A Patient of Crimean-Congo Hemorrhagic Fever Manifested with Pericardial Tamponade

Perikardiyal Tamponad ile Prezente olan bir Kırım Kongo Kanamalı Ateşi Olgusu

Yalın Tolga YAYLALI*, İbrahim SUSAM*, Özgür TAŞKÖYLÜ*, Ömer ÇAĞLAYAN*, Şükriye USLU*, Işık TEKİN*, Suzan SAÇAR**

*Pamukkale Üniversitesi Tıp Fakültesi, Kardiyoloji AD, Denizli
**Pamukkale Üniversitesi Tıp Fakültesi, Enfeksiyon Hastalıkları AD, Denizli

Abstract
Crimean-Congo hemorrhagic fever (CCHF) is an often fatal viral infection. Prompt diagnosis and treatment are critical both for patient survival and preventing the transmission of infection. It is characterized by a common dramatic progression with hemorrhage, myalgia, and fever. Atypical presentations of bleeding have been reported. However, pericardial tamponade has not been reported before. Here, we report an unusual case of CCHF infection with a very large hemorrhagic pericardial effusion manifested with tamponade.


Key words: Crimean-Congo hemorrhagic fever, pericardial tamponade

Özet

Anahtar sözcükler: Kırım Kongo kanamalı ateş, perikardiyal tamponad

Introduction
Crimean-Congo hemorrhagic fever (CCHF) is an often fatal viral infection caused by the CCHF virus from the genus Nairovirus in the Bunyaviridae family. Any infection, neoplasm, autoimmune, and inflammatory process causing pericarditis can cause a pericardial effusion [1]. Effusions with a high incidence of progression to tamponade are bacterial, fungal, and human immunodeficiency virus (HIV)-associated infections and neoplastic involvement. Patients with tamponade are almost always in acute distress with tachypnea, diaphoresis, cool extremities, peripheral cyanosis, and altered mental status. On physical examination Beck’s triad of hypotension, distant heart sounds, and elevated jugular venous pressure is a useful clue to the presence of severe tamponade [2]. Right ventricular diastolic collapse on echocardiographic examination is the most useful indicator of a hemodynamically significant pericardial effusion [3]. For most patients with actual or threatened tamponade, management should be directed toward urgent or emergent pericardiocentesis.

Here, we report a severe case of CCHF infection presenting with a large hemorrhagic pericardial effusion with tamponade.

Case
A 70 year-old male was transferred to our institution from a community hospital with the diagnosis of cardiac tamponade exhibiting cardiovascular collapse. He had been admitted to the surgical floor for abdominal pain with a tentative diagnosis of a paralytic ileus. An exploratory laparotomy was planned. While waiting for the procedure, he developed acute onset of shortness of breath, when a cardiologist was asked to see him. He was...
obtunded, tachycardic, and tachypneic with clear chest auscultation. His chest x-ray showed cardiomegaly with a globular shape to the heart silhouette with no acute infiltrates. His echocardiogram showed a very large pericardial effusion of at most 30 mm at the right atrial border, 15 mm at the right ventricular free wall, and 25 mm at the left ventricular posterior wall with early diastolic collapse of the right ventricle and collapse of the right atrium (Figure 1A and 1B).

Figure 1 A. Parasternal long-axis view of a large pericardial effusion.

Figure 1B. Apical four-chamber view of a large pericardial effusion with a marked right atrial collapse.

He was admitted to our institution for urgent pericardiocentesis. He was obtunded and in acute distress with shortness of breath on admission. His blood pressure was 70 mmHg/ systolic. His pulse rate was 130/min. His respiratory rate was 35–40/min. His jugular veins were distended, and heart sounds were muffled. His skin was diaphoretic. His extremities were cold and cyanotic. He was immediately taken to the cardiac catheterization lab where he underwent percutaneous pericardiocentesis with initial 500-600 ml of hemorrhagic fluid aspiration. Upon completion of the procedure his blood pressure normalized with relief of shortness of breath. He was transferred to the coronary care unit. An infectious diseases consultation was obtained because of increased WBC count. The diagnosis of CCHF was suspected because he had elevated liver enzymes, AST of 3651 IU/L, ALT of 2613 IU/L, LDH of 3159 IU/L, and increased prothrombin time to 26.4 s and INR of 2.11 suggestive of CCHF and he had been referred from a CCHF endemic region (Burdur) with a history of staying overnight in the field 10 days prior to admission. Later, he was isolated, barrier precautions were applied. He was initially put on broad spectrum antibiotics for the tentative diagnosis of paralytic ileus. Ribavirin was added to his regimen after the suspicion of CCHF infection was raised in addition to supportive therapy which included erythrocyte suspensions. His electrocardiogram showed a sinus tachycardia with reduced voltage. The rest of his laboratory work revealed wbc of 22.1k, hb of 9.6 g/dl, htc of 27.8%, plt of 202k, bun of 74.77 mg/dL, creatinin of 2.78 mg/dL, Na of 132 mmol/L, K of 6.4 mmol/L, total bilirubin 1.8 mg/dL, direct bilirubin 1.58 mg/dL, and CRP of 15.92. His thyroid function tests were normal. The pericardial fluid was grossly hemorrhagic and exudative in nature with protein of 3.2 g/dl, LDH of 402 IU/L, WBC count of 0.784k, Hb of 9.6 g/dl. There was no microorganisms or ARBs on microscopic examination. His culture results were negative. On the third day of his admission he developed cardiopulmonary arrest. He did not respond to resuscitative efforts and died. Refik Saydam Public Health Center (Ankara, Turkey) confirmed the diagnosis as CCHF by polymerase chain reaction method.

Discussion

Men become infected with CCHF virus through tick bites, by crushing infected ticks, or by contact with a patient with CCHF during the acute phase of infection. It is characterized by a common dramatic progression with hemorrhage, myalgia, and fever. The most common hemorrhagic manifestation sites are the nose, gastrointestinal system (haematemesis, melena, and intra-abdominal), uterus (menometrorrhagia) urinary tract (haematuria), and the respiratory tract (hemoptysis) [4]. Fatality from CCHF is due to severe gastrointestinal hemorrhage, cerebral hemorrhage, severe anemia and dehydration, and shock associated with prolonged diarrhea, myocardial infarction, lung edema, and pleural effusion [4-6]. Fatal illness in CCHF is characterized by rapid development of refractory shock, severe coagulopathy, and multifocal necrosis of the liver and other viscera [7]. Laboratory abnormalities include leukopenia, thrombocytopenia, elevated liver
and muscle enzymes. The pathophysiology of CCHF is not well described [4]. Heart functions have been hardly studied. Cardiac involvement may be seen especially in severe and fatal infections, and pericardial effusion may also be seen more frequently in severe and fatal cases [8]. The novel and important characteristics of the present case is that he had a very large hemorrhagic pericardial effusion with frank circulatory collapse. Urgent percutaneous pericardiocentesis was performed in the cardiac catheterization lab. Clinical improvement occurred after the drainage of few hundred mL of hemorrhagic fluid. There is currently no specific antiviral therapy for CCHF approved for use in humans by the FDA. Our case received ribavirin, broad spectrum antibiotics in addition to supportive measures. Unfortunately he died of cardiopulmonary arrest on the third day of admission. Clinicians taking care of critically ill patients with suspected or documented pericardial tamponade should also keep in mind the possibility of CCHF infection particularly if diagnosis is not evident on initial history and physical exam and if the patient is from an endemic area. Prompt procedures can be undertaken with extreme precautions not to transmit this contagious infection nosocomially.

References