Pericarditis due to an unusual microorganism in an immunocompromised patient

İmünsuprese hastada olağan dışı mikroorganizmaya bağlı gelişen perikardit

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Summary—A 77-year-old man with a past medical history of myelodysplastic syndrome, coronary artery disease, hypertension, and chronic atrial fibrillation presented at the hematology outpatient clinic with progressive shortness of breath, weakness, and chest and back pain. Echocardiography was performed and the patient was diagnosed with severe pericardial effusion near the right ventricle. Pericardial drainage was performed. *Erysipelothrix rhusiopathiae* was isolated from the pericardial fluid. Complications of respiratory and renal failure developed during follow-up. The clinical and laboratory findings of vegetation on the tricuspid valve, pericardial effusion, and atrial fibrillation with a low heart rate suggested possible pancarditis. A multidisciplinary treatment approach with the cardiology and infectious disease departments was critical to successful management of this case.


CASE REPORT

Pericarditis refers to an inflammatory response of the pericardium. [1] The diagnosis of pericarditis can be challenging due to diverse potential etiologies and presentations. *Erysipelothrix rhusiopathiae* (*E. rhusiopathiae*) is a recognized source of infection, most often occurring in poultry and pigs. In most human cases, the disease is acquired from animals through work-related exposure. [2] Three forms of human disease have been defined: a localized cutaneous form, known as erysipeloid; a generalized cutaneous form; and a septicemic form that is often associated with endocarditis. [3–5] A multidisciplinary approach is essential for optimal management. This case report describes an immunocompromised patient who presented with pericarditis caused by this unusual microorganism and developed endocarditis in follow-up.

CASE REPORT

A 77-year-old man with a past medical history of myelodysplastic syndrome (MDS), coronary artery disease, hypertension, and chronic atrial fibrillation presented with symptoms of progressive shortness of breath, weakness, and chest and back pain. An echocardiogram performed 8 months prior demon-
strated an ejection fraction of 30% with moderate mitral and mild aortic and tricuspid regurgitation and a localized, 15-mm pericardial effusion along the lateral wall of the left ventricle. Medical therapy consisting of an oral diuretic, a calcium channel blocker, beta-blocker, and antiaggregant therapy combined with a novel oral anticoagulant had been initiated.

The physical examination upon the current presentation yielded an arterial blood pressure of 120/46 mmHg and cardiac auscultation revealed muffled S1 and S2 sounds with an irregular heart rhythm. Auscultation of the lungs revealed decreased lung sounds in the left basal portion. There was no jugular venous distention. The electrocardiogram (ECG) demonstrated atrial fibrillation with a heart rate of 47 bpm. A chest X-ray revealed left pleural effusion and an enlarged cardiac shadow. Echocardiography showed severe pericardial effusion with a diameter of 2.5 cm behind the left ventricle, 2.1 cm adjacent to the right atrium, and a 2.3 cm effusion adjacent to the right ventricular lateral wall, accompanied by signs of right ventricular collapse (Fig. 1) and respiratory variation of more than 25% in mitral and tricuspid inflow velocities. Moderate mitral regurgitation, as well as mild aortic and tricuspid regurgitation were observed. Cardiac chambers and systolic pulmonary arterial pressure were normal. The ejection fraction was mildly decreased (50%).

A complete blood count indicated mild anemia, leukopenia, and thrombocytopenia consistent with MDS. His C-reactive protein (8.56 mg/L), troponin-T (30/91 ng/mL), and N-terminal-pro-brain natriuretic peptide (3748 pg/mL) levels were elevated. The creatinine value was 1.4 mg/dL. Liver function test results were within the normal range, but the total bilirubin level was slightly elevated (2.02 mg/dL).

Although the patient was clinically stable, due to the persistence of pericardial effusion and echocardiographic tamponade features, a pericardiocentesis was selected as the next management step, but the procedure was unsuccessful and the patient was referred to surgery for fluid drainage and sampling. A pericardial window was opened, and a left pleural catheter was inserted for thoracentesis. Postoperatively, the patient developed acute respiratory failure and acidosis secondary to central hypopnea and acute renal failure. The patient was extubated the following day and transferred to the coronary intensive care unit.

The pericardial fluid was classified as exudative according to the Light criteria\(^6\) and *E. rhusiopathiae*.
was isolated from the samples. The bacteria were sensitive to ceftriaxone and therapy was initiated. The isolates were identified as *E. rhusiopathiae* using the VITEK 2 system (BioMérieux, Marcy l’Etoile, France) (Fig. 2, 3).

Echocardiographic assessment was repeated every 2 days, since *E. rhusiopathiae* has been shown to cause endocarditis in human beings. Due to deteriorating renal function, dialysis treatment was also initiated. On the fourth day, the patient’s dialysis catheter became obstructed and dialysis was terminated earlier than expected. The patient experienced a bradycardic cardiopulmonary arrest, though the potassium level was normal. He was intubated, and after 10 minutes of cardiopulmonary resuscitation, his hemodynamic status was stabilized. The control echocardiography revealed mild pericardial effusion, normal left ventricular function, depressed right ventricular function with right ventricular and atrial dilatation, a pulmonary artery systolic pressure of 55–60 mmHg and suspected vegetation on the tricuspid leaflet. Transesophageal echocardiography (TEE) revealed filamentous vegetation on the atrial side of the tricuspid valve and a thrombus-like image attached to the tip of the dialysis catheter (Fig. 4). The dialysis catheter obstruction of the previous day and the thrombus seen in the catheter lumen suggested that the etiology of the cardiopulmonary arrest might have been a pulmonary embolism originating from the catheter. It was not possible to perform a pulmonary contrast angiography since the patient was in acute kidney failure. Thrombolysis for a possible acute pulmonary embolism was also not performed because though the patient was hemodynamically stable, severe anemia and thrombocytopenia were present. Dobutamine therapy was implemented to increase the contractility of the right ventricle.

Despite ceftriaxone therapy, the patient had a persistent fever of 38°C. Therefore, his antibiotic therapy was changed to meropenem and vancomycin empirically. Multidrug resistant *Acinetobacter baumannii* (*A. baumannii*) was detected in blood cultures of pleural and pericardial fluid, and colistin was added.
to the therapy. After the addition of colistin, he became afebrile and his C-reactive protein level gradually decreased. A follow-up echocardiography and control TEE revealed that the vegetation on the tricuspid valve had regressed. Once the infection was under control, his diuresis improved and dialysis was no longer necessary.

Antibiotic treatment was continued with only meropenem and colistin. A control blood culture was negative. Those two antibiotics were discontinued on the 14th day and ceftriaxone was re-initiated for *E. rhusiopathiae* for the next 6 weeks. The patient had no fever at the follow-up visit. A control echocardiography revealed mild-moderate pericardial effusion of 15 mm behind the left ventricular posterior wall. No vegetation or thrombus was seen on the tricuspid valve. Due to chronic kidney disease, colchicine therapy was initiated instead of non-steroid anti-inflammatory therapy.

**DISCUSSION**

To the best of our knowledge, this is the first case of *E. rhusiopathiae* pericarditis without signs of *E. rhusiopathiae* septicaemia. While it is important to note that the lack of matrix assisted laser desorption ionization-time of flight mass spectrometry and 16S RNA sequencing is a limitation, the isolates were identified as *E. rhusiopathiae* using the VITEK 2 automated microbiology system, and treatment was successfully managed according to this diagnosis. *Acinetobacter* bacteraemia was confirmed in our patient, but *Erysipelothrix* bacteraemia was not. *E. rhusiopathiae* is an immobile, pleomorphic, non-sporulating, aerobic or facultative anerobe, Gram-positive bacillus.[2]

*E. rhusiopathiae* is generally known as a cause of infection in animals. It can be found in pigs, sheep, rabbits, chickens, turkeys, ducks, emus, pigeons, cows, guinea pigs, cats, dogs, and fish. Human infection is typically acquired from animals through work-related exposure by those such as animal breeders, farmers and ranchers, veterinarians, furriers, butchers, fishermen and fishmongers, cooks, and grocers. Our patient had a history of contact with fowl. Three forms of human disease have been described: a localized cutaneous form, known as erysipeloid; a generalized cutaneous form; and a septicemic form that is often associated with endocarditis.[3] Rare manifestations of *E. rhusiopathiae* infection have also been reported in cases of persistent bacteremia, septic arthritis, osteomyelitis, pneumonia intra-abdominal abscess, and meningitis.[8–10]

In immunocompromised patients, bacteraemia without endocarditis is more common; there are no pericarditis cases in the literature thus far. We could provide no histological evidence or immunohistochemical confirmation of myocardial involvement according to the state of knowledge position statements of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. Cardiac magnetic resonance imagining was not available in order to provide other evidence of myocarditis.[11] Though there are no data indicating myocardial involvement of *Erysipelothrix* infection, considering the past history of pericardial effusion, atrial fibrillation with a fast ventricular rate, low ejection fraction 8 months prior, and an elevated troponin level at the current presentation might indicate a myo-pericarditis recurrence. It may be that the prior low ejection fraction was a result of myocardial cell infection by *E. rhusiopathiae*, or as a result of an inflammatory response to the pathogen which temporarily depressed the myocardium via inflammatory cytokines.[12] However, we do not have enough evidence to prove that in our case. Although penicillin and cephalosporins are the first-line choice for the treatment of *Erysipelothrix* infection, we preferred to implement ceftriaxone initially. Varied treatment periods have been applied for other rarely seen clinical entities.[10,13] However, the patient had a healthcare-associated blood stream infection of carbapenem-resistant *A. baumannii*. These infections are a serious problem in healthcare facilities because of the limited options for antibiotic treatment. Carbapenem resistance among *A. baumannii* blood stream infections was reported at 94% in a recent multicenter study conducted in Turkey.[14]

We attributed the deterioration of the patient during follow-up in the intensive care unit to early postoperative acute respiratory failure and hospital-acquired *A. baumannii* infection followed by renal failure. Considering the thrombus image on the dialysis catheter and motile hypoecogen mass on the tricuspid valve demonstrated with TEE in addition to right ventricular enlargement, an acute pulmonary embolism may have been responsible for the hemodynamic collapse in the follow-up period. Regression of the vegetation may have been a result of anticoagulant therapy or
We report this patient as the first proven case report of pericarditis and possible pancarditis due to *E. rhusiopathiae*. The administration of positive inotropic agents, the selection of appropriate antibiotics, and the timing of renal replacement therapy was essential in this complicated case.

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**Informed Consent:** Written informed consent was obtained from the patient for the publication of the case report and the accompanying images.


**REFERENCES**

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