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Heart failure misdiagnosed as acute cholecystitis: a case report

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Abstract

Background Heart failure is a clinical syndrome characterized by decreased cardiac output, leading to systemic organ hypoxia and resulting in dyspnea, pulmonary edema, organ congestion, and pleural effusion. Owing to the diverse clinical manifestations of heart failure, early diagnosis can be challenging, and misdiagnosis may occur occasionally. The use of echocardiography and blood brain natriuretic peptide can aid in obtaining a more accurate diagnosis.

Case presentation This article presents two case reports of patients who were misdiagnosed with acute cholecystitis. Both patients were young Mongolia males (age 26 and 39 years) who presented to the emergency department with acute upper abdominal pain, abdominal ultrasound revealed gallbladder enlargement, and blood tests suggested mild elevation of bilirubin levels. However, despite the absence of procalcitonin and C-reactive protein elevation, the patients were admitted to the general surgical department with a diagnosis of "acute cholecystitis." Both patients were given treatment for cholecystitis, but their vital signs did not improve, while later examinations confirmed heart failure. After treatment with diuretics and cardiac glycosides, both patients' symptoms were relieved.

Conclusion We aim to highlight the clinical manifestations of heart failure and differentiate it from rare conditions such as acute cholecystitis. Physicians should make accurate diagnoses on the basis of physical examinations, laboratory testing and imaging, and surveys while avoiding diagnostic heuristics or mindsets. By sharing these two case reports, we hope to increase awareness to prevent potential complications and improve patient outcomes.

Keywords Heart failure, Acute cholecystitis, Misdiagnosis, Case report, Diagnostic heuristics

Introduction

Heart failure (HF) is a clinical syndrome that often results from structural or functional abnormalities of the heart, leading to reduced cardiac output and other organ congestion [1]. The diagnosis of HF was based on guidelines provided by the European Society of Cardiology [2]. In some cases of HF, multiple pleural or peritoneal effusions caused by increased venous hydrostatic pressure can be detected. Owing to the diverse clinical manifestations of HF and individual differences among patients, misdiagnosis can occur at an early stage [3], with a prevalence rate as high as 16.1% in outpatient clinics. In community hospitals, this percentage may be even greater.

Acute cholecystitis (AC) is one of the most common causes of acute abdominal pain and is characterized by fever, pain, and jaundice, but its diagnosis also depends on radiological examinations such as ultrasound (US), computed tomography (CT), or magnetic resonance imaging (MRI) scans [4, 5]. Recently, an updated version of the Tokyo AC guidelines (TG13)12 was validated in a population similar to the derivation cohort, with a sensitivity of 87.6% and specificity of 77.7% [3, 6].

In most cases, the clinical manifestations of acute cholecystitis can be easily distinguished from those of congestive heart failure with a variety of symptoms, but

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when patients have no history of hypertension or cardiovascular disease or belong to a nontypical high-risk group for HF, it is possible that the two diseases may be misdiagnosed. In this situation, if a diagnosis is missed, treatment will be delayed, leading to further deterioration of the patient's condition and requiring clinicians' attention.

Case 1

The patient was a 26-year-old Chinese male (Mongoloid) with no history of hypertension or heart disease. He presented to the emergency department complaining of upper abdominal pain for 3 days. On examination, there was tenderness and rebound tenderness in the epigastric area with Murphy's sign. The abdomen CT revealed a hyperdense gallbladder, cardiac enlargement, and fluid in the chest, pericardial cavity, and abdomen (Fig. 1). An electrocardiogram (ECG) revealed ST segment depression with T wave inversion in V1–4, leading to a prolonged QT interval. Blood tests suggested mild jaundice, and routine blood tests revealed mildly elevated leucocyte counts. Procalcitonin (pct) and C-reactive protein (CRP) levels were normal. The patient was diagnosed with acute cholecystitis, admitted to the gastrointestinal (GI) surgery department, and sent for emergency laparotomy. During the operation, the surgeons did not find any evidence of bowel perforation, gallbladder enlargement, or cholangiogram infection. While there were intra-abdominal fluid collections, edema in the upper gastrointestinal tract with abdominal fluid was observed.

After decompression of the abdominal cavity with 1000 mL of peritoneal fluid, the patient was transferred to the intensive care unit (ICU). After a serial blood test, we found that the patient's blood test did not suggest infection, while the brain natriuretic peptide (BNP) level was significantly elevated (Fig. 3), and his blood pressure decreased to 83/57 mmHg. Bedside echocardiography revealed an enlarged right atrium (47 × 53 mm), an enlarged right ventricle (36 mm), and moderate pulmonary artery hypertension (53 mmHg) with normal left ventricle output (Fig. 2). The patient

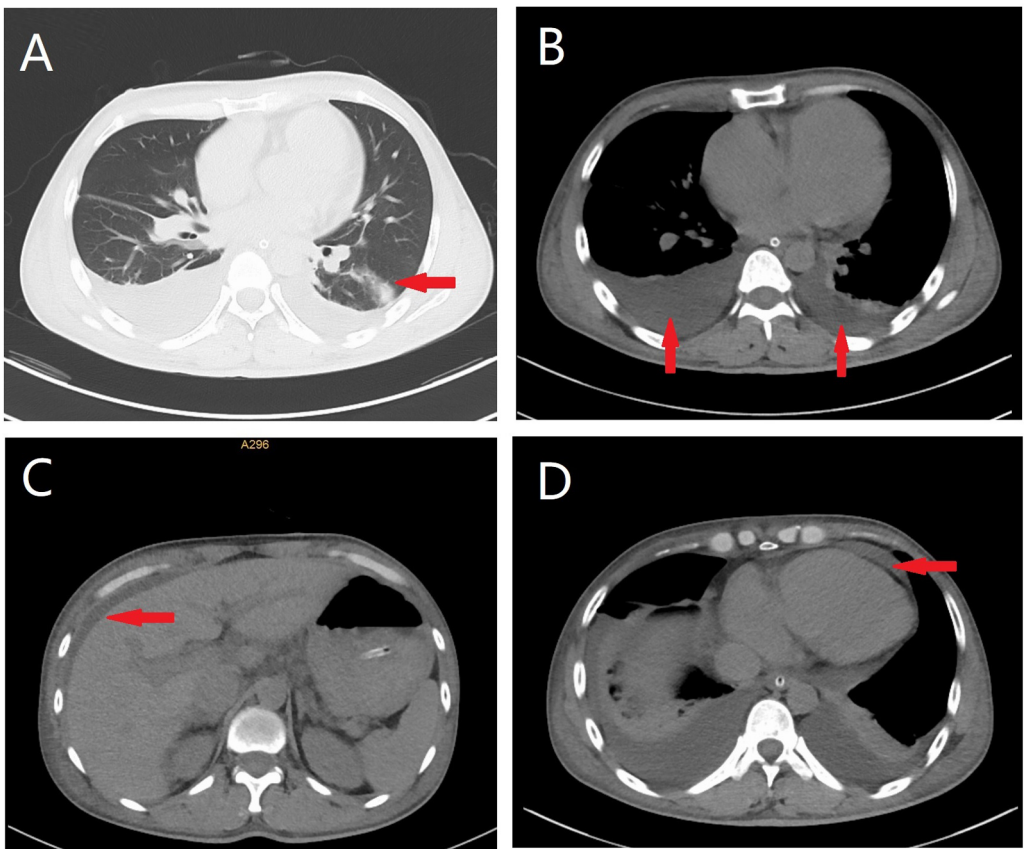

Fig. 1 **A** Chest computed tomography revealed minimal pleural effusion (red arrow). **B** Bilateral thoracic computed tomography imaging demonstrated an accumulation of fluid in both hemithoraces, with the right side exhibiting greater involvement than the left (red arrows show the pleural fluid accumulation on both sides). **C** The liver periphery showed evidence of abdominal fluid accumulation (red arrow), accompanied by swelling of the gallbladder. **D** The heart demonstrated enlargement with concomitant accumulation of fluid within the pericardial sac (red arrow)

was then diagnosed with HF on day 2 after hospitalization based on blood tests, echocardiography, and worsened vital signs [7–9].

Instead of AC, upper abdomen pain might be caused by the large quantity of abdominal–pleural fluid that irritates the peripheral nerves. A CT scan of the pulmonary arteries revealed negative signs of pulmonary embolism. There was also no evidence of blood clots in the deep veins of his lower limbs on Doppler ultrasound. The patient was given strict fluid therapy and diuretics and cardiac glycosides. This led to a progressive decrease in BNP levels, and the patient's abdominal pain was relieved on day 3. Throughout the treatment, the blood tests revealed no evidence of infection (Fig. 3). He was on fluid restrictions with diuretics until discharge. After 2 weeks, the patient improved and was discharged with a description of diuretics. At the 3-month follow-up, the patient had no further episodes of abdominal pain.

Case 2

A 39-year-old Chinese male (Mongoloid race) presented to the emergency department with upper abdominal pain that radiated to the shoulder and back for 3 days, accompanied by nausea and vomiting, with no obvious jaundice or fever. The patient had undergone surgery for congenital mitral stenosis 10 years prior and had been prescribed warfarin daily. Physical examination in the emergency department revealed mild tenderness in the upper abdomen, suspicious Murphy's sign, a heart rate of 110–120 beats/minute, and a blood pressure of 85/60 mmHg. A CT scan revealed pericholedochal effusion (Fig. 4). After a diagnosis of AC, the patient was admitted to the gastrointestinal (GI) surgery department for further treatment. At admission, routine blood tests revealed mildly elevated leucocyte counts and a normal neutrophil percentage of 63.2% (Fig. 5). The bilirubin level was elevated by 41.3 μmol/L (normal range 3–22 μmol/L), and the indirect bilirubin level was 34.6 μmol/L (normal range 0–19 μmol/L). The GI doctors prescribed ceftriaxone for infection, meperidine for abdominal pain, and fluid resuscitation therapy for hypotension. Seven hours after

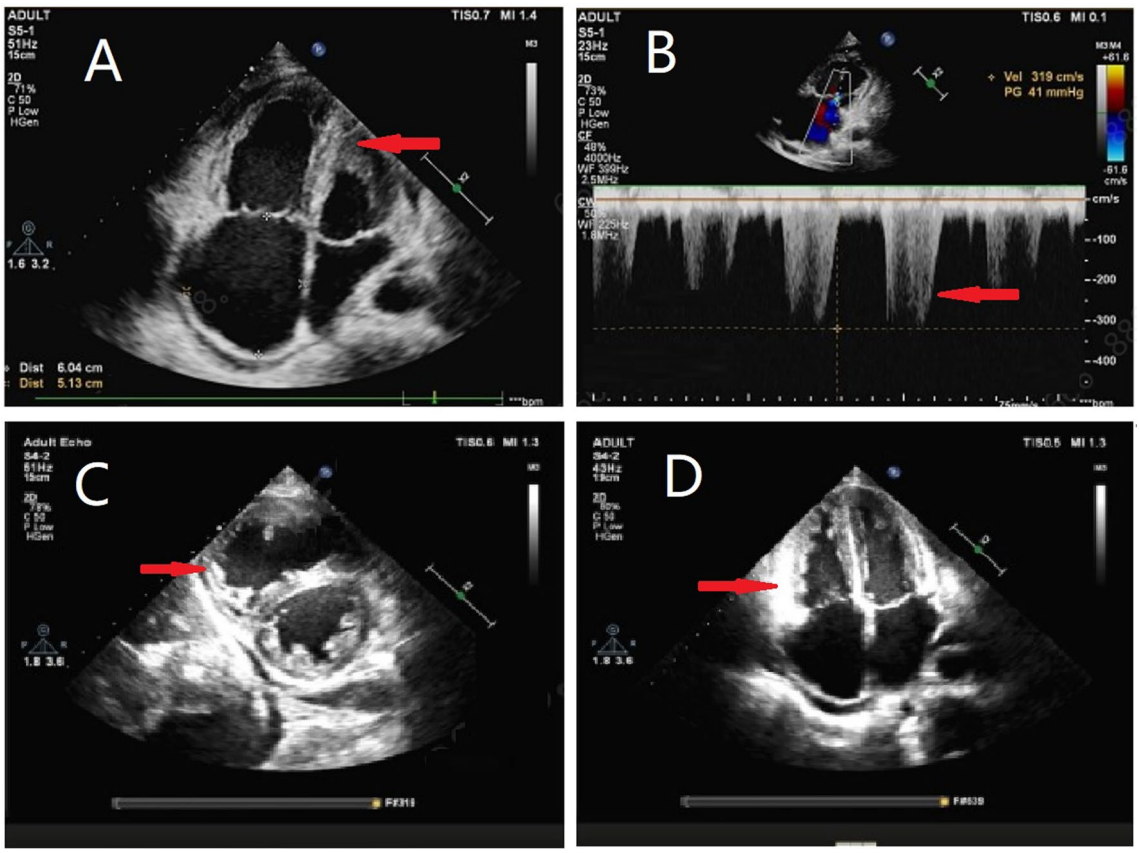

Fig. 2 **A** Dilation of the right atrium and right ventricular (red arrow). **B** Pulmonary artery hypertension and severe tricuspid regurgitation (red arrow shows elevated speed of tricuspid regurgitation). **C**, **D** The right ventricular wall showed remarkable thickening (red arrow)

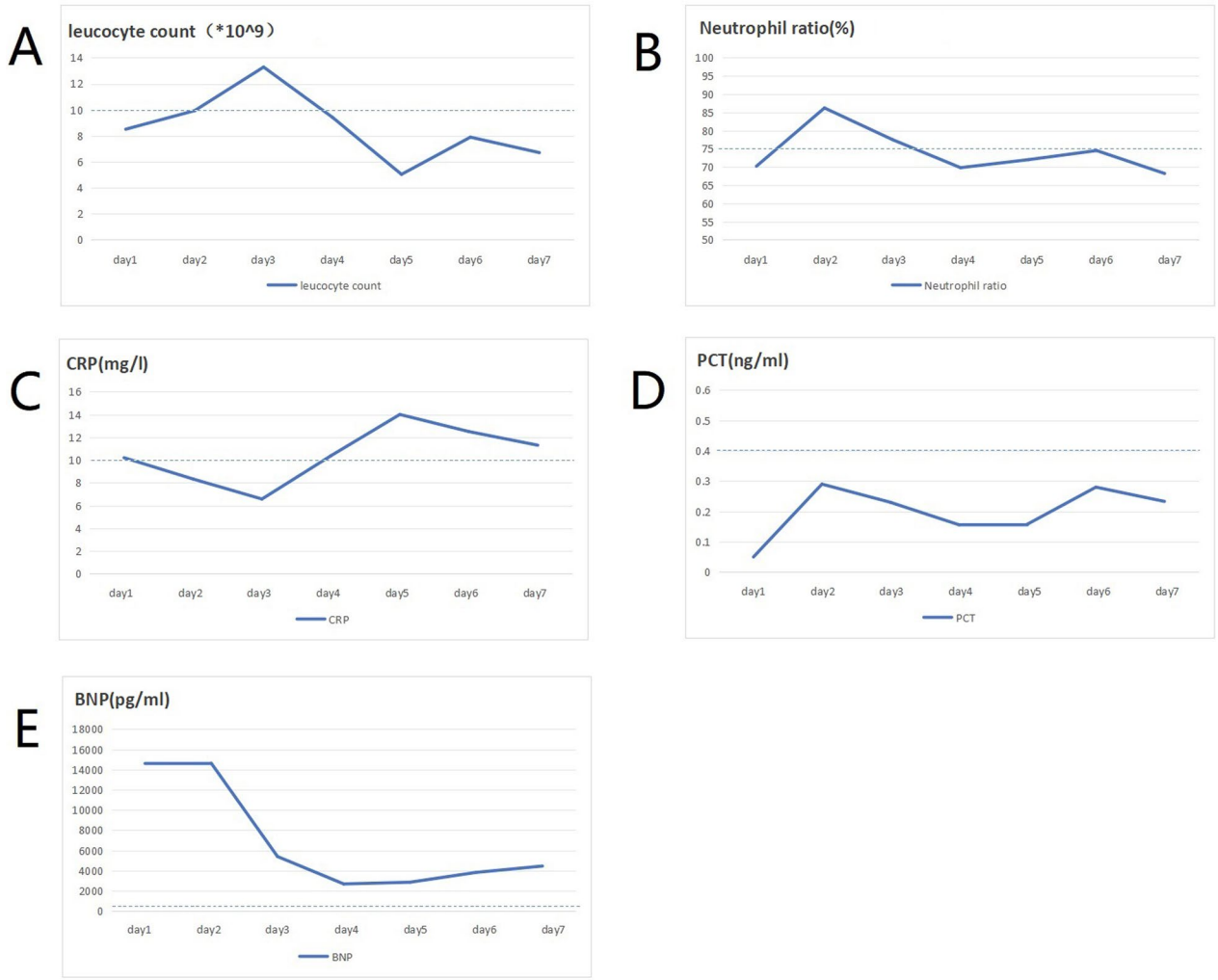

Fig. 3 Following hospitalization, serial blood samples were obtained over a period of 5 days to facilitate the monitoring of infection-related biomarkers and cardiac function indices. The normal ranges for each parameter are denoted by blue dashed lines. **A** The patient's white blood cell count surpassed the normal range only on day 3. **B** The neutrophil ratio exhibited a transient elevation above the upper limit of normal on day 2, while remaining within the normal range for the remainder of the observation period. **C** The C-reactive protein (CRP) level demonstrated a relatively stable profile, with only minor elevations observed on days 1, 5, and 7. **D** The patient's pct levels remained within the normal range throughout the 7 day observation period. **E** The BNP level exhibited an initial elevation following hospitalization on days 1 and 2, which subsequently decreased in response to treatment, yet still exceeded the normal range

admission, the patient developed tachycardia, with a heart rate of 250–280 beats/minute, and an electrocardiogram revealed atrial flutter. After the administration of verapamil, the patient's blood pressure decreased to 70/50 mmHg, with bilateral pulmonary wet rales. The patient became unconscious and was given invasive mechanical ventilation before being transferred to the intensive care unit (ICU). Moreover, the blood tests revealed significantly elevated BNP levels, with values of 12,200 pg/mL. Echocardiography suggested mitral stenosis with incomplete closure, and left ventricular output was compromised (Fig. 6). On the basis of the patient's vital signs, pneumonary edema, BNP level and echocardiography results, he was soon diagnosed with HF [7–9] on day 2.

The patient received comprehensive medical therapy, including strict fluid therapy, digoxin, diuretics, and antibiotics. Throughout the treatment, the leucocyte count increased, but the neutrophil percentage and pct and CRP levels remained within normal ranges, and the BNP level decreased significantly after antiheart failure therapy (Fig. 5). The patient's abdominal pain and nausea was relieved on day 5. In light of the presence of structural heart disease, the

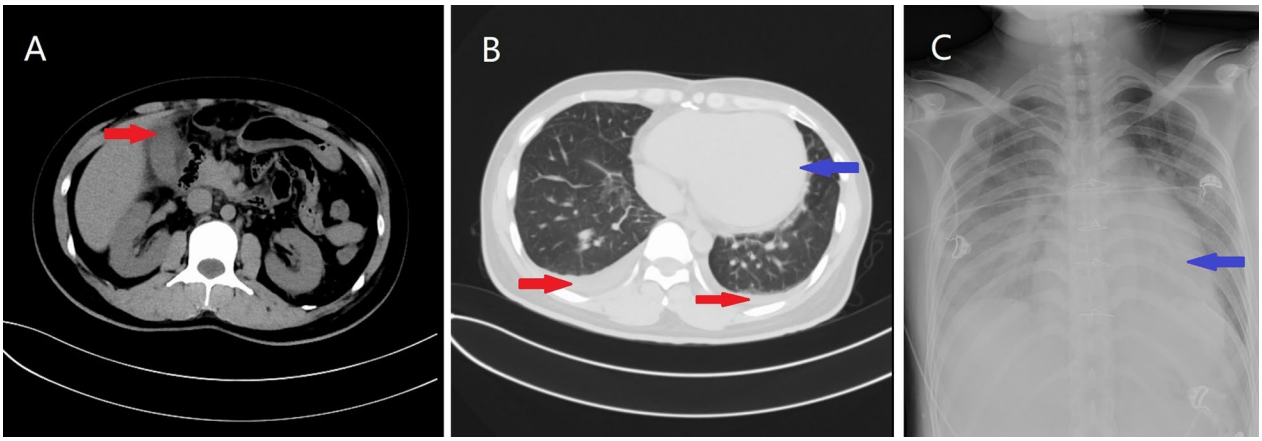

Fig. 4 **A** Abdominal computed tomography imaging revealed pericholedochal effusion and thickening wall of the gallbladder (red arrow). **B**, **C** Cardiac enlargement (blue arrows), accompanied by bilateral minimal pleural effusion (red arrows)

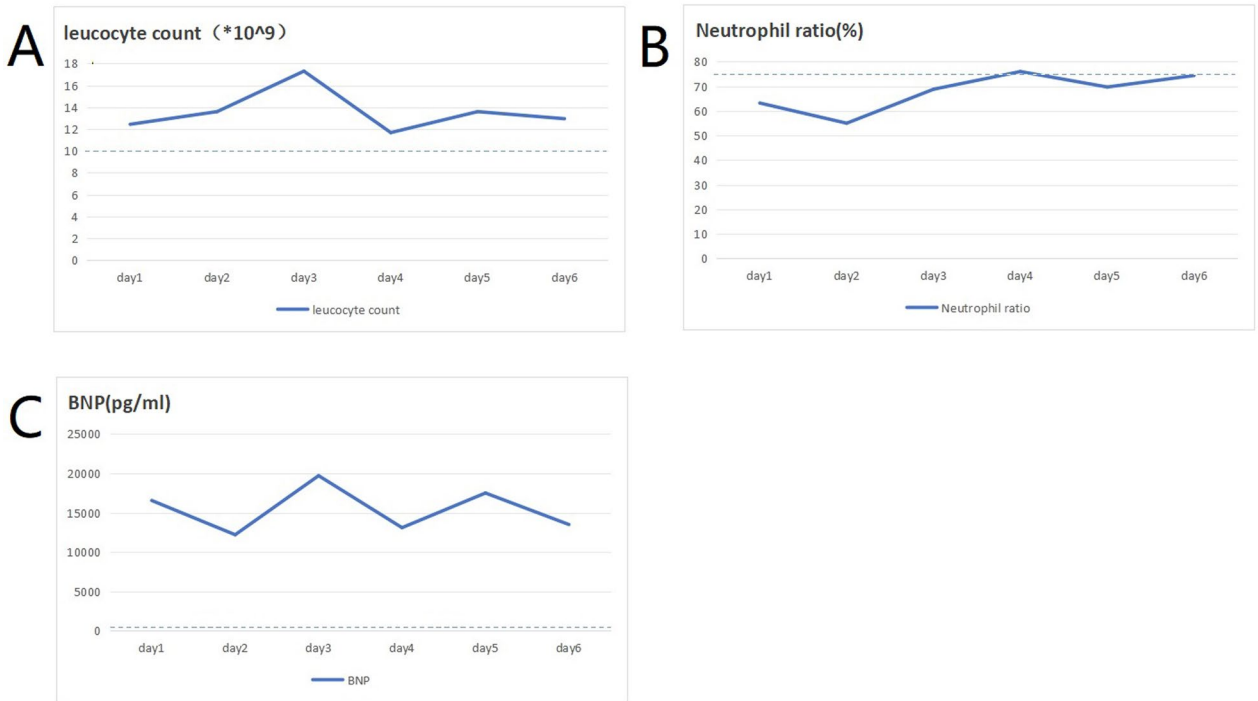

Fig. 5 The patient underwent daily blood tests, the blue dotted line represents the upper limit of normal values for each indicator. **A** The patient's white leucocyte count exceeded the normal range. **B** The neutrophil percentage remained normal. **C** The patient's brain natriuretic peptide level was elevated significantly, yet still exhibited fluctuations after anti-heart failure treatment

patient was deemed a candidate for cardiac surgery or even heart transplantation, and he was transferred to a major cardiology hospital on day 7. At the 3-month follow-up, the patient did not experience another episode of abdominal pain.

Discussion

Heart failure can be misdiagnosed as various other diseases, such as Acute exacerbation of chronic obstructive pulmonary disease (AECOPD) [7], asthma, and pneumonia [9]. However, cases of misdiagnosis as acute

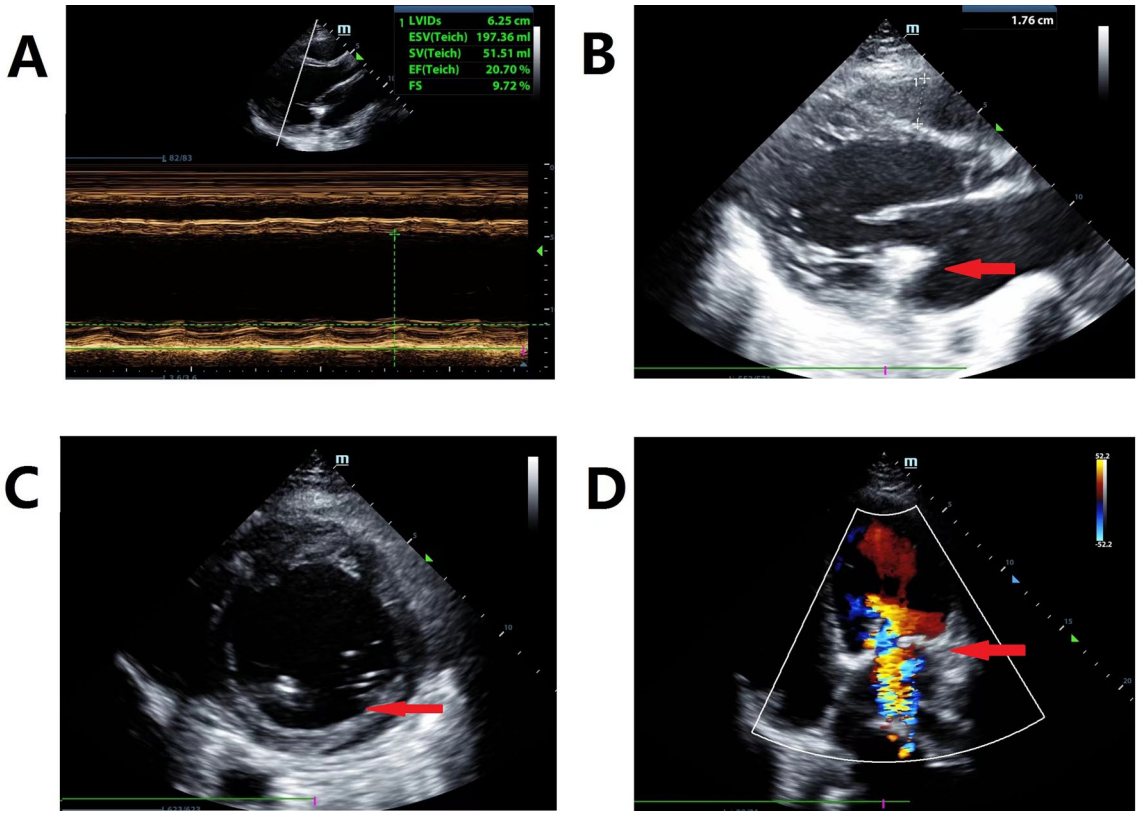

Fig. 6 The echocardiography was performed on day 2 after admission. **A** The left ventricular ejection fraction decreased to 25%, with left ventricular and atrial enlargement. **B** The density of mitral was increased (red arrow), indicating previous mitral valve repair or replacement surgery. **C** Enlarged left ventricle with an increase in the number of endocardial papillary muscles (red arrow). **D** Mitral stenosis with incomplete closure (red arrow) was present

cholecystitis are relatively rare. In the cases we presented here, both patients were young men with no history of hypertension or coronary heart disease and did not meet the high-risk group standard for acute heart failure. At admission, both patients presented with upper abdominal pain, and CT scans revealed edema in the gallbladder wall, with blood tests suggesting mildly elevated bilirubin levels. Therefore, the diagnosis of heart failure was overlooked.

Patient 1 presented with abdominal pain, elevated bilirubin levels, and multilocular pericardial effusion. However, the patient's leucocyte count, CRP level, and pct did not significantly increase, which is typical of general intra-abdominal infection. The surgeon also failed to perform a comprehensive examination, including BNP tests and echocardiography, in the presence of pericardial effusion. Therefore, the diagnosis of heart failure was delayed. After ICU admission, the patient's symptoms improved after reducing fluid overload and improving right ventricular function. However, echocardiograms on follow-up still revealed lung hypertension and tricuspid regurgitation, suggesting that the patient might have primary pulmonary hypertension leading to increased right ventricular load, resulting in tricuspid ring dilation and a decrease in right ventricular output and multiple noninfectious pericardial effusions [9, 10]. Multilocular pericardial effusion can also lead to symptoms of abdominal stimulation, while increasing static water pressure may cause stomach and gallbladder wall swelling, similar to acute abdomen symptoms, which ultimately leads to misdiagnosis as acute cholecystitis.

Patient 2 had a history of structural heart disease and had undergone mitral valve replacement surgery. He was admitted to the GI department despite his cardiac history, but the possibility that the abdominal pain was caused by cardiac disease was not taken into consideration. The GI doctors suspected that the patient's hypotension was caused by septic shock and provided fluid resuscitation with antibiotics, which further increased the left ventricular load and led to worsening cardiac output and acute heart failure during the process. During his treatment, the patient did not exhibit fever, and his blood tests did not support infection; therefore, this episode of abdominal pain and shock was more likely caused

by a worsening of structural heart disease, leading to HF rather than infection.

Abdominal pain is one of the most common chief complaints in the emergency department (ED), particularly at night, and patients with abdominal complaints are at the highest risk for cognitive errors in diagnosis [11]. The differential diagnosis includes acute cholecystitis, acute pancreatitis, acute appendicitis, and even vascular occlusion. Therefore, a comprehensive examination, imaging studies, and laboratory tests are often necessary. In clinical practice, CT scans have been widely used for the diagnosis of suspected cholecystitis. The main manifestations of CT include edema in the gallbladder wall, gallbladder enlargement, and sometimes gallstones. However, relying solely on imaging findings may lead to misdiagnosis, emphasizing the importance of combining symptoms and blood tests in the diagnostic process. Chacot's triad includes abdominal pain, fever, and jaundice, whereas blood tests often suggest elevated leucocyte count, pct, or CRP. By integrating multiple aspects of information, a precise diagnosis can be made. Fatemeh Mahmoudzadeh *et al*. demonstrated that combining clinical symptoms with imaging results can improve the sensitivity of acute cholecystitis diagnosis [12].

In China, emergency departments are divided into internal emergencies and surgical emergencies, and triage is often performed by senior nurses [13], who often refer patients with abdominal disease to the surgical emergency department, where surgeons may fall into the trap of diagnostic heuristics, attributing abdominal pain to common surgical diseases and increasing the possibility of misdiagnosis. Moreover, during the night shift in the ED, physicians are sometimes forced to complete a consultation, physical examination, and diagnosis within 30 minutes; thus, the lack of complete information, high workload, and pressure might also contribute to misdiagnosis [11, 14]. Diagnostic errors in the ED are frequent worldwide [15–17], and they can cause severe ethical, economic, or even legal problems. To minimize the chance of misdiagnosis, the first step is awareness of diagnostic error, which motivates physicians to implement preventive and corrective measures. Second, relevant information from patients and their families is crucial for understanding the progression of disease. Third, clinical decision support systems, which provide digital images, blood test information, consults, and second opinions from experts, allow physicians to obtain objective information to prevent clinical errors. Finally, reducing the clinical workload, clearing responsibilities and administrative duties can not only reduce the possibility of imprudent decisions but also protect medical staff from clinical burnout, which contributes to more efficient diagnoses [18–20].

Conclusion

For patients with suspected heart failure symptoms, performing objective examinations, such as echocardiography, BNP testing [21], and myocardial enzyme analysis, in the early stages is essential [22]. Echocardiography can quickly and directly detect structural heart disease or whether cardiac output is compromised [23]. Early diagnosis and standardized treatment of heart failure are related to increased survival rates and hospitalization times. By sharing these two cases, we recommend that physicians carry out comprehensive body examinations, blood tests, CT scans, and/or ultrasounds to make accurate diagnoses and avoid diagnostic heuristics. Night emergency physicians' and surgeons' burnout syndrome may also increase the risk of misdiagnosis, so it is even more important to pay attention to nontypical patients' differential diagnosis.

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Author contributions

YQ: conceptualization, writing, original draft preparation, and language review and editing. LW: data analysis, figure editing, and language review.

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Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

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Competing interests

The authors declare that they have no competing interests.

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