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Etiology, Clinical Features, and Cardiac Findings in Cardiac Tamponade Patients: A Tertiary Care Hospital Study

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Abstract

Background: Cardiac tamponade is a life-threatening condition resulting from pericardial fluid accumulation that compromises cardiac filling and output. The condition can arise from diverse etiologies, including infections, malignancies, and systemic diseases.

Aim: To emphasize the etiology, clinical characteristics, electrocardiographic, and echocardiographic features in patients with cardiac tamponade.

Material and Methods: This observational study included 80 patients diagnosed with cardiac tamponade in a tertiary care hospital. Data regarding etiology, past medical history, clinical presentation, ECG findings, echocardiographic parameters, and pericardial fluid volume were collected and analyzed.

Results: Tuberculosis and malignancy were leading causes, accounting for over 40% of cases. Dyspnoea, tachycardia, and tachypnoea were the most common symptoms and signs. Echocardiographic findings such as IVC plethora, transmitral flow variation, and chamber collapse were frequently observed. Most patients had aspirated fluid volumes between 500–1000 ml.

Conclusion: Cardiac tamponade presents with variable clinical features, but echocardiographic confirmation remains essential for diagnosis. The study reinforces the importance of integrating clinical, ECG, and echocardiographic findings for timely intervention.

Keywords: Cardiac tamponade, echocardiography, pericardial effusion, tuberculosis

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Introduction

Cardiac tamponade is a life-threatening clinical condition characterized by the accumulation of fluid, blood, or other substances in the pericardial sac, resulting in elevated intrapericardial pressure and subsequent impairment of cardiac filling and hemodynamic instability [1]. The etiology of cardiac tamponade varies widely across geographical and clinical settings, with causes ranging from infectious pericarditis and malignancy to trauma, post-cardiac surgery complications, and autoimmune disorders [2]. In developing countries, tuberculosis remains a significant cause of pericardial effusion leading to tamponade, whereas in developed regions, malignancy and iatrogenic complications predominate [3].

The clinical presentation of cardiac tamponade can be acute or subacute, depending on the rate of pericardial fluid accumulation and the compliance of the pericardium. Classical clinical findings include hypotension, elevated jugular venous pressure, muffled heart sounds (Beck's triad), and pulsus paradoxus; however, these signs may be absent in slowly progressive cases, necessitating reliance on imaging modalities for diagnosis [4]. Electrocardiography (ECG) findings such as low-voltage QRS complexes, electrical alternans, and nonspecific ST-T changes are often observed, but their sensitivity is variable [5].

Echocardiography remains the cornerstone of diagnosis, with features such as right atrial and right ventricular diastolic collapse, exaggerated respiratory variation in transvalvular flows, and inferior vena cava plethora serving as key indicators of hemodynamic compromise [6]. Additionally, advanced echocardiographic techniques, including Doppler assessment and tissue Doppler imaging, enhance diagnostic accuracy, especially in borderline cases [7].

The prognosis of cardiac tamponade is largely determined by the underlying etiology, the rapidity of diagnosis, and the timeliness of intervention, typically involving pericardiocentesis or surgical drainage. Malignant tamponade tends to have a poor prognosis despite intervention, while cases secondary to acute pericarditis often have a favorable outcome if treated promptly [8]. Identifying the etiology and characterizing associated clinical and echocardiographic features are essential for guiding therapy and preventing recurrence [9].

In tertiary care hospitals, where patients often present with complex comorbidities and advanced disease, a detailed evaluation of etiology, clinical characteristics, ECG changes, and echocardiographic findings can provide valuable insights for improving early recognition and

management strategies [10]. This study aims to emphasize the spectrum of causes, clinical profiles, and imaging features of cardiac tamponade in a tertiary care setting, thereby contributing to better diagnostic acumen and patient outcomes.

Material and Methods

This was a prospective observational study conducted in the Departments of Cardiology and Emergency Medicine of a tertiary care hospital in India over twelve months. A total of 80 consecutive adults (≥18 years) with suspected clinically significant pericardial effusion were screened, and those with echocardiographically confirmed cardiac tamponade were enrolled after written informed consent. Cardiac tamponade was defined by the presence of a circumferential pericardial effusion with at least one hemodynamic echo criterion—right atrial collapse in late diastole, right ventricular diastolic collapse, respiratory variation >25% in mitral inflow E-wave or >40% in tricuspid inflow, and dilated inferior vena cava (IVC) with reduced inspiratory collapse—together with clinical evidence of hemodynamic compromise such as hypotension, tachycardia, elevated jugular venous pressure, or pulsus paradoxus. Patients with isolated small effusions without hemodynamic compromise, those with prior pericardiotomy or pericardial window in the preceding three months, post–cardiac surgery within four weeks, or inability to provide consent were excluded.

At presentation, demographic data, symptom onset and duration, and clinical characteristics were recorded using a structured proforma. Vital signs included heart rate, blood pressure in both arms, respiratory rate, oxygen saturation, and bedside pulsus paradoxus assessment by sphygmomanometry. Physical examination captured jugular venous distension, heart sounds, peripheral perfusion, and signs of concomitant disease. Twelve-lead electrocardiography (ECG) was performed before any invasive procedure and evaluated for sinus tachycardia, low QRS voltage (limb leads <5 mm or precordial <10 mm), electrical alternans, PR-segment deviations, ST-T abnormalities, conduction blocks, and arrhythmias. Chest radiography was obtained when feasible prior to drainage.

Comprehensive transthoracic echocardiography was performed by level-II/III operators using standardized parasternal long and short axis, apical, and subcostal views. Effusion size was measured in end-diastole at standard windows and categorized as small (<10 mm), moderate (10–20 mm), or large (>20 mm). Echo hemodynamic parameters included right atrial late-diastolic

collapse duration as a fraction of the cardiac cycle, right ventricular free-wall diastolic collapse timing relative to the QRS, respiratory variation in mitral and tricuspid inflow (pulsed-wave Doppler), hepatic vein expiratory diastolic flow reversal, IVC diameter and collapsibility, and qualitative right ventricular systolic function. Tissue Doppler imaging of the septal and lateral mitral annulus (e′ velocities) and pericardial thickening or fibrin strands were noted when image quality permitted.

Etiology was determined by integrating clinical context, laboratory investigations, imaging, and pericardial fluid analysis when drainage was performed. All patients underwent baseline hematology, biochemistry, high-sensitivity C-reactive protein, thyroid profile where indicated, and serologies as clinically relevant. Pericardiocentesis was undertaken urgently in patients with hemodynamic compromise or progressive echocardiographic signs using subxiphoid or apical approach under ultrasound guidance and continuous ECG monitoring. Pericardial fluid was analyzed for appearance, cell counts, protein, LDH, glucose, Gram stain and culture, acid-fast bacilli smear and culture, GeneXpert/NAAT for Mycobacterium tuberculosis where available, and cytology for malignant cells; additional tests such as ADA were ordered per protocol. Etiologic categories were predefined as malignant, tuberculous, uremic, autoimmune/connective tissue disease–related, post-procedural/iatrogenic, bacterial (non-TB), hypothyroid, traumatic, and idiopathic/viral when no cause was identified after workup and three-month follow-up. Primary outcomes were the distribution of etiologies and the frequency of clinical, electrocardiographic, and echocardiographic features at presentation. Secondary outcomes included in-hospital need for repeat drainage, escalation to surgical pericardial window, and all-cause in-hospital mortality. Time from door to echocardiographic confirmation and to pericardiocentesis was recorded to explore workflow performance. Data quality was ensured by dual entry and random audit of 10% of cases.

Statistical analysis was performed using SPSS version 25.0. Continuous variables were examined for normality with the Shapiro–Wilk test and summarized as mean ± standard deviation or median (interquartile range) as appropriate; categorical variables were presented as counts and percentages. Group comparisons across etiologies used one-way ANOVA or Kruskal–Wallis tests for continuous data and chi-square or Fisher’s exact tests for categorical data. Associations between key echocardiographic tamponade indicators (e.g., RV diastolic collapse, IVC plethora,

Doppler respiratory variation) and clinical severity (presence of pulsus paradoxus, systolic blood pressure <90 mmHg) were evaluated by odds ratios with 95% confidence intervals using univariable and multivariable logistic regression adjusting for age, sex, effusion size, and heart rate. A two-sided p value <0.05 was considered statistically significant. The study protocol received approval from the institutional ethics committee and complied with the Declaration of Helsinki; all data were de-identified and stored on secure servers with access restricted to study personnel.

Results

In the present study of 80 patients diagnosed with cardiac tamponade, an evaluation of past medical, surgical, and drug history revealed tuberculosis to be a contributory factor in a notable proportion of cases, with some patients actively on antitubercular therapy. Malignancy was another significant underlying condition, with all affected patients undergoing chemo- or radiotherapy. Other identified etiologies included hypothyroidism, chronic kidney disease (CKD), connective tissue disorders such as systemic lupus erythematosus (SLE), and a small number with recent myocardial infarction or percutaneous coronary intervention. The detailed distribution is presented in Table 1.

Clinical symptom analysis demonstrated that dyspnoea was the most common presenting feature, followed by fatigue, fever, and cough. Chest pain and weight loss were observed in a smaller proportion, while palpitations and abdominal pain were rare. Clinical signs were consistent with hemodynamic compromise, with tachypnoea, tachycardia, and hypotension being frequent findings. Pulsus paradoxus, elevated jugular venous pressure, hypoxia, and Beck's triad were observed in a significant subset, whereas peripheral edema, hepatomegaly, and pericardial rub were less common. The detailed breakdown is shown in Table 2.

Electrocardiographic assessment revealed sinus tachycardia as the predominant abnormality, followed by low voltage QRS complexes and electrical alternans. A subset of patients demonstrated a combination of all these findings, emphasizing the diagnostic value of ECG in tamponade detection (Table 3).

Transthoracic echocardiography (TTE) findings confirmed the diagnosis in all cases, with inferior vena cava (IVC) plethora and transmitral flow variation being the most frequent, followed closely by right atrial and right ventricular collapse. Swinging heart motion was present in a substantial

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proportion, and strands in the pericardium were noted in some cases. Most patients maintained normal left ventricular systolic function (Table 4).

Analysis of aspirated pericardial fluid volume demonstrated that the majority of patients had fluid volumes between 500–1000 ml, followed by smaller proportions with volumes between 1000–1500 ml, and very few with either <500 ml or >1500 ml (Table 5).

Table 1: Past medical/surgical/drug history attributed for cardiac tamponade (n=80)

Past history/diagnoses	No. of patients	%
Tuberculosis	8	10
TB on ATT	5	6.25
Malignancy	18	22.5
On chemo/radiotherapy	18	22.5
Hypothyroidism	3	3.75
CKD	3	3.75
CTD (SLE)	3	3.75
Recent MI / PCI	2	2.5

Table 2: Clinical signs and symptoms of cardiac tamponade (n=80)

Variables	No. of patients	%
Clinical symptoms		
Dyspnoea	70	87.5
Fatigue	55	68.75
Chest pain	19	23.75
Fever	48	60
Weight loss	8	10
Cough	32	40
Palpitation	2	2.5
Abdominal pain	3	3.75
Clinical signs		
Tachycardia	67	83.75

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Hypotension	58	72.5
Pulsus paradoxus	48	60
Elevated JVP	51	63.75
Hypoxia	32	40
Tachypnoea	77	96.25
Beck's triad	34	42.5
Edema	16	20
Hepatomegaly	10	12.5
Pericardial rub	3	3.75

Table 3: ECG findings of cardiac tamponade cases (n=80)

ECG parameters	No. of patients	%
Sinus tachycardia	67	83.75
Low voltage QRS	55	68.75
Electrical alternans	35	43.75
All of the above	29	36.25

Table 4: TTE findings (n=80)

Parameters	No. of patients	%
RA collapse	64	80
RV collapse	55	68.75
IVC plethora	70	87.5
Transmitral flow variation	70	87.5
Swinging heart	58	72.5
Strands in pericardium	24	30
Normal LV function	77	96.25

Table 5: Categorization according to aspirated fluid volume (n=80)

Aspirated fluid quantity (ml)	No. of patients	%

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<500	8	10
500–1000	51	63.75
1000–1500	16	20
>1500	5	6.25

Discussion

The present study highlights the diverse etiologies, clinical presentations, and diagnostic features of cardiac tamponade in a tertiary care setting, aligning with emerging literature emphasizing the multifactorial nature of this condition. Tuberculosis remains a significant cause in developing countries, with our findings corroborating previous reports that link high endemicity of TB to pericardial involvement, often leading to large effusions and tamponade physiology [11]. Malignancy accounted for a considerable proportion of cases, particularly those undergoing chemo- or radiotherapy, consistent with recent studies that demonstrate an increased incidence of malignant pericardial effusions in advanced oncologic cases, where both direct tumor invasion and therapy-related inflammation contribute to pericardial fluid accumulation [12].

Clinical presentations in our cohort were dominated by dyspnoea, tachypnoea, and tachycardia, which are consistent with the pathophysiologic compromise caused by intrapericardial pressure on cardiac filling. The presence of pulsus paradoxus, hypotension, and elevated jugular venous pressure mirrors classical teaching but is variably present in real-world cases, as supported by studies showing that subacute or slowly accumulating effusions may present with subtler hemodynamic signs [13]. ECG findings such as sinus tachycardia, low voltage QRS complexes, and electrical alternans remain hallmark features, yet their absence does not exclude the diagnosis, highlighting the necessity of echocardiography as the primary diagnostic tool.

Echocardiographic markers like right atrial collapse, right ventricular collapse, and IVC plethora were observed with high frequency, reaffirming their diagnostic utility as described in recent consensus recommendations [14]. Notably, swinging heart motion and transmitral flow variation also had high prevalence, underscoring their role as supplementary diagnostic indicators. The aspirated fluid volumes in this study largely fell within the 500–1000 ml range, in keeping with global data suggesting that symptomatic tamponade most often occurs when pericardial effusion volume exceeds 500 ml, although rate of accumulation remains a key determinant of symptom onset [15].

Overall, the findings reinforce the need for early recognition and targeted management, particularly in high-risk groups such as TB patients, cancer patients on active treatment, and those with underlying systemic diseases. Integration of clinical, ECG, and echocardiographic findings remains crucial for timely diagnosis and intervention.

Conclusion

Cardiac tamponade in our cohort demonstrated varied etiologies, with tuberculosis and malignancy emerging as leading causes. Classical symptoms and signs were prevalent, but not universal, reinforcing the importance of echocardiographic evaluation in all suspected cases. The predominance of moderate fluid volumes in symptomatic patients suggests that clinical severity is determined more by the rate of accumulation than absolute volume. These findings underscore the value of prompt, multimodal diagnosis and targeted intervention in improving patient outcomes.

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