Fatal Acute Purulent Pericarditis in a Patient with Renal Transplant: A Case Report

Nabil Mohsin, Mohammad Budruddin, Jha Amitabh, Mohammad Ehab, Pakkayara Abbas

Abstract

Objectives: **Acute purulent pericarditis is a life-threatening disease, although it is becoming uncommon in the era of antibiotics.**

Materials and Methods: **We present a case of fatal acute massive purulent pericarditis in a kidney transplant recipient.**

Results: **A 46-year-old woman had an unrelated commercial renal transplant in 2003. She had a history of diabetes mellitus and hepatitis C infection. Kaposi sarcoma developed in the posttransplant period. Her last admission was prompted by the development of acute rejection confirmed by transplant biopsy, and she was treated with intravenous methylprednisolone. Three days before her death, thrombophlebitis of the right forearm was noted. We postulate that this could have been the source of the fulminant purulent pericarditis, as the organism in the pericardial fluid was *Staphylococcus aureus*, a common pathogen in thrombophlebitis. She was initially resuscitated after cardiac arrest but died shortly after.**

Conclusions: **Severe purulent pericarditis in the immunocompromised patient can occur abruptly. The source of infection may show minimal signs and symptoms. Thrombophlebitis and other apparently minor infections should not be overlooked in such patients.**

Key words: *Thrombophlebitis;* Staphylococcus *species, immunosuppression.*

From the Department of Nephrology, Royal Hospital, Muscat.
Address reprint requests to: *Nabil Mohsin, Royal Hospital, Department of Nephrology, P.O. Box 1331, PC 111, Muscat, Oman*
***Phone**: +968 24 599 451 **Fax:** 968 24 599 856 **E-mail:** nabmoh@omantel.net.om*

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Purulent pericarditis, although not very common in the era of antibiotics, may still be seen in particular clinical situations, such as patients on immunosuppressive drugs. The clinical presentation may also be misleading in the immunosuppressed patient, as the symptoms and signs of inflammation may be minimal, and the clinical picture is often smoldering. The source of infection can be from any part of the body. Purulent pericarditis carries a poor prognosis especially if the presentation is acute and without obvious inflammation, as this will delay treatment. At the time of diagnosis, the patient may be in tamponade or in shock. Even survivors of acute pericarditis may have high mortality because of the development of constrictive pericarditis.

Case Report

A 46-year-old Omani woman received a commercial kidney transplant in 2003 because of end-stage kidney failure. She had a history of diabetes mellitus and hepatitis C infection. The immediate posttransplant period was complicated by acute tubular necrosis, chest infection, and extensive oropharyngeal candidiasis, which were treated and had favorable outcomes at the expense of an altered general condition. Maintenance immunosuppression consisted of cyclosporine, mycophenolate mofetil, and prednisolone. She received regular follow-up at the renal transplant clinic. Renal function was satisfactory with serum creatinine of 71 μmol/L. In August 2005, almost 2 years after transplant, she developed Kaposi sarcoma of the left lower limb. This condition was treated with reduction of immunosuppression and conversion to a non-calcineurin inhibitor regimen; that is, the cyclosporine was stopped and she was started on sirolimus. Under this regimen, the Kaposi sarcoma regressed impressively within 6 weeks. Her serum creatinine was 77 μmol/L in March 2006.

In September 2006, she presented to the Royal Hospital with severe renal function impairment. The serum creatinine level was 475 μmol/L; the urea nitrogen level was 19.7 mmol/L. Physical examination was unremarkable, and the Kaposi sarcoma had regressed completely. A biopsy of the graft kidney was performed. The biopsy results showed acute cellular rejection along with changes suggestive of chronic allograft nephropathy. She was prescribed intravenous methylprednisolone for 3 days. The pretreatment chest radiograph was normal (Figure 1). There were no obvious signs of infection. The white blood cell count was within normal limits. On the second day of treatment, early signs of thrombophlebitis at the intravenous cannula site on the right forearm were noted. The cannula was promptly removed and a new one was inserted in the other arm, through which the patient received the third dose of methylprednisolone. The erythema and tenderness associated with the thrombophlebitis improved without the administration of antibiotics. The graft rejection proved to be steroid resistant, and renal function further worsened. The serum creatinine level increased to 554 μmol/L, and the urea nitrogen level increased to 47.7 mmol/L. The patient also developed some uremic manifestations. The patient was advised to undergo dialysis, but she refused.

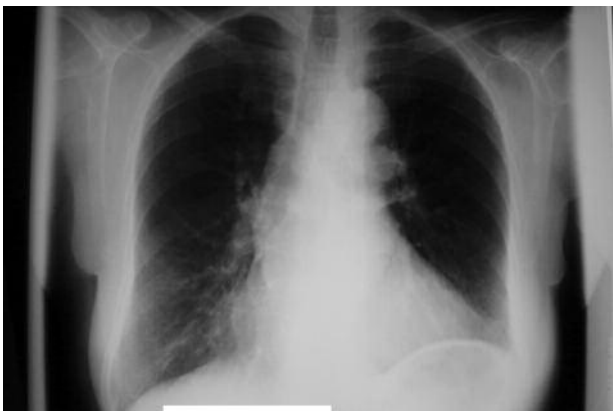

Figure 1. Chest radiograph before initiation of steroid treatment for acute graft rejection.

The following day, the patient developed sudden severe shortness of breath. Clinically, her physical examination showed dyspnea, orthopnea, and tachypnea. She was hypotensive. She remained afebrile. Her jugular venous pressure was increased. Chest auscultation demonstrated extensive wet crepitations. Results of blood gas testing showed hypoxia and severe acidosis with pH 7.1. Urgent hemodialysis was performed; at the same time, inotropic support was initiated, and she required high levels of inotropic support to maintain her blood pressure.

After receiving 2 hours of hemodialysis, her chest seemed less congested, and the results of repeat blood gas testing showed that the pH had improved to 7.29. However, she remained dyspneic, and the jugular venous pressure remained raised. Results of an electrocardiogram showed low voltage complexes. At this point, cardiac tamponade was suspected, and an urgent bedside chest radiograph was performed (Figure 2). Before the radiograph could be reviewed, she developed cardiac arrest while still receiving hemodialysis. Immediate resuscitation revived her, and she was mechanically ventilated and transferred to the intensive care unit (ICU). She continued to require high inotropic support.

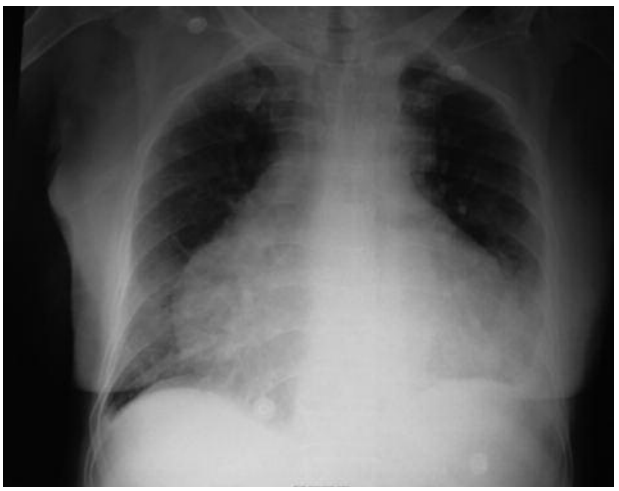

Figure 2. Chest radiograph 5 days later, showing enlargement and globular shape of the heart, suggesting pericardial effusion.

The bedside chest radiograph depicted a huge globular heart, suggestive of pericardial effusion. An urgent echocardiogram performed in the ICU showed massive pericardial effusion. An echocardiography-guided subxiphoid pericardiocentesis was attempted; however, only a few milliliters of thick purulent fluid were removed. Broad-spectrum antibiotics were started. Urgent pericardiotomy was performed, and more than 1000 mL of frank pus was drained from the pericardium. The patient remained hypotensive despite intravenous fluids and high doses of inotropics, and she died shortly after. It is noteworthy that the patient

never had fever or any signs of acute inflammation. The results of the pericardial fluid culture, received postmortem, revealed *Staphylococcus aureus*.

Discussion

Pericardial effusion and pericarditis are diseases known since antiquity. Ibn-Zuhr Avenzoar of Andalusia described serous, fibrinous, and purulent pericarditis (1). The era of antibiotics has helped to reduce the incidence of purulent pericarditis. Nevertheless, it still occurs, especially in the immunocompromised host (2-6). The classic clinical signs may not be present, and the clinical presentation can even be misleading. In immunocompetent individuals, signs and symptoms of purulent pericarditis are usually clear and consist of chest pain, fever, dyspnea, pulsus paradoxus, and distended internal jugular veins. The source of infection is often but not always obvious. In the immunocompetent host, fever and pericardial rub may not be common signs (7). However, signs and symptoms of purulent pericarditis may not be clear in persons who are immunocompromised, including patients with chronic kidney disease, (4,6) patients after organ transplant, and patients under immunosuppression for other medical conditions such as rheumatoid arthritis treated with biological agents and cytotoxics like tumor necrosis factor alpha antagonist and methotrexate (2), or steroids (3). In such patients, the clinical presentation may be smoldering or even misleading, for example, mimicking septic shock (1,8). Purulent pericarditis may even be the first indication of colon cancer, as reported by Kim and colleagues (5).

The source of infection can be diverse and include the mediastinum, lungs, prostate, urinary tract, colon, cutaneous abscess, and septic arthritis (1,8-10). Some cases of purulent pericarditis are reported to be spontaneous (9). It is possible that in these cases, the source was a mild infection, probably cutaneous, that was not noticed. The bacterial species most often involved is *Staphylococcus* (4,8,10,11). Nevertheless, other bacterial species have been reported, including *Proteus mirabilis* (4), *Streptococcus* (5,12), *Actinobacteria* (12), *Enterococcus* (12), *pneumococcus* (13), and *Haemophilus influenzae* (6).

Treatment should be instituted promptly. It includes broad-spectrum antibiotics and pericardial drainage through pericardiocentesis or pericardiotomy (11). Other measures include saline irrigation (5) and streptokinase instillation (12) into the pericardial sac. The best approach consists of prompt institution of antibiotics along with surgical drainage of the pericardial effusion (5,9,11,12).

The prognosis is often bleak. This is mainly due to inadvertent delay in diagnosis and treatment, because of the misleading presentation that can occur in a debilitated host. Acute development, female sex, and the volume of pericardial effusion have been reported as poor prognostic factors (14). In the patient under discussion, hemodynamic instability was initially thought to be due to acute pulmonary edema and severe acidosis. The persistence of dyspnea in spite of hemodialysis, raised jugular venous pressure, and low voltage on the electrocardiogram suggested pericardial effusion with tamponade. The diagnosis was further supported by the results from an urgent bedside chest radiograph and echocardiogram.

The case under discussion highlights the fact that immunosuppressed patients may develop very severe and even fatal infection without many clinical symptoms and signs. Also, the severity of infection may be inconsistent with its physical signs. Skin lesions and thrombophlebitis should not be overlooked, as these may be the cause of severe infection. A high index of suspicion for purulent pericarditis should be present when dealing with immunocompromised patients.

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