



Tuberculous Pericarditis Mimicking a Malignant Pericardial Tumor: A Case Report

악성 심막 종양으로 오인한 결핵성 심막염: 증례 보고

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Tuberculous pericarditis is an extrapulmonary manifestation of tuberculosis that is most commonly associated with pericardial thickening, effusion, and calcification. We present a case of tuberculous pericarditis mimicking a malignant pericardial tumor in a 77-year-old male. CT revealed an irregular and nodular pericardial thickening. MRI revealed high signal intensity on T1-weighted fat-suppressed images and peripheral rim enhancement after gadolinium administration. MRI can be helpful in determining the differential diagnoses in cases of tuberculous pericarditis with nonspecific imaging findings.

Index terms Tuberculosis; Tuberculous Pericarditis; Pericardial Abscess; Magnetic Resonance Imaging

INTRODUCTION

Tuberculous pericarditis (TBP) is a pericardial infection caused by tuberculosis bacilli and a significant extrapulmonary manifestation of tuberculosis. TBP is present in both the immunocompromised and immunocompetent individuals. TBP typically manifests as pericardial effusion, pericardial thickening that causes constrictive pericarditis, or both. Herein, we

report a rare case of TBP mimicking a malignant pericardial tumor in a 77-year-old male with CT and MRI findings.

CASE REPORT

A 77-year-old male was referred to our hospital after experiencing dyspnea and bilateral leg edema for more than 6 months. The patient was taking medications for hypertension. Upon admission, his laboratory test results, including cardiac enzyme tests, were found to be normal. Initial chest radiography revealed mild cardiomegaly and bilateral pleural effusions (Fig. 1A).

Contrast-enhanced chest CT revealed infiltrative nodular and mass-like lesions along the pericardium, with heterogeneous enhancement, pericardial thickening, and moderate bilateral pleural effusion (Fig. 1B). Several prominent or enlarged lymph nodes were observed in the mediastinal and cardiophrenic spaces.

MRI of the heart revealed multiple nodular lesions along the pericardium that were isointense to hyperintense on T1- and T2-weighted images (Fig. 1C). The lesions showed high signal intensity on T1-weighted fat-suppressed images, peripheral rim enhancement after gadolinium administration, high signal intensity on a higher b-value diffusion-weighted image ($b = 800 \text{ s/mm}^2$), and low signal intensity on the apparent diffusion coefficient map, indicating diffusion restriction (Fig. 1C).

Therefore, a malignant pericardial tumor, such as malignant mesothelioma, lymphoma, or metastasis of unknown origin, was diagnosed preoperatively. PET/CT scans were performed to rule out the presence of other malignancies and nodal diseases; no evidence of a hypermetabolic focus of the primary malignancy except for the pericardium was found (Fig. 1D).

The patient underwent an excisional biopsy of the pericardium. The lesion was confirmed to be nodular, and resection was performed accordingly. A substantial amount of caseous material was observed in the specimen. An intraoperative frozen section revealed granulomatous inflammation with necrosis, suggestive of tuberculosis. Histopathological examination of the sections stained with hematoxylin and eosin revealed the infiltration of epithelioid cells and lymphocytes around the confluent necrosis (Fig. 1E). The patient had a positive polymerase chain reaction (PCR) test for *Mycobacterium tuberculosis* (*M. tuberculosis*) (TB-PCR), and the final diagnosis was TBP.

The patient was prescribed an antituberculous medication. At the latest follow-up examination, the patient was well, although he still presented with mild dyspnea. A follow-up chest CT scan performed 4 months later revealed an overall decrease in the diffuse infiltrative nodular lesions and mediastinal lymphadenopathy (Fig. 1F).

Written informed consent for this case report was obtained from the patient's parents. This study was performed according to the latest ethical principles in the Declaration of Helsinki.

DISCUSSION

Tuberculosis is an airborne communicable disease caused by *M. tuberculosis*. It is primarily a pulmonary disease, although *M. tuberculosis* can affect any organ of the body. Heart in-

Fig. 1. Imaging and pathologic findings of tuberculous pericarditis mimicking a malignant pericardial tumor in a 77-year-old patients.

A. The chest radiography shows the presence of mild cardiomegaly and bilateral pleural effusion.

B. CE axial and coronal chest CT images with mediastinum window setting show an infiltrative nodular and mass-like lesion along the pericardium with peripheral enhancement. Pericardial thickening and moderate bilateral pleural effusion are also noted. Several prominent or enlarged lymph nodes were observed in the mediastinal and cardiophrenic spaces (not shown).

C. Axial MR images of the heart show multiple nodular lesions along the pericardium, isointense to hyperintense on T1- and T2-weighted images. On the T1-weighted FS image, they show high signal intensity and peripheral rim enhancement after gadolinium administration. The lesions show diffusion restriction on a higher b-value DWI ($b = 800 \text{ s/mm}^2$) and the ADC map.

ADC = apparent diffusion coefficient, CE = contrast-enhanced, DWI = diffusion-weighted image, FS = fat-suppressed

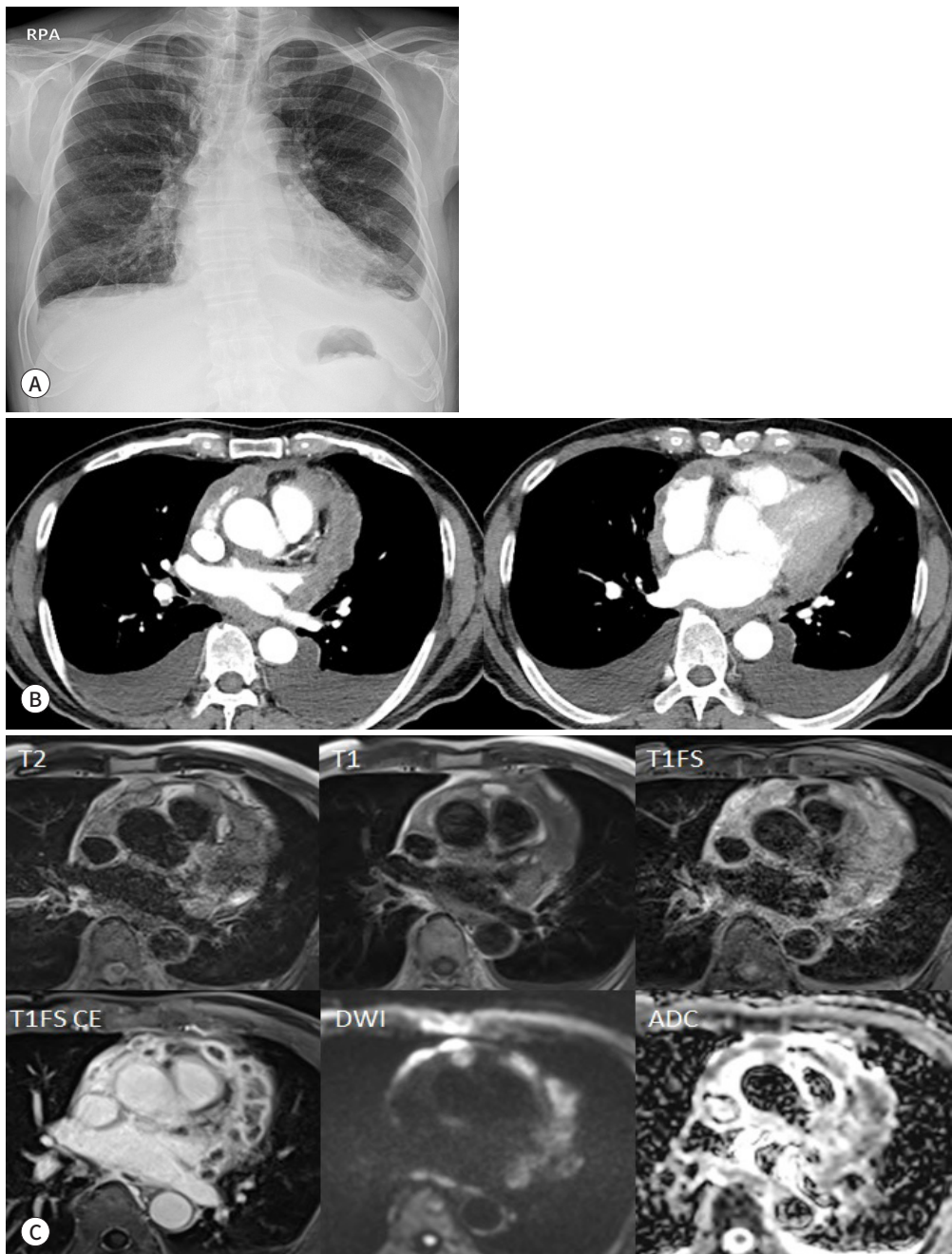


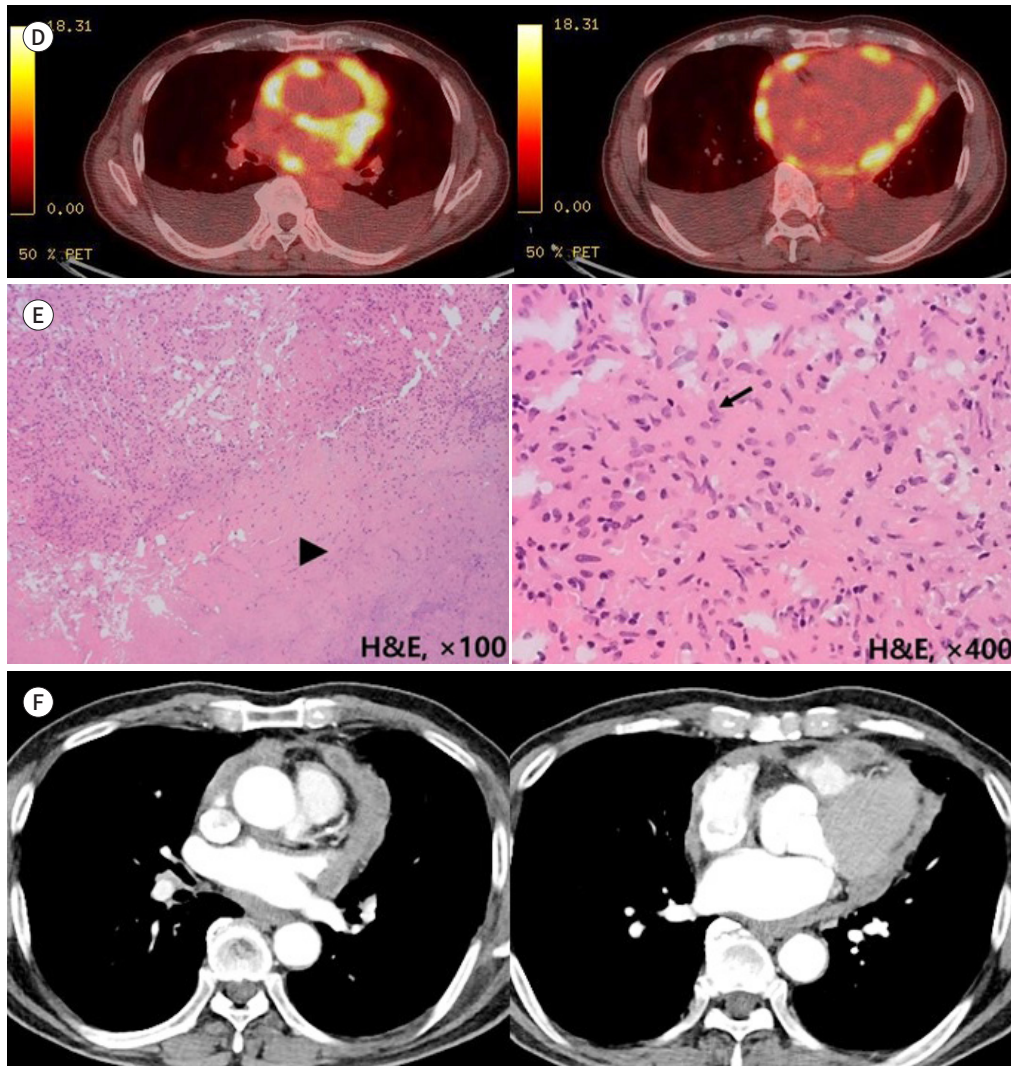
Fig. 1. Imaging and pathologic findings of tuberculous pericarditis mimicking a malignant pericardial tumor in a 77-year-old patients.

D. PET/CT transaxial fusion images show avid FDG uptake in the pericardium.

E. Histopathological examination shows inflammatory infiltration around necrosis (arrowhead) (left). There are diffuse infiltration of epithelioid cells (arrow) and lymphocytes in fibrous tissue (right).

F. Follow-up chest CT axial images after 4 months show an overall decrease in the extent of diffuse infiltrative nodular lesions. The overall size of several prominent or enlarged lymph nodes was also decreased (not shown).

H&E = hematoxylin and eosin



involvement in tuberculosis can manifest as TBP, myocarditis, and aortitis (1). The manifestations of TBP can be divided into pericardial effusion, constrictive pericarditis, or both (2). In developing countries, tuberculosis is the most common cause of constrictive pericarditis, accounting for 40%–70% cases of large pericardial effusions (3).

TBP frequently presents with insidious and systemic signs, such as fever, weight loss, and night sweats, usually preceding cardiopulmonary symptoms including cough, dyspnea, and chest pain.

Since the lungs are the principal entrance for the bacillus, pericardial involvement can oc-

cur by direct spread from the lungs and pleura, and lymphatic spread can occur from the mediastinal, paratracheal, and peribronchial lymph nodes. Hematogenous spread is also possible (1). There are four pathological stages of TBP. The first stage is a dry stage with an early immune response and exudation of fibrinous material. The second stage is an effusive stage with presence of serosanguineous fluid. The third stage is an absorptive stage with pericardial thickening and granulomatous caseation, and the fourth stage is a constrictive stage caused by scarring (2).

The typical imaging finding of TBP is pericardial thickening of > 3 mm. Pericardial effusions or localized calcifications have been reported in $< 20\%$ of the patients (4). Distension of the inferior vena cava to a diameter > 3 cm, pleural effusions (typically bilateral), and deformities of the intraventricular septum have also been reported in literature.

A few reports of MRI findings in TBP include pericardial thickening with isointensity to the myocardium on T1-weighted images, whereas hypointense lesions may be observed on the inner surface of the thickened pericardium, reflecting ferromagnetic elements after hemorrhage and pericardial fibrosis. Linear low signals in the pericardial sac on T2-weighted images represent strands of granulation tissue in past reports. Uniform tramline-like enhancement at the sites of fibrous hypertrophic parietal and visceral pericardia has also been reported (5). A hypointense core on the T2-weighted images is a common finding that can occur in the caseous part of intracranial tuberculoma due to fibrosis, gliosis, and macrophage product; it has been reported in a past report, although rarely with extracranial manifestation (6).

Over time, TBP may lead to irregular adhesions between the visceral and parietal layers of the pericardium, resulting in loculation of the pericardial fluid. If the fluid contains pus and debris, it is referred to as an abscess. The right atrioventricular groove is a common location for TBP abscesses, whereas lesions on the left side are uncommon (7). The characteristic imaging finding is a localized biconvex fluid-filled pocket within the pericardial space, compressing the adjacent cardiac chambers (8). A CT scan shows the presence of thin-rim enhancement with central low attenuation. The presence of high signal intensity on T1-weighted images strongly suggests high protein content, indicating an exudate (7). Based on these imaging findings, our case might also have been a type of TBP abscess. Furthermore, MRI is a more sensitive diagnostic imaging technique for detecting tuberculous pericardial abscesses, which may be misdiagnosed as malignant if the typical CT findings are not evident.

In this case, TBP did not manifest as pericardial effusion but instead as an infiltrative nodular-enhancing mass-like lesion along the pericardium on the CT scan, leading to an initial suspicion of malignancy. Diffusion restriction was achieved on MRI; however, no contrast enhancement inside the mass and only peripheral rim enhancement were observed, suggesting the possibility of an abscess rather than a malignancy.

One case report had an asymptomatic 26-year-old female presenting diffuse irregular pleural thickening in the left hemithorax with mediastinal and hilar lymph node enlargement, suggestive of pleural lymphoma or malignant pleural mesothelioma (9). In this case, a trans-thoracic needle biopsy of the pleural lesion revealed an epithelioid cell granuloma, and the specimen was positive for tuberculosis on both acid-fast staining and PCR. To date, few case reports have presented a tumor-like mass and a large amount of effusion in the pericardial cavity, finally diagnosed as TBP (10).

In conclusion, we presented an unusual case of TBP manifesting as infiltrative nodular pericardial thickening mimicking a pericardial mass. In cases of TBP presenting as irregular pericardial thickening or focal mass-like lesions, the differential diagnosis of primary pericardial malignancy on CT becomes difficult.

Therefore, MRI can help radiologists evaluate TBP and differentiate it from primary malignant pericardial tumors, such as mesothelioma or lymphoma, when it presents with rim enhancement and diffusion restriction on MRI, especially in tuberculosis-endemic countries.

Author Contributions

Conceptualization, K.D.S.; data curation, P.J.Y., H.J., K.M.S., K.D.S.; formal analysis, P.J.Y., K.D.S.; methodology, K.D.S.; project administration, K.D.S.; supervision, H.J., P.J., S.G.W., K.M.S., K.D.S.; visualization, all authors; writing—original draft, P.J.Y.; and writing—review & editing, K.M.S., K.D.S.

Conflicts of Interest

The authors have no potential conflicts of interest to disclose.

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악성 심막 종양으로 오인한 결핵성 심막염: 증례 보고

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결핵성 심막염은 폐외 결핵으로 나타나는 질환으로 대부분 심막 비후, 심막 삼출과 석회화를 동반한다. 우리는 77세 남성에서 발견된 악성 심막 종양으로 오인된 결핵성 심막염에 대해 보고하려 한다. CT상 불규칙적이고 결절성 심막 비후로 관찰된 병변은 MRI T1 지방 억제 영상에서 고신호강도 및 테두리 조영증강을 보였다. 비특이적 영상 소견을 보이는 결핵성 심막염의 경우 MRI가 감별진단에 도움이 될 수 있다.

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