Etiology and Clinical Profile of Patients Presenting with Pericardial Effusion in a Tertiary Care Centre

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Abstract

Background: Pericardial effusion is a condition evidenced by the presence of fluid in the pericardial sac. Various etiologies, including infections, autoimmune disorders, malignancies, and metabolic disruptions, cause it. Timely diagnosis and etiological classification are key to successful management. **Material and Methods:** The aim was to assess the clinical presentation, laboratory results, and etiologies underlying pericardial effusion in a tertiary care centre. This observational study included 34 patients diagnosed with pericardial effusion over a period of three months. Data collected included demographics, clinical presentation, comorbidities, echocardiographic findings, pericardial fluid analysis (CBNAAT, cytology, cell block), laboratory parameters (TSH, renal function), and ANA status. Etiological classification and clinical outcomes were noted. **Results:** The mean age was 40.6 years, with female predominance. The most common presenting features included systemic lupus erythematosus (29.4%), tuberculosis (23.5%), and altered sensorium. The most common etiologies were systemic lupus erythematosus (29.4%) and tuberculosis (23.5%). Cytology and cell block helped diagnose malignancy-related effusions using pericardial fluid. All SLE cases were ANA positive. Mortality was highest among patients with malignancy and renal failure. **Conclusion:** The most common causes of pericardial effusion in this cohort were SLE and tuberculosis. A systematic evaluation of autoimmune, infective, and malignant causes is essential for diagnosis. Malignancy and uremic causes were associated with worse outcomes and the need for early intervention.

Keywords: Cholesterol, Ischemic Stroke, Hemorrhagic Stroke, Dyslipidemia, Mortality.

INTRODUCTION

Pericardial effusion is the abnormal accumulation of fluid in the pericardial sac. It represents an important clinical condition with a wide etiological spectrum, including infectious, inflammatory, malignant, and metabolic causes. Clinical manifestations range from asymptomatic incidental findings to life-threatening cardiac tamponade, depending on the volume, rate of accumulation, and underlying pathology. Early identification of the etiology is critical for appropriate management and improved outcomes.

Pericardial effusion shows significant geographical variation in epidemiology. In developing countries such as India, infectious causes—especially tuberculosis—remain predominant in tertiary care settings.[1,2] In contrast, malignancy and post-procedural causes are more common in developed regions. Autoimmune diseases such as systemic lupus erythematosus (SLE), along with metabolic conditions like uremia, hypothyroidism, and cardiovascular diseases such as congestive cardiac failure (CCF), also contribute significantly.

Pathophysiology and Diagnosis

Pericardial effusion results from an imbalance between fluid production and resorption. Mechanisms include increased capillary permeability due to inflammation or malignancy, impaired lymphatic drainage, and altered hydrostatic or oncotic pressures. The biochemical nature of the fluid (transudate vs exudate) provides important diagnostic clues.

Advancements in echocardiography and pericardial fluid analysis—including cytology, CBNAAT, and autoimmune profiling—have improved the identification of etiology. However, in resource-limited settings, a structured clinical approach remains essential.

This study aims to evaluate the demographic and clinical profile, determine the etiological spectrum, and correlate clinical outcomes with laboratory parameters in patients presenting with pericardial effusion in a tertiary care setting.

MATERIALS AND METHODS

This cross-sectional observational study was conducted over three months in a tertiary care centre and included 34 patients

diagnosed with pericardial effusion by echocardiography. All eligible patients were enrolled after obtaining informed consent.

The study protocol was approved by the Institutional Ethics Committee and conducted in accordance with the Declaration of Helsinki. Patient confidentiality was maintained, and data were anonymized before analysis.

Data collection included demographic details (age, sex, comorbidities) and detailed clinical evaluation focusing on presenting symptoms such as dyspnea, fever, orthopnea, arthralgia, and altered sensorium.

Baseline laboratory investigations included:
* Complete blood count (hemoglobin levels)
* Thyroid function tests (TSH)
* Renal function tests (blood urea, serum creatinine)

Etiological evaluation included:
* Pericardial fluid analysis (when feasible)
* CBNAAT for tuberculosis
* Cytology and cell block for malignancy detection
* ANA testing for suspected autoimmune cases

All patients underwent transthoracic echocardiography to assess:
* Presence of effusion
* Size (mild, moderate, massive)
* Hemodynamic impact and signs of cardiac tamponade

Clinical outcomes, including discharge status and in-hospital mortality, were recorded.

Data were analyzed using descriptive statistics. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. Due to the small sample size, no inferential statistical analysis was performed.

RESULTS

A total of 34 patients were included, with a mean age of 40.6 ± 20.1 years (range: 13–75 years). There was a female predominance.

Clinical Presentation

Common presenting symptoms included:
* Breathlessness
* Fever
* Arthralgia
* Altered sensorium
* Orthopnea (in some cases)

Comorbidities
* Hypertension
* Hypothyroidism
* Type 2 diabetes mellitus

Laboratory Findings
* Mean hemoglobin: 9.0 g/dL (suggesting anemia)
* Median TSH: 18.04 mIU/L
* ❖ One patient had severe hypothyroidism (TSH: 171 mIU/L)
* Mean blood urea: 51.2 mg/dL
* Mean serum creatinine: 1.67 mg/dL (with some patients showing marked elevation)

Echocardiographic Findings
* Massive effusion: 12 patients
* Moderate effusion: 15 patients
* Mild effusion: 7 patients
* No cases of cardiac tamponade were observed

Pericardial Fluid Analysis
* CBNAAT positive in 2 cases
* Cytology and cell block detected malignant cells/blasts in 4 patients
* Lymphocyte predominance is seen in tuberculosis and SLE cases
* All SLE patients were ANA positive

The etiological distribution of pericardial effusion is summarized in [Table 1].

Table 1: ?

Etiology	Frequency(n=34)	Percentage (%)
Systemic Lupus Erythematosus (SLE)	10	29.4%
Tuberculosis (PTB)	8	23.5%
AcuteMyeloid Leukemia (AML)	3	8.8%

Table 2: ?

Etiology	Frequency(n=34)	Percentage (%)
CarcinomaLung	3	8.8%
Congestive Cardiac Failure (CCF)	3	8.8%
Myxedema Coma	2	5.9%
UremicPericarditis	2	5.9%
Hypothyroidism	1	2.9%
RheumatoidArthritis	1	2.9%
Viral Pericarditis	1	2.9%

Results (Continued)

Out of the total study population, 28 patients (82.3%) were discharged, while 6 patients (17.6%) died due to the illness.

DISCUSSION

The current research highlights the heterogeneous etiological spectrum of pericardial effusion in a tertiary care setting, with autoimmune and infectious causes being the most prominent. Systemic lupus erythematosus (SLE) emerged as the leading etiology, followed by tuberculosis, reaffirming a shift in disease trends while still emphasizing the significant burden of infectious etiologies in endemic regions.[2]

The predominance of SLE, especially among young female patients, aligns with well-established epidemiological trends, where pericardial involvement is observed in up to 25–50% of cases.[5] Uniform ANA positivity in this cohort further

substantiates the diagnostic utility of autoimmune profiling in patients presenting with unexplained effusions.

Tuberculous pericarditis remains a significant causative factor, consistent with previous literature from developing countries.[1,2] However, relatively low CBNAAT positivity highlights limitations in diagnostic sensitivity and underscores the need for adjunctive tools such as ADA estimation and histopathological evaluation. The exudative, lymphocyte-predominant nature of pericardial fluid in these cases is consistent with established pathophysiological mechanisms, including granulomatous inflammation.[3]

Malignancy-related effusions, including those due to acute myeloid leukemia and pulmonary carcinoma, accounted for a notable proportion of cases. These effusions are often hemorrhagic and recurrent. Cytology and cell block techniques proved highly useful for diagnosis, as also demonstrated in this study.[6] The higher mortality observed in this group is consistent with known adverse prognostic indicators.[7]

Metabolic causes, particularly hypothyroidism and uremia, were also significant contributors. Severe hypothyroidism, including cases of myxedema coma, was associated with moderate to large effusions, reflecting increased capillary permeability and impaired lymphatic drainage. Uremic pericarditis in patients with renal dysfunction further emphasizes the importance of renal evaluation in such cases. Echocardiographic findings in this study showed that most effusions were moderate to massive in size, yet no cases of overt cardiac tamponade were observed. This may be explained by the gradual accumulation of fluid, allowing compensatory pericardial stretching.

This study has several limitations. The small sample size and short duration limit the generalizability of findings and restrict the ability to perform robust statistical analyses or establish significant associations. Being a single-center study in a tertiary care hospital, referral bias is likely, with overrepresentation of severe or complex cases. Additionally, not all patients underwent standardized advanced diagnostic testing (such as pericardial biopsy, ADA levels, or detailed viral studies), which may have led to underdiagnosis or misclassification of certain etiologies. The absence of long-term follow-up also limits assessment of recurrence and long-term outcomes. Despite these limitations, the study provides valuable insights into the clinical and etiological profile of pericardial effusion in a resource-constrained setting.

Overall, a systematic diagnostic approach integrating clinical evaluation, imaging, laboratory parameters, and pericardial fluid analysis is essential. This is particularly important in resource-limited settings, where early etiological identification can significantly influence management and outcomes.

Conclusion

Pericardial effusion is a multifactorial and heterogeneous clinical condition. Autoimmune disorders—especially systemic lupus erythematosus—and tuberculosis were the most common causes in this study, followed by malignancy, renal, and metabolic etiologies. A comprehensive evaluation including echocardiography, pericardial fluid analysis, autoimmune profiling, and thyroid and renal function assessment is essential for accurate diagnosis.

Malignancy-related effusions and uremic pericarditis were associated with poorer outcomes, highlighting the need for early recognition and prompt management. Early identification of the underlying cause is crucial for targeted therapy and improved patient prognosis.

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Conflicts of interest

There are no conflicts of interest.

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