Successful Treatment of Staphylococcal Pericarditis

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We describe a case of a 26-year-old man with cardiac tamponade. Prompt needle pericardiocentesis revealed purulent fluid and established the diagnosis of staphylococcal pericarditis. The patient was treated successfully by percutaneous catheter drainage combined with antibiotics. One year later he remains in good condition without echocardiographic evidence of constriction. Our case shows that percutaneous catheter drainage of pericardial fluid is an easy, safe and effective technique and should be considered as first choice treatment in purulent pericarditis.

Purulent pericarditis, once a fatal disease, can now be treated successfully if an appropriate treatment is administered. Predisposing factors include pneumonia, previous cardiac or thoracic surgery, immunosuppression, pre-existing aseptic pericarditis and infective endocarditis. Staphylococcus Aureus, Pneumococcus, Streptococcus and gram-negative organisms are the main causative agents. The administration of antibiotics alone has a small impact on patients’ survival. However, when appropriate antibiotic treatment is combined with early complete surgical drainage the outcome is dramatically better. Experience with long-term catheter drainage of the pericardial space is very limited, but it seems a promising, easier and safer technique compared to surgical treatment.

We describe a 26-year-old man with staphylococcal pericarditis who was treated successfully by percutaneous catheter drainage combined with antibiotics.

Case report

A 26-year-old man with cardiac tamponade was admitted to the coronary care unit. He had known idiopathic thrombocytopenic purpura and for the past 2 years had been receiving steroids. Five weeks before admission he was treated in our Cardiology Department because of acute pericarditis. The laboratory tests (biochemical, autoimmune, thyroid function tests) were normal and in view of the favorable response to the combination of prednisolone 75 mg and indomethacin 200 mg daily, he was discharged with the presumptive diagnosis of acute idiopathic pericarditis. Before his current admission he gradually reduced prednisolone to the level of 50 mg daily, the usual maintenance dose for idiopathic thrombocytopenic purpura.

The patient developed worsening dyspnea the last 3 days prior to admission. Remarkably, there was no chest pain or pyrexia. On physical examination the patient was found to be orthopnoic, with a heart rate of 135 beats/minute, blood pressure of 95/60 mm Hg with pulsus paradoxus, distended neck veins and normal lung auscultation. Electrocardiogram showed sinus tachycardia, low QRS voltage, inverted T waves and electrical alternans. Chest x-ray revealed marked cardiomegaly (Figure 1, top) and the clinical suspicion of cardiac tamponade was confirmed by an echocardiogram, which demonstrated a large amount of pericardial fluid (Figure 2, top) with diastolic collapse of the right cavities. Immediately after-
wards, a needle pericardiocentesis was performed in the coronary care unit and 350 ml of purulent fluid were removed, resulting in a remarkable improvement in his clinical condition. Culture of the pericardial fluid, as well as the blood isolated methicillin resistant Staphylococcus Aureus, was performed. Biochemical tests were normal, apart from mildly affected liver enzymes. Hematocrit was 44%, leukocytes 20410/mm³ with 76% neutrophiles and platelets 110000/mm³. Pericardial fluid contained 30000/mm³ leukocytes with 90% neutrophiles and total protein was 3.5 g/dl. The patient was administered vancomycin, gentamycin, rifampicin and methylprednisolone (32 mg daily).

On the second day, a pigtail catheter (7 Fr) was introduced using the Seldinger technique (subxiphoid approach) and placed under fluoroscopic guidance in the pericardial cavity. Initially, 1100 ml of purulent fluid were removed. The pericardial cavity was flushed intermittently using 200 ml of saline within 30 minutes every 2 hours. After 48 hours the fluid appeared clear and cultures were sterile. The drainage catheter remained for 8 days, and was removed after the echocardiographic demonstration of only a small pericardial effusion (Figure 2, bottom).

On the third day the patient developed fever (38.1°C) and chest x-ray revealed multiple nodular infiltrations in both lungs with occasional pneumatoceles, suggestive of Staphylococcal pneumonia (Figure 1, bottom). Surprisingly, there was no further deterioration in the patient’s general condition. The patient received the antibiotic regime for 4 weeks and then was discharged in good general condition. On the follow-up echocardiograms one, six and twelve months later there was neither pericardial fluid nor evidence of constriction. In addition, computed tomography 1 year later did not detect pericardial thickening.

Figure 1. Chest x-ray on admission, showing marked cardiomegaly (top), and three days later, with the findings of pneumonia (bottom). The pigtail catheter (arrow) is placed in the pericardial cavity.

Figure 2. Two-dimensional echocardiogram (four-chamber view) displaying massive pericardial effusion on admission (top) and small amount of fluid after catheter drainage (bottom).
Discussion

Purulent pericarditis is a severe disease with fulminant course. There is general agreement that the combination of antibiotics and pericardial drainage has improved survival. However, controversy still remains concerning the type of pericardial drainage. Percutaneous subxiphoid drainage with a catheter is an easy procedure performed under only local anesthesia at low risk, while pericardiectomy in a critically ill patient carries additional operative morbidity. The theoretical advantage of the removal of the pericardium is the prevention of late constrictive pericarditis, although few cases of constriction have been reported. The possibility of dispersion of the infection with pericardiectomy, which was first raised by Cameron, has not been confirmed in other series. Early diagnosis of purulent pericarditis is vital. However, diagnosis can easily be missed since the classic signs of pericarditis (chest pain, pericardial friction rub, ST segment elevation) may be absent and clinical features such as fever, dyspnea or tachycardia may be attributed to the underlying infectious disease. Our patient was immnosuppressed due to the chronic administration of steroids. This occasionally poses additional difficulties in early diagnosis by masking the clinical presentation. In our case the patient was afebrile on admission. The preceding aseptic pericarditis could have further facilitated the transition from the aseptic to the purulent state. It has been postulated that rich fibrinous exudates in the pericardial space provide a fertile culture medium for bacterial growth, particularly in an immunosuppressed patient. The primary site which caused bacteremia, the probable common denominator for pericarditis and pneumonia, was not identified in our patient. Among the commonest septic foci causing direct extension of infection to pericardium are pneumonia, infective endocarditis (valvular ring abscess or myocardial abscess) and cardiac or thoracic surgery. In some cases of seriously ill patients it can be caused by hematogenous spread, mainly by gram-negative organisms.

The prompt needle pericardiocentesis resulted in early diagnosis, followed by appropriate antibiotic treatment and insertion of a pigtail catheter for continuous drainage. It is essential to keep the catheter unobstructed in order to evacuate the pericardial cavity completely. We achieved it by using a 7 Fr pigtail catheter in association with meticulous aseptic intermittent flushing. However, if the causative organism is Haemophilus influenzae, early pericardiectomy is recommended, since the exudate in the pericardium is described as having the density of “scrambled eggs” and can be hardly drained with a catheter. In our case the catheter remained for 8 days and when it was removed there was only a small amount of sterile pericardial fluid remaining, without any loculated effusion.

Pericardiocentesis is theoretically associated with a higher potential for constriction in comparison with pericardiectomy. However, the incidence of this complication appears low. In a series of 22 children treated with subxiphoid pericardiostomy tube draining none developed signs of constriction during a 3-year follow-up. In our case the echocardiogram after drainage did not show loculated pericardial effusion or evidence of adhesions. If this happened then surgical drainage along with extensive pericardiectomy would be the subsequent treatment to prevent the possibility of late pericardial constriction. Recently, there are encouraging data regarding the intrapericardial administration of streptokinase in purulent pericarditis in order to achieve better drainage. Although only a small number of patients have received this treatment, it appears that intrapericardial infusion of streptokinase enhances the complete drainage of pericardial fluid by dissolving its fibrinous components and therefore minimizing the risk of constrictive pericarditis.

In general the index of suspicion for staphylococcal pericarditis should be high in immunosuppressed patients with pericardial effusion, especially if there is a prior history of pericarditis. Diagnostic pericardiocentesis should be performed early, followed by a percutaneously placed catheter via a subxiphoid route along with the appropriate antibiotic treatment. Evacuation of the pericardial fluid is essential to minimize the risk of subsequent development of constrictive pericarditis.

We conclude that percutaneous catheter drainage of pericardial fluid is an easy, safe and effective technique and should be considered as first choice treatment in purulent pericarditis. If complete evacuation is not feasible or late constriction develops, then pericardiectomy should be considered.

References