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CARDIAC INVOLVEMENT AS FIRST MANIFESTATION OF PERIPHERAL T-CELL NOT OTHERWISE SPECIFIED LYMPHOMA

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ABSTRACT

Cardiac involvement as the first manifestation of peripheral T-cell lymphoma not otherwise specified (PTCL-NOS) is extremely rare. We report on a 72-year-old man with hypertension, dyslipidaemia and prediabetes who presented with acute pleuropericarditis, pericardial effusion and conduction abnormalities. Despite initial treatment, his condition worsened. Imaging revealed pericardial/myocardial infiltration and a right atrial mass. Biopsy confirmed PTCL-NOS (CD3+, CD5+, CD7+, high Ki-67). Complications included ileus, pulmonary embolism and neutropaenia. CHOP failed; GemOx was ineffective; he died from hypernatremia. This reported case emphasises the aggressive course, diagnostic challenges and poor treatment response of cardiac PTCL-NOS.

KEYWORDS

Cardiac lymphoma, atrioventricular block, chemotherapy

LEARNING POINTS

- This case represented a significant diagnostic challenge; the clinical problem was documented by multiple imaging modalities, none of which were sufficient to establish a definitive diagnosis.
- This case is a clear example of how the most challenging cases require a multidisciplinary team, involving professionals from very different specialties (for example cardiologists, cardiac surgeons, general surgeons, haematologists, radiologists, pathologists), to achieve the correct diagnosis and provide the best possible care for the patient.

INTRODUCTION

A 72-year-old man with hypertension, dyslipidaemia, prediabetes and a smoking habit presented with two-week history of progressive abdominal distension, bilateral lower limb oedema and chest discomfort. On admission, he had an acceptable general condition with a mucocutaneous pallor and mild tachypnoea. Blood pressure was 153/69 mmHg; heart rate was 54 bpm and O_2 saturation was 95%. A

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pulmonary examination revealed decreased breath sounds over the right lung base; he also had pitting oedema up to the mid-tibia in both legs. Laboratory evaluation showed mild renal dysfunction (eGFR 44 ml/min), leukocytosis (10,210/µl) with neutrophilia and monocytosis, elevated C-reactive protein (47.5 mg/dl), and elevated NT-proBNP (2,536 pg/ml) and D-dimer (3.16 mg/l). A chest X-ray confirmed right pleural effusion and mild cardiomegaly, and computed tomography (CT) pulmonary angiogram ruled out pulmonary embolism but showed pericardial effusion. Transthoracic echocardiography revealed a circumferential, moderate-to-severe pericardial effusion with right atrial diastolic collapse and ventricular interdependence but preserved biventricular function. No clinical signs of cardiac tamponade were noted. An ECG demonstrated sinus rhythm at 100 bpm with a 2:1 atrioventricular block *(Fig. 1A)*. The patient was admitted to the coronary care unit and started on colchicine, corticosteroids and fluid therapy.

CASE DESCRIPTION

Despite initial clinical improvement the patient deteriorated within 48 hours, with worsening shortness of breath, desaturation to 88% and increased respiratory rate. Blood pressure was maintained at 150/80 mmHg. Repeat imaging revealed signs of heart failure and worsening pericardial effusion with fibrinous content *(Fig. 1B)*. A pericardial window drained 600 ml of haemorrhagic fluid, with mononuclear-predominant exudate. Microbiological culture grew *Peptoniphilus spp.*, prompting intravenous penicillin G therapy.

Transoesophageal echocardiography revealed a non-pedunculated, hyperechoic mass (26 × 16 mm) along the interatrial septum extending to the superior vena cava *(Fig. 1C and 1D)*. Positron emission tomography computed tomography (PET-CT) showed pathological hypermetabolism in the pericardium and adjacent lymph nodes, with focal uptake in the appendix and right parietocolic recess *(Fig. 2A)*. Cardiac magnetic resonance imaging (MRI) identified altered epicardial fat signals extending along coronary grooves, a pseudonodular lesion in the interatrial septum and circumferential pericardial enhancement, supporting pericarditis and raising suspicion for malignancy *(Fig. 2B)*.

Median sternotomy revealed friable, haemorrhagic pericardial inflammation and white infiltrative tissue over the right atrium and pericardial recesses *(Fig. 3, left)*. A biopsy revealed diffuse lymphoid infiltration of cardiac muscle and fat, composed of intermediate-to-large size cells with vesicular nuclei, prominent nucleoli and frequent mitoses *(Fig. 3A and 3B)*. Immunohistochemistry showed positivity for CD3, CD5 and CD7; the Ki-67 index was 95% *(Fig.3C and 3D)*. CD4, CD8 and CD30 were negative. Bone marrow biopsy showed erythroid hypoplasia without infiltration. These findings confirmed the diagnosis of peripheral T-cell lymphoma, not otherwise specified (PTCL-NOS), stage IV-A. Postoperatively, the patient developed secondary distributive shock requiring permanent pacemaker implantation, paralytic ileus with

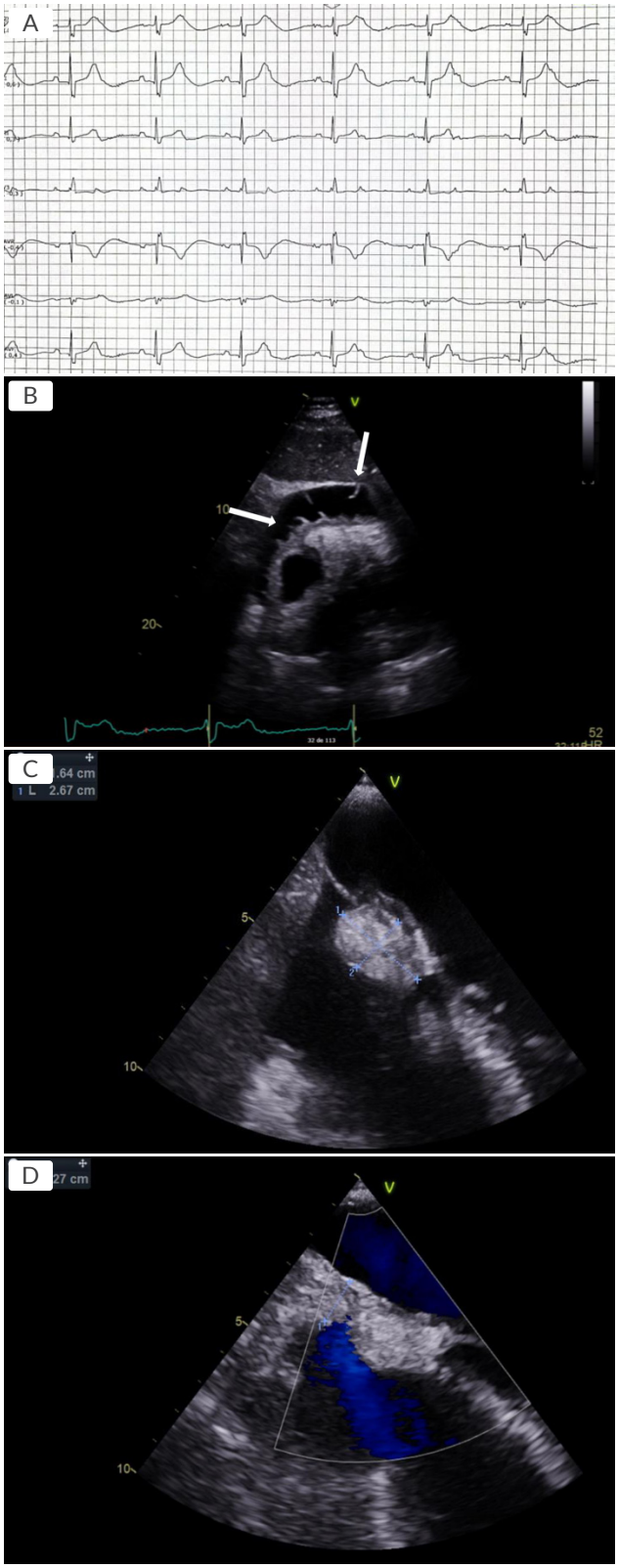


Figure 1. *A) ECG showing sinus rhythm with 2:1 AV block; B) Transthoracic echocardiogram, subcostal view: severe pericardial effusion with fibrinous content, filamentous appearance (white arrows), attached to both parietal and visceral pericardium; C, D) transoesophageal echocardiogram, 0 degrees: hyperechoic, heterogeneous, non-pedunculated mass infiltrating the interatrial septum.*

multifocal right hemicolon perforation (necessitating right hemicolectomy and ileostomy). It was considered secondary to the paralytic ileus vs mesenteric ischaemia related to low cardiac output because of the distributive shock, as no evidence of lymphoma infiltration was found in that area on the previous PET scan. He also had a segmental left pulmonary embolism that was managed with low molecular weight heparin and febrile neutropaenia. Antibiotic therapy with meropenem was initiated. He completed one month of IV penicillin and received systemic chemotherapy with six CHOP cycles (cyclophosphamide, doxorubicin, vincristine and prednisone) from October 2023 to March 2024. Due to lack of response and tumour progression, second-line therapy with gemcitabine and oxaliplatin (GemOx) was initiated. In May 2024, the patient was readmitted with marked asthenia and a decreased level of consciousness, along with abnormal laboratory results including leucocytosis, hyperuricaemia, hypernatremia, impaired renal function and abnormal liver function tests. Intracranial pathology and diabetes insipidus were ruled out. The clinical and laboratory abnormalities, in the context of a poor tumour response to first-line chemotherapy, were linked to the progression of the lymphoproliferative disorder. The patient remained in very poor general condition with drowsiness and discomfort despite correction of the hypernatremia and symptomatic treatment, experiencing episodes of restlessness and agitation that necessitated supportive care. He died three days after admission.

DISCUSSION

Cardiac masses comprise a broad spectrum of entities, including thrombi (the most common), vegetations, calcifications and tumours. Their clinical presentation is highly variable and depends primarily on size, location, friability and embolic potential, growth rate and invasiveness rather than on histologic subtype. They may cause pericarditis or cardiac tamponade when involving the pericardium, arrhythmias or conduction disturbances when infiltrating the myocardium, or heart failure when producing left-sided obstruction. Proper differential diagnosis requires integrating the mass with the patient's clinical context (such as age, atrial fibrillation, valvular prosthesis, fever). Echocardiography is fundamental as an initial diagnostic tool, and CT and MRI provide further characterisation; however, for many tumours – primary or metastatic – definitive diagnosis relies on histopathology.

Cardiac tumours are uncommon in clinical practice, with metastatic involvement far more frequent than primary tumours. The estimated prevalence of primary cardiac tumours is 1 in 2,000 autopsies, compared with 1 in 100 for secondary tumours, yielding a secondary-to-primary ratio of approximately 20:1. Cardiac metastases occur in 2.3 to 18.3% of patients with extracardiac malignancies, most commonly from lung and breast cancers, melanoma and lymphomas (typically B-cell lymphomas)[1]. PTCL-NOS is a rare, aggressive subtype of non-Hodgkin lymphoma (NHL) that represents about 30% of PTCLs and 4% of all NHL cases in Western countries[2,3]. It predominantly affects

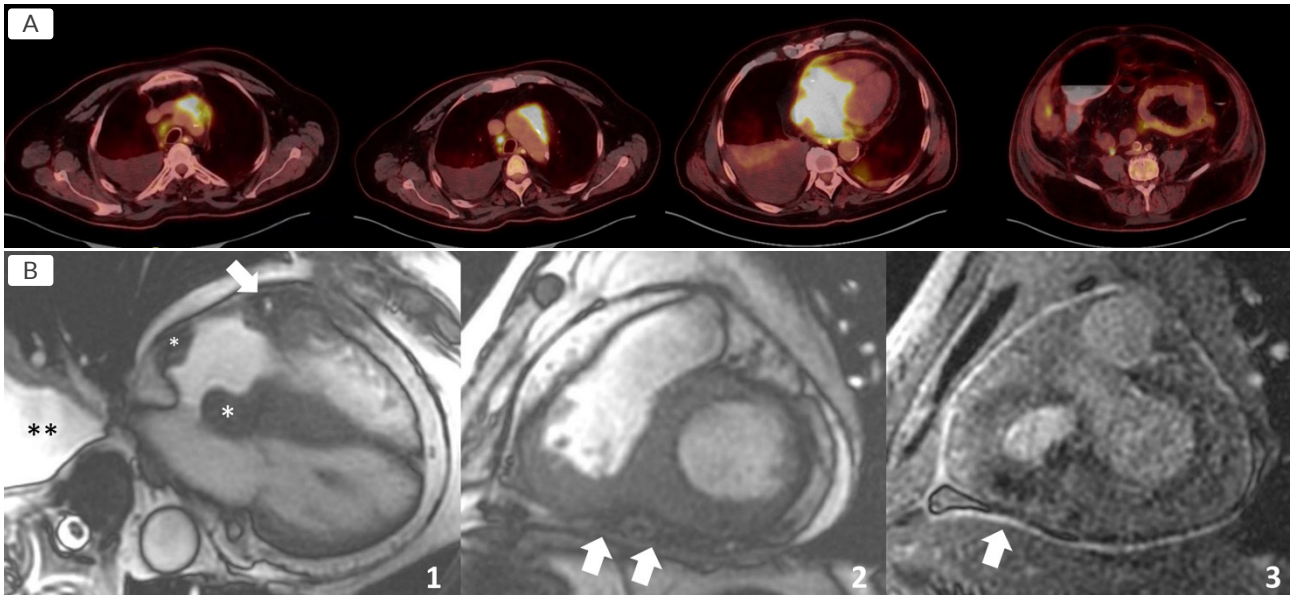


Figure 2. A) PET-CT images showing lymphadenopathy surrounding the supra-aortic trunks as well as the right paratracheal nodes. There is also intense radiotracer uptake in the pericardium, predominantly in the pericardial recesses. Additionally, mildly hypermetabolic retroperitoneal lymphadenopathy and subtle uptake along the paracolic gutters are observed. B) Cardiac MRI with cine images in four-chamber view (A) and short-axis view (B), and post-contrast short-axis view (C). Infiltrative lesion occupying the epicardial space within the right atrioventricular groove, encasing the right coronary artery without causing occlusion (arrow in A; compare with normal epicardial fat in the left atrioventricular groove). The lesion extends along the inferior wall, following the atrioventricular groove (arrows in B). Associated thickening of the interatrial septum and a small nodule in the posterior wall of the right atrium are noted (white asterisks in A). The lesion is isointense to the myocardium; in the post-gadolinium images (C), there is minimal enhancement of the lesion. Additionally, diffuse linear pericardial enhancement is observed (arrow), suggestive of pericarditis. Associated right pleural effusion is also seen (double asterisks in A).

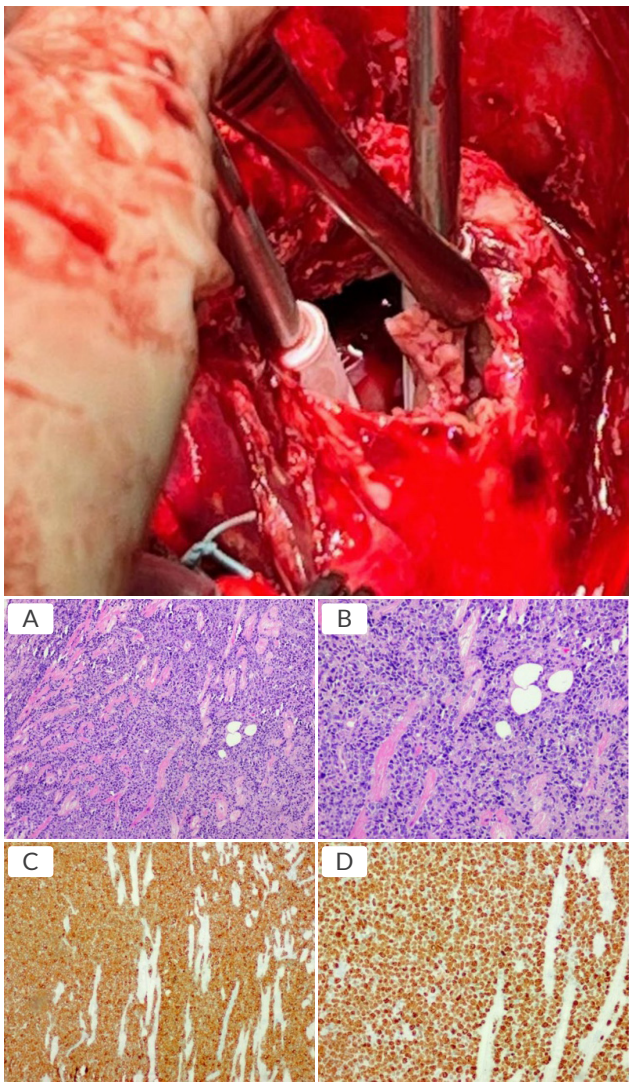


Figure 3. Left) Surgical field showing haemorrhagic inflammation of the pericardium as well as thickening of the right atrial wall; A) T-cell lymphoma infiltrating myocardial muscle cells (H&E, 10×); B) Neoplastic cellularity is composed of intermediate-to-large size cells with vesicular and irregular nuclei (H&E, 20×); C) Immunohistochemistry evaluation shows intense and diffuse expression of CD3, compatible with T phenotype (CD3, 10×). D) The cell proliferation index measured with Ki67 is 95% (Ki67, 20×).

males, with a median age of onset around 60 years. Most patients with PTCL-NOS present both nodal and extranodal involvement with the skin, gastrointestinal tract and bone marrow being the most frequently affected areas. Lung or central nervous system involvement is less common, and cardiac involvement is extremely rare.

Advanced disease at diagnosis is the most frequent form of presentation, with 60% of patients presenting with stage IV disease[3,4].

Histologically, PTCL-NOS is characterised by a pleomorphic population of atypical lymphocytes containing irregular, vesicular or hyperchromatic nuclei with prominent nucleoli and high mitotic rate. Pan-T cell antigens expression is so variable, at least one must be expressed: CD2, CD3, CD5 or CD7[3,5]. The predominant immunophenotype is CD3+ and CD4+ without cytotoxic markers. The Ki-67 proliferation index is typically high and may carry prognostic significance. Immunophenotypic confirmation is crucial, as diagnosis relies on exclusion of other mature T-cell lymphomas, following the WHO 5th edition classification[2,6,7].

Treatment typically involves anthracycline-based chemotherapy (CHOP), though outcomes remain poor, with a five-year progression-free survival below 30%[3,4,7]. Therapy selection now considers CD30 expression: if ≥1% of tumour cells express CD30, BV+CHP (brentuximab vedotin plus cyclophosphamide, doxorubicin, prednisone) may be used; otherwise, CHOP is the standard. In patients under 65 in good condition, CHOEP (CHOP + etoposide) is preferred to intensify treatment, while older or frail patients receive standard CHOP to limit toxicity. Evidence supporting radiation or stem cell transplant remains controversial[8,9].

The patient received CHOP but showed no response; second-line treatment with GemOx – typically used for refractory NHL – was also ineffective, resulting in death[10].

Although cardiac involvement has been described in extranodal PTCL-NOS, this is the first reported case in an adult patient with simultaneous pericardial and myocardial involvement documented by multiple imaging modalities (echocardiography, PET-CT and cardiac MRI), none of which were sufficient to establish a definitive diagnosis. The case underscores the need for early tissue diagnosis in atypical or rapidly progressing pericardial syndromes and broadens the known manifestations of extranodal PTCL-NOS.

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