An Unusual Cardiac Tamponade

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Abstract

Bacterial pericarditis with purulent pericardial effusion evolving into cardiac tamponade is rare in the modern practice. The present report describes an unusual presentation of a meningococcal infection: a cardiogenic shock. A fifty-year-old immunocompetent man exhibited signs and symptoms of infection accompanied by exacerbating chest pain mimicking a cardiovascular emergency e.g. aortic dissection or myocardial infarction or pulmonary embolism. A computed tomography scan performed in the Emergency Department was negative. Suddenly, patient’s conditions worsened rapidly for a cardiac tamponade, developed in only 24 hours, presenting as cardiogenic shock. Clinical findings improved with urgent pericardiocentesis. Neisseria Meningitidis was identified in the pericardial fluid and it was successfully treated with antibiotics. In such instances, rapid diagnosis and treatment are important to save the patient. This case emphasizes the challenge in making the diagnosis of an extremely rare occurrence of meningococcal pericarditis, especially in a low risk host. It is a rapidly life-threatening infection, and it is a medical emergency that could mimic several other pathological conditions.

ABBREVIATIONS

ED: Emergency Department; CRP: C-Reactive Protein; CT: Computed tomography; ECG: Electrocardiogram; ABG: Arterial Blood-Gas analysis; US: UltraSound; DMP: Disseminated Meningococcal Disease; IRP: Immuno Reactive Pericarditis; PMP: Primary Meningococcal Pericarditis

CASE PRESENTATION

A fifty years-old previously healthy man presented to the Emergency Department (ED) with two hours history of severe oppressive chest pain and fever over 38. On admission, he was agitated, and febrile. His physical examination was normal; his peripheral pulses were intact, palpable, and present bilaterally. Laboratory tests were notable for anemia (hemoglobin 11.4 g/dL), leukocytosis (white blood cell count 17,010/mm³). The procalcitonin was 29.48 ng/mL (n.v. 0.02-0.06 ng/ml), D-dimer 1580 mcg/mL and the C-reactive protein (CRP) level 29.9 mg/dL (n.v.<0.5). HIV test and cardiac enzymes resulted negative. Computed Tomography (CT) of the chest and abdomen revealed neither pulmonary embolism nor aortic dissection; no parenchymal abnormalities suggestive of infection or pericardial effusion were seen. Electrocardiogram (ECG) showed ubiquitous ST-segment elevation and a segment depression in lead aVR (Figure 1), arterial blood-gas analysis (ABG) while breathing ambient air showed only a slight hypocapnic hypoxemia with no acidosis (lactate levels 0.8 mmol/L). Oxygen therapy was started in addition to supplementation of water, electrolytes, and acid-base balancing. Empirical antibiotic therapy with ceftriaxone and levofloxacin intravenously had been started. During his hospitalization in the ED, the patient was persistently febrile despite antipyretics, and showed a rapid clinical deterioration. The patient was pale, sweating, hypotensive (blood pressure, 80/50 mmHg), suffering from pain and tachypnea with intercostal retraction despite oxygen treatment. ABG was repeated and revealed persistent hypocapnic hypoxemia and a respiratory acidosis. Point-of-care lung and heart ultrasound (US) evidenced the existence of a small consolidation in the right lung and a slight pericardial effusion with an impaired left ventricular systolic function (ejection fraction of 25-30%), indicating a myocardial dysfunction [1]. The patient did not show any signs of cardiac tamponade at this stage. ABG while improved oxygen therapy showed hypoxemia and hyperlactatemia (4.6 mmol/L). Intravenous morphine was used to reduce pain, a central venous catheter was placed, aggressive hydration with crystalloids was maintained and dobutamine infusion was initiated. The patient was admitted to the intensive care unit, because of severe septic shock. Vancomycin and nadroparin prophylaxis were added. After about 24 hours, a rapid decline of the conditions was demonstrated, even if he always remained conscious without a neurological involvement. An urgent transthoracic echocardiogram was done demonstrating a large circumferential pericardial effusion with right atrial...
diastolic collapse suggesting life-threatening cardiac tamponade (Figure 2). Emergent pericardiocentesis was performed with about 445 mL of purulent fluid drained with restoration of hemodynamic stability. A drainage was positioned for a few days. Neisseria Meningitidis serogroup W-135 was detected on culture of blood samples and pericardial fluid. Only treatment with ceftriaxone was maintained. In the following days, the patient also showed a concomitant respiratory failure. A chest X-ray detected bilateral pleural effusion. He was then transferred to the department of emergency medicine with the diagnosis of cardiogenic shock depending on cardiac tamponade in purulent pericarditis and right basal pneumonia caused by Neisseria Meningitidis. Antibiotic therapy was continued, later associated with gentamicin with a consequent complete defervescence and reduction of inflammatory markers (CRP 291 mg/dL). A checking with US was done, and showed a progressive reduction in the size of the pericardial effusion, but a worsening of his left pleural effusion. Thoracentesis was performed. Only 100 mL of blood-tinged fluid was obtained and its bacterial culture resulted negative. Steroid therapy was initiated with further improvement in gas exchange and complete absorption of pleural and pericardial effusion. The echocardiogram performed before discharge documented only a pericardial minimum detachment, it excluded the possibility of valvular dysfunction, or valvular vegetation. After discharge, this patient was in excellent health, and had a complete resolution of pleural and pericardial effusion after 2 weeks of antibiotics.
DISCUSSION

Purulent pericarditis is rare, accounting for 0.1% of pericarditis [2]. In Western cases, the most common organisms are staphylococci and streptococci [3]. A low pericardial: serum glucose ratio and elevated pericardial fluid neutrophils count differentiate purulent from tuberculous and neoplastic pericarditis. Culture of pericardial fluid is mandatory [4]. Death is inevitable if purulent pericarditis results untreated. Therapy consists of periocardiocentesis, and intravenous antimicrobial therapy at first empirically then based on microbiological results.

Meningococcal infection was first described by Vieusseaux in 1805 as epidemic cerebrospinal fever, and it is related to significant mortality, morbidity and long term sequelae. Neisseria meningitides, the causative agent, is a Gram-negative diplococcus, a common commensal organism of the human nasopharynx (8-25% of people) [5]. The disease caused by N. Meningitidis typically presents three main clinical manifestations: meningitis, sepsis and pneumonia. There may be rapid onset and progression of disease, and death may follow within hours. In about 10–15% of survivors, there are persistent sequelae including neurological defects, deafness, amputation of limbs or digits or skin scarring [6]. Acute meningococcal pericarditis is exceedingly rare clinical disorder, accounting for 5.9% of purulent pericarditis. Approximately 25 cases were reported in literature since the first case described in 1939 [7].

Meningococcal pericarditis can be classified into three categories: a local manifestation of disseminated meningococcal disease (DMP) that is purulent, culture-positive, associated with meningococcal bacteraemia; an immunoreactive pericarditis (IRP) that is immunological, late-onset, culture-negative, resembling post-viral pericarditis; and a primary meningococcal pericarditis (PMP) that is purulent, culture-positive but without signs of meningeval or other clinical systemic involvement, and it could evolve into cardiac tamponade and require pericardial drainage [8,9].

It is essential that clinicians recognize the various states of the disease. PMP is elusive at presentation, and is typically caused by Neisseria Meningitidis of serotype C (88% of cases), or, less commonly, B or W135 or more rarely Y [10]. Several mechanisms were proposed to explain the susceptibility to PMP, given its rarity: the lack of circulating antibodies against the pathogenic strains, the presence of blocking antibodies, complement system defects, and disease states associated with decreased complement levels [11].

In the present report, acute cardiac tamponade revealed a life-threatening rare case of Neisseria Meningitidis infection in a non-immunocompromised patient, without any other specific signs.

REFERENCES