Primary Methicillin-Resistant \textit{Staphylococcus aureus} Pericarditis in a Patient Undergoing Hemodialysis

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We report a case of a 41-year-old man undergoing hemodialysis who presented with a sudden fever and dyspnea. He developed a severe pericardial effusion due to methicillin-resistant \textit{Staphylococcus aureus}, which was identified in both blood and pericardial fluid cultures. He was successfully treated with intravenous vancomycin for 6 weeks. Although such cases are very rare in Korea, the current case describes a primary purulent pericarditis without any other potential infectious foci.

Keywords: Pericarditis; Methicillin-resistant \textit{Staphylococcus aureus}; Renal dialysis

INTRODUCTION

Pericarditis, defined as pericardial sac inflammation, has both infectious and noninfectious etiologies. Whereas idiopathic and viral pericarditis are usually benign and self-limiting conditions, purulent bacterial pericarditis requires prompt adequate antimicrobial treatment and is associated with significant morbidity and mortality [1]. The pericardium is rarely a primary infection site, especially with regard to bacterial infection. However, 4 infection spread pathways to the pericardium have been described with respect to secondary bacterial pericarditis: the direct extension of an intrathoracic process, local extension, a perforating injury to the chest wall (e.g., penetrating injury or cardiothoracic surgery), and hematogenous spread from a distant focus [2,3]. Pericarditis in patients undergoing chronic hemodialysis might be related to various causes such as uremic toxins, which are most commonly responsible for the manifestation of pericarditis. Although hemodialysis is a well-known risk factor for \textit{Staphylococcus aureus} (S. aureus) infection [4], primary bacterial pericarditis is uncommon and has serious outcomes [5]. We describe a case of infectious pericarditis caused by healthcare-associated methicillin-resistant \textit{Staphylococcus aureus} (MRSA) in a hemodialysis patient.

CASE REPORT

A 41-year-old man was referred to the infectious diseases division following the diagnosis of a dark, bloody pericardial effusion. He had a history of hypertension and advanced gastric carcinoma (Bormman’s type IV). He was currently undergoing hemodialysis via a tunneled cuffed catheter consequent to chronic kidney disease. He was initially admitted for the treatment of an intestinal obstruction due to a small bowel adhesion. During the previous 95 days, the patient had taken ceftriaxone for 10 days and cefotiam for 7 days 1 month ago. On the 95th day in hospital he presented with dyspnea, developed a high fever (38°C), and exhibited a pulse rate of 130/min, a respiratory rate of 25/min, and a blood pressure of 100/60 mm Hg. He also had jugular vein distention. Laboratory results revealed elevated white blood cell counts (23,200/µL), with a neutrophil percentage of 89%, a mildly elevated Troponin-T concentration (0.117 ng/mL; reference range, 0 to 0.1 ng/mL), and an elevated myoglobin concentration (222 ng/mL; reference range, 27 to 75 ng/mL). Chest radiography revealed a cardiac silhouette with a ‘water bottle’ configuration and sequelae of pulmonary tuberculosis with bilateral calcified lung granulomas (Fig. 1). Electrocardiography revealed sinus tachycardia with left ventricular hypertrophy. A transthoracic echocardiogram (TTE) demonstrated a large...
circumferential pericardial effusion with increased echogenicity and septation without tamponade physiology (Fig. 2). A chest computed tomography (CT) scan was performed to evaluate the possibility of tuberculosis reactivation and advanced gastric cancer metastasis. The scan revealed a moderate amount of pericardial effusion with a suspicious enhancing pericardium (Fig. 3). Echocardiography-guided pericardiocentesis was performed to drain 550 mL of dark bloody fluid from the pericardial space. The pericardial fluid analysis revealed elevated red (518,400/µL) and white blood cell counts (10,800/µL) with a neutrophil percentage of 93%, and the following concentrations: glucose, 50 mg/dL; total protein, 6.3 g/dL; lactate dehydrogenase, 659 U/L; and adenosine deaminase, 49 U/L. A *Mycobacterium tuberculosis* polymerase chain reaction-based DNA analysis (TB/NTM real time PCR kit; LG Life-science, Seoul, Korea) and acid-fast bacilli pericardial fluid stain and mycobacterial culture were all negative. The pericardial fluid cytology was negative for malignant cells. Percutaneous drainage and an empiric cefazolin plus piperacillin/tazobactam antibiotic treatment based on an epidemiological suspicion of causative organisms were immediately initiated. The antimicrobial agent was switched to intravenous vancomycin upon receiving an interim report of gram-positive cocci in the blood. The fever subsided 5 days after switching to intravenous vancomycin. The final blood and pericardial fluid culture results indicated MRSA with a minimum inhibitory vancomycin concentration of 2 mg/dL. Blood cultures from the central venous catheters and the central venous catheter tip cultures were negative. The catheter insertion sites exhibited no signs of local inflammation. A chest radiography and abdominal and pelvic CT indicated no evidence of infection. Because there was no clear evidence of MRSA infection at other sites, we assumed primary MRSA pericarditis with bacteremia. Ten days after the pericardiocentesis and initiation of antimicrobial therapy, a second TTE revealed only minimal pericardial effusion. The patient received 6 weeks of intravenous vancomycin therapy and was discharged without any further complications during a 10-month follow-up period.

**DISCUSSION**

Pericarditis is a common complication among uremic patients undergoing hemodialysis. Uremia and/or inadequate dialysis have been identified as major causes of pericarditis in hemodialysis patients and intensifying the dialysis process in such patients could improve the outcomes. However, purulent pericarditis is not a common manifestation [5]. Instead, this condition occurs mostly from the direct invasion of an infection from an adjacent pneumonia, mediastinitis, or distant infectious focus or subsequent to bac-
teremia [6]. Common organisms responsible for purulent pericarditis include *S. aureus*, *Streptococcus pneumoniae*, gram-negative bacilli (e.g., *Proteus, Escherichia coli, Pseudomonas, Klebsiella*), fungal pathogens in patients with predisposing factors, and *Mycobacterium tuberculosis* in developing countries. Most *S. aureus* infections that induce purulent pericarditis are hospital-acquired. Community-acquired MRSA has rarely been associated with purulent pericarditis [7]. Hemodialysis patients have an increased *S. aureus* nasal carriage rate, and probable predisposing factors for nasal carriage include immunodeficiency, the presence of prosthetic material, and frequent skin breaches associated with venipuncture. Staphylococcal translocation from colonized mucosal sites, including the nasal mucosa, to the bloodstream has been considered a potential source of bacterial invasion in cancer and hemodialysis patients because of the defective mucocutaneous barriers and concomitant immunodeficiencies exhibited by these patients. Furthermore, hemodialysis patients have frequent and direct contact with hospital environments, another factor that contributes to *S. aureus* acquisition [4]. In the current case, the patient had a medical history of advanced gastric carcinoma, end-stage renal disease treated with hemodialysis, and prior tuberculosis sequelae on a chest radiograph. Therefore, we considered the possible pericarditis causes, including metastasis, tuberculosis, and distant bacterial infection. However, this case did not present with any obvious septic foci, concomitant pulmonary sepsis, or mediastinal or intra-abdominal pathology. A diagnosis of acute primary MRSA pericarditis was made, despite that this is an extremely rare entity in hemodialysis patients.

**REFERENCES**