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Case Report

Primary pericardial abscess caused by *Staphylococcus aureus* infection without a predisposing condition

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ABSTRACT

A 75-year-old man presented to the hospital with a low-grade fever and worsening dyspnea. Transthoracic echocardiogram and contrast-enhanced computed tomography revealed a 20 × 20 mm lesion adjacent to the left ventricle with pericardial effusion. We suspected pericardial abscess, but no bacteria were detected even after 6 consecutive blood cultures. Ultimately, we drained 500 mL serosanguinous fluid from the pericardial effusion on the 4th hospital day; a subsequent culture grew methicillin-sensitive *Staphylococcus aureus*. Although we performed percutaneous and surgical drainage and intravenous administration of antibiotics, he developed constrictive pericarditis, and died due to multi-organ failure on the 21st hospital day. On histological examination, neutrophil infiltration was noted in the thickened pericardium and the myocardium. To our knowledge, a purulent pericarditis complicated pericardial abscess can occur without bacteremia, and early diagnosis and aggressive management are necessary for a good prognosis.

<**Learning objective:** Pericardial abscess (PA) is a rare but serious life-threatening illness. We report the case of a patient with primary PA induced by *S. aureus* infection without a predisposing condition. A purulent pericarditis complicated PA can occur without bacteremia being detected from sequential blood cultures. Early diagnosis and aggressive management are vital to ensure a good prognosis.>

Introduction

Pericardial abscess (PA) is a rare but serious life-threatening illness. Since the advent of antibiotic therapy, the incidence of PA has decreased, while the etiologic agents have changed from predominantly pneumococcus and streptococcal species to staphylococcus, anaerobes, and fungus [1]. Nowadays, PA is observed as a secondary complication of immunosuppression in hosts, chronic inflammation, drug users, or following trauma. We report the case of a patient with primary PA induced by *Staphylococcus aureus* infection without bacteremia, which developed to constrictive pericarditis.

Case report

A 75-year-old man presented to our hospital with a low-grade fever and a one-month history of worsening dyspnea. His medical history was myelodysplastic syndrome (MDS) without excess blasts, indicating good prognosis (international prognostic scoring system: scored as very low risk). On physical examination, the blood pressure was 113/61 mmHg, the pulse rate 53 beats per minute, and the temperature 37.3 °C. Laboratory tests revealed a white blood cell count of 10,300/μL (neutrophils, 81%), hemoglobin 7.5 g/dL, C-reactive protein 17.84 mg/dL, creatine kinase 69 U/L, creatine kinase myocardial band 16 U/L, and troponin T 0.038 ng/mL, which indicated inflammation, infection, and refractory anemia of MDS, but no sign of myocardial injury. His chest radiograph showed cardiomegaly without congestion. A twelve-lead electrocardiogram showed normal sinus rhythm and ST elevation in leads I, II,

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III, aV_F, and V_2–V_6. Bedside transthoracic echocardiogram (TTE) revealed that the left ventricle showed mild hypertrophy (interventricular septal thickness 11 mm, left ventricular posterior wall thickness 9 mm) with normal left ventricular diameter and good left ventricle function (ejection fraction 67%). In addition, surprisingly, TTE also demonstrated a 20.5 mm × 19.5 mm low echoic tissue adjacent to the left ventricle with a large amount of pericardial effusion and no evidence of infective endocarditis (Fig. 1A). Furthermore, there was no sign of cardiac tamponade including right ventricular collapse, and constrictive pericarditis was diagnosed based on the clinical condition and TTE. Contrast-enhanced computed tomography (CT) also demonstrated a roughly 20 × 20 mm abnormal mass between the left ventricle apex and pericardium, which had not been observed one month previously (Fig. 1B). Although pericardial abscess was suspected, no bacteria were detected from the sputum or 6 consecutive blood cultures and no primary lesion was observed. To determine the diagnosis, a 5-French pigtail catheter was inserted into the pericardial space and 500 mL of serosanguinous fluid was removed on the 4th hospital day (Fig. 1C). Laboratory data on serosanguinous fluid examination revealed elevated polynuclear leukocyte rate (94%) and lactate dehydrogenase level (601 U/L). Subsequent pericardial fluid culture grew methicillin-sensitive *S. aureus*. Although intravenous administration of cefazolin 4.0 g/day was initiated, severe drug eruptions appeared. Therefore, we changed the drug to vancomycin 1.5 g/day. As the patient's clinical condition was progressively worsening despite the vancomycin that was controlled within the range of 10–20 μg/mL(the target blood concentration was the range of 15–20 μg/mL), and a follow-up contrast-enhanced CT on the 9th hospital day showed that a roughly abnormal mass persisted and the thickness of the pericardium was increasing (Fig. 1D). Hence, surgical subxiphoid pericardial drainage was needed on the 17th hospital day. A repeat TTE on the 19th hospital day showed classic features of constrictive pericarditis such as increased pericardial thickness, septal bounce motion, dilatation of the inferior vena cava with diminished inspiratory collapse, moderate biatrial enlargement and worsening transmitral flow pattern (from abnormal relaxation pattern to restrictive pattern). Despite maximal inotropic therapy with an artificial respirator, his symptoms did not ameliorate and he developed cardiogenic shock. He developed disseminated intravascular coagulation, which indicated bacteremia. On the 21st hospital day, the patient finally died due to multiorgan failure from sepsis and constrictive pericarditis. Autopsy findings showed that the abnormal mass adjacent to the left ventricular was an abscess without infection of other organs (Fig. 2A). On histological examination, significant neutrophil infiltration was noted in the thickened pericardium and the myocardium around the abscess. However, there was less myocyte necrosis and inflammatory cell infiltration in the myocardial interstitial tissue (Fig. 2B–D).

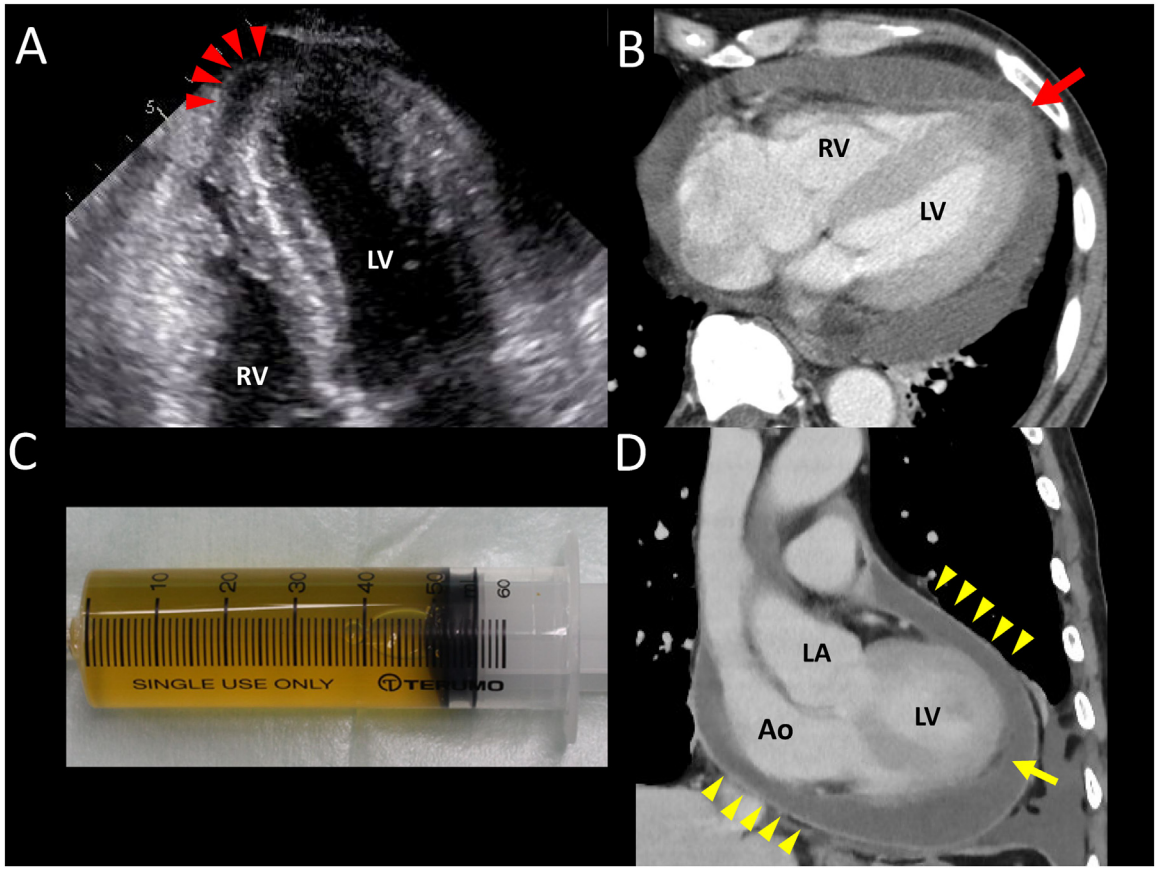

Fig. 1. (A) Transthoracic echocardiography shows a large amount of pericardial effusion and a 20.5 × 19.5 mm low echoic tissue adjacent to the left ventricle (red arrowheads). (B) Contrast-enhanced CT on admission shows pericardial effusion and a roughly 20 × 20 mm abnormal mass between the left ventricle apex and pericardium (red arrow). (C) The appearance of pericardial fluid shows serosanguinous. (D) Contrast-enhanced CT on the 9th hospital day shows thickened pericardium (yellow arrowheads) and a roughly abnormal mass (yellow arrow).
CT, computed tomography; LV, left ventricular; RV, right ventricular, Ao; aorta.

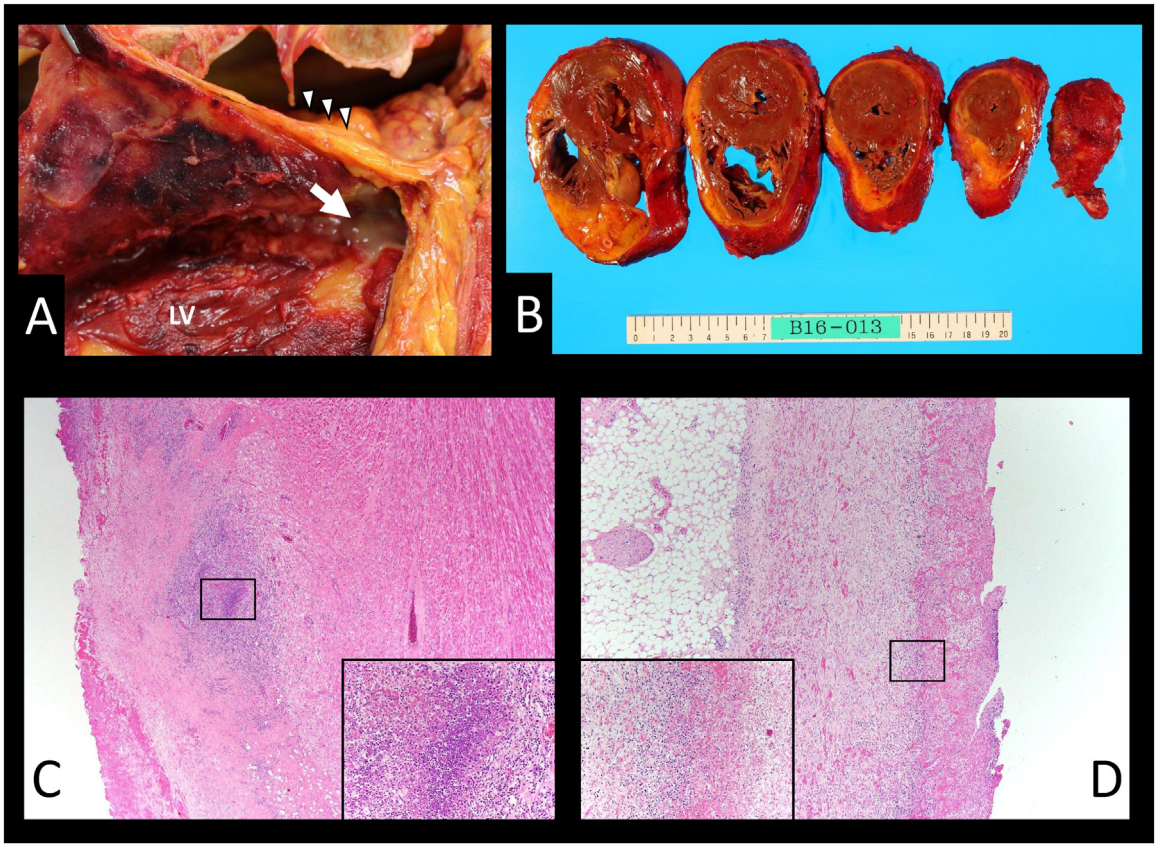

Fig. 2. (A) Autopsy finding shows an abscess (white arrow) adjacent to the left ventricle (LV) and thickened pericardium (white arrowhead). (B) Macroscopic findings show no abscess in the endocardium and no myocarditis. (C), (D) Histologic findings show significant neutrophil infiltration between the myocardium and pericardium, and slightly in myocardial interstitial tissue. We expanded and showed the black square part.

Discussion

PA is an extremely rare complication of *S. aureus* bacteremia, with only a few reported cases [1,2]. The possible explanations of the mechanism that can lead to invasion of the pericardial space in secondary purulent pericarditis are: (1) hematogenous spreading, (2) direct extension from a pulmonary, pleural, mediastinal, or diaphragmatic focus, (3) contiguous spreading from a cardiac infection (myocardial abscess, endocarditis), and (4) perforating injury of the chest wall (trauma or surgery) [3,4]. In this case, there was no history of chest trauma, chest surgery, sepsis, other organ abscess, or cardiac infection. The pathogenesis of infection route is therefore uncertain, but we consider that the PA may have been caused by transient *S. aureus* bacteremia, because *S. aureus* is more often involved in hematogenous spread. It is an interesting point that localized pericardium infection progressed to PA in a patient without any predisposing condition within one month. Pericardiocentesis is the simplest and fastest method to treat symptomatic pericardial effusion, but it is often ineffective to drain for localized fibrinous fluid or abscess. Besides, pericardiocentesis also frequently leads to development of constrictive pericarditis [5]. Intrapericardial infusion of fibrinolytics or pericardiotomy can prevent both constrictive and persistent purulent pericarditis [6]. Therefore, it might be necessary for patients with purulent pericarditis-complicated PA to undergo early aggressive management such as intrapericardial infusion of fibrinolytics or pericardiotomy. In our case, repeated laboratory tests did not show elevation of creatine kinase, creatine kinase myocardial band, and troponin T. Besides, post-mortem autopsy showed less myocyte necrosis and inflammatory cell infiltration in the myocardial interstitial tissue. Although PA did not progress to acute cardiac myocarditis, the patient developed sepsis and constrictive pericarditis. To the best of our knowledge, a purulent pericarditis-complicated PA can occur without bacteremia detected from sequential blood cultures; early diagnosis and aggressive management are necessary for good prognosis.

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Disclosure

None.

Conflict of interest

None declared.

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None.

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