Original Article

A Study on Aetiology and Clinical Profile of Significant Pericardial Effusion at a Tertiary Care Hospital in Eastern India

Indranil Sen[1], Tanmoyee Samanta[2], Dipanjan Bandyopadhyay[3], Debleena Pal[2]

[1]Assistant Professor, Department of General Medicine, North Bengal Medical College and Hospital, West Bengal, India. [2]Junior Resident, Department of General Medicine, North Bengal Medical College and Hospital, West Bengal, India. [3]Professor and Head of the Department, Department of General Medicine, North Bengal Medical College and Hospital, West Bengal, India

Abstract

Background: This study was done to determine the aetiology in 25 patients who underwent pericardiocentesis due to cardiac tamponade, large pericardial effusion or for diagnostic purpose through analysis of their echocardiographic findings, biochemical and cytological test results, and imaging methods in a tertiary care centre in sub-Himalayan West Bengal. **Material and Methods:** 25 consecutive patients selected by purposive sampling underwent USG guided pericardiocentesis and their echocardiograms, biochemical test results, imaging results, pericardial fluid, culture, and cytology results. All data of the patients were recorded and analyzed. **Results:** The most common indication was for diagnostic dilemma in 56% patients whereas 28% had tamponade. Tubercular pericardial effusion was the commonest aetiology in 32% patients and this was also the commonest cause among those done for diagnostic purpose while tamponade indications appeared across CKD, neoplastic, autoimmune, and idiopathic categories. Serosanguinous/haemorrhagic fluid was sensitive but less specific for tuberculosis because haemorrhagic fluid also occurred in non-tubercular aetiologies. Malignant cells were positive in 75% of the neoplastic effusions. **Conclusion:** This study was the first one to be carried out in this geographical region and the high prevalence of tuberculosis may contribute to the aetiology of extrapulmonary TB in this setting. Dyspnoea was present in majority of cases and Chest X ray should be done in these cases to exclude cardiomegaly with suspicion of pericardial effusion as a cause. There are no specific echocardiographic features indicating cause but macroscopic nature including fibrin strands, cytology and ADA has high diagnostic performance.

Keywords: Pericardial effusion, pericardiocentesis, Extrapulmonary Tuberculosis, ADA, tamponade.

Received: 17 July 2026 **Revised: 05 August 2026** **Accepted: 22 August 2026** **Published: 29 August 2026**

Introduction

Pericardial effusion is defined as pathological accumulation of fluid in the pericardial cavity. The normal pericardium is a dual-layered structure that encases the heart and the stems of the significant arteries. It is made up of two varied layers; the superficial layer is the fibrous parietal pericardium, whilst the deep layer is the visceral pericardium.[1] It performs important anatomical & physiological function like preventing contiguous infections from pleural space, facilitation of atrio-ventricular coupling and interdependence and limitation of deleterious effects of volume overload and valvular regurgitation by creating a reserve volume. This volume of approximately 50 ml can accommodate modest intrathoracic pressure alterations but in large or rapid accumulations there is increased right heart filling during inspiration concurrently decreasing left heart filling and symptomatic tamponade. The parietal pericardium has a nonlinear relationship between pressure and volume. The pressure-volume relationship has an initial flat segment where little alterations in pericardial volume result in negligible or no variation in pericardial pressure. Once the pericardial reserve volume is surpassed, there is an increase in pressure. As volume grows, the curve steepens quickly, leading to significant alterations in pericardial pressure with minor increments in pericardial fluid.

The aetiology of pericardial effusions is diverse, encompassing neoplasia, infections, congestive heart failure, iatrogenic factors such as post-procedural complications, radiation, trauma, connective tissue disorders, pericardial injury, and metabolic conditions like uremia and hypothyroidism; notably, a significant proportion of effusions remain idiopathic.[2] Pericardiocentesis is warranted for cardiac tamponade, symptomatic moderate to large effusions unresponsive to pharmacological treatment, and in cases of suspected unknown, mostly bacterial or neoplastic origins. The current investigation aimed to evaluate the clinical profile and aetiology of pericardial effusion, as well as to ascertain the relationship between aetiology and echocardiographic and biochemical features.

Address for correspondence: Dr. Indranil Sen, Assistant Professor, Department of General Medicine, North Bengal Medical College and Hospital, West Bengal, India.
E-mail: docindranilsen@gmail.com

DOI:
10.21276/amit.2026.v13.i2.989

How to cite this article: Sen I, Samanta T, Bandyopadhyay D, Pal D. A Study on Aetiology and Clinical Profile of Significant Pericardial Effusion at a Tertiary Care Hospital in Eastern India. Acta Med Int. 2026;13(2):1956-1962.

Materials and Methods

This observational cross-sectional study was done in a period of 6 months in North Bengal Medical College & Hospital in Darjeeling, West Bengal. 25 consecutive patients selected by purposive sampling who had clinical or echo Doppler features of cardiac tamponade, large effusions with haemodynamic compromise and symptomatic patients with pericardial effusion whose cause had not been determined were recruited. Effusions of any size who were haemodynamically stable and the diagnosis could be made with other non-invasive methods were excluded.

Methodology: The recorded patient information included demographic attributes, symptoms, clinical indicators, a comprehensive physical examination focusing on the cardiovascular system, routine laboratory tests, and analysis of pericardial effusion. Echocardiography was done and the parasternal long axis and subcostal views were used to differentiate from pleural effusion and epicardial pad of fat. Effusions were categorized according to depth into <10 mm as mild, 10-20 mm as moderate and >20mm as large. Tamponade was identified by Doppler characteristics such as early diastolic collapse of the right ventricle, late diastolic collapse of the right and left atria, loss of respiratory variations in the inferior vena cava, a marked increase in the tricuspid E wave during deep inspiration, and a marked decrease in mitral E velocity.

The approach and hardwire to USG guided pericardiocentesis were done according to indication. A subxiphoid route was utilised for therapeutic purposes; a 10-cc injector was utilised to apply negative pressure while an 18-G puncture needle was placed through the right side of the xiphoid and diverted up to the right shoulder after local anaesthetic was infiltrated. A 6F sheath was inserted and the floppy guidewire was advanced as soon as the liquid entered the injector. The pigtail catheter was then advanced for drainage and left in place. For smaller effusions with diagnostic dilemma 18 G spinocaine needle was used in paraapical or subxiphoid route depending on location of fluid and 10-20 cc of fluid was aspirated under continuous USG guidance. On all occasions fluid was analysed for cytology, biochemistry to differentiate transudative from exudative effusions, ADA levels, malignant cells, culture sensitivity. According to modified Light's criteria, the fluid was deemed exudate if any of the following conditions were met: fluid cholesterol concentration >45 mg/dL, fluid total protein >3 gr, fluid/serum protein ratio >0.5, fluid LDH >200 IU/dL, and fluid/serum LDH ratio >0.6.[4] All ancillary investigations and previous hospital records were reviewed to reach a diagnosis, and all these were documented and analysed.

Statistical methods: All of the data was first gathered and documented on a Microsoft Excel sheet before being imported into the SPSS software for statistical analysis. The acquired qualitative data was represented as frequency and percentage. Continuous variables are presented as median (IQR), because the sample size is small and distributions are unlikely to be reliably normal within etiological subgroups. Kruskal–Wallis test was used for continuous-variable comparisons across aetiology groups. Pearson chi-square test was used for exploratory categorical comparisons across aetiology groups.

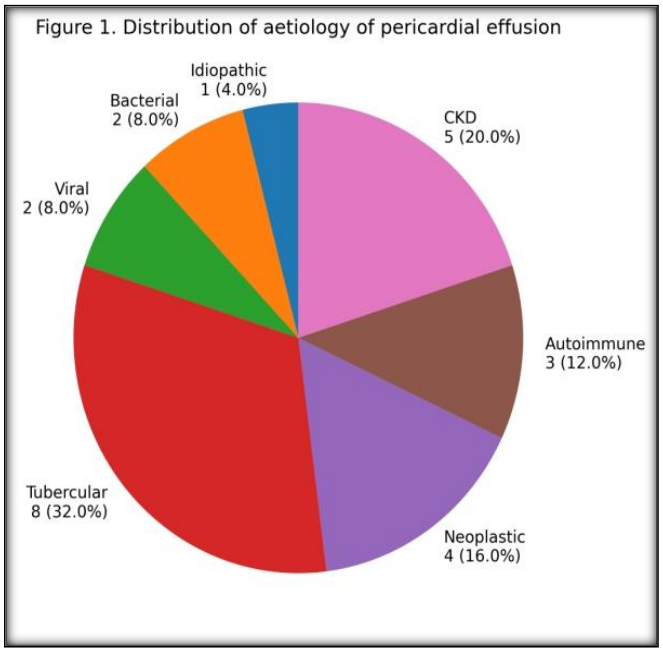
Fisher's exact test p-values, sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) were computed for 2x2 diagnostic validity analyses. The ADA levels in tubercular versus non-tubercular effusion were compared between the two groups using the Mann-Whitney U test.

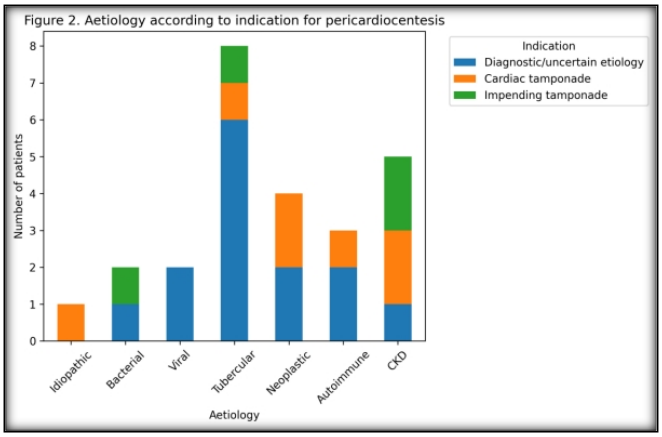
Ethical issues: Prior to starting the study, the institutional ethics committee gave its clearance (memorandum number IEC/NBMC/M-24/002/2026). Every patient who took part in the study provided written, informed consent. Patient privacy and data confidentiality were guaranteed at every level.

Results

The results of all the 25 patients were interpreted and elaborated. The median age was 50 years with a female preponderance. Since this medical college had a catchment area compromising hill-stations and tea gardens most of the patients 40% came from rural areas. The highest percentage of patients were of lower- middle socio-economic group. [Figure 1] showed the different aetiology of pericardial effusion (PE) in our study where tubercular effusion was the largest etiological category, followed by CKD and neoplastic effusion. Most tubercular cases were tapped for diagnostic/uncertain aetiology, while tamponade indications appeared across CKD, neoplastic, autoimmune, and idiopathic categories as shown in [Figure 2]. Based on the aetiology the age of the patients was categorized and older ages clustered in tubercular and CKD categories, while viral and autoimmune cases were comparatively younger in this dataset as seen in [Figure 3]. The clinical features and ECG changes were described in [Table 1] where dyspnoea was common across most categories. Cardiac examination often showed muffled heart sounds and pericardial rub, but no single clinical feature clearly separated aetiologies. The biochemical parameters were enumerated in [Table 2]. Renal parameters were higher in CKD-related effusions, as expected. CRP and liver enzymes varied substantially across cases. Because each subgroup had few patients, biochemical differences was to be considered descriptive rather than definitive. Routine and special investigations were carried out on all pericardial fluid samples as shown in [Table 3]. ADA was higher in tubercular effusion than in non-tubercular effusion [median 57.0 vs 23.0 U/L; Mann–Whitney U p=<0.001]. As there is very low yield of acid-fast tubercular bacilli on pericardial fluid, diagnosis depends upon lymphocytic leukocytosis, exudative according to Light's criteria and high ADA. Diagnostic significance of ADA was seen in [Table 4] with ADA ≥40 U/L showed high specificity and PPV in this dataset, while ADA ≥20 U/L maximised sensitivity but produced more false positives. ADA ≥30 U/L gave a balanced sensitivity/specificity profile. The echocardiographic haemodynamic and doppler parameters were shown in [Table 5] where it was seen large effusion was highly sensitive for tamponade indication, but not fully specific. IVC distension without respiratory variation and systolic RA/RV collapse were more specific indicators. The macroscopic features of fluid also are an indicator as shown in table 6 where fibrin strands showed

strong diagnostic performance for tubercular effusion. Serosanguinous/haemorrhagic fluid was sensitive but less specific because haemorrhagic fluid also occurred in non-tubercular aetiologies. The procedure was successfully completed in all cases, 5 patients had shock which resolved after drainage along with inotropes and 2 patients developed arrhythmia which resolved on medication.





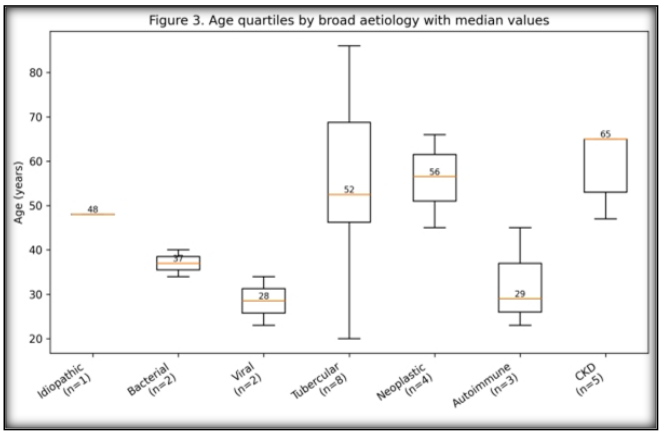


Table 1: Demographic and clinical characteristics by aetiology

Characteristic	Idiopathic	Bacterial	Viral	Tubercular	Neoplastic	Autoimmune	CKD	p-value	Test
Age, years, median (IQR)	48.0 (48.0–48.0)	37.0 (35.5–38.5)	28.5 (25.8–31.2)	52.5 (46.2–68.8)	56.5 (51.0–61.5)	29.0 (26.0–37.0)	65.0 (53.0–65.0)	0.076	Kruskal–Wallis
Female sex	0 (0.0)	1 (50.0)	2 (100.0)	4 (50.0)	4 (100.0)	3 (100.0)	5 (100.0)	0.080	Pearson χ^2
Dyspnoea	1 (100.0)	1 (50.0)	2 (100.0)	7 (87.5)	3 (75.0)	3 (100.0)	5 (100.0)	0.560	Pearson χ^2
Chest pain	0 (0.0)	1 (50.0)	1 (50.0)	5 (62.5)	2 (50.0)	2 (66.7)	1 (20.0)	0.731	Pearson χ^2
Fever	0 (0.0)	1 (50.0)	2 (100.0)	8 (100.0)	3 (75.0)	3 (100.0)	3 (60.0)	0.138	Pearson χ^2
Pallor	1 (100.0)	1 (50.0)	2 (100.0)	4 (50.0)	1 (25.0)	2 (66.7)	5 (100.0)	0.238	Pearson χ^2
Oedema	1 (100.0)	0 (0.0)	1 (50.0)	2 (25.0)	1 (25.0)	3 (100.0)	5 (100.0)	0.030	Pearson χ^2
Raised JVP category	1 (100.0)	0 (0.0)	0 (0.0)	1 (12.5)	1 (25.0)	2 (66.7)	3 (60.0)	0.176	Pearson χ^2
Muffled S1	1 (100.0)	1 (50.0)	1 (50.0)	3 (37.5)	2 (50.0)	2 (66.7)	3 (60.0)	0.920	Pearson χ^2
Muffled S2	1 (100.0)	0 (0.0)	0 (0.0)	2 (25.0)	2 (50.0)	2 (66.7)	1 (20.0)	0.343	Pearson χ^2
Pericardial rub	1 (100.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	2 (66.7)	4 (80.0)	0.007	Pearson χ^2
ECG: PR depression	0 (0.0)	1 (50.0)	0 (0.0)	4 (50.0)	1 (25.0)	0 (0.0)	1 (20.0)	0.555	Pearson χ^2
ECG: ST elevation	1 (100.0)	1 (50.0)	0 (0.0)	2 (25.0)	1 (25.0)	1 (33.3)	4 (80.0)	0.283	Pearson χ^2
ECG: T inversion/ST depression	1 (100.0)	1 (50.0)	1 (50.0)	3 (37.5)	1 (25.0)	2 (66.7)	4 (80.0)	0.596	Pearson χ^2
ECG: Sinus tachycardia	1 (100.0)	2 (100.0)	2 (100.0)	4 (50.0)	4 (100.0)	1 (33.3)	3 (60.0)	0.300	Pearson χ^2
ECG: Electrical alternans	0 (0.0)	0 (0.0)	0 (0.0)	1 (12.5)	1 (25.0)	1 (33.3)	1 (20.0)	0.920	Pearson χ^2

Table 2: Biochemical values by aetiology [median (IQR)]

Characteristic	Idiopathic	Bacterial	Viral	Tubercular	Neoplastic	Autoimmune	CKD	p-value	Test
Haemoglobin (g/dL)	6.40 (6.40–6.40)	11.2 (10.5–12.0)	6.60 (6.50–6.70)	9.45 (8.55–13.5)	10.3 (9.53–11.0)	10.0 (8.10–11.1)	7.70 (6.20–8.20)	0.169	Kruskal–Wallis
Total leukocyte count (/mm³)	6400 (6400–6400)	6345 (6172–6518)	40850 (25275–56425)	7350 (4650–7912)	8120 (7850–10540)	6500 (5275–8250)	7300 (6800–9500)	0.225	Kruskal–Wallis
Platelet count	1.50 (1.50–1.50)	2.24 (2.19–2.29)	2.29 (1.74–2.83)	1.65 (1.22–2.75)	3.14 (1.99–4.12)	1.67 (1.34–1.88)	1.58 (1.10–1.88)	0.630	Kruskal–Wallis
Total bilirubin (mg/dL)	0.80 (0.80–0.80)	0.75 (0.58–0.93)	0.50 (0.45–0.55)	1.30 (0.80–1.95)	0.60 (0.55–0.65)	0.60 (0.45–0.75)	0.50 (0.50–0.80)	0.167	Kruskal–Wallis
SGPT (U/L)	84.0 (84.0–84.0)	82.5 (69.8–95.2)	60.5 (49.2–71.8)	75.0 (40.0–106)	37.0 (25.2–101)	18.0 (14.0–58.0)	28.0 (26.0–28.0)	0.415	Kruskal–Wallis
SGOT (U/L)	92.0 (92.0–92.0)	58.5 (53.8–63.2)	35.5 (34.8–36.2)	59.5 (48.0–135)	59.5 (25.0–104)	48.0 (36.0–50.0)	38.0 (30.0–60.0)	0.401	Kruskal–Wallis
Creatinine (mg/dL)	1.50 (1.50–1.50)	0.75 (0.58–0.93)	1.10 (0.95–1.25)	1.05 (0.74–1.25)	0.95 (0.82–1.02)	0.90 (0.70–1.10)	3.47 (1.30–4.60)	0.216	Kruskal–Wallis
Urea (mg/dL)	50.0 (50.0–50.0)	28.0 (22.0–34.0)	75.0 (70.0–80.0)	38.5 (28.5–45.2)	36.0 (30.5–51.0)	54.0 (43.0–67.0)	75.0 (34.0–210)	0.393	Kruskal–Wallis
Sodium (mmol/L)	136 (136–136)	136 (134–138)	132 (132–133)	134 (133–136)	133 (127–134)	137 (134–138)	134 (130–136)	0.873	Kruskal–Wallis
Potassium (mmol/L)	5.00 (5.00–5.00)	3.95 (3.92–3.98)	5.05 (4.82–5.28)	4.05 (3.80–4.43)	3.85 (3.35–4.45)	3.60 (3.30–3.80)	3.80 (3.30–4.50)	0.316	Kruskal–Wallis
FBS (mg/dL)	112 (112–112)	107 (98.0–116)	102 (95.8–109)	132 (121–144)	134 (122–143)	95.0 (92.0–158)	191 (119–210)	0.417	Kruskal–Wallis
TSH	7.00 (7.00–7.00)	2.88 (2.69–3.07)	2.90 (2.90–2.90)	2.80 (2.02–3.68)	2.60 (1.97–3.00)	3.80 (3.05–3.90)	6.00 (4.20–8.00)	0.071	Kruskal–Wallis
T3	114 (114–114)	108 (107–110)	126 (123–128)	92.5 (86.0–114)	117 (106–127)	100 (95.0–106)	110 (109–112)	0.341	Kruskal–Wallis
T4	1.40 (1.40–1.40)	1.45 (1.12–1.78)	2.15 (1.88–2.43)	1.35 (1.20–1.42)	1.80 (1.48–2.15)	0.90 (0.85–0.90)	1.50 (1.24–1.90)	0.127	Kruskal–Wallis
CRP	17.0 (17.0–17.0)	6.70 (4.55–8.85)	170 (148–193)	11.5 (2.75–78.5)	29.5 (17.7–41.0)	22.7 (12.3–45.2)	1.20 (0.80–3.00)	0.108	Kruskal–Wallis

Table 3: Pericardial fluid analysis by aetiology

Characteristic	Idiopathic	Bacterial	Viral	Tubercular	Neoplastic	Autoimmune	CKD	p-value	Test
Drained volume (mL)	200 (200–200)	135 (77.5–192)	20.0 (20.0–20.0)	20.0 (20.0–288)	210 (17.5–425)	20.0 (15.0–260)	400 (300–450)	0.734	Kruskal–Wallis
Pericardial fluid cell count	70.0 (70.0–70.0)	115 (82.5–148)	30400 (17100–43700)	1100 (531–2200)	475 (320–700)	100 (90.0–150)	102 (100–150)	0.003	Kruskal–Wallis
Pericardial fluid sugar	108 (108–108)	63.0 (52.5–73.5)	25.0 (17.5–32.5)	74.0 (44.2–99.8)	69.0 (55.2–77.2)	105 (71.5–106)	101 (93.0–108)	0.299	Kruskal–Wallis
Pericardial fluid protein	2.60 (2.60–2.60)	4.80 (4.50–5.10)	3.55 (2.92–4.17)	5.70 (5.15–5.90)	4.98 (4.35–5.77)	3.80 (2.80–3.84)	3.10 (2.80–4.50)	0.050	Kruskal–Wallis
Pericardial fluid LDH	2.60 (2.60–2.60)	248 (234–262)	2837 (2050–3624)	1580 (920–2622)	715 (395–1320)	240 (200–414)	571 (342–842)	0.017	Kruskal–Wallis
Pericardial fluid ADA	10.0 (10.0–10.0)	25.0 (24.0–26.0)	24.5 (23.8–25.2)	57.0 (47.0–67.2)	20.0 (16.0–24.5)	24.0 (18.1–29.5)	18.0 (11.0–28.0)	0.042	Kruskal–Wallis
Serous fluid	1 (100.0)	2 (100.0)	0 (0.0)	0 (0.0)	0 (0.0)	3 (100.0)	5	<0.001	Pearson χ^2

							(100.0)		
Serosanguinous fluid	0 (0.0)	0 (0.0)	0 (0.0)	3 (37.5)	1 (25.0)	0 (0.0)	0 (0.0)	0.485	Pearson χ^2
Haemorrhagic fluid	0 (0.0)	0 (0.0)	2 (100.0)	5 (62.5)	3 (75.0)	0 (0.0)	0 (0.0)	0.029	Pearson χ^2
Fibrin strands present	0 (0.0)	1 (50.0)	0 (0.0)	7 (87.5)	0 (0.0)	0 (0.0)	0 (0.0)	0.005	Pearson χ^2
Malignant cells positive	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	3 (75.0)	0 (0.0)	0 (0.0)	0.090	Pearson χ^2
Culture/MTB positive	0 (0.0)	0 (0.0)	2 (100.0)	1 (12.5)	0 (0.0)	0 (0.0)	0 (0.0)	0.010	Pearson χ^2

Table 4: Diagnostic validity of ADA for tubercular pericardial effusion

Feature / cut-off	TP	FP	FN	TN	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	Fisher p
ADA ≥20 U/L	8	10	0	7	100.0	41.2	44.4	100.0	0.057
ADA ≥30 U/L	7	2	1	15	87.5	88.2	77.8	93.8	<0.001
ADA ≥40 U/L	7	0	1	17	87.5	100.0	100.0	94.4	<0.001
ADA ≥50 U/L	5	0	3	17	62.5	100.0	100.0	85.0	0.001
ADA ≥60 U/L	4	0	4	17	50.0	100.0	100.0	81.0	0.006

Table 5: Diagnostic validity of echocardiographic findings according to cardiac tamponade indication

Feature / cut-off	TP	FP	FN	TN	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	Fisher p
Large effusion on echo	7	4	0	14	100.0	77.8	63.6	100.0	<0.001
Doppler respiratory variation positive	6	3	1	15	85.7	83.3	66.7	93.8	0.003
Early RV diastolic collapse	0	4	7	14	0.0	77.8	0.0	66.7	0.294
Late diastolic indentation of RA	1	13	6	5	14.3	27.8	7.1	45.5	0.021
Late diastolic RA collapse	2	9	5	9	28.6	50.0	18.2	64.3	0.407
Exaggerated respiratory variations	7	7	0	11	100.0	61.1	50.0	100.0	0.008
Systolic RA/RV collapse	7	2	0	16	100.0	88.9	77.8	100.0	<0.001
IVC distended/no respiratory variation	6	0	1	18	85.7	100.0	100.0	94.7	<0.001

Table 6: Diagnostic validity of pericardial-fluid features for tubercular pericardial effusion

Feature / cut-off	TP	FP	FN	TN	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	Fisher p
Fibrin strands present	7	1	1	16	87.5	94.1	87.5	94.1	<0.001
Serosanguinous fluid	3	1	5	16	37.5	94.1	75.0	76.2	0.081
Haemorrhagic fluid	5	5	3	12	62.5	70.6	50.0	80.0	0.194
Serosanguinous or haemorrhagic fluid	8	6	0	11	100.0	64.7	57.1	100.0	0.003

Discussion

The lengthy survival of cancer patients, dialysis treatment for CRF, frequent use of anticoagulants, and radiation therapy for tumors are some of the factors contributing to the rising occurrence of pericardial effusions in recent years.[5]

In a tertiary care hospital in sub-Himalayan West Bengal, India, 25 patients with pericardial effusion participated in the current study to determine their aetiology and the relationship between the aetiologies and PE complications. Pericardial diseases can have a wide range of clinical signs, from asymptomatic to potentially fatal. The most common presenting symptom in our study was shortness of breath, which was reported by 88% of patients. This is consistent with the findings of studies by Desale et al., Neupane et al., and Khanal et al., where patients reported shortness of breath at rates of 93.3%, 85.3%, and 95%. [6-8] 48% of the individuals in our study reported having a temperature. In the study by Neupane et al. a somewhat lower percentage of patients (10.1%) reported having a fever.[7]

A significantly larger percentage of patients (54.5%) had fever, according to Uddin et al. [9] Similar to our results, Desale et al. and Khanal et al. also found a higher rate of fever (50% and 47.6%). [6,7]

Eighty percent of our patients reported having chest pain, which is a greater percentage. However, this is because the study cohort was primarily made up of symptomatic patients. JVP increased by 32%, which is comparable to Desale and Khanal's increases of 30% and 26.9%, respectively. However, a recent South Indian study found a significantly higher percentage of 73.9%.[10]

The causes of pericardial effusion in affluent countries differ from those in developing or underdeveloped Asian and African countries, according to existing literature. According to the current study, TB was the most common cause of PE among the patients (32%). One to two percent of all cases of tuberculosis are tuberculous pericarditis (TBP), a rare form of extrapulmonary tuberculosis. In low-income countries, TBP is the most common cause of substantial pericardial effusion.[11]

In miliary TB patients, the pericardium may be seeded; nevertheless, other organ systems will predominate in these cases. Direct extension from an infected visceral pleura or rib is quite rare. The breakdown of infection in the mediastinal nodes, which subsequently directly spreads into the pericardium, especially in the nodes at the tracheobronchial bifurcation, is the most frequent mode of transmission.[12]

According to Pradhan et al., TB was the most common cause of pericardial effusion in their patients (94.5%).[13]

Like Pradhan et al., Uddin et al., Khanal et al., and Desale et al. discovered that TB was the primary cause of PE (27.3%, 36.5%, and 50%).[6,8,9]

The echocardiogram of tuberculous PE has shown fibrin threads and thickened fluid.[14]

These characteristics were present in the majority of PE patients (> 50%) with tuberculous aetiology in our study. Similar to studies by Singh et al. (2019) (24.3%) and Gupta et al,[15,16] we discovered that 28% of patients experienced cardiac tamponade in our study, with CKD and neoplastic reasons being the most common causes. In a similar vein, Sagristà Sauleda et al. found a correlation between effusion brought on by cancer and tamponade in patients who showed no signs of inflammation.[17]

This finding contradicts that of Başar et al,[18] who studied 104 individuals and discovered that idiopathic reasons were the most prevalent. Idiopathic factors accounted for 31.1% of the aetiology in another Turkish investigation involving 123 individuals with pericardial effusion.[19]

Another study by Strobbe et al,[20] that included 269 patients who had pericardiocentesis within the previous ten years revealed that the aetiology was related with malignancy in 25% of instances and idiopathic in 26% of cases. Echocardiographic and Doppler hemodynamic measures did not significantly correlate with any specific aetiology.

Limitations: The following were the limitations of the current study: Our findings have limited external validity and generalizability because the study was conducted in a single institution. Another drawback is that a small sample size restricts the results' internal validity. It was unable to determine the patients' long-term results, such as mortality. The findings can only be applied to PE caused by medical conditions, which restricts their applicability to traumatic PE. The cross-sectional study design, in which all patients had pericardial effusion and no control group (no-PE patients) was included, made it impossible to investigate the relationship between comorbidities and the pericardial effusion. To confirm the validity of our findings, enhance their relevance in different contexts, and investigate additional problems like death, more multi-centric, long-term cohort studies must be conducted. Future research should include a comparative group of non-PE patients to investigate the relationship between comorbidities and pericardial effusion.

Conclusion

The geographic location is a crucial factor in comprehending the disease's epidemiology. Evaluation is not always necessary to identify the source of pericardial effusions. They are primarily classified as idiopathic in affluent nations, where they are thought to be connected to transient viral infections. Malignancies and illnesses are the second and third most frequent causes. On the other hand, tuberculosis continues to be the leading cause of the illness in poorer nations. Health decision-makers can approach illness prevention and treatment with the help of these epidemiological parameters. The three primary diagnostic pillars are echocardiography, physical examination, and history.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Little WC, Freeman GL: Pericardial disease. Circulation. 2006, 113:1622-32.
2. Cubero G, Rubin J, Martín M ...Pericardial effusion: Clinical and analytical parameters clues. International Journal of Cardiology, 108, 404-405
3. Munt BI, Moss RR, Grewal J: Pericardial disease. The Practice of Clinical Echocardiography, 4th Edition. Otto CM (ed): Saunders/Elsevier, Philadelphia; 2011. 565-78.
4. Becit N, Unlü Y, Ceviz M, Koçogullari CU, Koçak H, Gürlertop Y. Sub xiphoid pericardiostomy in the management of pericardial effusions: case series analysis of 368 patients. Heart 2005; 91(6): 785–90.
5. Guberman BA, Fowler NO, Engel PJ, Gueron M, Allen JM. Cardiac tamponade in medical patients. Circulation 1981; 64(3): 633–40.
6. Desale SS, Sharma SY, Chaudhary CK, Surnar SG. Study of etiological, and clinical profile outcome of patients, with pericardial effusion in a BKL Walawalkar Rural Medical College Dervan, Maharashtra. Int J Acad Med Pharm. 2022;4:772–6.
7. Neupane KR, Simkhada R, Manandhar R, Kansakar S, Yadav D, Kadel A, et al. Clinical profile of patients admitted with pericardial effusion in Shahid Gangalal National Heart Centre, Kathmandu, Nepal. Nepal Heart J. 2023;20(1):35–8. https:// doi. org/ 10. 3126/ njh. v20i1. 55003.
8. Khanal RR, Gajurel RM, Sahi R, Shrestha H, Poudel CM, Devkota S, et al. Study of etiological profile, clinical profile and short term outcome of patients presenting with Pericardial Effusion in a Tertiary Care Center. Nepal World J Cardiovasc Dis. 2019;9:12.
9. Uddin MJ, Singh MP, Mehdi MD. Study of etiological and clinical profile of pericardial effusion in a tertiary care hospital in Kosi region of Bihar. India Int J Adv Med. 2016;3:514–8.
10. Shaik et al. BMC Cardiovascular Disorders (2024) 24:714
11. Wang S, Wang J, Liu J, Zhang Z, He J, Wang Y. A case report and review of literature: Tuberculous pericarditis with pericardial effusion as the only clinical manifestation. Front Cardiovasc Med. 2022;9:1020672.
12. Cherian G. Diagnosis of tuberculous aetiology in pericardial effusions. Postgrad Med J. 2004;80:262–6.
13. Pradhan A, Vishwakarma P, Bhandari M, Sethi R, Snigdha B, Narain VS, et al. Demographic, clinical and etiological profile of pericardial effusion in India: a single centre experience. Indian J Tuberc. 2022;69:220–6.
14. Liu PY, Li YH, Tsai WC, Tsai LM, Chao TH, Yung YJ, et al. Usefulness of echocardiographic intrapericardial abnormalities in the diagnosis of tuberculous pericardial effusion. Am J Cardiol. 2001;87(1133–5):A10
15. Singh A, Kumar S, Himanshu D, Sethi R, Pradhan A. Clinico-epidemiological study of pericardial effusion in Northern India. Indian J Community Heal. 2019;31 3 SE-Original:322–30
16. Gupta M, Prasad M, Lal A, Mishra J, Ali I, Bhatta P, et al. Clinical profile and etiology of patients with pericardial effusion. J Natl Hear Lung Soc Nepal. 2023;2:97–102.
17. Sagristà-Sauleda J, Mercé J, Permanyer-Miralda G, Soler-Soler J. Clinical clues to the causes of large pericardial effusions. Am J Med. 2000;109:95–101.
18. Basar N, Turak O, Malçok Gürel Ö, Çağlı K, Özcan F, Ekizler A, et al. Pericardial effusion: etiology, diagnose and management. Düzce Med J 2012; 14(2): 23–7.
19. Ercan S, Özer O, Yavuz F, Kaplan M, Alıcı MH, Günsoy B, et al. Clinical, laboratuary and echocardiographic features of patients with pericardial effusion in Gaziantep region. Gaziantep Med J 2013; 19(2): 81–5

20. Strobbe A, Adriaenssens T, Bennett J, Dubois C, Desmet W, McCutch eon K, et al. Etiology and long-term outcome of patients undergoing pericardiocentesis. J Am Heart Assoc 2017; 6(12): e007598.