Challenges in Management of Pericardial Effusion in Patients with HIV/AIDS

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Abstract

The presentation of pericardial effusion is variable relative to the effects on the hemodynamics; hence the diagnosis can be made with certainty by echocardiography. Reaching the etiological cause may be challenging, particularly in HIV/AIDS patients due to their susceptibility to variety of uncommon pathogens as well as several malignancies. In this brief article, two major challenges are highlighted; first, the benefit versus the risk of diagnostic aspiration of pericardial fluid. Secondly: the use of oral corticosteroids in the settings of tuberculous pericarditis.

Keywords: Pericardial effusion; HIV; AIDS; Aspiration; Corticosteroids; Tuberculosis

Introduction

Pericardial effusions (PE) are not uncommon findings in patients with HIV/AIDS reportedly affecting nearly 5% of these patients [1-4]. The quantity of PE has been classified into mild, moderate and large [5,6]. It has been demonstrated that cardiac tamponade, a serious hemodynamic medical emergency as a result of pericardial effusion, is not necessary associated with large effusions but rather with the rapid rate of PE accumulation and lack of compensatory compliance within the pericardial space [7-10]. Moreover, the severity of PE in HIV/AIDS patients has been shown to correlate with poor prognosis before the widespread use of highly active antiretroviral therapy (HAART), nonetheless, prognostic implications of PE cannot be determined as HAART has altered the natural history of PE in these patients [11,12].

The management of PE in patients with HIV/AIDS has three major steps; first: clinical suspicion of PE based on history and physical examination. Second: reaching all aspects of diagnosis including assessing the quantity of PE, the hemodynamic effects of PE, and the etiology of PE. The third step in the management is essentially treatment decisions as well as vigilant monitoring of patient’s response. Benefits versus the risk of inserting a needle in the pericardial space for aspiration may be challenging. Additionally, in the settings of tuberculous pericardial effusion, the question of adding corticosteroids needs to be addressed in the presence of HIV/AIDS as well as HAART.

Aspiration of PE

In patients with HIV/AIDS, the presentation of PE may follow the recognized symptoms and signs or it may be atypical [13]. An echocardiography that detects the presence of PE is frequently obtained for further evaluation of cardiomegaly seen on a plain chest radiograph with very high sensitivity and specificity [14]. Asymmetrical pericardial deposits of variable echogenicity and size ranging from 4 to 8 millimeters may suggest a diagnosis of tuberculous PE (TPE) when visualized by echocardiography. Moreover, adhesive echogenic strands between the parietal and visceral pericardium in TPE can be missed unless looked for carefully during echocardiographic imaging acquisition. Transesophageal echocardiography is necessary for proper assessment of the posterior pericardium that is not clearly seen on transthoracic windows. The common causes of PE in patients with HIV/AIDS are shown in table 1. Additional information gained by echocardiographic evaluation that aid in making the likely diagnosis, are summarized in table 2.

Percutaneous aspiration of pericardial fluid for diagnostic purpose may be unnecessary in patients with HIV/AIDS specifically for small effusions with high clinical index of the underlying cause of PE. Pericardiocentesis is an invasive procedure with considerable complications in patients with HIV/AIDS that carries minor but definite mortality [15-17]. Hazardous complications associated with pericardiocentesis may include hemopericardium, pneumopericardium, cardiac perforation, introduction of infections, and arrhythmias.

Furthermore, the yield of establishing the etiology of PE from samples obtained by pericardiocentesis such as tuberculosis may not outweigh the potential risk. Cardiac imaging by computed tomography (CT) and magnetic resonance imaging (MRI) may have a higher

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Table 1: Etiology of pericardial effusion in HIV/AIDS.

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diagnostic efficiency than pericardiocentesis in PE particularly, when loculated or pericardial thickening are suspected by echocardiography [18]. CT and MRI provide images of mediastinal lymph nodes that are characteristically enlarged in patients with TPE, in addition to other abnormalities in the lung fields. CT attenuation measurements are helpful in differentiating transudates, exudates and solid masses in the pericardium. Pericardial calcification is quite clear on CT images in patients presenting with constrictive effusive pericarditis”.

**Corticosteroids Therapy for Tuberculous Pericarditis**

Studies have demonstrated the rationale of initiating oral corticosteroids along with anti-tuberculous therapy to prevent development of constrictive pericarditis [19]. Another study questioned the beneficial effect of steroids on re-accumulation of pericardial effusion or progression to constrictive pericarditis [20].

Nonetheless, corticosteroids use is risky in patients with HIV/AIDS as reported exacerbation of AIDS-associated Kaposis sarcoma in the setting of iatrogenic Cushing syndrome caused by an interaction between protease inhibitor and steroids [21]. Moreover, severe adrenal suppression has been reported as well [22].

Pericardial constriction as a result of tuberculous pericarditis in patients with HIV/AIDS has been reported to occur less frequently than in immunocompetent patients [23,24]. Conversely, another study showed that adjunctive corticosteroids for tuberculosis pericarditis produced a marked reduction in mortality and suggested the addition of steroids to treat HIV related effusive tuberculous pericarditis [25].

It remains controversial whether adjunctive corticosteroids are essential effective in treatment of tuberculous pericarditis in patients with HIV/AIDS; nonetheless, it is imperative to monitor the potential harm of steroids in these patients when administered.

**References**