

# World Journal of *Clinical Cases*

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Editorial Board Member of *World Journal of Clinical Cases*, Rajesh Kumar Rajnish, MBBS, MS, Assistant Professor, Department of Orthopaedics, All India Institute of Medical Sciences, Bilaspur, Bilaspur 174001, Himachal Pradesh, India. [duktiraj@gmail.com](mailto:duktiraj@gmail.com)

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CASE REPORT

## Pseudomonas aeruginosa-related effusive-constrictive pericarditis diagnosed with echocardiography: A case report

Jin-Ling Chen, Dan-E Mei, Cai-Gui Yu, Zhi-Yu Zhao

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### Abstract

#### BACKGROUND

Effusive-constrictive pericarditis (ECP) is an uncommon pericardial syndrome. Careful echocardiographic examination may provide helpful information not only for diagnosing but also for managing ECP. ECP has various etiologies; however, *Pseudomonas aeruginosa* (*P. aeruginosa*) infection has not been reported as a cause to date. Herein, we present a rare case of ECP caused by *P. aeruginosa* infection, which was followed up using echocardiography.

#### CASE SUMMARY

A 30-year-old man was admitted to our hospital with a 2-mo history of cough, dyspnea, bloating, palpitations, and lower-extremity edema. The patient was initially diagnosed with pericardial effusion by transthoracic echocardiography. Drainage of pericardial effusion was performed to relieve the clinical symptoms. A follow-up echocardiogram showed that the pericardial effusion had decreased; however, the right atrial pressure continued to increase, and signs of constrictive pericarditis were observed upon a more comprehensive inspection. Therefore, the diagnosis of ECP was established based on the comprehensive pre- and post-pericardiocentesis echocardiographic findings. An urgent pericardectomy was subsequently performed, which significantly relieved the patient's clinical symptoms, and the signs of pericardial constriction on echocardiography improved. Pericardial effusion and pericardial culture showed growth of *P. aeruginosa*.

#### CONCLUSION

ECP induced by *P. aeruginosa* infection remains a rare disease. The presence of echocardiographic features of constrictive pericarditis after pericardiocentesis therapy is highly indicative of ECP.

**Key Words:** Effusive-constrictive pericarditis; *Pseudomonas aeruginosa* infection; Echocardiography; Case report

**Core Tip:** Effusive-constrictive pericarditis (ECP) is an uncommon clinical syndrome with varied causes, and *Pseudomonas aeruginosa* (*P. aeruginosa*) infection is an extremely rare etiology of ECP. This report presents a rare case of *P. aeruginosa*-induced ECP that was diagnosed based on comprehensive post-pericardiocentesis follow-up echocardiography. The patient was initially misdiagnosed with pure pericardial effusion. This emphasizes the importance of sensitive identification of ECP echocardiographic features and the advantage of follow-up echocardiography after pericardiocentesis for the diagnosis and treatment of ECP.

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## INTRODUCTION

Pericardial effusion is a common clinical condition that can be accurately diagnosed using echocardiography. However, when acute pericardial effusion gradually transforms into chronic constrictive pericarditis, which is defined as effusive-constrictive pericarditis (ECP), the diagnosis becomes confusing and may lead to misdiagnosis and inappropriate treatment. As an uncommon clinical syndrome, ECP is characterized by the coexistence of pericardial effusion and constriction of the heart by the visceral pericardium[1]. The persistence of elevated central venous pressure after pericardiocentesis is the hemodynamic characteristic of ECP. The etiologies of ECP include purulent pericarditis, tuberculosis, trauma, and neoplastic involvement[2], of which *Pseudomonas aeruginosa* (*P. aeruginosa*) infection represents an extremely small percentage. Follow-up echocardiography is a useful tool for diagnosing and treating ECP. Herein, we report a rare case of ECP caused by *P. aeruginosa* infection, diagnosed using echocardiography before and after pericardiocentesis.

## CASE PRESENTATION

### Chief complaints

A 30-year-old man was admitted to our hospital with a 2-mo history of cough, dyspnea, bloating, palpitations, and lower extremity edema (Table 1).

### History of present illness

He was diagnosed with atrial fibrillation, heart failure, and multiple serous effusions (pleural, pericardial, and abdominal effusions) at a local hospital.

### History of past illness

The patient had no history of any previous disease.

### Personal and family history

The patient had no previous or family history of similar illnesses.

### Physical examination

The patient's vital signs on admission were stable. Cardiac auscultation revealed muffled heart sounds. Palpation revealed mild edema in both the lower extremities. Electrocardiography revealed atrial fibrillation and a decreased voltage.

### Laboratory examinations

Blood tests revealed slightly elevated thyroid-stimulating hormone levels, moderately elevated inflammatory markers, and significantly increased N-terminal-pro hormone brain natriuretic peptide levels. The liver and kidney functions were within the normal range. The T-SPOT test yielded a negative result.

### Imaging examinations

Two-dimensional transthoracic echocardiography (2D-TTE), on admission, revealed a significantly

**Table 1 Timeline**

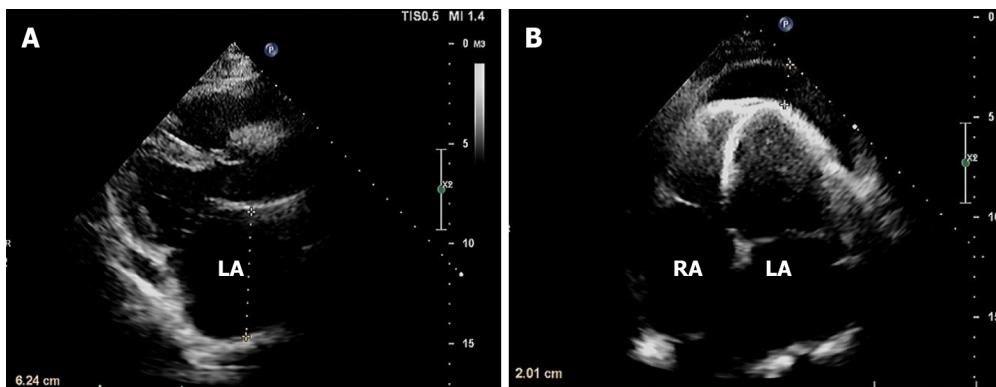
|                     |  |
|---------------------|--|
| Day 1               | A 30-yr-old man was admitted to our hospital with a 2-mo history of cough, dyspnea, bloating, palpitation, and lower extremity oedema  |
| Day 2               | The patient was taken to the ultrasound department for an echocardiogram showing a moderate-sized localized pericardial effusion, with an increase in the RAD and LAD (Figure 1)   |
| Day 5               | The patient underwent pericardiocentesis to relieve the clinical conditions  |
| Day 6               | The patient's clinical symptoms were slightly relieved   |
| Day 8               | A repeat echocardiogram showed decreased pericardial effusion compared with the first examination and characteristic manifestations of pericardial constriction. A diagnosis of ECP was established (Figure 2)               |
| Day 10              | A CT scan demonstrated a small amount of pericardial effusion with thickened pericardium   |
| Day 15              | PET-CT examination ruled out neoplastic and connective tissue diseases   |
| Day 17              | The patient underwent pericardectomy, which led to a rapid improvement in the clinical symptoms  |
| Day 19              | Histologic examination performed on the pericardial tissue obtained during pericardectomy showed increased thickening with the proliferation of collagen fibers, hyaline degeneration, and calcification (Figure 4)          |
| Day 20              | Cytologic examination of the pericardial fluid obtained during pericardectomy showed many inflammatory cells and few mesothelial cells. Cultures of the pericardial fluid and pericardium grew <i>Pseudomonas aeruginosa</i> |
| Day 24              | A repeat 2D-TTE 1 wk after pericardectomy revealed improvement in the biatrial diameter, Doppler features of pericardial constriction, and the right atrial pressure (Figure 3)  |
| Day 26              | The patient was discharged home in stable condition  |
| 1 mo post-discharge | A follow-up echocardiogram revealed that the right atrial pressure assessed using echocardiography had significantly decreased in the absence of pericardial constriction echocardiographic features (Figure 5)              |

RAD: Right atrium dimension; LAD: Left atrium dimension; ECP: Effusive-constrictive pericarditis; 2D-TTE: two-dimensional transthoracic echocardiography; PET: Positron emission tomography; CT: Computed tomography.

enlarged diameter of the left atrium (LAD) and right atrium (RAD) and a moderate pericardial effusion that was predominantly along the apical wall, measuring up to 2 cm (Figure 1). The diagnosis of biatrial enlargement due to atrial fibrillation and localized pericardial effusion was established according to echocardiographic manifestations and history. Emergency surgical pericardial drainage was performed by removing 500 mL purulent fluid to relieve the clinical presentation. Despite the removal of most of the pericardial fluid, which indicates a significant decrease in intrapericardial pressure, the patient's clinical symptoms showed no significant improvement. Subsequent 2D-TTE (Figure 2) failed to show normalization of LAD and RAD. In addition, echocardiography revealed other information with significant diagnostic value, such as abnormal movement of the interventricular septum ("interventricular septum bounce") and increased pericardial thickness. Notably, pulsed-wave Doppler revealed significant respiratory variation (greater than 25%) in the peak mitral inflow velocity (E) and an increase in the peak velocity blood flow from left ventricular relaxation in early diastole to peak velocity flow in late diastole caused by atrial contraction (E/A) ratio ( $> 2$ ). Tissue Doppler imaging of the mitral annulus revealed "annulus reversus," as the lateral mitral e' velocity was lower than the medial mitral e' velocity. M-mode echocardiography revealed a dilated inferior vena cava (IVC) with almost no respiratory variation, according to which the estimated right atrial pressure was elevated (20 mmHg). Thus, a diagnosis of ECP as the underlying cause of the clinical presentation was established according to the pre- and post-pericardiocentesis echocardiographic performance. Computed tomography (CT) scan also revealed a small amount of pericardial effusion with a thickened pericardium. Positron emission tomography-CT (PET-CT) ruled out connective tissue and neoplastic diseases. Clinical and imaging tests revealed the need for urgent surgical removal of the residual pericardial effusion and visceral pericardium. A repeat 2D-TTE 1-wk after pericardectomy revealed that the biatrial diameter and echocardiographic features of pericardial constriction improved, and the right atrial pressure assessed using echocardiography also decreased (Figure 3 and Table 1).

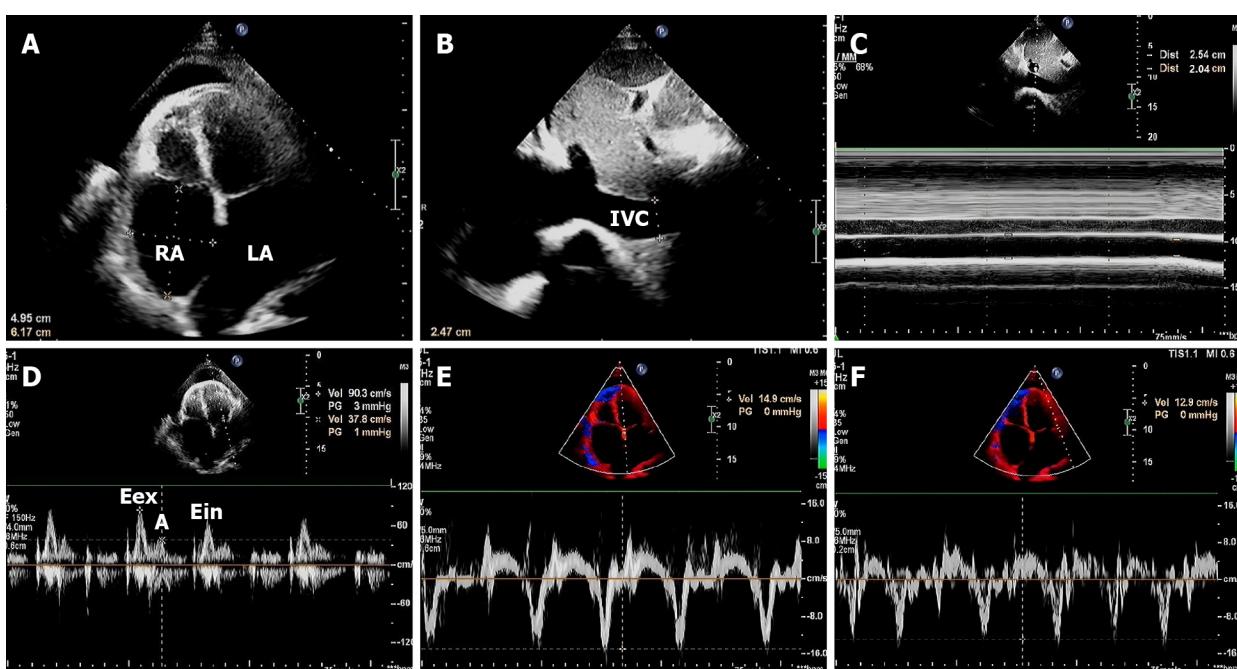
### Postoperative biochemical examination and pathological examination

Cytological examination of the pericardial fluid revealed many inflammatory cells and few mesothelial cells. Cultures of the pericardial fluid and the pericardium grew *P. aeruginosa*. Histological examination of the pericardial tissue obtained during pericardectomy revealed increased thickening with the proliferation of collagen fibers, hyaline degeneration, and calcification (Figure 4 and Table 1).



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**Figure 1 Echocardiographic examinations before pericardiocentesis.** A: The left atrium was significantly enlarged, with an anteroposterior diameter of 6.2 cm; B: The diameters of the left and right atrium were 5.9 cm × 6.1 cm and 5.0 cm × 6.0 cm, respectively. A moderate pericardial effusion that was predominantly along the apical wall, measuring up to 2.0 cm. LA: Left atrium; RA: Right atrium.



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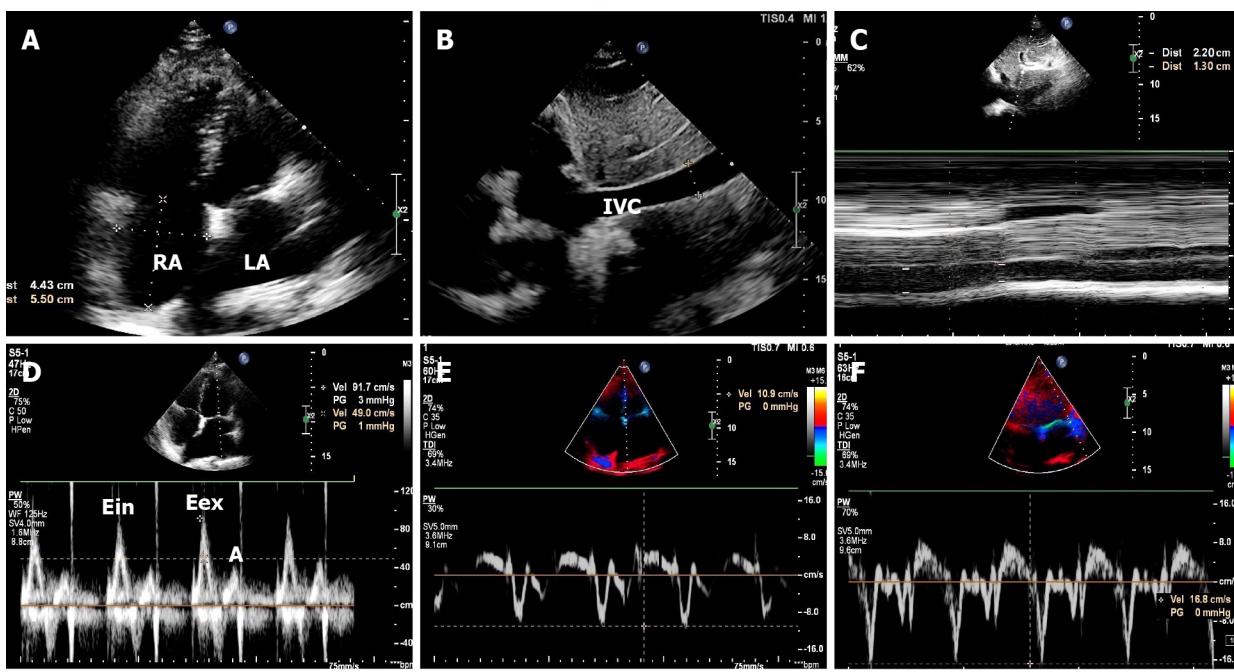
**Figure 2 Echocardiographic examinations after pericardiocentesis.** A: Right atrial (50 mm × 62 mm) and left atrial (61 mm × 61 mm) enlargement, increased pericardial thickness, and pericardial effusion that was located predominantly along the apical wall (measuring up to 1.2 cm); B and C: Dilated inferior vena cava (up to 2.5 cm) almost without any respiratory variation (20%), indicating an elevated right atrial pressure of approximately 20 mmHg; D: Pulsed wave doppler of the mitral valve showed that the peak mitral E and A inflow velocity were 90 cm/s and 38 cm/s, respectively. The E/A ratio was > 2, indicating restricted mitral inflow velocity. Respiratory variation in the peak mitral E inflow velocity was 28% (peak E velocity during expiration and inspiration were 90 cm/s and 65 cm/s, respectively); E and F: Tissue Doppler imaging showed “annulus reversus” with the lateral mitral e' velocity (12.9 cm/s) abnormally lower than the medial mitral e' velocity (14.9 cm/s). IVC: Inferior vena cava; LA: Left atrium; RA: Right atrium.

## FINAL DIAGNOSIS

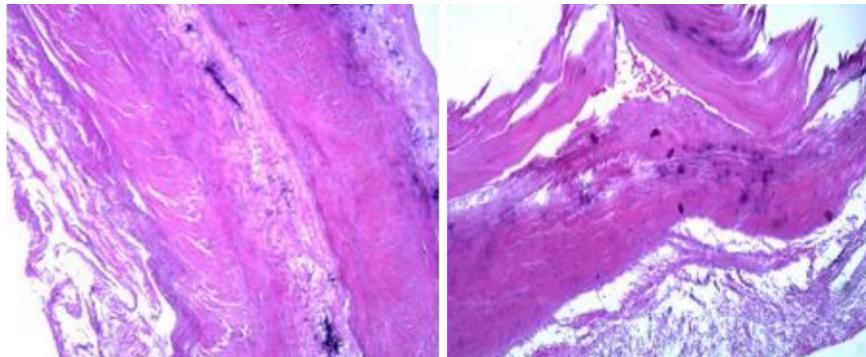
ECP caused by *P. aeruginosa* infection.

## TREATMENT

Emergency surgical pericardial drainage was performed to relieve the clinical presentation by removing 500 mL purulent fluid according to the initial diagnosis of moderate pericardial effusion established by echocardiography. Antibiotics and diuretics were subsequently administered to relieve the symptoms. After the diagnosis of ECP was established via follow-up echocardiography, pericardectomy was



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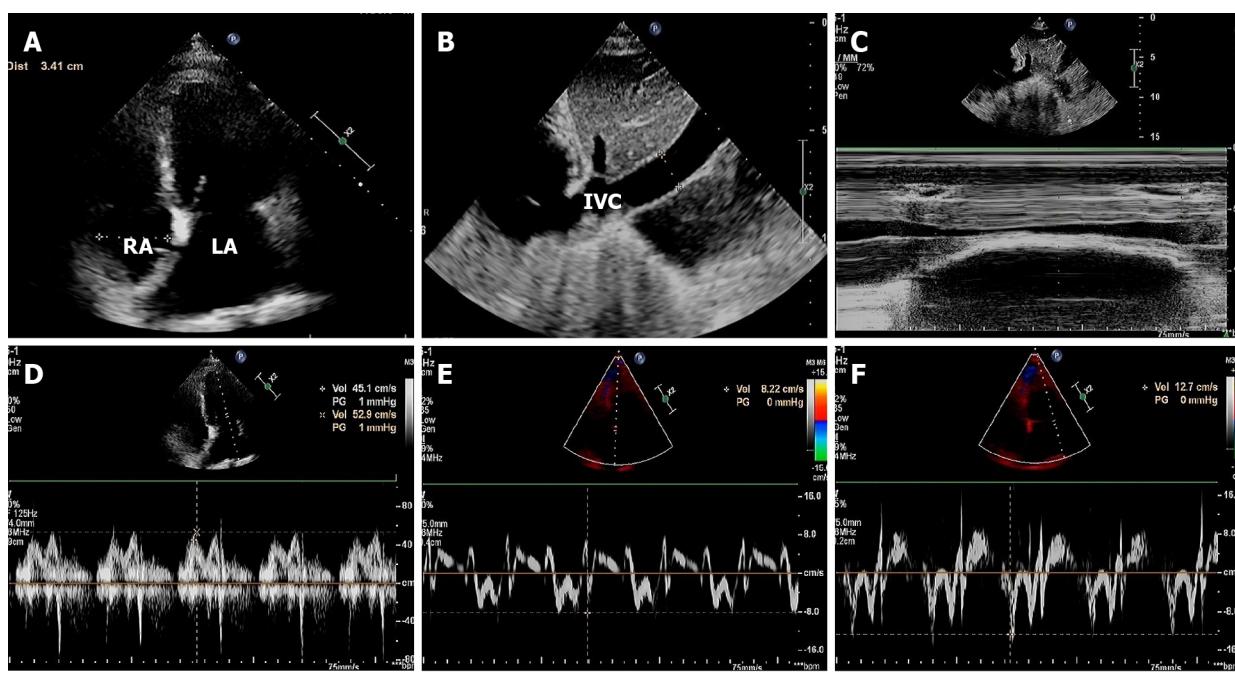
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**Figure 4** Histologic examination performed on the pericardial tissue revealed increased thickening with the proliferation of collagen fibers, hyaline degeneration, and calcification.

performed. During pericardectomy, we observed that the heart was covered by a 2 mm inflammatory membrane, which eventually led to the loss of elasticity of the pericardium, external compression, and diastolic restriction of the heart. Antibiotic therapy was administered to the patient after surgery (Table 1).

## OUTCOME AND FOLLOW-UP

It was only after the careful removal of the constricted visceral pericardium that a reduction in the right atrial pressure was observed, and the echocardiographic patterns of constrictive physiology improved. The patient's clinical symptoms were significantly relieved. Echocardiography performed 1 mo after surgery demonstrated an obvious improvement in the echocardiography data and hemodynamics. It showed that the pericardial effusion was resolved, the RAD and LAD decreased, and the right atrial pressure was also significantly decreased (Figure 5 and Table 1).



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**Figure 5 The follow-up echocardiography 1 mo after discharge.** A: The size of the left atrium was smaller than before ( $5.3 \text{ cm} \times 5.5 \text{ cm}$ ), and the right atrium was approximately normal ( $3.4 \text{ cm} \times 4.2 \text{ cm}$ ) in the absence of pericardial effusion; B and C: The diameter of the inferior vena cava (1.9 cm) and respiratory variation (greater than 50%) were normalized, based on which the calculated right atrial pressure was within the normal range, approximately 5 mmHg; D: Pulsed-wave Doppler of the mitral valve showed that the peak mitral E and A inflow velocities were 45 cm/s and 53 cm/s, respectively. The E/A ratio was < 1; E and F: Tissue Doppler imaging showed that the medial mitral e' velocity (8.2 cm/s) was lower than the lateral mitral e' velocity (12.7 cm/s). IVC: Inferior vena cava; LA: Left atrium; RA: Right atrium.

## DISCUSSION

ECP is a rare pericardial disease characterized by the coexistence of pericardial constriction and effusion. Its characteristic feature is a persistently elevated right atrial pressure of  $> 10 \text{ mmHg}$  or a reduction of less than 50% from baseline despite pericardiocentesis[3]. The etiology of ECP varies, including idiopathic origin, viral origin, post-procedural origin, radiation, drug-induced, connective tissue disease, malignancy, and tuberculosis. *P. aeruginosa* is a rare etiological agent of ECP. Given the growth of *P. aeruginosa* in the pericardial fluid and tissue, this was a case of ECP induced by *P. aeruginosa*, which is rarely reported in the literature. Infections with *P. aeruginosa* occur mainly in immunocompromised and neutropenic patients and are associated with the presence of indwelling catheters and disruption of the mucocutaneous barriers. Prior hospitalization and prior use of antibiotics were the most significant risk factors for the acquisition of antibiotic-resistant *P. aeruginosa* [4]. *P. aeruginosa* causes a polymorphonuclear reaction that may progress to fibrotic thickening of the pericardium, fibrin deposition and non-specific inflammation, eventually leading to constrictive pericarditis and purulent pericardial effusion[5].

The prevalence of ECP varies from 1.4% to 14% in patients presenting with pericardial effusion[6]. Most pericardial effusions are accurately recognized using echocardiography and do not require drainage with pericardiocentesis; pericardiocentesis is urgently needed to relieve symptoms only in symptomatic patients with cardiac tamponade. For patients with ECP, the clinical condition cannot be improved by drainage of the pericardial effusion alone, and pericardectomy is a life-saving intervention. Therefore, sensitive identification of pure pericardial effusion and ECP is of vital significance for clinical treatment.

The gold standard for the diagnosis of ECP is invasive hemodynamic evaluation using cardiac catheterization. Echocardiography can provide a potential non-invasive diagnostic tool with high sensitivity and specificity, which shows the coexistence of effusive and constrictive pericardial features[2]. Identification of ECP using echocardiography requires comprehensive observation with 2D, Doppler, and M-mode echocardiography[7]. 2D imaging has an excellent ability to detect effusion, enlarged atrial size, interventricular septum bounce, and a dilated IVC, which are sensitive and specific signs for the initial diagnosis of ECP. The echo-Doppler findings are diagnostically helpful, and it has been proposed that the presence of constrictive pericarditis echocardiographic Doppler features after pericardiocentesis would help clarify the diagnosis of ECP[8,9]. The key characteristic performance features of ECP include “annulus reversus,” restrictive mitral inflow (E/A > 2), greater than 25% respiratory variation in the peak mitral inflow velocity (E)[10]. The M-mode echocardiography can demonstrate a persistently

dilated IVC and the posterior motion of the septum during early diastole on inspiration[7,11], providing additional diagnostic information. In the present case, echocardiographic signs of pericardial effusion were observed during the initial examination. However, ECP is relatively rare and cannot be easily diagnosed. As one of the most notable characteristics of pericardial constriction, pericardial thickness was ignored because of a lack of comprehensive understanding of ECP echocardiographic features, leading to a misdiagnosis of localized pericardial effusion. Although pericardial effusion decreased after pericardiocentesis, repeat echocardiography demonstrated that the right atrial pressure evaluated using echocardiography remained high, and the characteristics of pericardial constriction were present, providing sufficient evidence for the appropriate diagnosis of ECP.

The present case of ECP associated with *P. aeruginosa* infection demonstrated that in patients with localized pericardial effusion who have typical risk factors, such as infection, prior use of antibiotics, and prior hospitalization, the presence of ECP should be highly suspected[3]. Additionally, this case highlighted the significance of sensitive recognition of the echocardiographic features of ECP, including pericardial effusion combined with atrial enlargement, which is the most intuitive feature of constrictive pericarditis. A comprehensive study with 2D imaging, Doppler, and M-mode echocardiography is required to avoid misdiagnosis. More importantly, for patients initially diagnosed with pericardial effusion, the echocardiographic features of constrictive pericarditis after pericardiocentesis should raise a suspicion of ECP[12]. Therefore, when a localized pericardial effusion is detected, follow-up echocardiographic examinations may assist in clinical diagnosis and treatment.

## CONCLUSION

We highlight a rare case of ECP associated with *P. aeruginosa* infection that was initially misdiagnosed as pericardial effusion using echocardiography. Comprehensive echocardiography, including 2D, M-mode, and Doppler echocardiography, should help avoid misdiagnosis, especially during the follow-up after pericardiocentesis.

## FOOTNOTES

**Author contributions:** Chen JL designed the report and revised the manuscript; Mei DE analyzed the data, reviewed the literature and drafted the manuscript; Yu CG and Zhao ZY collected the patient's clinical data; all authors have read and approved the final manuscript.

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**Country/Territory of origin:** China

**ORCID number:** Jin-Ling Chen 0000-0001-5997-131X; Dan-E Mei 0000-0002-8910-0395; Cai-Gui Yu 0000-0003-2690-588X; Zhi-Yu Zhao 0000-0002-1619-5258.

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