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Primary Purulent Pericarditis with Cardiac Tamponade due to Oropharyngeal Polymicrobial Infection: A Case Report and Literature Review

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Cardiac tamponade due to purulent pericarditis with a characteristic greenish fluid is rare in this antibiotic era. It is highly fatal despite early diagnosis and advanced treatment. Gram-positive cocci are the leading cause of purulent pericarditis, which usually results from a direct or hematogenous spread of organisms to the pericardium from the primary foci of infection. We describe an index case of rapidly developing pericardial tamponade caused by oropharyngeal polymicrobial infection in the absence of a primary source of infection in a 62-year-old man, who was successfully managed with emergency large-volume pericardiocentesis followed by pericardiectomy.

Key words: 1. Purulent pericarditis
2. Cardiac tamponade
3. Coinfection
4. Pericardiectomy

CASE REPORT

A 62-year-old male with a history of tobacco abuse and newly diagnosed small-cell lung cancer (T1N1M0) two months earlier was readmitted in the hospital with shortness of breath for one day. He had been on chemo-radiation for his lung cancer over the course of the previous month. Meanwhile, four days prior to this admission, he was diagnosed with acute idiopathic pericarditis for which he was started on Colchicine 0.6 mg daily and Naproxen 500 mg three times a day; the patient was discharged on the same day.

In this presentation, he had a temperature of 100°F. His blood pressure was 80/40 mmHg, heart rate was 120 beats/min, and respiratory rate was 22 breaths/min. He was slightly hypoxic and was placed on 3 L/min of oxygen by nasal cannula with 96% of SpO_2. The physical exam was significant for marked jugular venous distension without Kussmaul's sign and pulsus paradoxus. His breath sounds were clear, but he had distant heart sounds without murmur, rub, or gallops. His peripheral extremities were cool and mottled. His labs were significant; the total white count was 1,600/μL, 61% segmented cells and absolute neutrophil count was 1,040/μL, creatinine was 2.9 mg/dL (from a baseline of 0.8 mg/dL), blood urea nitrogen was 62 mg/dL, and lactic acid was 6 mmol/L (normal lactic acid level, 0.67 to 1.8 mmol/L). An electrocardiogram showed sinus tachycardia having normal voltage with diffuse ST-segment elevations. Urgent trans-

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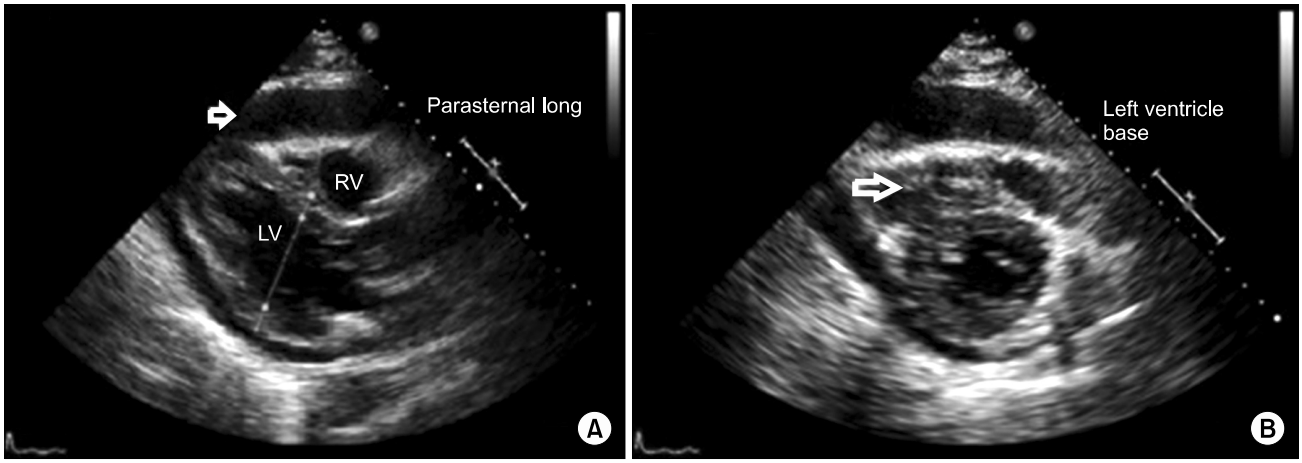

Fig. 1. (A) TTE along the parasternal long axis: the arrows show a large pericardial effusion. (B) TTE along a short axis showing the right ventricular collapsed in diastole, suggestive of cardiac tamponade. LV, left ventricle; RV, right ventricle; TTE, transthoracic echocardiogram.

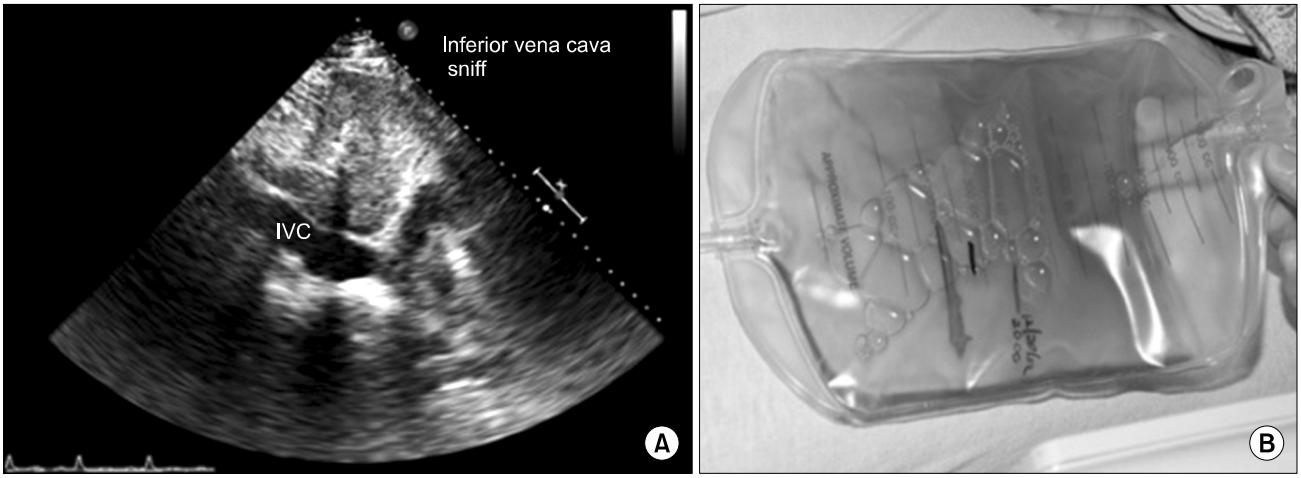

Fig. 2. (A) Transthoracic echocardiogram showing dilated inferior vena cava (IVC), which is often seen with tamponade physiology. (B) Green pericardial fluid obtained after emergency pericardiocentesis that grew multiple microorganisms.

thoracic echocardiography (TTE) revealed large pericardial effusion causing tamponade physiology (Fig. 1A, B) and right ventricular collapse in diastole and dilated inferior vena cava (Fig. 2A). Emergency pericardiocentesis was performed and substantially drained 800 mL of the green fluid (Fig. 2B). The patient was started on vancomycin and cefepime empirically for purulent pericarditis (PP). Repeat TTE on the next day showed loculated pericardial effusion for which he underwent partial pericardiectomy. Cultures from the pericardial fluid grew *Prevotella melaninogenica* and *P. intermedia, Fusobacterium* species, and *Streptococcus* group C. His antibiotics were tailored to intravenous (IV) ertapenem. The source of the polymicrobial pericarditis was unclear despite an extensive work-up including a computed tomography scan of the abdomen and the chest. The patient also had negative blood, sputum, and urine cultures. Subsequently, his acute renal failure and sepsis resolved. He was then discharged home on the eighth day of hospitalization; later, he completed the course of IV ertapenem for two more weeks. There was no recurrence of pericardial effusion according to the TTE 4

weeks following discharge. The patient eventually resumed chemo-radiation therapy 2 months after hospital discharge.

DISCUSSION

PP is rare in this modern antibiotic era [1,2]. It is usually caused by gram-positive cocci, mostly *Staphylococcus aureus* and *Streptococcus pneumoniae*, in the presence of the primary foci of infection [1,3]. Bacterial pericarditis is associated with considerable morbidity and death despite the advances in diagnosis and treatment [2].

Most of the common etiologies of acute pericarditis and pericardial effusion in developed countries are idiopathic or viral [3,4]. The other common causes of pericardial effusion include neoplasm (36%) and uremia (20%) [3]. However, in 30% of the patients of non-infectious pericardial effusion, the cause cannot be determined in spite of the pericardial fluid and tissue analysis [5]. Bacterial PP has become very rare since the advent of antibiotic usage, particularly in the developed world [2,4,5]. The incidence of bacterial pericarditis accounts for less than 1% of pericarditis, the incidence of which has decreased further with the availability and widespread use of effective antibiotics [2,4]. Gram-positive cocci are the major causes of bacterial pericarditis. Before the introduction of antibiotics, *Streptococcus pneumoniae* was responsible for most cases of PP. Penicillin availability has decreased not only the rates of pneumococcus infection in general but also the rates of PP [2]. The other common causes of bacterial pericarditis are *Staphylococcus aureus, Haemophilus influenzae, Streptococcus viridans,* and Group A *Streptococcus* [1,2]. Further, certain gram-negative anaerobic organisms such as *Bacteroides fragilis, Prevotella, Porphyromonas,* and *Fusobacterium* are discussed in the literature as causes of PP [6]. The other rare anaerobes isolated from patients with pericarditis are *Peptostreptococcus, Clostridium, Bifidobacterium, Actinomyces* species, and *Propionibacterium* [6].

Our patient developed PP due to rare polymicrobial infection with *Streptococcus* group C, *Prevotella melaninogenica* and *P. intermedia,* and *Fusobacterium* species. Combinations of such pathogens have never been discussed in the literature as the causes of pericardial effusion. The *Prevotella* species and the *Fusobacterium* species are predominantly gram-negative anaerobic bacteria and are indigenous flora on all mucosal surfaces predominantly of the oral cavity [7]. *Streptococci* group C are facultative anaerobic gram-positive cocci that occur in pairs or chains. They are part of the endogenous microbial flora of the pharynx and are considered to be common causes of infection in several animal species. There is little information about their overall importance as a cause of human infection [8].

Unlike our case, bacterial pericarditis rarely occurs as a primary site of infection. The organisms usually invade as opportunistic pathogens through a break in the mucosa and cause infection. They have an opportunity to penetrate tissues and then to set up infection under certain circumstances such as surgical or other trauma or when tumors arise at the mucosal surface [7]. This results as a complication of an infection originating elsewhere in the body, arising by direct spread or hematogenous dissemination [1]. Pneumonia was the primary source of infection for a majority of the patients (72%) in the pre-antibiotic era, compared with only 22% of the patients in this modern era [2]. Other primary sources of infection include empyema, myocarditis, suppurative mediastinal lymphadenitis, and infective endocarditis, which cause direct bacterial seeding to the pericardial space and pericarditis [1]. The other known primary sources of infection causing direct infection to the pericardium are trauma and contiguous infections such as traumatic endotracheal intubation in nasopharyngeal carcinoma, cardio-thoracic surgery, or catheter drainage, by spread from an intrathoracic, myocardial, or subdiaphragmatic focus [4,7]. In our case, the source of polymicrobial pericarditis was unclear. The organisms retrieved were normal flora that resides inside the oropharynx as mentioned earlier. In our case, we believe that purulent pericarditis was spontaneous in the setting of an immunocompromised state via a hematogenous spread of polymicrobial agents. The most likely sequence of events to explain our patient's condition was that he developed a transient polymicrobial bacteremia from a mucosal breech in the oropharynx or proximal esophagus without frank perforation. After getting seeded into the pericardial space, it developed into a polymicrobial empyema. However, there is no report of an immunocompromised patient who developed poly-

microbial pericarditis in the absence of any other primary infections.

Purulent pericarditis is a life-threatening condition with a fatality rate of 100% if untreated. The mortality rate even with advance treatment varies from 40% to 75%, and death is mostly due to cardiac tamponade, systemic toxicity, cardiac decompensation, and constriction [9]. Therefore, it is important to recognize this condition early for urgent intervention. Unfortunately, purulent pericarditis is so rare and fulminant that in more than half of the cases the diagnosis is made postmortem [2].

Therefore, once purulent pericarditis is suspected clinically, TTE needs to be performed urgently for the assessment of pericardial effusion because these patients might require prompt echocardiography-guided drainage and placement of an indwelling pericardial catheter [5]. However, loculation with fibrin accumulation may occur resulting in a pericardial empyema, making percutaneous drainage incomplete and ineffective. In such cases, a pericardial window or extensive pericardiectomy usually through subxiphoid pericardiotomy is essential to achieve adequate drainage [2,3]. Constrictive pericarditis occurs over the course of purulent pericarditis in at least 3.5% of the cases, as shown in a large review [10]. However, there is no existing consensus on early pericardiectomy in order to prevent constrictive pericarditis. In addition, the exact timing and type of surgery is still debatable [10]. Moreover, few authors propose intrapericardial fibrinolysis, a less invasive procedure, as an alternative to surgery for PP management. It is believed that frequent irrigation of the pericardial cavity with urokinase or streptokinase using large catheters might liquefy the purulent exudate, but there is a lack of definitive evidence as a treatment for PP and for prevention of persistent PP and constrictive pericarditis [10]. Meanwhile, in retrospective studies, as compared to simple drainage, systematic pericardiectomy led to the prevention of constrictive pericarditis with a better clinical outcome [1,10]. Our patient underwent emergency pericardiocentesis with the placement of a pericardial drain and later underwent pericardiectomy relieving the effusion without the use of intrapericardial fibrinolysis.

In addition to surgery, antimicrobial therapy is equally important for treating bacterial infection. Identification of the pathogens and determination of their antimicrobial susceptibility and beta-lactamase production are essential for adequate selection of an antibiotic therapy effective against these organisms [6,7]. Recent data have shown that increasing numbers of anaerobes are showing resistance to penicillin due to β-lactamase production [6,7]. Our patient received ertapenem, which is a broad-spectrum antibiotic for serious infection caused by both aerobic and anaerobic gram-positive and gram-negative organisms. Furthermore, because of a lack of guidelines on the duration of antibiotic therapy in the case of PP and by analogy with empyema, a treatment of at least 3 weeks seems reasonable [10]. Based on this review, our patient received 3 weeks of antibiotics, resulting in a complete resolution of the infection. Despite advances in diagnostic and treatment modalities, PP with its complications remains a challenging problem in clinical practice even in the developed world [3,5].

To conclude, oropharyngeal bacteria may seed into the pericardium and lead to acute fulminant pericarditis were the primary source of infection cannot be identified. Because of the rarity and high fatality of such a case, a high index of suspicion is needed to make an early diagnosis in an immunocompromised patient who is suspected of having acute viral or idiopathic pericarditis. The immediate surgical treatment and thoughtful selection of antibiotics can prevent the fatality.

We report a case of purulent pericarditis causing cardiac tamponade due to rare polymicrobial infection in a patient with lung cancer who was recently started on chemo-radiation therapy. The patient was successfully managed in a tertiary care center with a combination of antibiotic therapy, emergency pericardiocentesis, and pericardiectomy.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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