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*Case Report*

# Liver abscess caused by *Bacteroides thetaiotaomicron* complicated by purulent pericarditis: a case report

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**Abstract:** Background: Liver abscess complicated by purulent pericarditis is a rare clinical condition. To date, no cases of liver abscess caused by *Bacteroides thetaiotaomicron* with concomitant purulent pericarditis have been reported. We present a rare case of this dual pathology to improve clinicians' understanding of liver abscess-related complications and uncommon pathogens. Case Presentation: A 33-year-old female was admitted with hypothermia, chest tightness, and vomiting. Upon admission, laboratory tests showed elevated inflammatory markers. Ultrasound imaging revealed both pericardial effusion and a hepatic abscess, leading to a diagnosis of liver abscess complicated by purulent pericarditis. Metagenomic next-generation sequencing (mNGS) of pericardial fluid identified *Bacteroides thetaiotaomicron*. The patient received anti-infective therapy with meropenem and ornidazole, combined with pericardial effusion drainage. During the later stage of treatment, the patient developed thoracic empyema, necessitating thoracic drainage. Following treatment, the inflammatory markers significantly improved, the liver abscess reduced in size, and the pericardial effusion nearly resolved. At the 8-week follow-up after discharge, clinical and imaging findings were normal. Conclusion: This case highlights the importance of recognizing atypical manifestations in immunocompromised patients and emphasizes the critical role of early comprehensive examination, prompt and effective anti-infective therapy, and puncture drainage for improving patient outcome. Pathogen identification methods such as mNGS can guide more precise treatment strategies, thereby shortening hospitalization and reducing complications.

**Keywords:** Liver abscess, purulent pericarditis, *Bacteroides thetaiotaomicron*, metagenomic next-generation sequencing

## Introduction

Pyogenic liver abscess (PLA) is the most common visceral abscess in clinical practice. Purulent pericarditis is an uncommon but serious complication of PLA. *Bacteroides thetaiotaomicron*, an intestinal commensal bacteria, is a rare pathogen responsible for both liver abscess and purulent pericarditis. We describe a case of PLA complicated by purulent pericarditis caused by *Bacteroides thetaiotaomicron*. The patient's condition improved with early diagnosis, prompt initiation of effective anti-infective therapy, and timely drainage. This case highlights the importance of recognizing rare pathogens and complications in PLA to avoid misdiagnosis and delayed treatment.

## Case presentation

A 33-year-old woman employed in e-commerce was admitted on February 21, 2024, with a history of progressive weight loss for 3 months (about 15 kg) and chest tightness with vomiting for 1 day. She had a 6-year history of microcytic, hypochromic anemia, with a baseline hemoglobin level of 90-110 g/L, but remained largely asymptomatic and had not sought medical evaluation due to limited health awareness. On February 8, 2024, routine outpatient blood test revealed leukocytosis (white blood cell [WBC] count 24.3 × 10^9/L) and elevated C-reactive protein (CRP, 119.9 mg/L). The patient denied fever, cough, sputum production, chest tightness, shortness of breath, abdominal pain,

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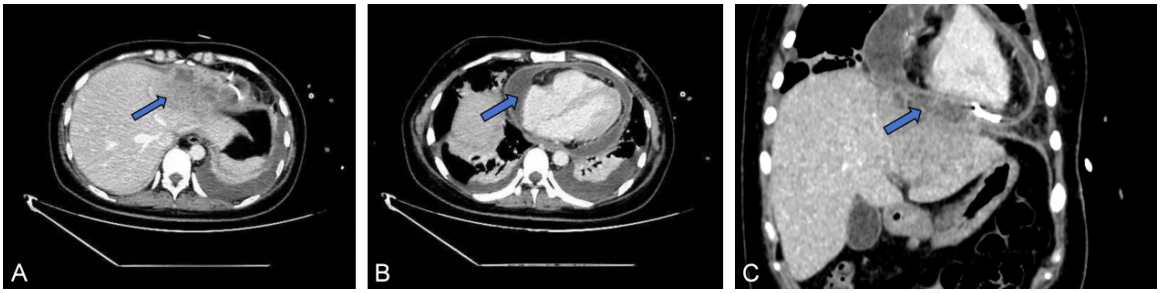


**Figure 1.** A. Axial CT scan at the upper abdominal level: The blue arrow indicates the left lateral segment of the liver, with unclear boundaries and portions extending beyond the liver margin, growing outward. After contrast enhancement, the lesion shows heterogeneous enhancement. B. Chest CT: The blue arrow highlights the Pericardial effusion. C. Coronal CT reconstruction of the abdomen: The lesion has invaded the diaphragm, and the pericardial effusion is not septated.

diarrhea, or urinary symptoms at that time. She received empirical treatment in the emergency department with intravenous ceftriaxone sodium (2 g once daily for 5 days) and oral cefuroxime axetil tablets (0.25 g twice daily for 3 days). However, inflammatory markers showed minimal improvement. One day prior to admission, she developed chest tightness, shortness of breath, and recurrent vomiting. On admission, laboratory results showed WBC of 57 × 10^9/L, CRP of 266.4 mg/L, random blood glucose of 11.36 mmol/L, hemoglobin A1c of 6.3%, brain natriuretic peptide (BNP) of 268.2 pg/mL, and procalcitonin (PCT) >100.000 ng/mL. Cardiac ultrasound demonstrated pericardial effusion, while hepatobiliary ultrasound revealed a mixed echogenic mass in the left hepatic lobe, suggestive of a liver abscess. Following cardiology consultation, ultrasound-guided pericardial puncture and drainage were performed. The patient was admitted with a final diagnosis of liver abscess complicated by purulent pericarditis.

### Family and physical examination

The patient's mother had a history of chronic lymphocytic leukemia. On admission, the patient's temperature was 36.8°C, respiratory rate 18 breaths per minute, pulse 119 beats/min, and blood pressure 126/84 mmHg. She appeared fatigued and adopted an upright position to ease breathing. No jaundice of the skin or sclera was observed. Lung auscultation revealed coarse breath sounds bilaterally, without crackles or rales. The heart rhythm was regular, with no pathologic murmurs. Percussion tenderness over the liver region was positive.

### Laboratory and imaging findings

February 21, 2024, pericardial fluid analysis showed a positive Rivalta test, nucleated cell count of 6240 × 10^6/L (86% neutrophils), and CRP of 130.4 mg/L. Cardiac ultrasound revealed pericardial effusion (1.7 cm posterior to the left ventricular posterior wall, 1.4 cm anterior to the right ventricular anterior wall, and 1.7 cm at the apex). Liver ultrasound revealed a mixed echogenic mass in the left hepatic lobe (8.26 × 7.23 cm), suggestive of a liver abscess. On February 22, 2024, laboratory tests showed WBC of 41.8 × $10^9$/L, CRP of 291.7 mg/L, PCT of 71.243 ng/mL, ferritin of 17871.1 ng/mL, and interleukin-6 of 152.05 pg/mL. Autoimmune tests revealed no significant abnormalities in antinuclear antibodies. Blood culture and abscess fluid culture were negative. Enhanced abdominal computed tomography (CT) (**Figure 1A**) demonstrated a suspected left liver abscess and possible caudate lobe infection. Chest CT (**Figure 1B**) revealed pericardial effusion, bilateral pulmonary infiltrates, and pulmonary edema. The lesion has invaded the diaphragm, and the pericardial effusion is not septated (**Figure 1C**).

### Treatment

Upon admission, the patient received empirical broad-spectrum anti-infective therapy consisting of intravenous meropenem (1 g every 8 h, February 21-March 18, 2024) combined with intravenous omadacycline tosylate (0.1 g once daily, February 22-23, 2024). Imaging revealed that the liver abscess had not liquefied, making percutaneous drainage unfeasible. Pericardial

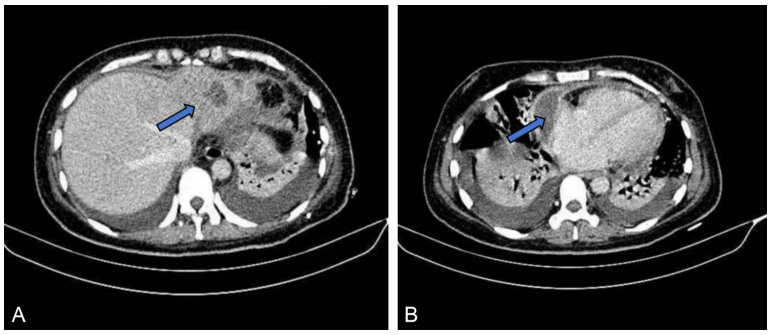


**Figure 2.** Follow-up contrast-enhanced CT images after treatment. A. Axial CT scan demonstrating a significant reduction in the size of the hepatic abscess (blue arrow) compared with the previous scan. B. Axial CT scan showing a marked reduction in pericardial effusion (blue arrow) compared to the previous examination.

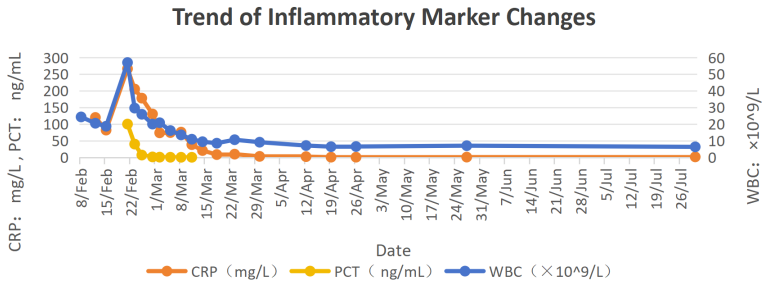


**Figure 3.** Trends in inflammatory marker changes.

drainage was continued, yielding a total of 220 ml of yellow purulent fluid. Metagenomic next-generation sequencing (mNGS) of the pericardial fluid identified *Bacteroides thetaiotaomicron* (383 sequence reads, with a relative abundance of 45.11%). Based on these findings, omadacycline was discontinued, and intravenous ornidazole (0.5 g every 12 hours, 2024.2.23-2024.3.15) was added to enhance anti-anaerobic bacterial therapy. Thymalfasin was administered as adjunctive immunomodulatory therapy.

Following treatment, the pericardial effusion decreased, and the patient's chest tightness improved, allowing removal of the pericardial drainage tube on February 29, 2024. On March 4, 2024, the patient developed fever (37.3°C to 37.9°C), accompanied by fatigue and exertional chest tightness. Chest ultrasound revealed a left-sided pleural effusion measuring 10.40 cm. A pleural effusion drainage was performed, and pleural fluid analysis revealed increased nucleated cells. Symptomatic treatment, including diuretics, was provided. A total of 1540 ml of pale-yellow fluid with flocculent material was drained. Follow-up CT on March 7, 2025, and ultrasound on March 11, 2024, confirmed significant reduction of pleural effusion, and the chest drainage tube was removed (**Figure 2A**, **2B**). During hospitalization, blood glucose levels remained within the normal range. The patient adhered to a strict diabetic diet, which maintained adequate glucose control. Owing to the transient nature of hyperglycemia at admission and absence of persistent abnormalities, no further HbA1c testing was performed.

After anti-infective treatment, follow-up ultrasound showed that the liver abscess had shrunk to 4.69 × 3.96 cm. Given the incomplete liquefaction, satisfactory infection control with antibiotics, and the patient's reluctance to undergo invasive procedures for physical reasons, percutaneous drainage was not performed. The pericardial effusion had also significantly decreased, and inflammatory markers had mostly returned to normal. The patient was discharged on March 18, 2024, with instructions to continue oral faropenem and ornidazole. Inflammatory markers (WBC, PCT) were regularly monitored and demonstrated a steady decline, with normalization by March 30, 2024. At that time, the liver abscess had further decreased to 2.16 × 2.0 cm. Since the lesion persisted, antimicrobial therapy was extended until complete resolution was confirmed. Antibiotics were discontinued after completing an 8-week course. Follow-up evaluations on April 26, May 27, and July 30, 2024 revealed normal inflammatory marker levels (**Figure 3**), with near-complete resolution of both the pericardial effusion and the liver abscess. The timeline of treatment and medication is shown in **Figure 4**.

## Discussion

Pyogenic liver abscess (PLA) is characterized by bacterial invasion of the liver, leading to inflammation, liquefaction, and necrosis of

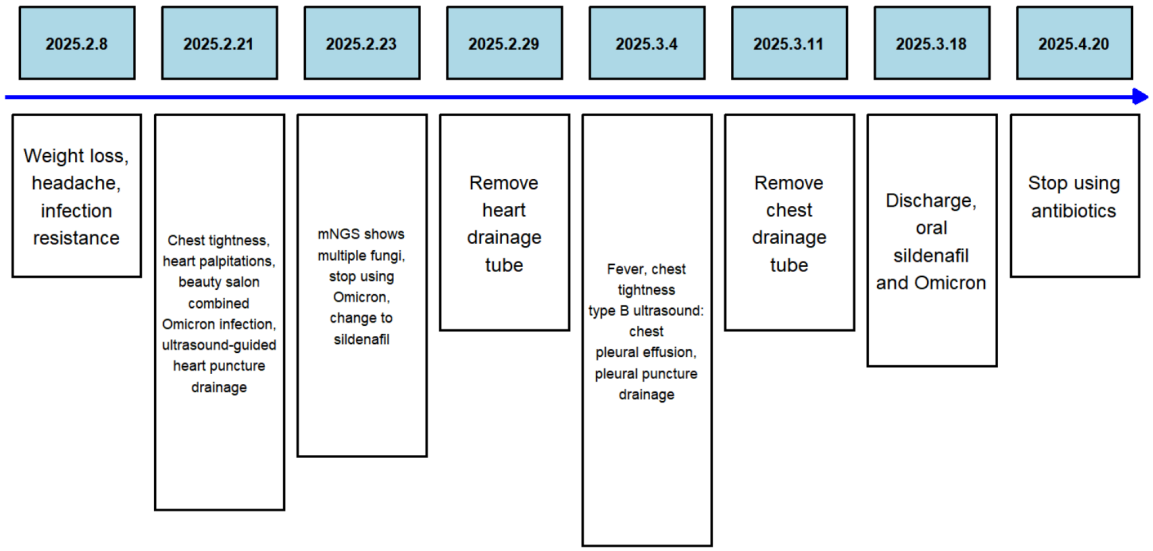


**Figure 4.** Treatment and medication timeline.

hepatic tissue, ultimately resulting in the formation of a purulent cavity. The common sources of PLA include intra-abdominal infections, biliary tract infections, hematogenous infections, and trauma or surgery. Additionally, there have been reports of foreign body-induced liver abscesses, such as those caused by fish bones or toothpicks penetrating the digestive tract, resulting in liver abscesses [1, 2]. Risk factors for PLA include diabetes, biliary diseases, malignancies, and liver cirrhosis [3]. Chronic anemia may impair both innate and adaptive immune cell functions, thereby predisposing individuals to pyogenic infections. While this mechanism could have contributed to the patient's vulnerability, causality cannot be established in this single case because complete iron studies and etiologic evaluation were not performed at admission. Furthermore, infection-related hyperglycemia can occur during acute illness, and persistent hyperglycemia may impair immune function, possibly contributing to immunocompromise. In this case, the elevated blood glucose on admission was likely secondary to infection rather than undiagnosed diabetes, as levels normalized with strict dietary control. However, transient hyperglycemia during infections can still exacerbate disease severity by impairing host immune responses.

PLA has a rapid and severe onset, and if not diagnosed and treated promptly, the condition may further deteriorate, potentially leading to invasive syndromes with high mortality and recurrence rates. With the widespread use of techniques such as percutaneous liver abscess drainage, the mortality rate of PLA has significantly decreased. However, in recent years, the incidence of PLA has shown a rising trend, which may be associated with an aging population, an increased incidence of diabetes, and a higher incidence of hepatobiliary system diseases [4].

The most common pathogen causing PLA is *Klebsiella pneumoniae*, followed by *Escherichia coli* and other gram-negative bacilli, gram-positive cocci, and anaerobic bacteria [5, 6]. Pathogens vary depending on the source of infection and are also influenced by underlying conditions such as diabetes or biliary system diseases. In patients with impaired immunity or disruption of the intestinal mucosal barrier, *Klebsiella pneumoniae*, a common intestinal colonizer, can invade the liver through the portal vein system, leading to the formation of a liver abscess. In cases of biliary tract stenosis or obstruction, bile stasis and increased intrabiliary pressure facilitate bacterial overgrowth and retrograde migration into the liver, ultimately leading to abscess formation.

*Bacteroides fragilis* is the most common anaerobic bacterium causing liver abscesses. Numerous reports have described cases of *Bacteroides fragilis*-associated PLA in the settings of gastrointestinal infections, post-appen-

dectomy, and cholangitis, with the portal venous route considered the most likely pathway of infection [7-9]. Like *B.fragilis*, *B.thetaiotaomicron* is a gram-negative, obligate anaerobe and one of the most prevalent symbiotic bacteria in the human gut. When the intestinal mucosal barrier is compromised, *B.thetaiotaomicron* can act as an opportunistic pathogen, producing adhesion factors and toxins that promote tissue invasion and dissemination. In a microbiological study of 103 PLA cases, only one was attributed to *B.thetaiotaomicron* [10]. In this case, mNGS of pericardial effusion identified *B.thetaiotaomicron*. Although infections caused by Bacteroides typically originate from intestinal barrier damage (e.g., perforation, biliary tract disease), abdominal CT and gastrointestinal evaluation did not reveal an obvious infection source. A gastrointestinal origin remains the most plausible explanation, but this hypothesis cannot be confirmed in the absence of direct evidence. Furthermore, the patient's transient hyperglycemia likely contributed to impaired immunity defenses, as elevated glucose levels can suppress neutrophil chemotaxis and phagocytic function, thereby facilitating bacterial proliferation and dissemination. Once bloodstream invasion occurs, colonization of the liver ensues, leading to the development of PLA.

PLA typically presents with fever and hepatic region pain. Nonspecific symptoms such as chills, nausea, vomiting, anorexia, and weight loss may occur [11]. On physical examination, patients may exhibit tenderness and rebound tenderness in the right upper abdomen, and some may present with jaundice. However, a subset of patients, especially the elderly and immunocompromised (e.g., those with HIV/AIDS, receiving chemotherapy or immunosuppressive therapy, or post-organ transplantation), may not exhibit typical fever or other classic signs of infection. In this case, the patient's predominant manifestations were anorexia and weight loss, without fever. Owing to these atypical clinical symptoms, the patient did not seek medical attention promptly. By the time chest tightness and vomiting developed, inflammatory markers were already significantly increased. Therefore, recognizing these risk factors is essential when diagnosing infections in such patients, even in the absence of fever or other typical symptoms. Compared to traditional culture methods, mNGS offers faster and more comprehensive pathogen identification. Conventional cultures may take several days and may fail to detect certain organisms, whereas mNGS can identify a broader range of pathogens, including rare or unculturable organisms, within a shorter time frame. Therefore, mNGS is particularly valuable in immunocompromised patients or patients with polymicrobial infections, where rapid and accurate pathogen identification is crucial. Clinicians should consider mNGS when traditional cultures fail to identify the pathogen or when a rapid diagnosis is necessary, especially in critically ill or immunocompromised patients.

PLA may also present with extrahepatic complications, including pulmonary infections, intracranial infections, endophthalmitis, intestinal infections, and abscesses in other parts of the abdominal cavity. This case reports a rare case of *Bacteroides thetaiotaomicron*-induced PLA complicated by infectious endocarditis. Studies have shown that the development of extrahepatic infections is influenced by various factors, including abscess characteristics, host immune status, infection severity, and the timeliness and effectiveness of treatment [12].

The mechanisms underlying pyogenic pericarditis caused by liver abscess are diverse, with the primary routes being spread from subdiaphragmatic abscesses or hematogenous dissemination. Reported cases include pyogenic pericarditis after hepatic resection [13] and pyogenic pericarditis with cardiac tamponade following diaphragmatic rupture caused by a liver abscess [14]. In another report, a bacterial liver abscess due to cholangitis caused by *Bacteroides fragilis* spreads through the diaphragm, eventually resulting in pyogenic pericardial effusion and cardiac tamponade [9]. To date, there have been no reported cases of PLA caused by *Bacteroides ovatus* or *Bacteroides thetaiotaomicron*, leading to pyogenic pericarditis.

This patient presented with a large liver abscess, with contrast-enhanced abdominal CT revealing extension of the lesion in the left lateral hepatic segment beyond the liver margin. The unclear boundary between the lesion and the pericardium suggested that pyogenic pericarditis might have resulted from direct invasion of the pericardium by the liver abscess. Additionally, the patient had a compromised

immunity, severe infection, and delayed hospital admission. The initial intensity of anti-infective treatment was insufficient, leading to further progression of the infection. Inflammatory markers significantly increased, and the patient's PCT levels exceeded the upper limit of detection, indicating possible bacteremia with bacterial dissemination to the pericardium, causing pyogenic pericarditis. Therefore, both direct invasion and hematogenous dissemination were plausible mechanisms in this case, though the imaging findings favored direct invasion. mNGS of pericardial pus identified *B.thetaiotaomicron*, while both blood and abscess fluid cultures were negative. The negative culture results may be explained by the collection of blood samples after antibiotic administration and the generally low positivity rate of anaerobic cultures. Consequently, hematogenous dissemination could not be completely ruled out.

Empirical treatment for PLA primarily targets gram-negative bacilli and anaerobes. Before susceptibility results are obtained, antibiotics are empirically chosen on the basis of the infection source and common pathogens. Commonly recommended regimens include β-lactams, nitroimidazoles, and fluoroquinolones. Percutaneous drainage guided by ultrasound or CT, combined with antimicrobial therapy, is the first-line treatment for PLA, particularly for large solitary abscesses or multiple interconnected, liquefied, and mature collection. The *Expert Consensus on Diagnosis and Treatment of Bacterial Liver Abscess in Emergency Medicine* recommends antibiotic therapy for 4-6 weeks for patients receiving only antimicrobial treatment. For those with effective drainage, the duration of antibiotic therapy is recommended to be 2-4 weeks [15]. In this case, the liver abscess had not yet liquefied on initial imaging, making percutaneous drainage infeasible. However, the pericardial effusion was promptly drained, combined with effective antimicrobial treatment. Given the persistence of the liver abscess despite treatment, the antibiotic course was extended to 8 weeks, beyond the consensus-recommended duration. This decision was made to ensure complete infection control and to minimize the risk of incomplete resolution in such complex infections.

Notably, β-lactam/enzyme inhibitor combinations, carbapenems, and nitroimidazoles all possess anti-anaerobic properties. In most cases, combination therapy is unnecessary, as studies have shown no improvement in clinical efficacy against anaerobes with combined regimens. Moreover, overuse of antianaerobic agents can even induce the production of degrading enzymes or modifications of target sites, leading to bacterial resistance [16, 17]. The *Emergency Expert Consensus on the Diagnosis and Treatment of Pyogenic Liver Abscess* recommends the use of carbapenems or β-lactam/enzyme inhibitor combinations, or third- or fourth-generation cephalosporins plus metronidazole, for severe infections, followed by de-escalation once symptoms improve [15]. Retrospective analyses of broad-spectrum antimicrobial agents combined with nitroimidazole in the treatment of obligate anaerobic intra-abdominal bacterial infections have demonstrated no significant difference in clinical outcomes compared to broad-spectrum monotherapy. However, owing to the complexity of intra-abdominal infections, direct comparisons remain challenging [18].

Emerging evidence also indicates that anaerobes are gradually developing resistance to nitroimidazole drugs, and resistance to carbapenems has been reported [19]. Therefore, in critically ill patients where antimicrobial susceptibility results are unclear, initial combination therapy may still be justified. In our case, the patient presented with significantly elevated inflammatory markers, multiple organ involvement, and poor general condition, consistent with severe infection. Meropenem was initiated as empirical therapy. mNGS of the pericardial effusion confirmed anaerobic bacteria, prompting the addition of a nitroimidazole for enhanced anaerobic coverage. The infection was ultimately controlled, and carbapenems were combined with a nitroimidazole throughout the treatment course. Whether combination therapy should be continued after infection control in such patients remains a matter for discussion. Based on this experience, we suggest that in critically ill patients with an identified pathogen, de-escalation to monotherapy could be considered after infection control.

## Conclusion

This rare case of *Bacteroides thetaiotaomicron*-induced pyogenic liver abscess complicated by purulent pericarditis highlights the diagnostic challenges posed by atypical presentations in immunocompromised patients, who

may lack fever or other classic signs of infection. Effective management of PLA with extrahepatic complications requires timely recognition, sustained and potent antimicrobial therapy, and prompt drainage to reduce the risk of sequelae such as constrictive pericarditis. Routine laboratory, biochemical, and microbiological analyses of drainage fluid are essential for pathogen identification. Moreover, in severe or refractory infections, mNGS offers rapid and comprehensive pathogen detection, enabling more precise therapy, shortening hospitalization, with an improved outcome.

## Disclosure of conflict of interest

None.

## Abbreviations

CT, Computed tomography; mNGS, Metagenomic next-generation sequencing; WBC, White blood cell; CRP, C-reactive protein; BNP, Brain natriuretic peptide; PCT, procalcitonin.

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