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Case Report

Purulent Pericarditis in an Immunocompetent Young Child

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ABSTRACT

Purulent pericarditis in children is a life-threatening condition causing cardiac tamponade and disrupting the hemodynamic status of the patient. It has been associated with high mortality if treatment is delayed. Furthermore, purulent pericarditis may lead to constrictive pericarditis in the long term if not fully treated. Acute purulent pericarditis should be seriously considered in every septic child presenting with signs of right heart decompensation. Echocardiography is important for diagnosis. Diagnostic pericardiocentesis should be performed. Recent experience shows that excellent results can be obtained when adequate surgical drainage and antibiotic therapy are combined. We report a case of purulent bacterial pericarditis caused by methicillin-resistant *Staphylococcus aureus* in an immunocompetent young child presenting with sepsis. The patient was successfully treated with a combined medical and early surgical approach.

Keywords: Purulent pericarditis, *staphylococcus aureus* sepsis, pericarditis, pericardial tamponade

Introduction

Purulent pericarditis is rare and accounts for less than 1% of all cases of pericarditis. Purulent pericarditis usually presents with sepsis clinic accompanied by refractory fever. Suspicion of purulent pericarditis is an indication for emergency pericardiocentesis. Purulent pericarditis should be treated aggressively because death is inevitable if left untreated.

In this case report, we present a 2-year-old immunocompetent girl with purulent bacterial pericarditis caused by methicillin-resistant *Staphylococcus aureus* (MRSA) who presented with sepsis and pericardial tamponade.

Case Report

A 20-month-old female patient was admitted to another center with complaints of vomiting and diarrhea two weeks before presentation to our center. She was diagnosed with gastroenteritis and hospitalized with oral intake disorder and persistent fever (39 °C). Intravenous hydration support was given at this center. Acute phase responses were previously negative [C-reactive protein (CRP) 2 mg/L, white blood cell (WBC): 9,710/mm^3 and 82% neutrophil predominance]. Rotavirus antigen 3+ was detected in stool examination and oral probiotic support was given. On the 3rd day of hospitalization in the other center, the patient developed tachycardia, high fever,

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decreased oxygen saturation and tachypnea. Investigations revealed leukocytosis and increased acute phase response (CRP: 146 mg/L; normal upper limit is 5 mg/L, WBC: 9,920/mm^3, 74% pnl) and increased cardiothoracic index on chest radiography. Pediatric cardiology consultation was requested. During the evaluation of the patient by the cardiologist, diffuse pericardial effusion was observed on echocardiography. The patient was referred to the intensive care unit of another university hospital with the diagnosis of pericardial effusion and sepsis. The patient was hospitalized; ampicillin sulbactam and amikacin combination was started as antibiotic therapy for sepsis. The patient's respiratory virus panel was negative. The Pro-BNP value was 3,264 ng/L (normal value <125 ng/L for reference laboratory) in the tests performed during intensive care unit hospitalization and ibuprofen, aldactazide and colchicine treatments were started due to diffuse pericardial effusion.

Echocardiography performed at this center revealed a 10 mm diffuse pericardial effusion without evidence of tamponade. Pericarditis was considered. The ventricular ejection fraction was normal. Antibiotic treatment was revised to a meropenem+vancomycin combination on the 5th day of hospitalization. There was no growth in the peripheral blood culture and urine culture obtained from the tests performed for the etiology of pericarditis during intensive care unit hospitalization, and the anti-strephtolyzine-O value sent was within the normal limits at 20 IU/mL (normal value <150 IU/mL for reference laboratory). C3 and C4 values were normal; anti-neutrophil cytoplasmic antibody and anti-nuclear antibody values were negative. Viral serology, quantiFERON test, and purified protein derivative of tuberculin were negative.

Pericardial fluid and acute phase reactants of the patient, who did not show clinical improvement during follow-up, increased and an 18 mm thick effusion between the pericardial leaves was detected on thorax computed tomography examination. The patient, who was referred to the pediatric intensive care unit of our hospital on the 7th day of hospitalization, had tachypnea and tachycardia at the first evaluation in our hospital. Her respiratory sounds were deep and her fever was persistently high. Milrinone and dobutamine were started as inotropic treatment because the patient's blood pressure value was at the lower limit of normal (mean arterial pressure MAP 50-55 mmHg). During follow-up, we also observed that the pulse pressure of our patient was narrowed to 25 mmHg. Bilevel positive airway pressure (BiPAP) support was given to the patient due to accompanying respiratory distress. Echocardiographic evaluation performed on admission to our intensive care unit revealed an 18 mm fibrinous pericardial effusion, thickened pericardium, normal left ventricular systolic function and no evidence of cardiac tamponade (Figure 1). After obtaining parental consent, pericardiocentesis was performed and 70 cc of effusion was drained. A pigtail catheter was placed due to the presence of purulent effusion. Bacteriologic, mycotic cultures and ARB were obtained from pericardial fluid samples. The patient's antibiotic therapy was revised as vancomycin+piperacillin tazobactam and fluconazole until the culture results were received. Ibuprofen as an anti-inflammatory agent and pantoprazole as a gastroprotective agent were also initiated. Blood tests obtained from the patient after pericardiocentesis showed a significant decrease in acute phase reactant values. Clinically, fever was controlled, tachycardia and tachypnea regressed, and nasal oxygen support was started since BiPAP was no longer needed. Hemodynamically, the patient was normotensive. After pericardiocentesis, the patient was followed up with daily echocardiography. The next day, it was observed that the amount of fluid coming from the 24-hour drain was 100 cc and 9 mm pericardial effusion continued on the echo and cardiovascular surgery was requested. Surgical drainage and subtotal pericardiectomy were performed (Figure 2) and a pericardial drainage catheter was placed. The removed pericardial tissue was very thick and fibrous. Pathologic evaluation of the surgically removed pericardial tissue was reported as chronic fibrinopurulent pericarditis.

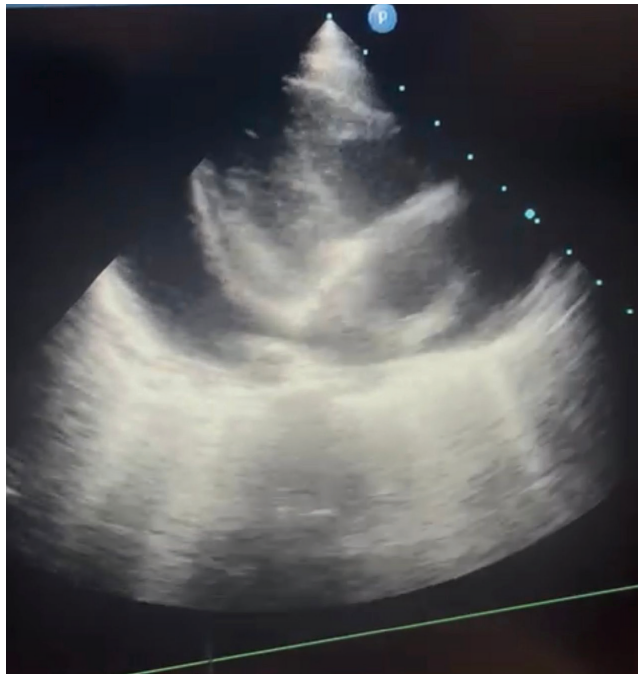

Figure 1. Echocardiographic image of patient (pericardial effusion)

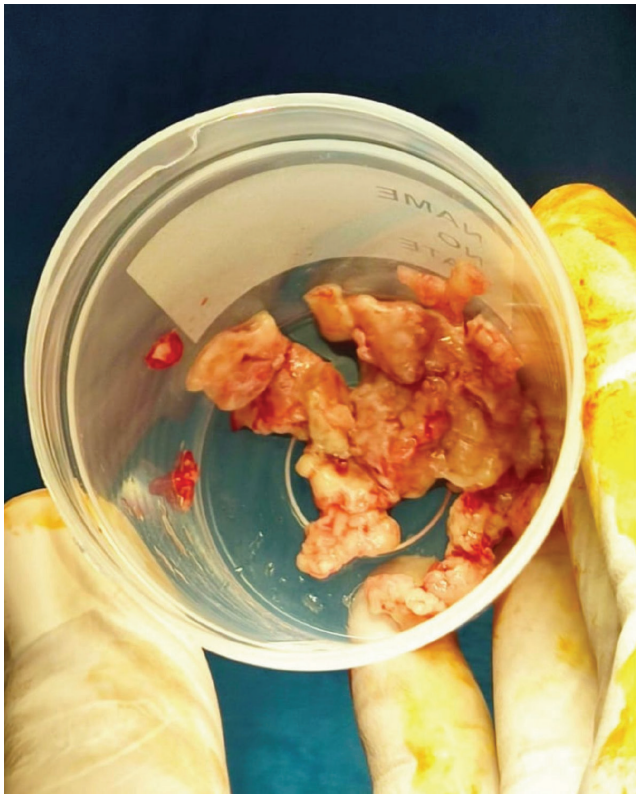

Figure 2. Removed pericardial tissue

MRSA growth was detected in the sample sent from the first pericardiocentesis material. Blood cultures were negative.

In the 2nd week of intensive care unit follow-up, the patient, who no longer needed nasal oxygen and was stabilized, was transferred to our pediatric cardiology service for further treatment. We completed and discontinued 14-day fluconazole and piperacillin-tazobactam treatment in our patient whose vitals were stable in room air in the ward follow-up.

The 6-week antibiotic therapy was completed and the patient was discharged from the hospital with planned outpatient follow-up.

Discussion

Purulent bacterial pericarditis is a rare condition in children (1). It presents as acute pericarditis with symptoms of systemic inflammation, fever, chest pain and dyspnea (2-6). Physical examination findings of purulent pericarditis include increased jugular venous pressure, tachycardia, tachypnea, fever and hepatomegaly. An enlarged heart shadow on chest radiography is one of the most important findings suggesting the diagnosis (5). Pericardial friction sound is not a common finding in purulent pericarditis (7,8). ST segment elevation due to epicardial damage, which is the classical electrocardiographic feature of pericarditis, has also been reported to be rare in purulent pericarditis (9,10). In our patient, there was no pathologic finding on electrocardiogram except for diffuse low voltage. In cases of purulent pericarditis in pediatric patients reported in the literature (2-10), pericardial drainage (temporary pericardial drainage was left in place in some cases) was required in all cases for both diagnostic and therapeutic purposes. Furthermore, all cases were medically treated with antibiotics of different durations (range: 2-8 weeks) and routes [oral/intravenous (IV)/combination].

An older study showed that early intervention with pericardiectomy is indicated in purulent pericarditis if tamponade occurs after initial pericardiocentesis or if the fever persists despite appropriate antibiotic therapy (9,10). It is known that patients with bacterial pericarditis have a higher risk of constrictive pericarditis after pericarditis. Surgical intervention should be considered in purulent bacterial pericarditis in order to reduce the risk of constrictive pericarditis and to decrease morbidity and mortality. The mortality rate in purulent pericarditis is reduced to 20% or less when medical and surgical treatments are combined (9,10).

The spectrum of microorganisms causing purulent pericarditis varies in different parts of the world. In reports in the Western literature, *Streptococcus pneumonia* was the most common cause in the pre-antibiotic era, but *Staphylococcus aureus* has become the most important agent during the antibiotic era (9,10). When a specific focus of infection occurs, the pericardium may become infected by direct spread from septic emboli or pulmonary infection.

In our patient, purulent pericarditis was due to *Staphylococcus aureus* sepsis as a secondary bacterial infection due to rotavirus gastroenteritis.

There are two major complications of purulent pericarditis: cardiac tamponade and septicemia. Cardiac tamponade may develop early due to a rapid accumulation of purulent effusion fluid and it requires early treatment. Sepsis is still a serious complication despite effective antibiotic regimens (10). Antibiotic agent selection should be based on culture antibiograms; however, it is not always possible to culture the agent. Initial empirical antibiotic therapy initiated by the referring clinician sometimes prevents the microorganism from being produced.

Conclusion

Purulent pericarditis in children is a life-threatening condition causing cardiac tamponade. Early diagnosis and early treatment are very important. Emergency pericardiocentesis is life-saving and has diagnostic value. Complete surgical drainage and appropriate parenteral antibiotic therapy are crucial in order to prevent constrictive pericarditis in the long term.

Ethics

Informed Consent: Parental consent was obtained.

Authorship Contributions

Surgical and Medical Practices: Ş.Ş.Ö., F.E., K.C., O.N.T., P.Y.Ö., B.K., Concept: Ş.Ş.Ö., E.D., Z.Ü., Design: Ş.Ş.Ö., E.D., Z.Ü., Data Collection and/or Processing: Ş.Ş.Ö., A.B., B.K.B., Analysis and/or Interpretation: Ş.Ş.Ö., Z.Ü., Literature Search: Ş.Ş.Ö., B.K.B., K.C., O.N.T., Writing: Ş.Ş.Ö., A.B.

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