Clinical Etiological and Imaging Profile of Pericardial Effusion

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Abstract- Pericardial effusion is especially important clinically when it develops within relatively short time as it may lead to cardiac tamponade. Cardiac Tamponade is common medical emergency which can be fatal if un recognized in acute medical care units.

The pericardium may be involved in large number of systemic disease or may be diseased as an isolated process. The principle manifestations of pericardial disease are pericarditis and pericardial effusion while these are distinct phenomena most patients with pericardial disease present with both manifestations to greater or lesser degree. In our study pericardial effusion, predominant cause was tuberculosis in 28 patients (56%) followed by uremic pericarditis in 8 patients (16%) hypothyroidism was responsible in 5 patients (10%), bacterial pericarditis in 4 patients (8%), viral / idiopathic pericarditis in 3 patients (6%), trauma in 1 patient 2%, post myocardial infarction in 1 patient (2%).

Index Terms- Pericardial effusion, cardiac tamponade, tuberculosis, pericardiocentesis

I. INTRODUCTION

The pericardium is a fibrous sac surrounding the heart, the thicker, outer parietal pericardium and an inner, thinner visceral layer. The two layers are separated by small amount of pericardial fluid (25 to 50 mL) produced by the visceral pericardium. It is an ultrafiltrate of the plasma and is resorbed by the lymphatics. The pericardium prevents friction between the heart and surrounding structures, acts as a Mechanical and immunological barrier, and limits distention of the heart thereby maintaining a relatively fixed maximal heart volume. The most common forms of pericardial diseases include acute and recurrent pericarditis, isolated pericardial effusion with or without cardiac tamponade, and constrictive pericarditis.

Pericarditis

Pericarditis is inflammation of the pericardium with or without an associated pericardial effusion.

It can be of three types:

1. Acute (dry or effusive) pericarditis
2. Chronic (effusive, adhesive, or constrictive)—lasting three months or more.
3. Recurrent pericarditis[1]

1) Acute pericarditis, defined as symptoms and / or signs resulting from pericardial inflammation of no more than 1 to 2 weeks duration, can occur in a variety of these diseases, but most cases are considered idiopathic[2,3,4]. Most idiopathic cases are presumed to be viral in cause, but testing for specific viruses is not routine because of cost, low yield, and negligible impact on management[5].
2) Chronic pericarditis - Pericardial inflammation (>3 months) includes effusive, adhesive, and constrictive forms[6,7]. It is important to differentiate chronic inflammatory effusions from non-inflammatory hydropericardium (congestive heart failure). The detection of the curable causes (e.g., tuberculosis, toxoplasmosis, myxedema, autoimmune, and systemic diseases) allows specific therapy with high success rate[8].
3) Recurrent pericarditis the term recurrent pericarditis encompasses (1) the intermittent type (widely varying symptom-free interval without therapy) and (2) the incessant type (discontinuation of anti-inflammatory therapy always ensures a relapse).

II. MATERIAL & METHODS

PATIENTS: All patients presenting with pericardial effusion from 2007 to 2009 at Department of Medicine, cardiology and nephrology were studied. Total 50 patients were studied in this study.

Inclusion criteria

Patients who presented with pericardial effusion including cardiac tamponade based on clinical criteria and confirmed by echocardiography were included in the study.

Methods: In all 50 patients detailed history was taken and complete clinical examination was done. In addition to regular investigations were done for all patients.

Hemogram ; ESR ; Complete urine examination ; Blood urea ; Sr. creatinine; X ray chest PA view; ECG

2 D echocardiogram and Doppler studies were done for all patients and studied for homodynamic changes suggestive pericardial effusion. Pericardiocentesis was done in cardiac cath lab under fluoroscopic guidance. Pericardial fluid was analyzed for protein, sugar, cell count, ADA levels, gram stain, smear for AFB and sensitivity. Miscellaneous investigations ELISA for HIV 1 & 11 and computed tomogram of chest, serum TSH and T3 & T4 were done in selected patients.

III. OBSERVATIONS & RESULTS

Showing percentage distribution of symptoms & Percentage distribution of vital data

Symptoms	No. of Patients	Percentage
SOB	42	84%
Symptoms S/o CHF	36	72%
Fever	34	68%
Chest pain	5	10%
Pulse rate > = 100 / mt < 100	15 35	30% 70%
Systolic blood pressure > = 100 / mm Hg < 100 mm Hg	 38 12	 76% 24%
Pulse paradoxus 20 – 40 without pulses paradoxus	12 18	24% 76%

42 (84%) patients had shortness of breath, 36 (72%) patients had symptoms suggestive of congestive heart failure, 34 (68%) patients had fever, 5 (10%) had chest pain, and 15 (30%) had cough. Pulse rate was greater than 100 / mt in 15 (30%), less than 100/mt in 35 (70%) patient, systolic blood pressure less than 100 / mt in 12 (24%) patients, pulses paradoxus seen in 12 (24%) due to cardiac tamponade.

Showing percentage distribution of electrocardiographic and radiological changes

ECG & X ray changes	No. of patients	Percentage
ECG Low voltage complexes ST / T changes Electric alternans	 42 8 3	 84% 16% 6%
X Ray Chest PA View Cardiomegaly Findings S/o pulmonary Koch's Pleural effusion	 50 6 4	 100% 12% 8%

Electrocardiographic changes in order of frequency were low voltage complexes in 42 patients (84%), ST / T Changes in 8 patients (16%), and electric alternans in 3 patients (6%).

Radiological changes in x ray chest PA view showed predominately cardiomegaly in 50 patients, findings suggestive of pulmonary Koch's in 6 patients (12%), pleural effusion in 4 patients (8%).

Echocardiographic findings of tamponade	No. of patient	Percentage	TB	CRF	Viral	Pyogenic	Traumatic	Hypothyroidism	Post MI
Echo free space Anterior -1-3 cm >3 cm posterior 1-3 cm > 3 cm	 38 12 36 14	 76% 24% 72% 28%	 20 8 20 8	 6 2 6 2	 3 - 3 -	 3 1 3 1	 - 1 - 1	 5 - 3 2	 1 - 1 -
Right Atrial Collapse	12	24%	7	2	-	1	1	1	-
Right ventricular Diastolic collapse	12	24%	7	2	-	1	1	1	-
Left atrial collapse	6	12%	3	1	-	-	1	1	-
Fibrin strands	18	36%	15	2	1	-	-	-	-
Amount of fluid large moderate mild	 12 24 14	 24% 48% 28%	 8 15 5	 2 4 2	 - 1 2	 1 1 2	 - 1 -	 1 2 3	 - - 1

Echocardiographic evidence of tamponade indicated by right atrial collapse, right ventricular diastolic collapse was seen in 12 patients (24%), left atrial collapse was seen in 6 patients (12%) most of the patients with large fluid collections as in tuberculosis, uremia, hypothyroidism and pyogenic pericardial effusion. Fibrin strands were seen in 18 patients (36%) predominantly in tuberculosis, uremia and viral pericardial effusions the amount of fluid was large in 12 patients (24%), especially in tuberculosis, uremic, pyogenic and myxedematous pericardial effusion, moderate pericardial effusion seen in 24 patients (48%), mild pericardial effusion seen in 14 patients (28%).

Showing percentage distribution of etiology in pericardial effusion

Etiology	No. of patients	Percentage
Tuberculous pericarditis	28	56%
Uremic Pericarditis	8	16%
Hypothyroidism (myxedematous pericardial disease)	5	10%
Bacterial pericarditis	4	8%
Viral / Idiopathic pericarditis	3	6%
Traumatic	1	2%
Post MI	1	2%

IV. CONCLUSION

- Pericardial effusion is not an uncommon medical emergency in acute medical care wards that is nearly curative if recognized early.
- Pericardial tamponade is medical emergency condition if not treated early it may lead to death.
- Shortness of breath was the most common symptoms, and clinical signs of cardiac tamponade are raised jugular venous pressure, hypotension, pulses paradoxus and diminished heart sounds seen in most of the cases of pericardial effusion.
- Echocardiography is the method of choice for evaluation most pericardial diseases. When competently performed in patients with good acoustic windows, echocardiography accurately detects all pericardial effusion and provides clinically relevant information about their size and homodynamic importance.
- Most of the chest X – ray's shown enlargement of the cardiac silhouette.
- The ECG findings suggestive of low voltage QRS complexes (low voltage is usually defined as QRS complexes < 5 mm (0.5 mV) in all of the limbs leads, and QRS amplitude of < 10 mm in V1 to V6) seen in most of the cases with pericardial effusion and cardiac tamponade.
- The biochemical, cytological, and bacteriologic characteristics of an effusion are established by examination of the pericardial fluid.
- In Our 50 patients with pericardial effusion, predominant cause was tuberculosis in 28 patients, ADA levels of > 40 U/Lit was seen and diagnostic of tuberculosis pericardial effusions. (56%) followed by uremic pericarditis in 8 patients (16%) hypothyroidism was responsible in 5 patients (10%), bacterial pericarditis in 4 patients (8%), viral / idiopathic pericarditis in 3 patients (6%), trauma in 1 patients 2%, post myocardial infarction in 1 patients (2%).

V. DISCUSSION

Pericardial effusion may appear as transudate (hydropericardium), exudate, pyopericardium or haemopericardium. Large effusions are common with neoplastic, tuberculous, cholesterol, uremic pericarditis, myxedema, and parasitoses[9]. Effusions that develop slowly can be remarkably asymptomatic, while rapidly accumulating smaller effusions can present with tamponade. Loculated effusions are more common when scarring has supervened (e.g., postsurgical, post trauma, post purulent pericarditis). Massive chronic pericardial effusions are rare (2–3.5% of all large effusions).

Cardiac Tamponade

The accumulation of fluid in the pericardial space in a quantity sufficient to cause serious obstruction to the inflow of blood to the ventricles results in cardiac tamponade. This complication may be fatal if it is not recognized and treated promptly. The three most common causes of tamponade are neoplastic disease, idiopathic pericarditis, and renal failure. Tamponade may also result from bleeding into the pericardial space after cardiac operations, trauma, and treatment of patients with acute pericarditis with anticoagulants.

The three principal features of tamponade (*Beck's triad*) are hypotension, soft or absent heart sounds, and jugular venous distention with a prominent *x* descent but an absent *y* descent. There are both limitation of ventricular filling and reduction of cardiac output. The quantity of fluid necessary to produce this critical state may be as small as 200 mL when the fluid develops rapidly or >2000 mL in slowly developing effusions when the pericardium has had the opportunity to stretch and adapt to an increasing volume[10]

Cardiac tamponade is the decompensated phase of cardiac compression caused by effusion accumulation and the increased intra pericardial pressure. In "surgical" tamponade intra pericardial pressure is rising rapidly, in the matter of minutes to hours (i.e., Haemorrhage), whereas a low-intensity inflammatory process is developing in days to weeks before cardiac compression occurs ("medical" tamponade). The volume of fluid causing tamponade varies inversely with both parietal pericardial stiffness and thickness (150–2000 ml). In local compression, dyspnoea, dysphagia, hoarseness (recurrent laryngeal nerve), hiccups (phrenic nerve), or nausea (diaphragm) can occur. Heart sounds are distant. Compression of the base of the lung results in a dullness under the left scapula (Bamberger–Pins–Ewart's sign)[7]. In 60% of the patients, the cause of pericardial effusion may be a known medical condition[11]. Tamponade without two or more inflammatory signs (typical pain, pericardial friction rub, fever, diffuse ST segment elevation) is usually associated with a malignant effusion (likelihood ratio 2.9). Electrocardiography may demonstrate diminished QRS and T-wave voltages, PR-segment depression, ST-T changes, bundle branch block, and electrical alternans (rarely seen in the absence of tamponade)[12]. In chest radiography large effusions are depicted as globular cardiomegaly with sharp margins ("water bottle" silhouette). On well-penetrated lateral radiographies, or better on cine films, pericardial fluid is suggested by lucent lines within the cardiopericardial shadow (epicardial halo sign).

Diagnosis

Specific diagnosis are possible using visual, cytologic, and immunologic analysis of the pericardial effusion and pericardioscopic guided biopsy. Echocardiography is the procedure of chronic for the diagnosis of pericardial effusion. Although flask – shaped enlargement of the cardiac silhouette on chest radiography occurs with a moderate or large pericardial effusion. The diagnostic feature on M – mode echocardiography is the persistence of an echo-free space between parietal and visceral pericardium throughout the cardiac cycle. Pericardial effusions are easily detected by computed tomography. The size, geometry, and distribution of pericardial effusions can be obtained with this technique, and the attenuation coefficients for blood, exudate, chyle, and serious fluid are generally sufficiently characteristic to identify the nature of the effusion. Computed tomography may be useful in estimating the hematocrit of the pericardial effusion[17] identifying loculated and atypically loculated pericardial effusions, and in guiding pericardiocentesis[13].

Magnetic resonance imaging (MRI) detects pericardial effusion with high sensitivity and provides an estimate of pericardial fluid volumes.

Treatment

Drainage of a pericardial effusion is usually unnecessary unless purulent pericarditis is suspected or cardiac tamponade supervenes, although pericardiocentesis is sometimes needed to establish the etiology of haemodynamically insignificant pericardial effusion. Persistent (greater than 3 months) large or progressive effusion, particularly when the cause is uncertain, also warrant pericardiocentesis[18.] However, routine drainage of large pericardial effusion without tamponade or suspected purulent pericarditis has a low diagnostic yeile (7percent) and no clear therapeutic benefit. Anticoagulants should be discontinued temporarily, if possible, to reduce the risk of cardiac tamponade. In patients on chronic oral anticoagulation, heparin should be used, as its effect can be reversed rapidly. Large effusions may respond to nonsteroidal anti-inflammatory drugs, corticosteroids, or colchicine[14.]

Pericardiocentesis is lifesaving in cardiac tamponade (level of evidence B, class I indication)[15]. Aortic dissection is a major contraindication[16]. Relative contraindications include uncorrected coagulopathy, anticoagulant therapy, thrombocytopenia < 50,000/m3, small, posterior, and loculated effusions. Pericardiocentesis in acute traumatic haemopericardium and purulent pericarditis is probably less appropriate than surgical drainage[7]. In effusions causing no haemodynamic compromise pericardiocentesis is indicated in effusions >20 mm in echocardiography in diastole[8] or for diagnostic purposes if additional procedures are available (e.g., pericardial fluid and tissue analyses, pericardioscopy, and epicardial/pericardial biopsy) which could reveal the etiology of the disease and permit further causative therapy (level of evidence B, class IIa indication).

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