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Full Length Research Paper

Etiological Profile of Pericardial effusion in Kashmir: A Study from Northern India

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Abstract

Pericardial effusion is one of the common diseases presenting in emergency and outpatient departments of a tertiary care center. The etiology of pericardial effusion varies in different parts of the world and is related to the relative prevalence of different diseases known to cause it. Although there is abundant literature regarding the clinical and etiological profile of pericardial effusion and cardiac temponade in developed countries, it remains under reported in developing countries like India. This study was conducted to assess the etiological spectrum of pericardial effusion in northern India. This single center observational study included 102 patients presenting as moderate to severe pericardial effusion over a period of two years from March 2012 to February 2014. Iatrogenic and post traumatic cases were excluded. The diagnosis of pericardial effusion was established by echocardiography, seen as echo-free space (representing pericardial fluid) surrounding the heart , more than 10 mm deep in front of the right ventricle and beyond the left ventricle. Evaluation for the cause of PE included complete blood count with ESR, Blood urea, serum creatinine, tuberculin skin test, Chest X-ray, QuantiFERON TB-GOLD, Thyroid profile, ANA, Rheumatoid factor and imaging (CT chest or MRI). Pericardiocentesis and pericardial fluid analysis was done only in patients who were in temponade and those who had persistent/recurrent effusion extending beyond three months. Final diagnosis was based on history, examination and specific laboratory investigations. The major cause of pericardial effusion is tuberculosis (24.5%) followed by malignancy (16.6%), uremia (15.6%) and acute pericarditis (idiopathic/viral)(14.7). Other causes of significant pericardial effusion are collagen vascular disease (5.8%), hypothyroidism (3.9%), CCF (3.9), myocardial infarction (2.9), pyogenic infection (1.9%), mediastinal irradiation (1.9%) and HIV infection (1.9%). Six patients (5.8%) had significant effusion of more than three months duration in whom no cause could be established. The etiological profile of pericardial effusion in developing country differs from what is seen in developed countries. Tuberculosis is the most common cause in our study in contrast to studies from the developed region where malignancy remains the leading cause.

Keywords: Pericardial effusion; Temponade; Etiology; Pericarditis; Tuberculosis.

INTRODUCTION

Pericardium normally contains up to 50 ml of serous fluid within its two layers (Shabetai, 2003). Pericardial effusion (PE) is the presence of an abnormal amount of fluid in the pericardial space. It is caused by a variety of local and systemic disorders, or may be idiopathic. Pericardial effusions can be acute or chronic, and the

time course of development has a great impact on the patient's symptoms. The cause of abnormal fluid production depends on the underlying etiology, but usually is secondary to injury or insult to the pericardium called pericarditis. Transudative fluids result from obstruction to fluid drainage, which occurs through lymphatic channels. Exudative effusion occurs secondary to inflammatory, infectious, malignant or autoimmune processes within the pericardium.

Clinical manifestations of pericardial effusion are highly dependent on the rate of accumulation of fluid in the pericardial sac. Rapid accumulation of pericardial fluid may cause elevated intrapericardial pressures with as little as 80 mL of fluid, while as slowly progressing effusions can accumulate upto 2 liters without symptoms (Shabetai, 2003; LeWinter and Hopkins, 2014; Braunwald, 2011;Willerson and Cohn, 2007; Maisch et al., 2004; Dudzinski et al., 2012).

Commonly encountered causes of pericardial effusion are infectious/idiopathic pericarditis, malignancy, renal failure and collagen vascular disease. Pericardial effusion resulting from acute pericarditis of no more than 1 to 2 weeks duration is considered idiopathic. Most idiopathic cases are presumed to be of viral etiology, but testing for specific viruses is not routinely done because of the cost involved, low yield, and negligible impact on management. (Abu Fanne et al., 2011; Sagrista-Sauleda et al., 2000; Goland et al., 2000; Sagristà-Sauleda et al., 1999; Vithalani, 1992; Cheema et al., 1999; Colombo et al., 1988; Corey et al., 1993). Pericardial effusion leading to hemodynamic compromise should be subjected to pericardiocentesis. However it is recommended that asymptomatic and hemodynamically stable patients need close follow up and often there is no need for intervention in such cases. Idiopathic chronic pericardial effusion constitutes 15-20% and can even lead to large pericardial effusion and cardiac tamponade. It is defined as collection of pericardial fluid that persists for greater than three months with no apparent cause. Large idiopathic pericardial effusion is well tolerated for a long period by most patients but severe pericardial effusion can occur at any time. (Chong and Plotnick, 1995; Cherian et al., 2004; Sagristà-Sauleda et al., 2011; Adler et al., 1998; Mercé et al., 1998; Tsang et al., 2003)

We studied the clinical profile of pericardial effusion in our patients and also to determine the correlation with the reported literature.

MATERIAL AND METHODS

102 hospitalized patients were evaluated at our hospital, a tertiary care centre, over a period of two years from March 2012 to February 2014.

Iatrogenic (cardiac surgery, catherterization) and post traumatic cases were excluded. The diagnosis of pericardial effusion was established by echocardiography, seen as echo-free space (representing pericardial fluid) surrounding the heart , more than 10 mm deep in front of the right ventricle and beyond the left ventricle.

Evaluation for the cause of PE included complete blood count with ESR, Blood urea, serum creatinine, tuberculin skin test, Chest X-ray, QuantiFERON TB-GOLD (interferon-γ release assay used for diagnosis of tuberculosis), Thyroid profile, ANA, Rheumatoid factor, CT chest / MRI and pericardiocentesis. Pericardial fluid was analysed for cells, proteins, LDH, malignant cells, ADA, PCR (for mycobacterium tuberculosis), gram staining, AFB staining and cultures.

Final diagnosis was based on clinical history, examination, and specific laboratory investigations for tuberculosis, uraemia, malignancy, collagen vascular disease hypothyroidism etc. The size of the Effusion was categorized as moderate-sized when 10 to 20 mm and severe when more than 20 mm. Effusion measuring less than 10 mm in absence of tamponade was not included. Tamponade was defined as early diastolic right atrial and/or ventricular collapse or more than 25% change in mitral inflow velocities with respiration in presence of tachycardia, hypotension or significant paradox. The diagnosis of acute idiopathic/viral etiology was presumptive and was based on the clinical picture, and negative screening tests for other etiologies. Therapeutic Echo-guided percutenaous pericardiocentesis was performed by placing pigtail catheter in pericardial space through subxiphoid approach in 39 patients presenting with cardiac tamponade. Six patients having long standing (>3 months) and those with recurrent pericardial effusion were also subjected to pericardiocentesis as part of evaluation after all non invasive tests were inconclusive.

RESULTS

The study included 102 patients with age ranging from 7 to 80 years. Fifty three patients (51.6 percent) were males and forty nine (48.4 percent) were females. The most common symptom was shortness of breath (n=64; 62.9%) followed by chest pain (n=54; 53.2%), fever (n=45; 44.6%) and cough (n=29; 28%). Two patients (1.9%) were asymptomatic and were found to have pericardial effusion on echocardiography after having done routine chest X-ray which showed cardiomegaly. Tachycardia was present in 65 patients (64%), hypotension in 24 patients (23.5%), and significant paradox was present in 36 patients (35.3%). Electrical alternans was present in only 6 patients (5.8%) and cardiomegaly on chest radiograph was present in 64 patients (63.7%). (Table 1)

Out of 102 patients,65 patients (63.7%) presented with moderate pericardial effusion and 37 patients (36.3%) presented with severe pericardial effusion.

Table 1. Showing demographic, clinical and laboratory characteristics. (HR: Heart rate; ECG: electrocardiogram; CXR: Chest X-ray; PE: Pericardial effusion)

Clinical/laboratory characteristic	Number	Percentage
Total number of patients	102	100
Age range (in years)	7-80 (Mean=44.4 yrs)	
Males	53	51.6
Females	49	48.4
Symptoms		
Breathlessness	64	62.9
Chest pain	54	53.2
Fever	45	44.6
Cough	29	28.0
Other symptoms	16	15.6
Asymptomatic	2	1.9
Signs		
Tachycardia (HR>100 bpm)	65	64
Pulses paradoxes	36	35.3
Hypotension (systolic BP<90 mmHg)	24	23.5
Electrical alternans on ECG	6	5.8
Cardiomegaly on CXR	64	62.7
Echo		
Moderate PE (10-20 mm)	65	63.7
Severe PE (>20 mm)	37	36.3
Tamponade (clinical+echo)	39	38.2

Thirty nine patients (38.2%) had echocardiographic as well as clinical evidence of cardiac tamponade.

The most common etiology of pericardial effusion was tuberculosis (n=25; 24.5%), followed by malignancy (n=17; 16.6%), uremia (n=16; 15.6%), acute idiopathic/viral (n=15; 14.7%) collagen vascular disease (n=6; 5.8%) hypothyroidism (n=4; 3.9%), CCF (n=4; 3.9%), post MI (n=3; 2.9%), pyogenic (n=2; 1.9%), radiation (n=2; 1.9%) and HIV (n=2; 1.9%) respectively. Six patients (5.8%) had large pericardial effusion of more than 3 months duration in which no etiology could be established after extensive evaluation and emperical trial of antitubercular treatment to which they didn't respond (table 2). Among malignancies, carcinoma lung was the most common (n=8), followed by breast carcinoma (n=3) and lymphoma (n=2). Mediastinal germ cell tumor, esophageal carcinoma, mesothelioma and metastatic adrenal tumor were present in one patient each.

Malignancy was the most common cause in patients presenting with tamponade (n=13; 33.3%) followed by tuberculosis (n=12; 30.7%), uremia (n=5; 12.8%) and acute viral/idiopathic pericarditis (n=4; 10.2%) respectively (table 3). Pyogenic pericarditis was present in two patients and both presented with tamponade of which one died on third day due to multi-organ failure. SLE, hypothyroidism and CCF was the cause of temponade in one patient each.

Out of 102 patients, 13 patients (12.7%) died during hospital stay. The underlying etiology of these patients included malignancy (n=6), uremia (n=3), tuberculosis (n=1), postMI (n=1), pyogenic pericardial effusion (n=1) and CCF (n=1). Among these patients who died, 9 had cardiac tamponade and had undergone pericardiocentesis.

DISCUSSION

Pericardial effusion results from the abnormal accumulation of fluid within pericardial space as a result of insult to pericardium due to multiple causes. It can occur at any age but age specific etiologies may differ. The clinical presentation of pericardial effusion depends on the underlying etiology and the rapidity with which fluid accumulates within the pericardial space. Rapid accumulation of as little as 200 ml of fluid within pericardial space may result in impairment in ventricular filling and decreased cardiac output leading to clinical features of cardiac tamponade. On the other hand gradual accumulation of even 2000 ml of fluid within the pericardium may not produce cardiac tamponade as pericardium gets ample time to adapt to this slowly accumulating fluid. (Shabetai, 2003; LeWinter and Hopkins, 2014; Braunwald, 2011; Willerson and Cohn, 2007; Maisch et al., 2004; Dudzinski et al., 2012)

The commonest symptoms include shortness of breath, chest pain, fever and cough. Pericardial effusion may also present with hiccups (phrenic nerve stimulation), hoarsness of voice (involvement of recurrent laryngeal nerve), dysphagia (esophageal compression) or nausea (vagal stimulation). Commonest

Table 2. Showing etiological profile of moderate to large pericardial effusion. (CCF: Congestive cardiac failure; MI: Myocardial infection; HIV: human immunodeficiency virus)

Diagnosis	*Number*	*Percentage*
Tubercular	*25*	*24.5*
Malignant	*17*	*16.6*
Ureamia	*16*	*15.6*
Acute pericardial effusion (Idiopathic/ viral)	*15*	*14.7*
Collagen vascular disease	6	5.8
Chronic idiopathic pericardial effusion (etiology unknown)	*6*	*5.8*
CCF	*4*	*3.9*
Hypothyroidism	*4*	*3.9*
Post MI	*3*	*2.9*
Pyogenic	*2*	*1.9*
Radiation	*2*	*1.9*
HIV- infection	*2*	*1.9*
Total	102	100.0

Table 3. Number and percentage of patients presenting as tamponade in different etiological groups

Etiology	*Number*	*Percentage*
Malignancy	13	33.3
Tuberculosis	12	30.7
Uremia	5	12.8
Acute/idiopathic	4	10.2
Others	5	12.8
Total	39	100

symptoms in our patients were breathlessness, chest pain and fever.

Hemodynamically significant pericardial effusion may present with classical Beck's triad of hypotension, distended neck veins and distant heart sounds. (Shabetai, 2003; LeWinter and Hopkins, 2014; Braunwald, 2011). Thirty-nine (38.2%) of our patients had clinical features of cardiac tamponade.

Most common ECG findings in our patients were sinus tachycardia, non specific ST-T wave changes and low voltage QRS complexes.

In our study the leading cause of significant pericardial effusion was tuberculosis followed by malignancy, uremia and idiopathic/viral etiology. Tuberculosis remains the most common cause in developing countries. However, in industrialised countries; malignancy is the commonest cause of significant effusion and tamponade. Acute idiopathic pericardial effusion is mostly of viral etiology, however testing for specific viruses is not routinely done because of its disproportionate cost-benefit ratio, low yield and negligible impact on management. (Abu Fanne et al., 2011; Sagrista-Sauleda et al., 2000; Goland et al., 2000; Sagristà-Sauleda et al., 1999; Vithalani, 1992; Cheema et al., 1999; Colombo et al., 1988; Corey et al., 1993) Echocardiography is the procedure of choice for the diagnosis of pericardial effusion. 2D and M-mode echocardiography show the presence of echo free space between two pericardial layers in both phases of cardiac cycle. In addition it may be helpful in detecting localized effusion as well as septations within the pericardial space. It is also utilized as a useful tool to guide pericardiocentesis. Pericardial effusion is easily detected by computed tomography (CT). The attenuation coefficient of pericardial fluid may be helpful in depicting its characteristics like blood, serous fluid, chyle or hydropericardium. Mediastinal lymphadenopathy and thickened pericardium on CT chest has high sensitivity for detecting tuberculous pericardial effusion. (hong and Plotnick, 1995; Cherian et al., 2004; Sagristà-Sauleda et al., 2011). CT scan proved useful in arriving at the diagnosis in our patients having tuberculosis or malignancy.

Acute idiopathic or viral pericardial effusion without tamponade may respond to nonsteroidal anti-inflammatory drugs, steroids, or colchicine. All large pericardial effusions do not need drainage. However urgent pericardiocentesis should be done whenever there is actual or threatened tamponade and may prove

life saving. Purulent pericardial effusion should also be drained even in absence of tamponade, However surgical drainage has shown better outcome in such cases. Pericardiocentesis may occasionally be required to establish the etiology of hemodynamically insignificant pericardial effusion. However, it has been shown that analysis of pericardial fluid alone in general has low yield in providing specific diagnosis. Persistent (greater than 3 months) large or progressive effusion, particularly when the cause is uncertain, also warrants closed pericardiocentesis and in many such cases effusion may not re-accumulate. For most patients with clinically significant idiopathic pericardial effusion requiring intervention, echo-guided pericardiocentesis is the definitive treatment. Pericardiectomy is necessary for patients in whom effusion occurs in context of effusive constrictive disease, chronic relapsing pericarditis, or recurrent effusion despite pericardiocentesis. (Sagristà-Sauleda et al., 2011; Adler et al., 1998; Mercé et al., 1998; Tsang et al., 2003).

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