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Original Research Article

A study of etiology, clinical characteristics, electrocardiographic and echocardiographic findings in patients with cardiac tamponade in a tertiary care hospital in Western Odisha

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ABSTRACT

Background: Cardiac tamponade is a life-threatening condition characterized by elevated intracardiac pressure, limitation of diastolic filling, and reduction of cardiac output and mostly requires urgent therapeutic intervention. Pericardial disease in on the rise attributed to improved survival of malignancy, growing no of cardiac interventions, chronic kidney diseases with dialysis, advent of modern chemo-radiotherapy. Still, there are paucity of data on etiology, clinical characteristics, electrocardiogram (ECG) and echocardiographic features in patients with cardiac tamponade from Odisha. Aim of this study was to emphasize the etiology, clinical characteristics, electrocardiographic and echocardiographic features in patients with cardiac tamponade.
Methods: A prospective observational study of 1-year duration was undertaken for patients with cardiac tamponade admitted at VIMSAR, a cardiology unit. Data on etiology, clinical characteristics, Echocardiography, ECG findings were documented. Echo guided pericardiocentesis followed culture, cytological and biochemical analysis done.
Results: Most common symptom was dyspnoea (88%), clinical signs were tachypnoea (96%), raised jugular venous pulse (64%), tachycardia (84%), pulsus paradoxus (60%). Classical beck's triad was seen in 42% cases. X-ray showed cardiomegaly (94%) & pleural effusion (64%). Common ECG findings were sinus tachycardia (84%), low voltage QRS (68%) and electrical alternans (44%). Echocardiography showed 80% and 68% of patients had right atrium collapse and right ventricle collapse respectively. Both inferior vena cava plethora and transmitral flow variation were seen in 88% of cases. 96% had normal left ventricle function. 80% had large effusion. Pericardiocentesis yield was mostly in between 500-1000ml (mean 908 ml) with 72% with haemorrhagic aspirate. All malignancy cases and 76% tubercular cases had haemorrhagic effusion. Most common etiology was tubercular (50%), followed by malignancy (28%). 6% remained idiopathic. chronic kidney disease, systemic lupus erythematosus, post MI intervention, hypothyroid were some of the other causes.
Conclusions: Initial assessment with investigation and careful follow-up can yield a causal diagnosis in most cases. Early diagnosis and intervention by pericardiocentesis can be lifesaving.

Keywords: Cardiac tamponade, Observational study, Pericardial effusion, Clinical characteristics

INTRODUCTION

Cardiac tamponade is defined as a cardiac compression caused by pericardial fluid.[1] The compression may be blood, pus, fluid (transudate or exudate) or even air.[2] It is a life-threatening condition characterized by elevated intracardiac pressure with progressive limitation of diastolic filling and reduction of cardiac output. Mostly urgent therapeutic interventions are required. The principal hemodynamic alteration is a constraint on atrial

filling with a reduction of atrial diastolic volume, which results in an increase in atrial diastolic pressure.[3]

Pericardial disease has become much more common in recent years. Longer survival of patients with malignant disease, growing numbers of cardiac surgical operations, the treatment of chronic renal disease with dialysis, use of anticoagulant drugs and the advent of newer drugs and irradiation in tumor therapy are largely responsible. The causes of a cardiac tamponade also include an acute accumulation of pericardial fluid from a ruptured myocardium (following MI, blunt or penetrating trauma or cardiac perforation following cardiac catheterization), carcinomatous infiltrate of the pericardium and acute pericarditis or proximal dissecting aortic aneurysm.[4] Idiopathic or viral pericarditis, tuberculosis and bacterial infection, iatrogenic injury (post PCI, post-CABG), uremia, collagen vascular disease, may culminate to cardiac tamponade.[5] Patients with acute pericarditis may have cardiac tamponade as the presenting feature.

The classical presentation of a cardiac tamponade is an elevated venous pressure, decreased systemic arterial pressure and a quiet heart (i.e. Beck's triad).[6] Cardiac tamponade may be acute or chronic and should be viewed hemodynamically as a continuum ranging from mild (pericardial pressure <10 mmHg) to severe (pericardial pressure >15 to 20 mmHg). Tamponade on sudden occurrence may result in death without any symptoms. In less drastic circumstances, patients with acute cardiac tamponade may complain of severe shortness of breath accompanied by tightness of chest and dizziness.[7] The fluid accumulation is often more gradual in the medical patient with tamponade. Hence, the classic small, quiet heart with increasing venous pressure and decreasing blood pressure may not be found.

Early diagnosis of cardiac tamponade is essential to prevent hemodynamic impairment and fatal outcome.[8] Various modalities available for tamponade diagnosis include clinical signs and symptoms, chest X-ray, echocardiography (ECHO), ECG and other investigations. But all these have certain limitations.[9] Chest X-ray is non-invasive, cheap and easily available diagnostic tool in most of the hospitals whereas echocardiography is frequently performed for confirmation of tamponade but its value and early availability in emergency is questionable.[10]

Surgical placement of subxiphoid tube is the preferred technique for draining a small amount of effusion in patients with quickly developing pericardial tamponade such as those with acute traumatic hemopericardium.[11,12] Echocardiographic guidance increases the success rate of pericardiocentesis by reducing complications.[13] Echocardiography assisted pericardiocentesis is a simple, safe and effective primary management of significant Pericardial Effusion.[14]

There is very little published data on etiology, clinical characteristics, ECG and echocardiographic features in patients with cardiac tamponade from Odisha, a state on the eastern part of India. In this study we evaluated 50 consecutive patients of cardiac tamponade who underwent therapeutic pericardiocentesis in the cardiology department of VIMSAR, Burla, a tertiary care hospital in Odisha from January 2019 to January 2020. This study aims at emphasizing the etiologic background, clinical characteristics, electrocardiographic and echocardiographic features in patients with cardiac tamponade.

Aim and objectives

To categorize patients of cardiac tamponade in terms of etiologic background, clinical characteristics, electrocardiographic and echocardio-graphic findings in patients with cardiac tamponade secondary objective- to follow-up cases of successful pericardiocentesis for their clinical outcomes over time.

METHODS

A prospective and observational study was carried out in the cardiology department of VIMSAR, Burla, a tertiary care hospital in ODISHA between January 2019 and January 2020. After proper institutional ethics committee approval, written consent was taken from eligible patients to be included in the study. Fifty patients after fulfilling selection criteria were finally selected for final study to evaluate Etiology, clinical features, ECG, and ECHO and other relevant laboratory parameters.

Inclusion criteria

All consecutive patients with cardiac tamponade diagnosed by clinical and echocardiography criteria and admitted to our department were enrolled in this study.[15-18] Finally, those patients who had given consent for pericardiocentesis were included in this study.

Exclusion criteria

Vulnerable groups or critically ill with severe comorbidities were excluded from our study.

Pericardiocentesis and pericardial fluid analysis

An echocardiography guided pericardiocentesis was performed in patients with cardiac tamponade. Pericardiocentesis was done by subxiphoid approach. Pericardial fluid was drained and submitted for culture, cytological and biochemical analysis (Light's criteria).[20]

All patients were followed up at 1 month after discharge and thereafter at every three months intervals.

Data analysis and statistics

The medical records were reviewed and data on demography, medical history, hospital course, laboratory results and X-rays, ECG and echocardiographic characteristics were collected on a predesigned questionnaire. Data was entered and analyzed using SPSS statistical software package version 11.0. All categorical variables were described as percentages and all continuous variables were expressed as mean.

RESULTS

Out of 50 patients, 26 (52%) were male and 24 (48%) were females (Figure 1). The mean age was 43.86 years (ranging from 10 to 82 years). Maximum number of patients (34%) were in the age group of 31 to 45 years, followed by 20% in the age group of 16 to 30 years. Only 8% of patients were below the age of 15 years (Figure 2).

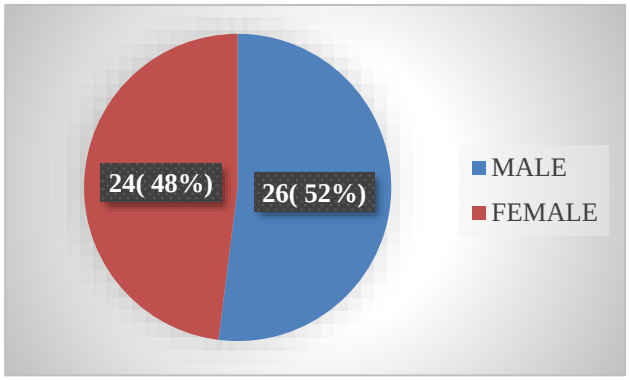

Figure 1: Gender distribution of study patients.

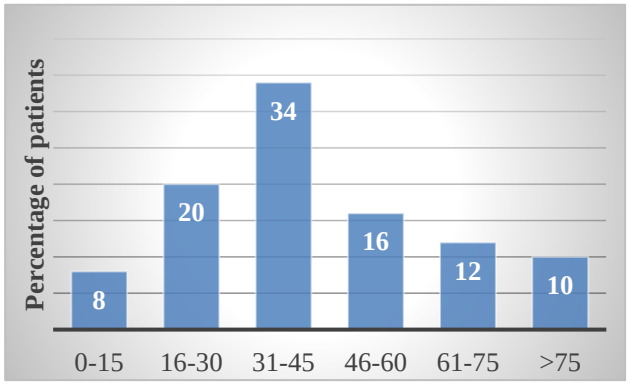

Figure 2: Age distribution of patients.

In terms of past medical history or diagnoses (Table 1), 22% (n=11) of patients had a history of malignancy and 22% were on either chemo or radiotherapy at the time of presentation of cardiac tamponade. 16% of patients had past history of TB and 3 of them were on ATT, but all of them had not completed ATT. Hypothyroidism, CKD and SLE were present in 4% (n=2 each) of cases each and were on treatment. Both CKD patients were having evidence of uremia and were on hemodialysis. One patient had recent MI who underwent PCI following which developed cardiac tamponade.

Table 1: Past medical/surgical/ drug history attributed for cardiac tamponade (n=50).

Past history/diagnoses	No. of patients	%
Tuberculosis	5	10
TB on ATT	3	6
Malignancy	11	22
On chemo/radio therapy	11	22
Hypothyroidism	2	4
CKD	2	4
CTD (SLE)	2	4
Recent MI /PCI	1	2

Common presenting symptoms were dyspnea (88%), fatigue (68%), fever (60%), cough (40%) and chest pain (24%). Abdominal pain and palpitation were the rarest of symptoms and were present in 4% and 2% of cases. In terms of clinical signs tachypnea, tachycardia (HR>100 bpm), hypotension (SBP<100mmHg), elevated JVP and pulsus paradoxus were documented in 96%, 84%, 72%, 64% and 60% of patients respectively. The mean SBP of patients was 94 mm Hg. The mean HR was 114 bpm. The mean elevated JVP was 12 cm of water. Only 4% had documented pericardial rub. Classical Beck's triad was seen in 42% of patients. Table 2 depicts all presenting features with clinical symptoms.

Table 2: Clinical signs and symptoms of cardiac tamponade (n=50).

Variables	No. of patients	Percentage
Clinical symptoms		
Dyspnoea	44	88
Fatigue	34	68
Chest pain	12	24
Fever	30	60
Weight loss	5	10
Cough	20	40
Palpitation	1	2
Abdominal pain	2	4
Clinical signs		
Tachycardia	42	84
Hypotension	36	72
Pulsus paradoxus	30	60
Elevated JVP	32	64
Hypoxia	20	40
Tachypnoea	48	96
Becks triad	21	42
Edema	10	20
Hepatomegaly	6	12
Pericardial rub	2	4

Radiographic evidence of cardiomegaly and pleural effusion were present in 94% and 42% of patients with

tamponade respectively. Common ECG findings were sinus tachycardia (84%), low voltage QRS (68%) and electrical alternans (44%). The mean heart rate was 114 bpm. Simultaneous presence of all the above ECG findings was seen in 36% of patients (Table 3).

Table 3: ECG findings of cardiac tamponade cases.

ECG parameters	Total cases	percentages
Sinus tachycardia	42	84
Low voltage QRS	34	68
Electrical alternans	22	44
All of the above	18	36

All the patients had a standard transthoracic echocardiogram (TTE) before Pericardiocentesis. 80% and 68% of patients had RA collapse and RV collapse respectively. Both IVC plethora and transmitral flow variation were seen in 88% of cases. Swinging heart was seen 72% of patients with cardiac tamponade. Strands in the pericardial space were seen in 30% of cases and all of them had tubercular aetiology. 96% of patient had normal LV function. Both the CKD patients (4%) showed mild LV dysfunction (Table 4).

Table 4: TTE finding (n=50).

Parameters	No. of patients	Percentage (%)
RA collapse	40	80
RV collapse	34	68
IVC plethora	44	88
Trans mitral flow variation	44	88
Swinging heart	36	72
Strands in pericardium	15	30
Normal LV function	48	96

Table 5: Categorization according to aspirated fluid volume.

Aspirated fluid quantity(ml)	No. of patients	Percentage (%)
<500	5	10
500-1000	32	64
1000-1500	10	20
>1500	3	6

In 64% of cases the amount of aspirated pericardial fluid was between 500 and 1000 ml and in 20% of cases the aspirated volume was between 1000 and 1500 ml. More than 1500 ml was aspirated in only 6% of cases. The average volume of aspirated pericardial fluid was 908ml (ranging from 300 ml to 1700 ml) (Table 5). The aspirated pericardial fluid was sent for cytological, microbiological and biochemical examination.

80% of patients had a large effusion (circumferential effusions with width of >2 cm) on echocardiography, while 16% had a moderate effusion (circumferential effusion with an arc width of >1 cm at its greatest) and 4% of the patients had mild effusion (posteriorly loculated effusions of 1 cm or less in width) (Figure 3). On 2D echocardiography 96% patients had circumferential and 4% had loculated effusion (Figure 4). As (Figure 5) depicts, pericardial fluid was haemorrhagic in 72%, serous in 16%, serosanguinous in 10% and purulent in 2%. All malignancy (100%) and tuberculosis (76%) cases had haemorrhagic aspirate. Serous fluid was commonly aspirated in patients mostly from CKD, SLE, hypothyroidism and some cases of TB.

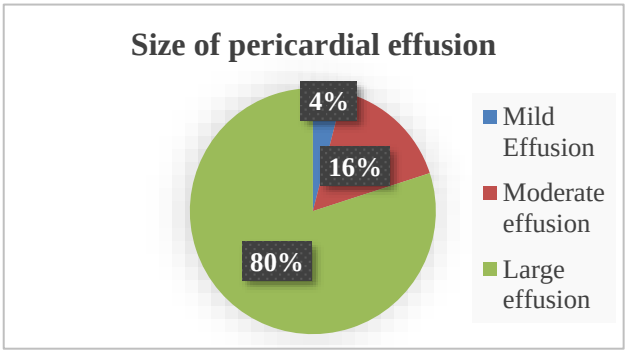

Figure 3: Categorization according to size of pericardial effusion.

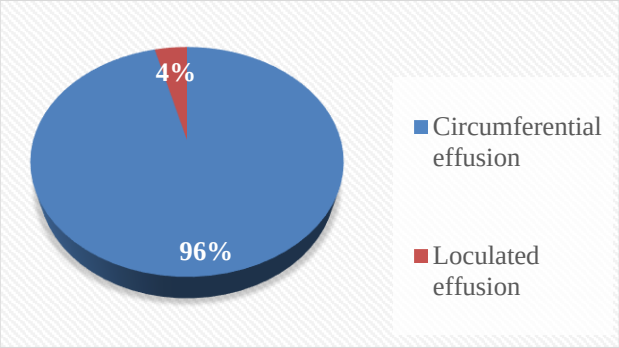

Figure 4: Distribution of pericardial effusion.

The most common causes of cardiac tamponade were tuberculosis (50%) and malignancy (28%). In three cases (6%) the aetiology could not be established (idiopathic). Pyopericardium due to bacterial infection and post PCI related tamponade were seen in 2% each. CKD, connective tissue disease (SLE) and Hypothyroidism accounted for 4% of cases each. Eighteen cases of tuberculosis were diagnosed either by histopathology or microbiology, while the rest seven patients had a presumptive diagnosis of tuberculosis based on radiological, clinical, biochemical (e.g. ADA, ESR, etc) and circumstantial evidences (Figure 6).

Malignancy was the cause of cardiac tamponade in 28% of patients. A wide variety of malignancies were

responsible for the development of cardiac tamponade. Carcinoma of lung (n=6/14) was the most common (42.8%) cause in our study, followed by carcinoma breast (21.4%) and haematological (14.2%). Non-Hodgkin's lymphoma was the commonest hematological malignancy. Metastatic tumors with unknown primary was seen in a patient (7.1%) (Figure 7).

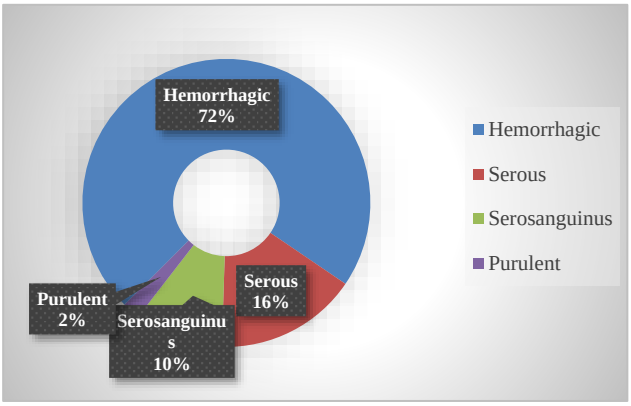

Figure 5: Nature of pericardial fluid.

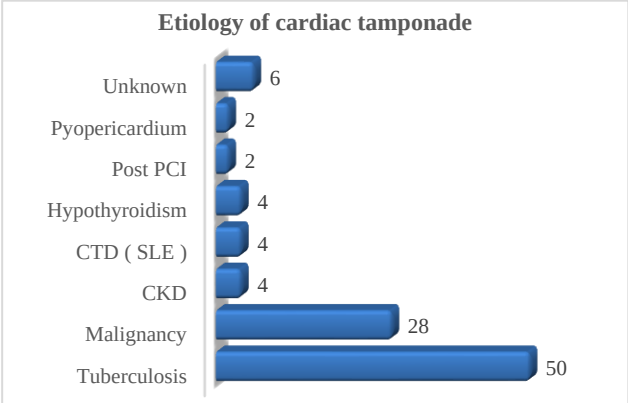

Figure 6: Percentage wise classification of etiology.

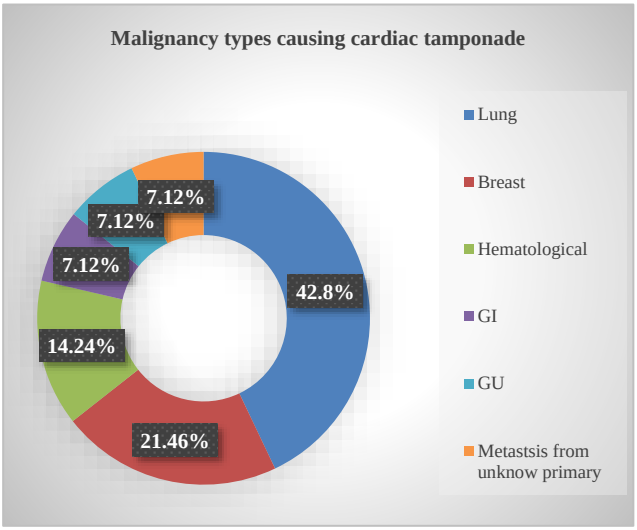

Figure 7: Categorization by malignancy causing cardiac tamponade according to site.

DISCUSSION

Cardiac tamponade can be life threatening unless intervened by pericardiocentesis. Initial assessment by clinical, serological, cytological, biochemical, ECG and echocardiography followed by careful monitoring can enable the discovery of a cause in most cases of pericardial tamponade. The aetiology in our patients with cardiac tamponade were dissimilar to those found by Colombo et al.[21] In their series, the most frequently encountered causal factors were neoplastic diseases (36%), idiopathic pericarditis (32%), and uremic pericarditis (20%). In another series by Natraj et al tuberculosis (35%) and malignancy (24%) were the most common causes of cardiac tamponade.[22] In our series, the most common causes of cardiac tamponade were tubercular (50%), malignancy (28%) and Idiopathic (6%). In studies by Quraishi et al and Natraj et al idiopathic aetiology contributed 4.5% and 3% respectively.[22-23]

In our study, all malignancy and 76% of tuberculosis associated tamponade were haemorrhagic. In the study by Quraishi et al, common causes of haemorrhagic effusion were malignancy (47%) and tuberculosis (19%) respectively.[23]

Uremic pericardial effusion is an expected complication in the course of chronic renal failure. In our series, 2 patients (4%) with end stage renal disease on hemodialysis had successful pericardiocentesis. The contribution of CKD with cardiac tamponade was comparable to studies by Quraishi et al (6.8%) and Natraj et al (3%).[22,23]

In connective tissue diseases, clinically insignificant pericardial effusion is common, but pericardial tamponade is rare. We documented the diagnosis of cardiac tamponade in 2 patients (4%) of systemic lupus erythematosus. In studies by Quraishi et al and Natraj et al, CTD contributed 4.5% and 4% of cases with cardiac tamponade respectively.[22,23]

Our study population comprised of patients who were acutely but not critically ill, a population somewhat similar to that described by Guberman et al.[24] All of these patients were symptomatic and resting dyspnea (88%) was the most frequent symptom which in accordance with findings from studies of Quraishi et al (88.6%) and Natraj et al (87%). Diagnoses of cardiac tamponade on clinical ground alone is often a challenge.[22,23]

In one series by Russo et al hypotension, pulsus paradoxus and raised JVP were present in 70%, 60% and 50% respectively of the patients with echocardiographic evidence of tamponade.[25] In another series by Yilmaz et al only 14% of patients who presented with late posterior tamponade had pulsus paradoxus and increased JVP.[26] In our study, hypotension (SBP<100 mmHg) was present in 72% of cases. Tachypnoea (96%), sinus tachycardia

(84%), raised JVP (64%) and pulsus paradoxus (60%) were frequent findings in our study. In the study by Quraishi et al tachypnea, sinus tachycardia, pulsus paradoxus and elevated JVP were seen in 89%, 88.6%, 59.1% and 40.9% of patients respectively.[23] In the study by Natraj et al tachypnea, tachycardia and elevated JVP were present in 100%, 49.6% and 43.5% of patients respectively.[22] The presence of pulsus paradoxus supports the diagnosis of cardiac tamponade, but it has a low specificity. In addition, with severe hypotension accompanying advanced tamponade, the pulsus paradoxus may be difficult to detect but is present in nearly every patient, and the diagnosis of cardiac tamponade is to be strongly suspected in every patient who has symptoms and findings suggestive of myocardial failure, but who also has a significant paradoxical arterial pulse.[24]

Significant PE as a result of myxedema was first reported by Martin and Spanthis and Ivy.[28,29] In our series, only two patient (4%) had cardiac tamponade as a result of hypothyroidism.[22]

Low voltage complexes in ECG and electrical alternans have been proposed as diagnostic of cardiac tamponade. However, normal voltage electrocardiogram doesn't necessarily rule out cardiac tamponade. We found sinus tachycardia in 84% (76.19% by Argula et al), low voltage QRS in ECG in 68% (55.9% by Argula et al of patients, however electrical alternans occurred in only 40% cases (22.62% by Argula et al).[30] In contrast with reports in literature, we did not find a strong association between electrical alternans and the presence of large PE, although it is suggested that electrical alternans is a sensitive and relatively specific sign for PE and cardiac tamponade. However, in our study we found that electrical alternance was absent in 60% cases of cardiac tamponade.

Enlarged cardiac silhouette on chest X-ray was a relatively common finding in our patients (94%). This is in concordance to earlier reports from Guberman et al and Eisenberg et al which suggested that cardiomegaly on chest X-ray is an important and frequent (95%) diagnostic finding in patients with cardiac tamponade.[24,31] Our study suggests that enlarged cardiac silhouette on Chest X- ray if present is a helpful diagnostic finding in patients with cardiac tamponade, but its absence does not rule out the diagnosis.

In our study, trans mitral flow variation of >25% and IVC plethora were the commonest echocardiographic abnormality (88% each), which was in contrast to the findings by Quraishi et al (31.8%).[23] RA diastolic collapse was seen in 80% of cases comparable to Natraj et al (93%), but in contrast to those by Quraishi et al (54.5%). RVdiastolic collapse was observed in 68% of cases as compared to 50% cases by Quraishi et al and 87% cases by Natraj et al.[22,23]

In our study 80% of patients had large pericardial effusion (>20 mm) as compared to 63.6% in the study by Quraishi et al (Only 4% patients had mild with loculated effusion which was comparable to 2.9% in the study by Quraishi et al.[23] On 2D-echo, 96% of patients had circumferential effusion (95.5% in study of Quraishi et al). Only two cases (4%) were having LV dysfunction and both of them were having CKD. In the study by Quraishi et al. 11.4% of patients were having LV dysfunction.[23]

In 64% of cases the amount of aspirated pericardial fluid was between 500 and 1000 ml and in 20% of cases the aspirated volume was between 1000 to 1500 ml. More than 1500 ml was aspirated in only 6% of cases. The average volume of aspirated pericardial fluid was 908ml (ranging from 300ml to 1700ml). These observations are similar to those of Natraj et al.[22]

A wide variety of malignancies were responsible for the development of cardiac tamponade in 28% of our patients. Carcinoma of lung was the most common (42.8%) cause, followed by carcinoma breast (21.4%) and hematological (14.2%). Non-Hodgkin's lymphoma was the commonest hematological malignancy. Metastatic tumors with unknown primary was seen in 7.1% of patients.

In the study by Quraishi et al out of all malignancy carcinoma breast, Non-Hodgkin's lymphoma, metastasis from unknown primary and carcinoma bronchus constituted 18%, 13%, 13% and 9% respectively as a cause of tamponade.[23]

Limitations

Our study was a small (sample size=50) prospective observational study which encompassed detailed records of echocardiography assisted pericardiocentesis procedures and the data acquired is subject to minimal bias. Referral bias may be another potential limitation of our analysis. Although this limitation cannot be eliminated, it can be analyzed, based on the sample area of our hospital. Further, a larger study with longer follow up will bring more conclusive evidence.

CONCLUSION

Cardiac tamponade is a life-threatening condition unless suspected, diagnosed and intervened early by pericardiocentesis. Initial assessment with clinical, electrocardiography, echocardiographic and other investigation and careful follow-up can yield a causal diagnosis in most cases. Echocardiographically guided pericardiocentesis is a safe and sufficient initial treatment for patients with cardiac tamponade. The prognosis depends chiefly upon the patient's underlying disease. We believe that our patient population is representative of both rural and urban community in this part of India.

Still, there is need of a larger study with a longer period of follow-up.

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