



STUDY OF ETIOLOGICAL AND CLINICAL PROFILE OF CHRONIC PERICARDITIS IN A TERRITORY CARE CENTER

Cardiology

Dr. P. Kannan

MD. DM., Professor, Department of Cardiology, Govt. Mohan Kumaramangalam Medical College, Salem, Tamilnadu

Dr. T. Munusamy*

MD. DM. Associate Professor, Department of Cardiology, Govt. Mohan Kumaramangalam Medical College, Salem, Tamilnadu *Corresponding Author

ABSTRACT

Introduction: The etiologic spectrum in different series largely depends on the source of the patients, the characteristics of the centre and on the frequency distribution of different etiologies in each geographic area. The aim of this paper to study etiological and clinical profile of patients with chronic pericarditis in a territory Care Center.

Materials and Methods: It is a single centre descriptive study of 50 consecutive patients who were hospitalized in the department of cardiology at Govt. Mohan Kumaramangalam Medical College between January 2015 to January 2018

Results: Out of 50 patients enrolled in our study, 56% were males. The mean age of our study population was 35 ± 18 years. Dyspnea was the common presentation in all three groups in our study. We found the most common etiology for all the three groups of chronic pericarditis was idiopathic.

Conclusion: Our study reported that patients with chronic pericardial effusion, CCP, CT specific etiology were found in 19 patients (48%) (18%) (24%) respectively. The most common cause in all the three groups of chronic pericarditis was idiopathic.

KEYWORDS

CCP: Chronic Constrictive Pericarditis, CT : Cardiac Tamponade, PE : pericardial Effusion

Introduction:

Pericardium is specialized structure to its complex active and passive functions. The pericardium is composed of parietal pericardium and visceral pericardium. The pericardium normally contains as much as 50 ml of an ultra filtrate of plasma. In many cases pericardial effusion is associated with a previous known condition or underlying cardiac diseases which are finally proves to be the cause of the pericardial effusion. In patients with no apparent cause of pericardial effusion, the presence of inflammatory signs of predictive of acute (idiopathic) pericarditis. Our aim of study is to evaluate etiological and clinical profile of patients with chronic pericarditis.

Materials and Methods:

This study was conducted at department of cardiology Govt. Mohan Kumaramangalam Medical College, Salem from January 2015 to January 2018. Informed consent obtained from every patients included in the study. The patients with clinical features of chronic pericarditis which lasted for more than three months were included. All patients with the diagnosis chronic pericardial effusion, chronic cardiac Tamponade and chronic constructive pericarditis were included in this study. All patients with clinical features of pericarditis which lasted for less than three month were excluded.

All patients were underwent a proper systematic clinical examination immediately after the enrollment. Complete Hemogram, Blood urea and serum creatinin were done in all patients. Chest x-ray was taken in all patients and lateral views in all patients. Surface ECG was taken in all patients. ECG was analyzed for presence of Low voltage complexes, atrial enlargement, arrhythmias. Low voltage complex was defined if QRS voltage in leads I, II, III each less than 0.5mv or V_1 to V_6 each less than 1.0mv.

In all patients 2D and M mode and Doppler examination was done. In M mode, we have analyzed the septal motion, septal notch, septal bounce, pericardial fluid quantification, pericardial thickness and posteriorly wall movement. The severity of pericardial effusion was classified based on the size of effusion 1.Small (echo free space in diastole < 10 mm) 2.Moderate (at least ≥ 10 mm posteriorly) 3.Large (≥ 20 mm) 4.Very large (≥ 20 mm with compression of heart). In 2D echo, we specifically looked for the presence of early diastole right atrial collapse and the presence of collapse of the free wall of right ventricle. The Doppler examination was done to find out the flow variation during the both phases of respiration. We analyzed the respiratory flow variation in mitral valve, tricuspid valve, hepatic vein and pulmonary veins.

Results:

Totally fifty patients were included in this study. Out of them 28 (56%) patients were male. The mean age of our study population was 35 ± 18 years. Dyspnea was the predominant symptom in our study population. 46 (92%) patients had dyspnea. Facial puffiness was present in 32 (62%) patients. Only 5 patients (10%) had previous history of Tuberculosis. 4 patients (8%) had evidence of chronic renal failure and 8 (16%) patients had evidence of hypothyroidism. 14 patients (28%) had pulsus paradoxus and 8 patients had pericardial knock.

Table: 1 Baseline Characteristics

Total no of patients	50
Age in years (mean \pm SD)	35 ± 18
Male	28 (56%)
Mean duration of symptoms (in months)	4.3 ± 4.1
Clinical Profile	
Dyspnea class II to IV	46 (92%)
Facial puffiness	32 (62%)
Chest pain	26 (52%)
Fever	24 (48%)
Hypothyroidism	8 (16%)
Chronic renal failure	4 (8%)
Previous H/o Tuberculosis	5 (10%)
H/o Rheumatic fever	1 (2%)
Acute myocardial infarction	1(2%)
H/o irradiation	Nil
Mean pulse rate (per min)	92 ± 18
Elevated Jugular venous pressure	34 (68%)
Pulsus paradoxus	14 (28%)
Pericardial knock	8 (16%)

Table-2: Patterns of Chronic Pericarditis

Periodical Disease	Total no of Patients
Periodical (effusion (PE) small	3 (6%)
Moderate	12 (24%)
Large	16 (32%)
Constrictive Pericarditis (CCP)	8 (16%)
Cardiac Tamponade (CT)	11(22%)

Table 2 describes the profile of our study population. 8(16%) had constrictive pericarditis and 11(22%) had cardiac tamponade. 31 (62%) patients had pericardial effusion. Out of 31 patients 16 (32%) patients had large pericardial effusion. 12 (24%) patients had moderate pericardial effusion and 3 (6%) patients had small pericardial effusion.

Table-3: Clinical Profile

	Pericardialeffusion (PE)	Constrictive Pericarditis (CCP)	Cardiac Temponde (CT)
Age in years (Mean \pm SD)	38 \pm 17	21 \pm 7	32 \pm 14
Male (%)	12 (42%)	6 (75%)	10 (90%)
Duration of Symptoms (months)	4.7 \pm 5.5	4.2 \pm 2.3	3.1 \pm 0.3
Previous evidence of Tuberculosis	2 (40%)	2 (40%)	1 (20%)
Dyspnea	28 (61%)	10 (22%)	8 (17%)
Facial Puffiness	18 (56%)	6 (19%)	8 (25%)
Pedal edema	18 (60%)	6 (20%)	6 (20%)
Chest pain	14 (54%)	8 (31%)	4 (15%)
Fever	8 (34%)	12 (50%)	4 (16%)
No of patients with pulsus paradoxus	5 (36%)	Nil	9 (64%)
Mean Jugular venous pressure (CM)	6.9 \pm 2.6	7.5 \pm 2.2	9.8 \pm 1.3
No of patients with pericardial knock	Nil	8 (90%)	Nil

Table 3 showed the clinical profile of our patients. We divide our study population into the three groups namely pericardial effusion, chronic constrictive pericarditis, cardiac Tamponade. The mean age of patients in three groups were 38, 21 and 32 respectively. The predominant study population is constrictive pericarditis and cardiac Tamponade were males and in pericardial effusion group predominantly they were females. Patients in constrictive pericarditis group were younger age group than the others. The mean duration of clinical presentation prior to the study were 4.8, 4.2 and 3.1 months respectively. 40% patients in the constrictive pericarditis group had previous evidence of Tuberculosis. 9 patients (64%) in cardiac tamponade and 5 patients (36%) in severe pericardial effusion group had pulsus paradoxus. The mean JVP were 6.9, 7.5 and 9.8 respectively. 8 patients (90%) in constrictive pericarditis group had pericardial knock.

Table-4: Etiological Profile

Diseases	PE	CT	CCP
Tuberculosis	1 (20%)	2 (40%)	2 (40%)
Rheumatic fever	1 (4%)	Nil	Nil
Irradiation	Nil	Nil	Nil
Myocardial infarction	1 (4%)	Nil	Nil
Chronic renal failure	4 (15%)	Nil	Nil
Hypothyroidism	8 (16%)	Nil	Nil
Idiopathic	16 (52%)	9 (82%)	6 (75%)

Table 4 shows the etiological profile of study population. Most common etiology in all the three groups were idiopathic. In CCP 6 patients (75%) belonged idiopathic cause and in 2 patients (40%) it was due to tuberculosis infection. In patients with cardiac tamponade in 9 (82%) patients, it was due to idiopathic cause and the rest of the 2 patients (40%), it was due to tuberculosis infection. In patients with pericardial effusion, 16 patients (52%) were idiopathic and 8 patients (16%) were hypothyroid and 4 patients (15%) were in chronic renal failure. The rest of 3 patients belonged to myocardial infarction Rheumatic fever and Tuberculosis etiology.

Table-5: Baseline Investigations

	PE	CCP	CT
ESR (mm/hour)	27 \pm 2.2	34.7 \pm 19	42.3 \pm 23.8
CTR (%) in Chest X-ray	61 \pm 7.5	53.4 \pm 3.2	62.4 \pm 3.4
Pericardial calcification in X-ray	Nil	3(25%)	Nil
No of patients with Low voltage complex in ECG	16(57%)	5 (50%)	10 (80%)

Table 5 showed the comparison of baseline investigations of our study population. ESR was higher in patients with cardiac Tamponade. The mean CTR was also high in patients with cardiac Tamponade. 3 patients (25%) showed evidence of pericardial calcification, 10 patients (80%) had low voltage complexes in ECG patients with cardiac tamponade.

Table-6: Echo Characteristics of Chronic Pericarditis

Variables	PE	CCP	CT
Right Atrial Collapse	Nil	Nil	11
Right Ventricular Collapse	Nil	Nil	11
Septal Bounce	Nil	10	Nil
Flat posterior wall	Nil	10	Nil
Pericardial Thickness (mm)	2.26 \pm 0.3	4.2 \pm 0.4	2.32 \pm 0.6
Pericardial effusion (mm)	18.17 \pm 4	7.2 \pm 2.7	24.6 \pm 2.6
MV flow velocity respiratory variation (mean%)	9.2 \pm 2.4	28.1 \pm 2.6	2.48 \pm 3.9
Total no of patients with prominent diastolic flow reversal in hepatic vein during expiration	15(55%)	9(90%)	11(100%)

Table 6 showed echocardiographic features of patients in chronic pericarditis. The mean pericardial Thickness in all the three groups were 2.26, 4.2 and 2.32 mm. The mean pericardial thickness was high in patients with constrictive pericarditis group. The mean MV flow velocity respiratory variation was high in patients with constrictive pericarditis.

Discussion:

We have analyzed totally fifty consecutive patients with chronic pericarditis. The principal aim of our study was to find the etiological profile in patients with chronic pericarditis. The study population were divided into three major groups namely Pericardial effusion, Chronic constrictive pericarditis and cardiac tamponade. 8 (16%) had constrictive pericarditis and 11 (22%) had cardiac Tamponade and 31 patients (62%) had chronic pericardial effusion. Out of 31 patients with chronic pericardial effusion 16 (32%) patients had large pericardial effusion. 12 (24%) patients had moderate pericardial effusion and 3 (6%) patients had small pericardial effusion.

In our study 56% were males. It was consistent with study by **Sagríst-Sauled J et al**¹¹. He analyzed 322 patients with severe pericardial effusion. He found that 166 patients were (52%) men. The mean age of our study population was 35 \pm 18 years. Our study population belongs to younger age group. In the study by **Sagríst-Sauled J et al**¹⁰, the mean age of his study population was 56 \pm 17 years. The mean age of patients in three groups were 39, 20 and 34 years respectively. The patients in constrictive pericarditis group were younger than the other groups.

Dyspnea was the common presentation in all three groups in our study. In our study population, only 5 patients (10%) had previous history of tuberculosis. Clinical evidence of chronic renal failure was found in 4 patients (8%) and 8 (16%) patients had evidence of hypothyroidism.

In our study population pulsus paradoxus was found in 9 patients (64%) had pericardial effusion. In contrast, the study by **Levine et al**¹ in patients with severe pericardial effusion with Tamponade showed pulsus paradoxus 36% patients. The mean Jugular venous pressure was 6.9, 7.5 and 9.8 cm in the three groups respectively. In contrast, the study by **Levine et al**¹ in patients with tamponade elevation of Jugular venous pressure was found only 34%. In our study population 8 patients (90%) in constrictive pericarditis group had pericardial knock.

In our study population 10 patients (80%) with cardiac Tamponade had low voltage complexes in ECG. In contrast in study by **Casala et al**⁶ using standard ECG criteria for low voltage (lead I III avf each less than 0.5mv, v₁ to v₆ each less than 1.0mv), the low voltage complexes was found in 12% patients with pericardial effusion with tamponade.

The mean pericardial thickness in the three groups were 2.29, 4.4 and 2.45 mm respectively. In our study, the mean MV flow velocity respiratory variation was high in patients with constrictive pericarditis. **Amuthan.V et al**⁷ in his study of 16 patients with chronic constrictive pericarditis found that Doppler diagnostic of constrictive pericarditis was correctly made in 90% patients. He used Doppler flow pattern of respiratory variation in ventricular filling (mitral and tricuspid forward flow) and central venous flow velocities (hepatic vein diastolic flow reversal) of more than 25% for the diagnosis of chronic constrictive pericarditis. Right atrial collapse and right ventricular collapse were found in all patients with cardiac tamponade. The mean amount by pericardial fluid in patients with cardiac tamponade was 650 \pm 92ml. 6 patients (54%) in CT group had hemorrhagic pericardial effusion.

We found the most common etiology for all the three groups of chronic

pericarditis was idiopathic. This was consistent with pre existing studies. In patients with chronic constrictive pericarditis 6 patients belonged to idiopathic cause and 2 patients (40%) it was due to tuberculosis infection. **Cacoub P et al**⁵ studied the etiology profile in patients with constrictive pericarditis in 13 patients. He found neoplasia in 4 patients pericarditis as a result of radiotherapy in 2 patients as a result of injuries in 2 patients.

In patients with chronic pericardial effusion, specific etiology was found only in 15 (48%) patients. Out of them 8 patients (16%) were found to have hypothyroidism and 4 patients (8%) had pericardial effusion due to chronic renal failure. 1 patient (2%) had first myocardial infarction pericarditis. 1 patients (2%) had pericardial effusion as a result of chronic rheumatic pericarditis and 5 of patients (10%) had tuberculosis etiology, the rest of the 31 (62%) patients were idiopathic. In a study by **Oliver Negue et al**¹⁰, 141 consecutive patients with unexplained pericardial effusion underwent pericardioscopy. After complete workshop a specific cause was found in 69 cases (48.6%) the other 73 cases were considered as idiopathic effusion.

Conclusion:

This single centre descriptive study provides that patients with chronic pericardial effusion (that patients with chronic pericardial effusion) specific etiology was found in 15 patients (30%). Out of them 8 patients (16%) had hypothyroidism. 4 patients (15%) had chronic renal failure. 1 patient (4%) had post myocardial infarction pericarditis. 1 patient (4%) had rheumatic pericarditis and 1 patient (20%) had tuberculosis etiology. In CCP, 2 patients (40%) had tuberculosis etiology and the rest (75%) were idiopathic. In cardiac tamponade, 2 patients (40%) were found to have tuberculosis infection and the remaining, 9 patients (82%) were idiopathic, the most common cause in all the three groups of constrictive pericarditis was idiopathic.

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