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【 CASE REPORT 】

Constrictive Pericarditis with Cardiac Ascites Caused Spontaneous Bacterial Peritonitis

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Abstract:
Patients with constrictive pericarditis (CP) typically present with symptoms related to right-sided heart failure, such as cardiac ascites. Spontaneous bacterial peritonitis (SBP) usually arises in association with ascites secondary to hepatic cirrhosis. We herein report a rare case of CP in which SBP developed due to cardiac ascites, even in the absence of cirrhosis. In this case, pericardiectomy improved both the hemodynamics and the ascites, while therapy with diuretics alone was insufficient. It is important to consider SBP in the differential diagnosis when any abdominal symptoms or an inflammatory response is found in patients with heart failure and cardiac ascites.

Key words: constrictive pericarditis, heart failure, cardiac ascites, spontaneous bacterial peritonitis, pericardiectomy

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Introduction

Constrictive pericarditis (CP) causes ventricular diastolic dysfunction due to organic changes in the pericardium, such as fibrotic thickening, adhesions, and calcification. The most common causes of CP are idiopathic or viral, post-cardiac surgery, post radiation therapy, connective tissue disorder, postinfectious (tuberculous or purulent pericarditis), and miscellaneous causes (malignancy, trauma, drug-induced, asbestosis, sarcoidosis, uremic pericarditis) (1). CP patients frequently complain of fatigue, breathlessness, and in particular, right-sided heart failure symptoms, such as lower leg edema and ascites (2). However, in principle, pericardiectomy is the first choice of treatment for CP, with diuretics used to reduce ascites and edema while awaiting surgery.

Spontaneous bacterial peritonitis (SBP) is defined as an ascitic fluid infection in the absence of a surgically treatable intra-abdominal source (3). It is a severe complication in cirrhotic patients with ascites (4).

We herein report a case of CP in which SBP developed due to cardiac ascites even in the absence of cirrhosis. In this case, diuretic therapy alone was insufficient but performing both pericardiectomy and the waffle procedure was able to improve the hemodynamics and eliminate ascites.

Case Report

A 50-year-old man without a history of any appreciable disease, presented with exertional breathlessness and a sense of abdominal fullness for the past 4 months. Chest X-ray and computed tomography (CT) showed pericardial calcification on the front side of the right ventricle (RV) (Fig. 1). Furthermore, abdominal CT showed ascites retention (Fig. 2A). The patient was hospitalized due to suspected heart failure associated with CP.

At admission, a physical examination showed mild bilateral lower leg edema without any other notable findings, and the intravenous injection of furosemide was started. Blood tests showed that the level of C-reactive protein (CRP) slightly increased to 1.71 mg/dL. No findings suggested autoimmune disease, the tumor marker levels were within the normal range, and the interferon gamma release assay in the diagnosis of *Mycobacterium tuberculosis* (the T-SPOT®.

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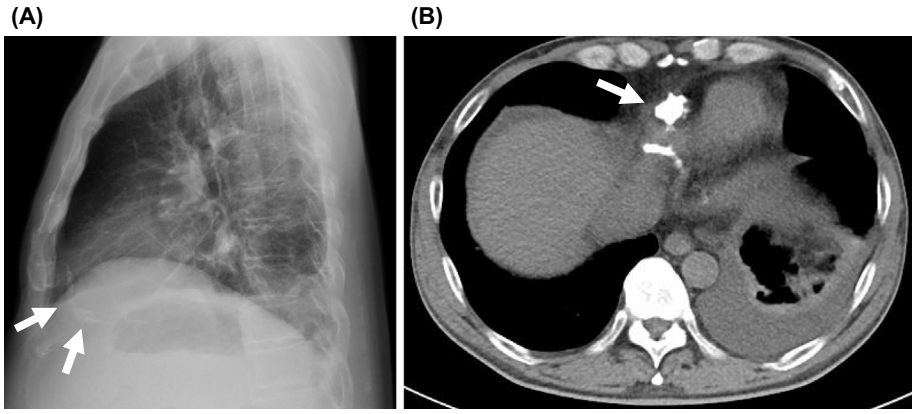

Figure 1. **Chest X-ray (A) and CT (B) showed pericardial calcification on the front side of the RV (white arrows). CT: computed tomography, RV: right ventricle**

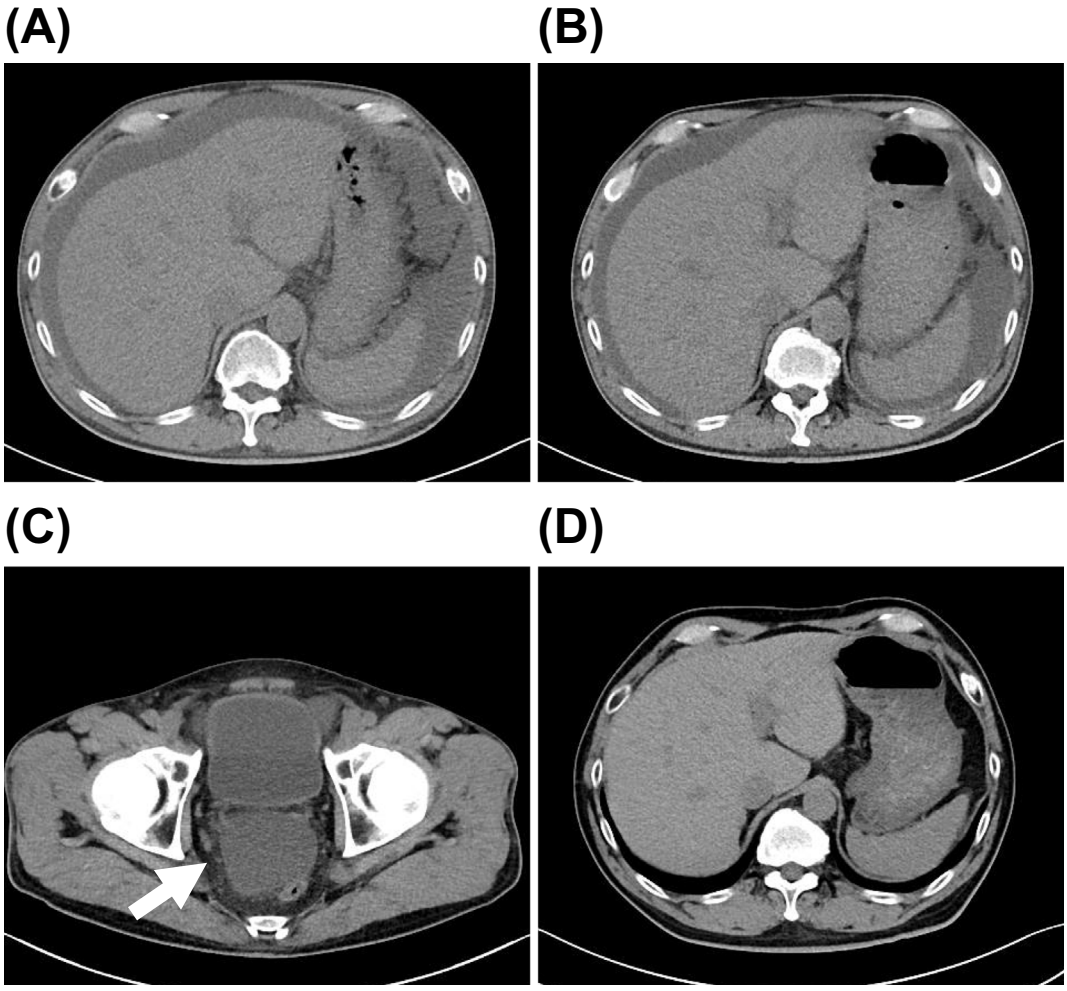

Figure 2. **At the first hospital admission, abdominal CT showed the retention of ascites (A). At two weeks after being discharged from the first hospitalization, cardiac ascites was slightly reduced owing to diuretic therapy, but the ascites seen in the pelvic floor area were exudative (white arrow) (B, C). At eight months after the surgical operation, cardiac ascites was eliminated (D). CT: computed tomography**

TB test) was negative. Transthoracic echocardiography (TTE) showed a normal cardiac function and we also detected septal bounce with abrupt transient rightward movement of the interventricular septum with expiration, an increased pericardial thickness (Fig. 3A, B), and a dilated inferior vena cava with diminished inspiratory collapse (Fig. 3C, D). Pulsed-wave Doppler echocardiography showed an 18% inspiratory decrease of the mitral E-wave

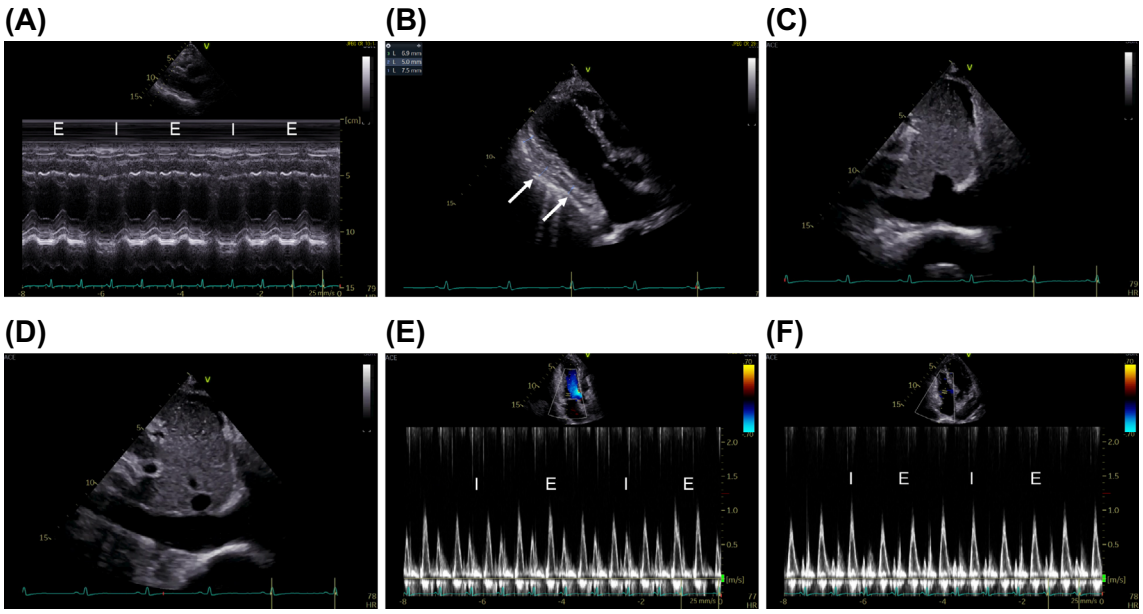

Figure 3. **TTE detected an abrupt transient rightward movement of the interventricular septum with expiration (septal bounce) (I: inspiration, E: expiration) (A), increased pericardial thickness (B), and a dilated inferior vena cava with diminished inspiratory collapse (C, D) (C: at the time of inspiration) (D: at the time of expiration). Pulsed-wave Doppler demonstrated an 18% inspiratory decrease in the mitral inflow E-wave velocity (E), and a 28% inspiratory increase in the tricuspid inflow E-wave velocity (F). TTE: transthoracic echocardiography**

velocity, and a 28% inspiratory increase of the tricuspid E-wave velocity (Fig. 3E, F). The early diastolic Doppler tissue velocity at the mitral annulus (e') demonstrated an elevation of the medial e' velocity relative to the lateral e' velocity (medial e'/lateral e'=1.1). Cardiac catheterization detected prominent Y descents of right atrial (RA) pressure tracings and an early diastolic dip followed by a plateau of the last stage of diastole just before contraction, the so-called dip and plateau, in the RV diastolic pressure tracings (Fig. 4A, B). Based on these results, the patient was diagnosed as CP.

Eventually, the subjective symptoms improved after the oral administration of diuretics (furosemide 80 mg per day and spironolactone 25 mg per day). The patient was thereafter discharged from the hospital, and elective surgery was scheduled. In the outpatient department 2 weeks later, the patients complained of lower abdominal pain, and his CRP level had increased to 14.55 mg/dL. Abdominal CT revealed exudative ascites (Fig. 2B, C). Although a culture of the ascites was negative, neutrophils in the ascites had increased to 1,400/μL, so the patient was diagnosed with SBP.

Although some data indicated that the patients had chronic liver damage (lymphocyte count 1,000/μL, type IV collagen 225 ng/mL), there was no evidence of any cirrhosis on abdominal ultrasonography or blood sampling [aspartate aminotransferase (AST) 26 U/L, alanine aminotransferase (ALT) 24 U/L, total bilirubin 0.8 mg/dL, albumin 3.8 g/dL, and platelet count 255×10^3/μL]. The patient had no history of an immunocompromised status (e.g., human immunodeficiency virus infection) and was not taking any immunosuppressive drugs. The patient was readmitted to the hospital, and antibiotic treatment was started. Upper and lower gastrointestinal endoscopy showed no major abnormalities (e.g., cancer or ulcer). After the peritonitis improved, pericardiectomy and the waffle procedure were subsequently performed without any major complications. The duration of the surgical procedure was 220 minutes, and the total amount of bleeding during surgery was 255 mL.

The patient left the intensive-care unit (ICU) the day after surgery and thereafter demonstrated an uneventful postoperative course. On postoperative cardiac catheterization (12 days after surgery), "the prominent Y descents" of RA pressure tracings and "dip and plateau" in the RV diastolic pressure tracings had disappeared (Fig. 4C, D). The patient was discharged from the hospital at 16 days after surgery. Further, CT performed eight months after surgery show the ascites to have disappeared without any recurrence of symptoms (Fig. 2D).

Discussion

We herein described a case of CP in which SBP developed due to cardiac ascites, even in the absence of hepatic cirrhosis. It is important to consider SBP in the differential diagnosis when any abdominal symptoms or an inflammatory response is found in patients with heart failure and cardiac ascites.

SBP usually arises in association with ascites secondary to hepatic cirrhosis (5). Cirrhosis predisposes patients to the development of bacterial overgrowth, possibly because of in-

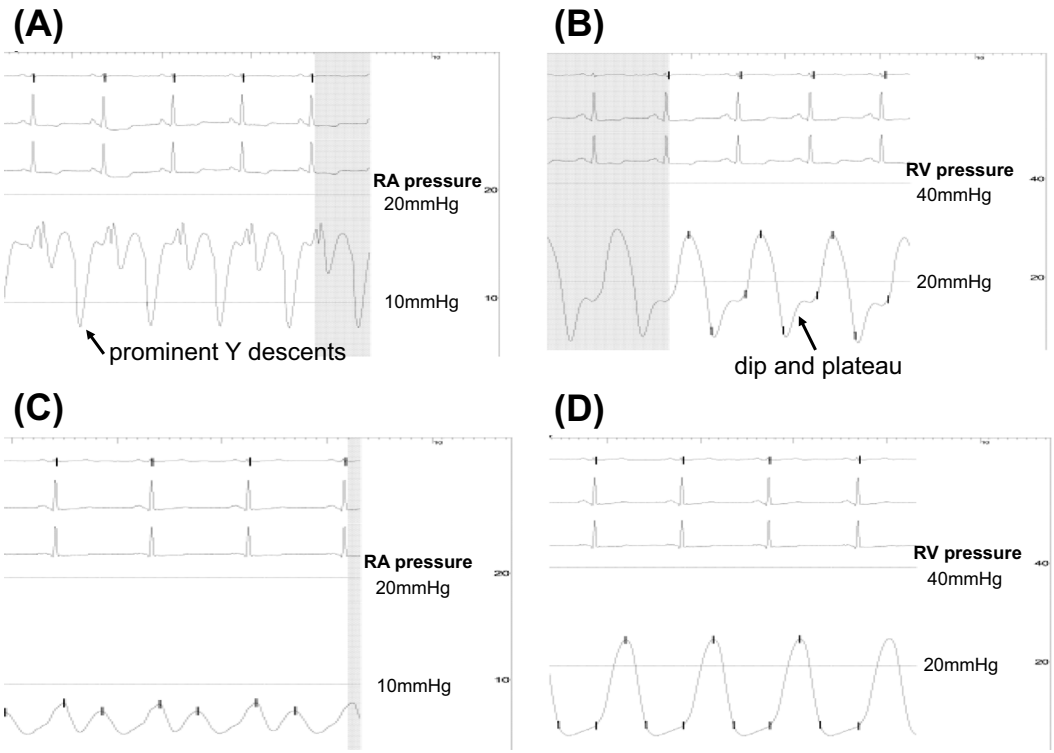

Figure 4. **Cardiac catheterization detected "prominent Y descents" of the RA pressure tracings (A) and an early diastolic dip followed by a plateau of the last stage of diastole just before contraction ("dip and plateau") in the RV diastolic pressure tracings (B). On postoperative cardiac catheterization (12 days after surgery), "prominent Y descents" in the RA pressure tracings and "dip and plateau" in RV pressure tracings disappeared (C, D). RA: right atrial, RV: right ventricle**

testinal hypomotility and local intestinal immunodeficiency (3). Furthermore, cirrhosis is associated with structural and functional alterations in the intestinal mucosa that increase the permeability and facilitates bacterial translocation, which is the most common cause of SBP (6). However, in the present case, SBP developed despite the absence of cirrhosis. Some reports have suggested that cardiogenic ascites with heart failure due to any type of heart disease (e.g., CP, ischemic heart disease, congenital heart disease, valvular heart disease, or arrhythmia) may be a possible cause of SBP, even in the absence of cirrhosis (5, 7-10). In patients with heart failure, substantial hemodynamic changes, such as hypoperfusion and congestion in the intestines, can alter the gut morphology, permeability, and function and induce the growth of microbiota that may ultimately stimulate bacterial translocation and endotoxin release (5, 11, 12). Particularly, since the risk of developing SBP increases in heart failure patients with cardiac ascites, it is important to consider SBP in the differential diagnosis when any abdominal symptoms or an inflammatory response is found in patients with heart failure and cardiac ascites.

In this case, chest X-rays and CT showed pericardial calcification on the front side of the RV. Even though there was no evidence of cirrhosis on abdominal ultrasonography or transaminases, some data (e.g., decreased lymphocyte count and elevated type IV collagen level) seemed to indicate chronic liver damage. Although subjective symptoms such as exertional breathlessness and a sense of abdominal fullness began to appear about 4 months prior to presentation, these findings of calcification and chronic liver damage suggested that the problem of right heart failure may have existed for even longer, thus leading to the development of cardiac ascites. Despite intravascular volume reduction by diuretic therapy, the ascites that leaked out to the extravascular space because of the long-term right heart failure required time to return to the intravascular space. This pathophysiology might have caused chronic intestinal damage that predisposed the patient to develop a translocation of the intestinal bacteria leading to SBP.

CP often develops due to malignant tumors, connective tissue disorders (systemic lupus erythematosus, etc.), infectious diseases (viral infection, tuberculosis, etc.), post-cardiac surgery, post-radiation therapy, and other conditions (1). Furthermore, some previous reports have shown that non-cirrhotic SBP was also caused by malignant ascites and systemic lupus erythematosus (13, 14). In the present case, there were no findings suggestive of a malignant tumor, connective tissue disorder, or infectious disease. In addition, there was no history of surgery or radiation therapy. Finally, an idiopathic cause was therefore suspected to be the etiology of pericarditis in this case.

A previous report demonstrated that, in CP cases, the mitral E-wave velocity showed an inspiratory decrease (≥25%) and the tricuspid E-wave velocity showed an inspiratory in-

crease (>40%) (15), while the respiratory variation of the ventricular inflow did not meet these criteria in this case. Previous studies also reported the absence of typical respiratory variation of mitral E-wave velocity in some CP patients, such as cases with a markedly elevated left atrial pressure, and a diagnosis of CP should therefore not be ruled out based on the absence of respiratory variation (15, 16).

Pericardiectomy is the definitive treatment option for patients with CP. Diuretic therapy is supportive and aimed at controlling the symptoms of congestion in advanced cases and when surgery is contraindicated or carries a high risk (2, 17). In the present case, diuretic therapy alleviated the symptoms before surgery, but ascites remained on CT. Pericardiectomy ultimately improved the hemodynamics and eliminated the ascites that had been observed on CT.

In conclusion, we encountered a rare case of CP in which SBP developed due to cardiac ascites, even in the absence of hepatic cirrhosis. It is important to consider SBP in the differential diagnosis when any abdominal symptoms or an inflammatory response is found in patients with heart failure and cardiac ascites.

The authors state that they have no Conflict of Interest (COI).

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