows). Delayed enhancement was seen in the hypertrophied apex (arrowheads).

posture and respiration. Although the electrocardiogram (ECG) showed inverted T waves in the anterolateral leads, acute coronary syndrome (ACS) was ruled out based on blood tests and echocardiogram. The patient was advised to rest at home. Three days later, the patient presented to our hospital because of persistent chest pain. Physical examination was normal, but laboratory tests revealed elevated white blood cell count 9280/μL (reference value 8600<μL), C-reactive protein 0.26 mg/dL (reference value 0.14<mg/dL), and brain natriuretic peptide (BNP) 25.5 pg/mL (reference value 18.4<pg/mL). Troponin I was within the normal limits. The ECG showed a heart rate of 54 bpm in sinus rhythm and a normal axis, with negative T-waves in leads I, II, III, aVf, aVL, and V1-6. The echocardiogram showed a normal left ventricular ejection fraction and no regional wall motion abnormalities. However, myocardial hypertrophy was observed at the left ventricular apex. Although his symptoms had improved by the 5th day from onset without any treatment, given his age, lifestyle history (smoking 20 cigarettes per day for 30 years), and the presence of abnormal ECG findings, it was deemed necessary to exclude coronary artery disease, and cardiac CT, including noncontrast CT for calcium scoring, coronary CT angiography, and delayed enhancement CT, was performed on the 11th day from onset. Cardiac CT demonstrated normal coronary arteries and hypertrophy of the left ventricular apex associated with delayed contrast enhancement consistent with apical HCM (Fig. 1). Additionally, CT revealed an ovoid fat attenuation lesion measuring 25 × 12 mm with a soft tissue attenuation rim in the pericardial fat tissue near the left ventricular apex. Mild thickening and contrast enhancement of the adjacent pericardium was also noted (Fig. 2). A diagnosis of EFN was made. There was no recurrence of symptoms and no follow-up was performed.

Discussion

EFN is the necrosis of adipose tissue in the mediastinum, particularly near the pericardium [1]. There are no known risk factors [7], although a slight male predominance has been reported [9]. Typical symptoms include intermittent chest pain that increases with postural changes and deep inspiration, which is usually self-limiting and lasts for several days to weeks [1,3,5]. ECG findings are often normal [5,10]. Conservative treatment, such as nonsteroidal anti-inflammatory drugs is usually sufficient to relieve symptoms [3,7,9].

CT is the main imaging modality used in the diagnosis of EFN [1,3,9]. CT allows not only the localization and characterization of the lesion but also differentiation from other diseases, such as lipoma, liposarcoma, and thymolipoma [3]. On CT, EFN typically appears as an ovoid fat attenuation lesion with a soft tissue attenuation rim in the anterior mediastinal pericardial fat, as seen in the present case [1,3]. Due to inflammation forming the rim [7], thickening of the adjacent pericardium and pleural effusion may also be seen [1,3,7], but

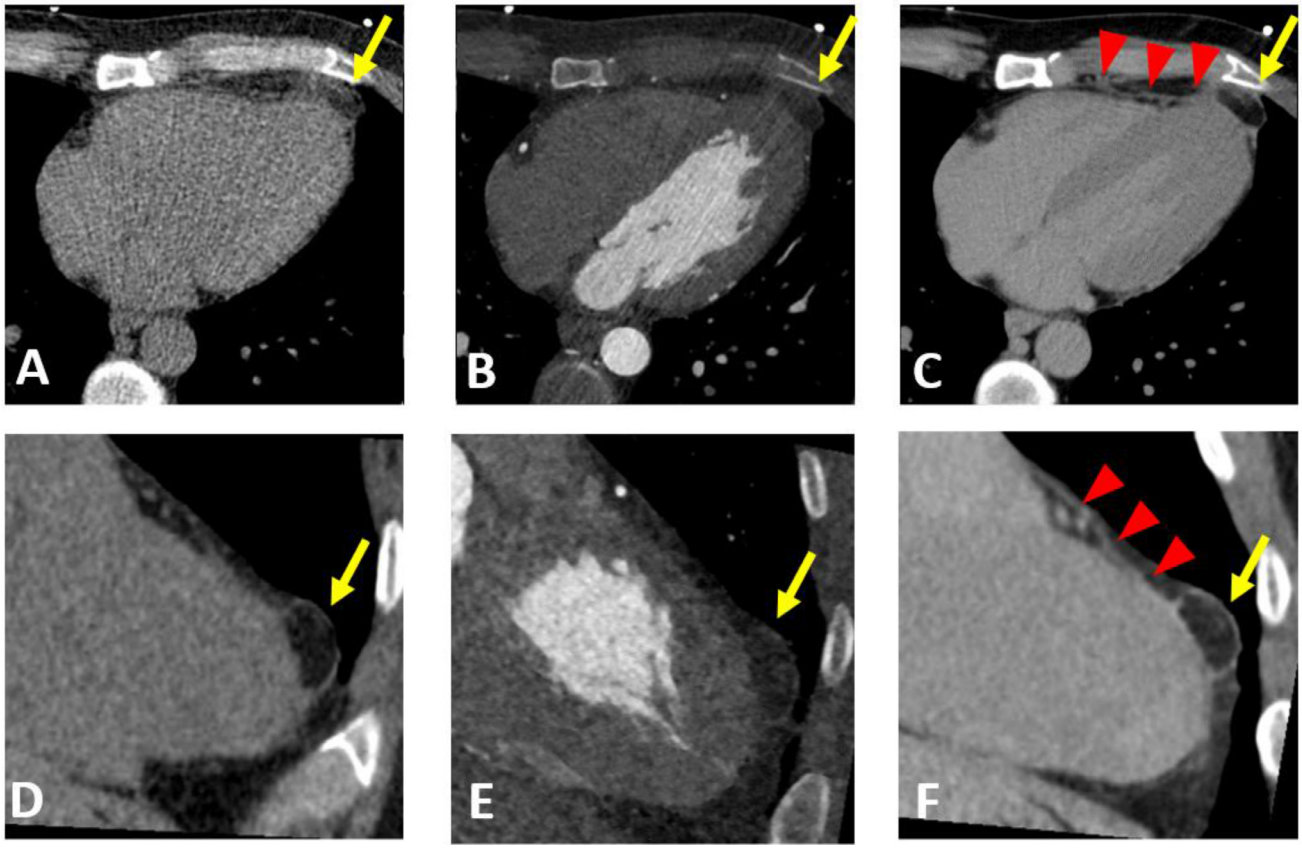

Fig. 2 – Cardiac CT axial images (A: noncontrast CT, B: arterial phase, C: delayed phase), Cardiac CT coronal images (D: noncontrast CT, E: arterial phase, F: delayed phase).
An anterior mediastinal ovoid fat attenuation lesion with a soft tissue attenuation rim (arrows) was seen, which was associated with a thickened pericardium on delayed phase images (arrowheads).

without invasion of the deeper cardiac structures or the chest walls [4]. EFN is more likely to occur on the left side of the mediastinum near the left ventricular apex, but it can also occur on the right side, and atypical locations may include adjacent interlobar fissures [3,5].

In the case presented, ACS has ruled out based on symptoms and blood test data. Cardiac CT showed no stenosis of the coronary arteries but characteristic imaging findings of EFN. It is important to note that these findings were most evident in delayed-phase images. EFN was more difficult to detect on coronary CT angiography images, which are optimized for high-contrast subjects (ie, coronary arteries). Without attention to mediastinal fat, EFN may have been missed, potentially leading to unnecessary investigation of other causes of chest pain, such as ischemia with non-obstructive coronary arteries. Specifically, in this case, chest pain may have been misattributed to apical HCM. It is known that approximately 14% of patients with HCM are known to complain of chest pain symptoms [11], which are thought to be caused by microvascular dysfunction, reduced capillary density, and myocardial bridging [12,13].

In conclusion, this case highlights the difficulty of diagnosing EFN on coronary CT angiography and emphasizes the importance of careful observation of mediastinal fat.

Patient consent

Written informed consent was obtained from the patient for this case report.

REFERENCES

[1] Pineda V, Cáceres J, Andreu J, Vilar J, Domingo ML. Epipericardial fat necrosis: radiologic diagnosis and follow-up. AJR Am J Roentgenol 2005;185(5):1234–6. doi:10.2214/AJR.04.1310.
[2] Pineda V, Andreu J, Cáceres J, Merino X, Varona D, Domínguez-Oronoz R. Lesions of the cardiophrenic space: findings at cross-sectional imaging. Radiographics 2007;27(1):19–32. doi:10.1148/rg.271065089.
[3] Bhatt MY, Martínez-Jiménez S, Rosado-de-Christenson ML, Watson KR, Walker CM, Kunin JR. Imaging manifestations of mediastinal fat necrosis. Case Rep Radiol 2013;2013:323579. doi:10.1155/2013/323579.
[4] Gayer G. Mediastinal (Epipericardial) fat necrosis: an overlooked and little known cause of acute chest pain mimicking acute coronary syndrome. Semin Ultrasound CT MR 2017;38(6):629–33. doi:10.1053/j.sult.2017.08.003.

[5] Giassi KS, Costa AN, Bachion GH, Kairalla RA, Filho JRP. Epipericardial fat necrosis: who should be a candidate? AJR Am J Roentgenol 2016;207(4):773–7. doi:10.2214/AJR.15.15934.
[6] Lee HH, Ryu DS, Jung SS, Jung SM, Choi SJ, Shin DH. MRI findings of pericardial fat necrosis: case report. Korean J Radiol 2011;12(3):390–4. doi:10.3348/kjr.2011.12.3.390.
[7] Kenyon SK, Wijesekera VA, Wesley AJ. Case report on the radiographic features of epipericardial fat necrosis in a patient presenting with acute chest pain. J Med Imaging Radiat Oncol 2023;67(5):509–13. doi:10.1111/1754-9485.13562.
[8] Jackson RC, Clagett OT, Mcdonald JR. Pericardial fat necrosis; report of three cases. J Thorac Surg 1957;33(6):723–9.
[9] Giassi Kde S, Costa AN, Bachion GH, Apanavicius A, Filho JR, Kairalla RA, et al. Epipericardial fat necrosis: an underdiagnosed condition. Br J Radiol 2014;87(1038):20140118. doi:10.1259/bjr.20140118.
[10] Berchuck JE, Patel S. An often-overlooked etiology of pleuritic chest pain. J Gen Intern Med 2016;31(1):138. doi:10.1007/s11606-015-3316-4.
[11] Doctorian T, Mosley WJ, Do B. Apical hypertrophic cardiomyopathy: case report and literature review. Am J Case Rep 2017;18:525–8. doi:10.12659/ajcr.902774.
[12] Stephenson E, Monney P, Pugliese F, Malcolmson J, Petersen SE, Knight C, et al. Ineffective and prolonged apical contraction is associated with chest pain and ischaemia in apical hypertrophic cardiomyopathy. Int J Cardiol 2018;251:65–70. doi:10.1016/j.ijcard.2017.09.206.
[13] Mohiddin SA, Begley D, Shih J, Fananapazir L. Myocardial bridging does not predict sudden death in children with hypertrophic cardiomyopathy but is associated with more severe cardiac disease. J Am Coll Cardiol 2000;36(7):2270–8. doi:10.1016/s0735-1097(00)00987-6.