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A Case of 25-Year-Old-HIV-Infected Male with Acute Pericarditis

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ABSTRACT

A cute pericarditis is defined as inflammation of the pericardium that surrounds the heart and the base of the great vessels. The classical presentation consists of chest pain, a pericardial friction rub and serial changes on electrocardiogram. We report a case of typical acute pericarditis in a 25-year-old male with HIV infection.

Keywords: Pericarditis; HIV infection

1. Introduction

A cute pericarditis is defined as inflammation of the pericardium that surrounds the heart and the base of the great vessels. The classical presentation consists of chest pain, a pericardial friction rub, and serial changes on electrocardiogram. Although data on the incidence of pericarditis are lacking, estimates indicate that it is the cause of at least 1% of emergency room visits among patients with ST-segment elevation and up to 5% of emergency room visits for nonischemic chest pain^{1,2}. The main pericardial syndromes encompass pericarditis (acute, subacute, chronic, recurrent), pericardial effusion, cardiac tamponade and pericardial masses. Pericarditis is the most common form of pericardial disease worldwide and is typically encountered in young and middle-aged people³. It represents 0.2% of all hospital admissions of cardiovascular aetiology and approximately 5% of patients with nonischaemic aetiology chest pain, presenting in the emergency departments of North America and Western Europe³. Acute pericarditis is the most common pericardial syndrome in clinical practice.

2. Case Presentation

A 25-year-old male patient was admitted to the hospital because of pericardial chest pain: sharp pain, increased when

breathing deeply and when lying down, decreased when sitting and bending forward. Past history of HIV infection. On review of systems, the patient reported no fever, chills, malaise, and a headache. He denied sore throat, nasal congestion, body aches, cough or ear pain. Further evaluation of the patient revealed the following vital signs: T 37°C Blood Pressure 120/60 mmHg Pulse 101 bpm Respiratory Rate 16 O₂ Sat 97% He did not appear toxic and his exam was normal. A rapid flu test was negative. His ECG (**Figure 1**) demonstrated diffuse concave-upward ST-segment elevation and PR-segment depression, ST-segment depression in aVR or V1. The patient's chest x-ray (**Figure 2**) showed an normal heart. Given the patient's history and clinical findings, he was referred to the emergency room for suspected pericarditis with pericardial effusion.

3. Discussion

Although viral infection is the most common identifiable cause of acute pericarditis, there are multiple other etiologies, as listed in (**Table 1**). The classic history of acute pericarditis begins with prodromal symptoms of fever, myalgia, and malaise. It is followed by acute onset of pleuritic, substernal chest pain that may radiate to the scapular ridge, neck, arms or jaw. The pain is usually relieved by leaning forward and made worse with

laying supine⁴. Other associated symptoms include low-grade intermittent fever, dyspnea, tachypnea, cough and dysphagia. A pericardial friction rub is the most specific physical exam finding in pericarditis (specificity approaching 100%), however, this exam finding is transient over time, has a low sensitivity, and may be present in only about 50% of cases^{4,5}. The rub is best heard over the left sterna border, during expiration with the patient leaning forward. It is characterized by a grating or rasping sound similar to leather rubbing together^{4,6}. A major life-threatening complication of acute pericarditis is cardiac tamponade. Pericardial effusion results from accumulation of fluid between the visceral and parietal layer of the pericardium. Tamponade occurs when the fluid pressure in the intrapericardial space alters cardiac filling. The classic signs as described by Beck's Triad are hypotension, jugular venous distension, and muffled heart sounds. Another important physical exam finding is pulsus paradoxus, a drop of at least 10 mmHg in arterial blood pressure on inspiration^{4,6}. Cardiac tamponade is a medical emergency and patients should be transferred to an emergency care setting for further evaluation. During acute pericarditis, ECG changes evolve through four stages as described in (Table 2)4,6-8. The hallmark ECG findings of diffusely concave upward ST elevation (not seen in V1 and aVR) with upright T waves, and a PR interval that deviates opposite of the P wave polarity are found during Stage I (Figure 1). Chest x-ray is usually normal in patients with pericarditis and minimal effusion. However, when a large amount of effusion is present (200- 250 mL), a chest x-ray will reveal a flask-shaped, enlarged cardiac silhouette, and a possible left-sided pleural effusion⁷. The chest x-ray taken of our patient demonstrated an normal cardiac silhouette. In this case, an echocardiogram would be warranted to further evaluate the significance of the effusion and assess cardiac function and there was no pericardial effusion. High sensivity cardiac troponin I were negative 2 times 3 hours apart.

4. Diagnosis

Presumed viral (HIV) pericarditis. Treatment for pericarditis is directed toward the underlying cause. For idiopathic and viral pericarditis, therapy should be directed toward symptom control. Nonsteroidal anti-inflammatory drugs (NSAIDS) are the mainstay of therapy⁶. Colchicine is a useful adjunct to NSAIDs and was once reserved for patient with recurrent or prolonged symptoms⁵. Data from the Colchicine for Acute Pericarditis Trial has led to its routine use by many practioners⁶. Corticosteroids are not recommended for first-line treatment unless indicated for the underlying disease or because of lack of response to NSAIDs or colchicine⁶. NSAIDs and steroids should not be used in pericarditis associated with acute myocardial infarction (MI). Pericardiocentesis is indicated when significant pericardial effusion is present, for both diagnostic and therapeutic purposes.

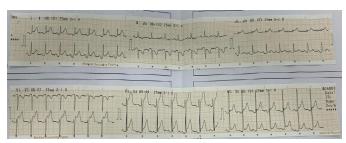


Figure 1: ECG show diffuse concave-upward ST-segment elevation and PR-segment depression, ST-segment depression in aVR or V1.



Figure 2: Chest Xray showed normal.

Table 1. Main Causes of Acute Pericarditis³

Categories	Causes	Frequency
A. Idiopathic	Unknown	Most frequent cause
B. Infectious Causes		
Viral	Epstein-Barr, influenza, hepatitis, human immunodeficiency virus, mumps, echovirus, adenovirus, cytomegalovirus, varicella, rubella, human herpesvirus, parvovirus, coxsackie	Most frequent cause in developed countries
Bacterial	Mycobacterium tuberculosis, Coxiella burnetii, streptococcus, staphylococcus, pneumococcus, legionella, salmonella, haemophilus	Rare (with the exception of mycobacterium tuberculosis)
Fungal	Candida, aspergillosis, histoplasmosis, blastomycosis	Very rare
Parasitic	Toxoplasma, echinococcus	Very rare
C. Non-Infectious Causes		
Neoplastic	Primary: pericardial mesothelioma Secondary tumours: leukemia, breast cancer, lung cancer, lymphoma, melanoma	Frequent as secondary metastasis
Metabolic	Hypothyroidism, renal failure, hypercholesterolaemia, gout, anorexia nervosa	Frequent
Cardiovascular	Acute myocardial infarction, Dressler's syndrome, aortic dissection	Frequent
Autoimmune	Rheumatoid arthritis, systemic lupus erythematosus, Sjogren syndrome, dermatomyositis, sarcoidosis, systemic vasculitides, Behçet's syndrome, familial Mediterranean fever	Frequent
Traumatic and iatrogenic	Catheterisation, surgery, chest trauma, radiation	Frequent

Categories	Causes	Frequency
Drug-related	Phenytoin, minoxidil, isoniazid, procainamide, hydralazine, methyldopa, doxorubicin, amiodarone, clozapine, streptomycin	Rare
Other	Congenital absence of pericardium	Rare

Table 2. Stages of ECG Changes During Acute Pericarditis^{3,4}

Stage I: Hallmark signs. Occurs in early stages of disease. Includes diffuse concave upward ST elevation, elevation not seen in leads aVR and V1, T waves are upright in the leads with ST

segment elevation, and PR segment deviates opposite of P wave polarity. **Stage II:** Occurs several days after onset of symptoms. ST segment return to baseline, and T waves flatten.

Stage III: T waves become inverted. No Q waves should be seen.

Stage IV: Weeks to months. EKG normalizes or if chronic pericarditis develops, T wave inversions may remain indefinitely

5. Conclusion

This case highlights several important issues for urgent care providers. First is the danger of "anchoring" to the diagnosis of a prior provider. All patients presenting to urgent care deserve a full investigation of their chief complaint with an open mind as to the cause. The clinical presentation of the patient in this case warranted further investigation to rule out other significant disease processes, such as MI and pulmonary embolism. The second important issue is that medical conditions are dynamic and evolve. While it may be tempting to criticize the first provider for having "missed" the diagnosis, we do not know if the key features of sharp positional chest pain, tachycardia were present 4 days prior. A third key issue is to make sure the clinical presentation is consistent with the patient's diagnosis. Several features make this case inconsistent with the original diagnosis of upper respiratory infection. The presence of positional pleuritic chest pain and subtle vital sign abnormalities and the absence of upper respiratory infection symptoms warranted the chest x-ray and ECG, which made the diagnosis obvious.

6. Author Contributions

The author wrote the manuscript. The author have read, reviewed, and approved the article.

7. Funding

No funding was received for this article.

8. Availability of Data and Materials

The datasets used during the current study are available from the corresponding author on reasonable request.

9. Declarations

Ethics approval and consent to participate

This study was performed in accordance with the Declaration of Helsinki. The patient gave informed consent, and the patient's anonymity was preserved.

Consent for publication

Written informed consent for publication was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

10. Competing Interests

The author declare that they have no competing interests.

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