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Evaluation of in-hospital and long-term outcomes of patients undergoing pericardiocentesis: A retrospective analysis

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Abstract

Aim: Pericardiocentesis is a critical invasive procedure for the diagnosis and treatment of patients with pericardial effusion, regardless of the etiological cause. We aimed to evaluate the in-hospital and long-term outcomes of patients undergoing pericardiocentesis due to pericardial effusion at a tertiary referral center.

Materials and Methods: A retrospective analysis of 204 patients who underwent pericardiocentesis between 2017 and 2022 was conducted. Patients were divided into two groups based on the development of mortality during long-term follow-up.

Results: The mean follow-up duration was 42.5 ± 22.7 months. The most commonly identified etiology was idiopathic, accounting for 44.1%, followed by neoplastic (33.3%) and infectious (10.8%) causes. In-hospital mortality occurred in 21 (10.2%) patients, while recurrence developed in 41 (20.1%) patients. During the follow-up period, 74 out of 204 participants experienced mortality. Multivariable Cox regression analysis identified left ventricular ejection fraction (LVEF) ($p=0.013$), malignancy ($p<0.001$), and hemodynamic instability ($p<0.001$) as independent determinants of long-term mortality.

Conclusion: Although pericardiocentesis has a low complication rate, mortality rates remain high in these patients due to additional comorbidities. LVEF, malignancy, and hemodynamic instability determine mortality. Careful management of this group can reduce further events.

Introduction

Pericardial effusion (PE), characterized by abnormal accumulation of fluid between the layers of the pericardium, emerges as a clinical condition either as a sign of systemic or cardiac disease. Depending on the rate, quantity, and etiological cause of fluid accumulation, PE can lead to pericardial tamponade, which can range from asymptomatic to potentially fatal clinical presentations. Infectious, neoplastic, autoimmune, and iatrogenic causes are the most common causes encountered in clinical practice [1]. The underlying aetiology and size of fluid accumulation are associated with prognosis.

Pericardiocentesis is a critical invasive procedure for the diagnosis and treatment of patients with PE, independent of the etiological cause. This procedure was first performed by Riolanus for cardiac tamponade [2]. Initially, blind pericardiocentesis procedures were associated with high morbidity and mortality rates. However, with the advent of echocardiography-guided procedures, these rates have decreased, making pericardiocentesis vital for the diagnosis and management of significant PEs [3]. In medium to large-sized PEs, the success rates of echocardiography-guided pericardiocentesis are above 95%. The morbidity rate is approximately 1-3%, and the procedure-related mortality rate is less than 1% [4]. While mild fluid accumulations may respond to medical treatment, severe fluid accumulations can lead to cardiac dysfunction, making pericardiocentesis the most beneficial treatment method in the absence of clinical contraindications [5]. The decrease in complication rates in pericardiocentesis has made it more attractive in diagnosis and treatment. However, comprehensive data regarding the profile and prognosis of patients undergoing pericardiocentesis are currently insufficient.

This study aims to describe our experience with percutaneous pericardiocentesis performed due to PE at our tertiary referral center, and to determine the clinical char-

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acteristics, complications, and factors associated with in-hospital and long-term mortality in patients undergoing this intervention.

Materials and Methods

Study population

This study represents an observational, retrospective analysis conducted at a single center. Adult patients aged 18 years and above who underwent percutaneous pericardiocentesis in the intensive care unit of our tertiary referral hospital due to a diagnosis of PE from January 2017 to June 2022 were included in the study. Patients with effusion occurring after cardiac or other intrathoracic surgeries, acute aortic syndromes, patients with known tuberculosis, iatrogenic PE following invasive cardiac procedures were excluded from the study. Following the application of inclusion and exclusion criteria, a cohort comprising 204 patients were included in the study. Each participant underwent follow-up assessments for an average duration of 42.5 ± 22.7 months. The baseline and follow-up data of patients were retrospectively reviewed through the hospital's electronic record system. Demographic and clinical characteristics of patients, etiological diagnosis of PE, status at clinical presentation (asymptomatic, tamponade, dyspnea, hemodynamic instability), in-hospital surgical requirement, complications related to pericardiocentesis, site of intervention, macroscopic and microscopic characteristics of pericardial fluid, date of death, as well as other variables were recorded retrospectively. All procedures were performed under sterile conditions in the coronary intensive care unit by an experienced invasive cardiologist under local anesthesia. Following the procedure, all patients were monitored in the coronary intensive care unit. Twenty-one patients (10.3%) were asymptomatic and the majority of patients (89.7%) were symptomatic. Asymptomatic patients were mostly incidentally detected with computed tomography at an external center and referred to us. The success of the procedure was defined as successful drainage of pericardial fluid until minimal fluid remained, no deaths during the procedure, and no requirement for open surgeries. The primary endpoint of the investigation was long-term all-cause mortality. For the study protocol received approval from the Scientific Research Ethics Committee of the University of Health Sciences, Mehmet Akif Ersoy Thoracic and Cardiovascular Surgery Training and Research Hospital (Decision no: 2024.01-10) and adhered to the principles stated in the Declaration of Helsinki. Written informed consent was obtained from all enrolled individuals prior to their participation in the study.

Definition

All patients underwent percutaneous pericardiocentesis under echocardiographic guidance, followed by pericardial drainage with sheath and pigtail until minimal fluid remained. Patients with pericardial fluid containing atypical cells on cytology, those with known neoplasia diagnosis, or those newly diagnosed with imaging methods showing pericardial involvement but with negative pericardial fluid cytology were considered to have neoplastic effusion. PE occurring during invasive cardiac procedures (coronary interventions, pacemaker insertion, percutaneous valvuloplasty, etc.) was defined as iatrogenic. Patients with known autoimmune disease and those with polyserositis were defined as having autoimmune effusion, and those with PE requiring dialysis without any other reason and BUN levels of 60 mg/dl were defined as having uremic effusion. Congestive heart failure was defined as EF $<$ 50%. A history of cerebrovascular events included stroke or transient ischemic attack. Transudate and exudate differentiation was made by simultaneously collected blood samples and evaluation of pericardial fluid using light criteria.

Hemodynamic instability was defined as cardiac arrest, systolic blood pressure below 90 mmHg despite adequate fluid replacement or the need for vasopressors to maintain blood pressure above 90 mmHg, and signs of end-organ hypoperfusion (altered mental status, oliguria/anuria, increased serum lactate) [6]. Emergent surgery was defined as the requirement for surgical intervention following pericardiocentesis (myocardial wall rupture requiring surgical repair, or requirement for pericardial window). Major complications were defined as acute myocardial infarction, myocardial wall perforation, intra-abdominal organ injury or diaphragmatic injury, pneumothorax and hemothorax. Minor complications were defined as arrhythmia, pericarditis, and acute pulmonary edema.

Follow-up and outcomes

All patients underwent echocardiography post-procedure and then at 1, 6, and 12 months before discharge for follow-up of PE. Annual evaluations were subsequently conducted. The primary endpoint of the study was the rate of all-cause mortality; patients were followed from the day of pericardiocentesis until death. The cause and time of death were obtained from hospital records and national death records. This paper was prepared in accordance to The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) Statement: guidelines for reporting observational studies.

Statistical analysis

The analysis was conducted using SPSS 26.0 software (SPSS, Chicago, IL). The normality of variables was assessed using Kolmogorov-Smirnov tests, histograms, and probability plots. Numerical variables were reported as mean ± standard deviation (e.g., age, hemoglobin, etc.) or median (interquartile range) (e.g., C-reactive protein, creatinine, etc.) depending on their distributions. Categorical variables such as gender, smoking status, etc., were expressed as percentages (%). Numerical variables between two groups were compared using Student's t-test or Mann-Whitney U test, while categorical variables were compared using Chi-square or Fisher's exact test. Kaplan-Meier modeling was used to depict the time until the cessation of events as an indicator of post-pericardiocentesis mortality. Statistical comparisons of time-to-event data for various interventions and controls were made using log-rank tests and reported as mean survival rates (years ± 95% CI). Additionally, a single-variable Cox proportional hazards model was used to calculate hazard ratios (HRs) and corresponding 95% confidence intervals (95% CIs) for

long-term mortality in patients undergoing pericardiocentesis. Multivariable Cox proportional hazard models were used to evaluate potential independent determinants of survival. A significance level of p<0.050 was set.

Results

A total of 204 patients were included in the study, with a mean age of 65.7 ± 15.6 years and 96 (47.1%) female participants. In-hospital mortality occurred in 21 (10.2%) patients, while recurrence occurred in 41 (20.1%) patients during the follow-up period. Throughout the follow-up, 74 (36.3%) participants experienced death. Participants were divided into two groups: survivors and non-survivors. The clinical, demographic characteristics, comorbidities, and echocardiographic data at presentation are detailed in Table 1. While demographic characteristics were comparable between the two groups, non-survivors had higher age (p<0.001), female gender (p=0.007), chronic heart failure (p = 0.002), and chronic obstructive pulmonary disease (COPD) (p<0.010).The main reason of higher mortality of female patient group could be increased frailty and cancer subgroup preogression.

Echocardiographic characteristics showed similarities between the groups, however, higher Left Ventricular Ejection Fraction (p=0.001) was found in the non-survivor group. Sinus tachycardia was the most common electrocardiographic finding at presentation, observed in 84 patients (41.2%). Dyspnea was the most common presenting complaint clinically, accounting for 59.3%, followed by chest pain at 25.5%. Hemodynamic instability was present in 35 patients (17.2%) at presentation, which was significantly higher in the non-survivor group (p<0.001). The mean length of hospital stay was 12.8 ± 9.7 days, with patients followed up for an average of 42.5 ± 22.7 months. Additionally, follow-up time was higher in survivors (p<0.001).

Patients' laboratory data at presentation are detailed in Table 2. Upon examination of laboratory parame-

Table 1. Demographic and clinical features of the patients.

	All patients (n=204)	Survivors group (n=130)	Mortality group (n=74)	P value
Age, year, mean (SD)	65.7 ± 15.6	62.6 ± 16	71.1 ± 13.2	**<0.001**
Gender (female), n (%)	96 (47.1)	52 (40)	44 (59.5)	**0.007**
Diabetes mellitus, n (%)	72 (35.5)	49 (37.7)	23 (31.5)	0.377
Hypertension, n (%)	123 (60.6)	82 (63.1)	41 (56.2)	0.333
Hyperlipidemia, n (%)	76 (37.4)	46 (35.4)	30 (41.1)	0.420
Coronary artery disease, n (%)	83 (40.7)	52 (40)	31 (41.9)	0.791
Chronic heart failure, n (%)	19 (9.3)	6 (4.6)	13 (17.6)	**0.002**
Chronic renal failure, n (%)	60 (29.6)	33 (25.4)	27 (37)	0.082
COPD, n (%)	53 (26)	26 (20)	27 (36.5)	**0.010**
Previous CVD, n (%)	21 (10.3)	12 (9.2)	9 (12.2)	0.508
Echocardiographic evaluation, n (%)				
LVEF, (%)	57.2 ± 7.7	58.5 ± 5.6	54.7 ± 10	**0.001**
LVEDD, mm	46.1 ± 4.7	46.4 ± 5	45.7 ± 4.1	0.268
LVESD, mm	30 ± 5.3	30 ± 5.6	30 ± 4.7	0.986
LAD, mm	36.6 ± 6.1	36.4 ± 6.2	36.9 ± 6.1	0.579
Effusion size, mm	2.93 ± 1.16	2.87 ± 0.71	2.9 ± 0.97	0.800
Electrocardiographic evaluation, n (%)				0.134
Tachycardia, n (%)	84 (41.2)	48 (36.9)	36 (48.6)	
Low voltage, n (%)	58 (28.4)	44 (33.8)	14 (18.9)	
Electrical alternans, n (%)	9 (4.4)	5 (3.8)	4 (5.4)	
Clinical presentation, n (%)				0.581
Asymptomatic	21 (10.3)	10 (7.7)	11 (14.9)	
Dyspnea	121 (59.3)	78 (60)	43 (58.1)	
Chest pain	52 (25.5)	35 (26.9)	17 (23)	
Syncope	7 (3.4)	5 (3.8)	2 (2.7)	
Peripheral edema	3 (1.5)	2 (1.5)	1 (1.4)	
Hemodynamic instability, n (%)	35 (17.2)	1 (0.8)	34 (45.9)	**<0.001**
Recurrence (n, %)	41 (20.1)	21 (16.2)	20 (27)	0.062
ICU length of stay, days	5.2 ± 3.6	5.1 ± 3.6	5.3 ± 3.7	0.782
Hospital length of stay, days	12.8 ± 9.7	13.6 ± 10.6	11.2 ± 8.4	0.086
Follow-up, months	42.5 ± 22.7	49.3 ± 20.7	30.5 ± 21.3	**<0.001**

Abbreviation: COPD: Chronic obstructive pulmonary disease; CVD:cerebrovascular disease; LAD: Left atrial diameter; LVEDD: Left ventricular end-diastolic diameter; LVEF Left ventricular ejection fraction; LVESD: Left ventricular end-systolic diameter; ICU: intensive care unit.

Table 2. Laboratory features of the patients.

	All patients (n=204)	Survivors group (n=130)	Mortality group (n=74)	P value
Hemoglobin (g/dL)	11.2 ± 2	11.3 ± 2	11 ± 2.1	0.358
Leukocyte (10^3/mm^3)	9.5 ± 4.2	9.3 ± 4.5	9.8 ± 3.8	0.432
Neutrophil, 10^9/L	9.1 ± 5.4	9.2 ± 5.6	9 ± 5.2	0.818
Lymphocyte, 10^9/	1.35 ± 0.78	1.41 ± 0.65	1.23 ± 0.95	0.097
L Monocyte, 10^9/L	0.78 ± 0.38	0.79 ± 0.42	0.77 ± 0.31	0.704
NLR	6.4 (4.1-11.5)	5.9 (4-9.5)	7.5 (4.3-14.7)	**0.040**
LMR	1.8 (1-2.6)	1.8 (1.3-2.8)	1.4 (0.7-2.5)	**0.024**
Platelet, (10^3/mm^3)	278.2 ± 119.6	267.2 ± 108.4	297.5 ± 135.7	0.082
C-reactive protein, mg/L	35.7 (10.4-86.7)	29.1 (7.3-90)	44.7 (16-80.2)	0.064
Fasting glucose (mg/dL)	136 ± 59.7	130.9 ± 57.2	145.1 ± 63.3	0.102
Albumin, g/dL	34.8 ± 8.7	35.6 ± 9.3	33.4 ± 7.5	0.081
Creatinine, mg/dL	0.93 (0.7-1.3)	0.85 (0.68-1.23)	1.04 (0.76-1.5)	**0.016**
Total cholesterol, mg/dL	140.2 ± 35.1	137.4 ± 33.2	145.2 ± 38	0.128
LDL, mg/dL	105.5 ± 37.7	104.6 ± 37	107 ± 39.3	0.665
HDL, mg/dL	38.4 ± 15.1	37.8 ± 14.8	39.6 ± 15.6	0.411
Triglyceride, mg/dL	108.3 ± 47.5	104.8 ± 44.6	114.6 ± 52	0.158
ALT (U/L)	21 (13-38)	20 (13.8-38)	22 (13-47)	0.347
AST (U/L)	26 (17-38)	26 (17-34.5)	26 (16-38)	0.756
TSH (mU/L)	1.78 (0.97-2.69)	1.82 (1.12-3.04)	1.28 (0.61-2.33)	0.181
Troponin, mg/L	16 (6.8-29.4)	13 (5-23)	20 (9.9-39.4)	**0.002**
Lactate, mmol/L	0.8 (0.7-1.04)	0.8 (0.7-0.9)	1.15 (0.77-1.8)	**<0.001**

Abbreviation: ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; HDL-C: High-density lipoprotein cholesterol; LDL-C: Low-density lipoprotein cholesterol; LMR, lymphocyte-monocyte ratio; NLR: neutrophil lymphocyte ratio; TSH: Thyroid Stimulating Hormone.

Table 3. Procedural data of the patients.

	All patients (n=204)	Survivors group (n=130)	Mortality group (n=74)	P value
Site of entry				0.772
Subksifoid	91 (44.6)	57 (43.8)	34 (45.9)	
Apical	113 (55.4)	73 (56.2)	40 (54.1)	
Catheter used				0.857
Pigtail	164 (80.4)	105 (80.8)	59 (79.7)	
Sheath	40 (19.6)	25 (19.2)	15 (20.3)	
Size of pericardial effusion				0.193
Large	179 (87.7)	117 (90)	62 (83.8)	
Medium	25 (12.3)	13 (109)	12 (16.2)	
Tamponade physiology				0.977
Yes	94 (46.1)	60 (46.2)	34 (45.9)	
No	110 (53.9)	70 (53.8)	40 (54.1)	
Distribution of pericardial effusion				0.432
Circumferential	168 (82.4)	105 (80.8)	63 (85.1)	
Loculated	36 (17.6)	25 (19.2)	11 (14.9)	
Appearance of effusion, n, (%)				0.226
Serous	72 (35.3)	46 (35.4)	26 (35.1)	
Serohemorrhagic	5 (2.5)	5 (3.8)	0 (0)	
Hemorrhagic	127 (62.3)	79 (60.8)	48 (64.9)	
Drainage volume (ml)	982.2 ± 468.2	1016.9 ± 451.4	921.4 ± 493.8	0.162
Duration of drainage (hour)	16.2 ± 10.8	14.4 ± 10.3	19.4 ± 10.9	**0.001**
Minor complication (n, %)	18 (8.8)	12 (9.2)	6 (8.1)	0.941
Major complication (n, %)	10 (4.9)	6 (4.6)	4 (5.4)	0.802
Etiologies				**<0.001**
Malignancy, n (%)	68 (33.3)	22 (16.9)	46 (62.2)	
Idiopathic, n (%)	90 (44.1)	73 (56.2)	17 (23)	
Infectious, n (%)	22 (10.8)	19 (14.6)	3 (4.1)	
Uremia, n (%)	6 (2.9)	2 (1.5)	4 (5.4)	
Thyroid diseases	3 (1.5)	1 (0.8)	2 (2.7)	
Post-MI conditions	7 (3.4)	6 (4.6)	1 (1.4)	
CNTD	8 (3.9)	7 (5.4)	1 (1.4)	

Abbreviation: CNTD: Connective tissue disease, MI: Myocardial infarction.

ters, higher levels of Neutrophil-Lymphocyte Ratio (NLR) (p=0.040), creatinine (p<0.016), troponin (p<0.002), and lactate (p<0.001) were observed in the non-survivor group, while the Lymphocyte-Monocyte Ratio (LMR) (p=0.024) value was higher in the survivor group. Other laboratory parameters were not comparable between the two groups.

Most procedures were performed in emergency or urgent settings. The intervention site was apical in 113 patients (55.4%). The most common indication for intervention was cardiac tamponade. Cardiac tamponade physiology was echocardiographically present in 94 patients (46.1%)when macroscopically evaluated, pericardial fluid was predominantly hemorrhagic in nature in most patients (62.3%). The most commonly identified etiology was idiopathic (44.1%), followed by neoplastic (33.3%) and infectious causes (10.8%). Iatrogenic effusions developed during cardiovascular interventions were not included in the study. Procedure-related data, including pericardial fluid characteristics and etiological factors, are detailed in Table 3.

Periprocedurally, minor complications developed in 18 patients (8.8%), while major complications occurred in 10 patients (4.9%). Major complications included myocardial wall perforation (n:3), intrabdominal organ injury or diaphragmatic damage (n:2), pneumothorax (n:4), and hemothorax (n: 1). Among patients with major complications, two developed in-hospital mortality, one due to myocardial perforation and the other due to sepsis following hepatic vein injury. Four patients developed entry site infection, with in-hospital mortality in two cases and recurrence during long-term follow-up in one case. Another finding of the study was the longer drainage duration in the group with mortality (p=0.001).

Univariate Cox regression analyses were conducted with all parameters to determine the predictors of long-term mortality; age, gender, COPD, LVEF, malignancy, drainage duration, hemodynamic instability, NLR, and creatinine were identified as parameters associated with mortality (Table 4). In the multivariate Cox regression analysis using these parameters, LVEF (p=0.013), malignancy (p<0.001), and hemodynamic instability (p<0.001) were found to be independent determinants of long-term mortality.

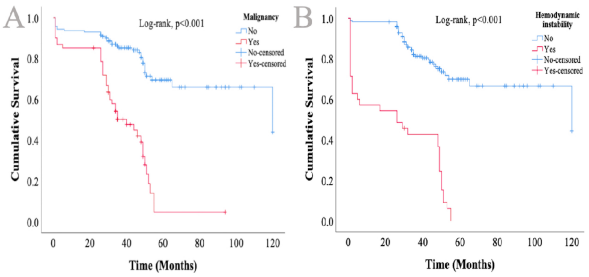

Figure 1. Kaplan-Meier survival curves for malignancy (A) and hemodynamic instability (B) in-long term mortality.

Kaplan-Meier survival analysis represented that (Figure 1), malignncy and haemodynamic unstability significantly increased long term mortality rates (Log-rank: p<0.001).

Discussion

We present the results of a retrospective cohort study evaluating the clinical characteristics and outcomes of adult patients undergoing pericardiocentesis due to pericardial effusion at our tertiary cardiovascular center. It also demonstrates the analysis of the patient population undergoing pericardiocentesis according to specific etiology, along with predictors of poor clinical outcomes and mortality, highlighting the safety and effectiveness of pericardiocentesis. The main findings of our study are summarized below:

Table 4. Univariate and multivariate Cox regression analysis to identify long-term predictors of mortality.

Variables	Univariate Analyses			Multivariate Analyses		
	HR	95%CI (lower–upper)	P value	HR	95%CI (lower–upper)	P value
Age	1.029	1.013-1.046	**<0.001**	1.018	0.998-1.038	0.080
Gender	0.486	0.305-0.774	**0.002**	0.650	0.380-1.112	0.116
COPD	1.931	1.200-3.108	**0.007**	1.066	0.637-1.785	0.807
LVEF	0.956	0.934-0.978	**<0.001**	0.966	0.940-0.993	**0.013**
Recurrence	1.632	0.975-2.730	0.062			
Drainage volume	1	0.999-1	0.076			
Malignancy	4.718	2.935-7.585	**<0.001**	3.525	1.946-6.386	**<0.001**
Drainage time	1.025	1.006-1.045	**0.011**	1.020	0.998-1.043	0.078
Hemodynamic instability	6.469	4.048-10.338	**<0.001**	2.858	1.623-5.034	**<0.001**
NLR	1.016	1.004-1.029	**0.011**	0.994	0.970-1.018	0.606
CRP	1.002	1.000-1.005	0.098			
Creatinine	1.222	1.037-1.439	**0.017**	1.196	0.960-1.490	0.110
Albumin	0.982	0.961-1.003	0.088			

Abbreviations: HR, hazard ratio; others, see Table 1.

** LVEF (p=0.013), malignancy (p<0.001), and hemodynamic instability (p<0.001) were identified as independent determinants of long-term mortality.

Pericardiocentesis is a preferred method for draining extensive PE, whether life-threatening cardiac tamponade is present or not. It is a life-saving procedure in diagnosis and treatment. The major complication rate in our study was 4.9%, which is considered quite safe. This rate is consistent with European data (4-10%) and larger studies (5.9%) [7]. In light of our study findings, we recommend performing pericardiocentesis under echocardiographic or fluoroscopic guidance to enhance safety.

In our study, when etiological factors were evaluated, the most frequently identified etiology was idiopathic, followed by neoplastic and infectious causes. Our study data were comparable to many studies in the literature. In a study by Andrea Pennachioni et al., the most common etiology was idiopathic (33.3%), followed by neoplastic (22.2%) [8]. Additionally, in several studies in the literature, when excluding cardiac surgeries, neoplastic etiology has been more predominant [9]. In another study conducted at a tertiary center, malignancy was identified as the most common etiological cause, which was found to be a predictor for increased long-term mortality [10]. The difference observed in our study could be attributed to our center being a tertiary cardiovascular center and possibly inadequate diagnostic tests for idiopathic etiology.

In this current study, advanced age and low LVEF were identified as independent rik factors for increased long term mortality. These two parameters have shown to be predictors for worse prognosis in numerous occasions. In a study by Frohlich et al., reduced LVEF was found to be associated with adverse clinical outcomes in patients with pericardial effusion, including those without hemodynamic decompensation or instability [11]. Additionally, congestive heart failure is a risk factor for both the cause of PE and hemodynamic decompensation in PE due to systemic inflammation. This condition could also explain the association between reduced LVEF and mortality.

Predictably, in our study, patients with hemodynamic instability at the time of admission had higher in-hospital mortality independent of the etiology of PE. The increased in-hospital mortality can be explained by acute fluid accumulation and acute cardiac decompensation, leading to the requirement of urgent life-saving procedures and thereby increasing the patient's clinical risk. In our study, the in-hospital mortality was 10.2%, which is consistent with many studies in the literature. A recent study originated from Italy represented in-hospital mortality of 14.8% in such patient group, and hemodynamic instability was identified as an independent predictor of mortality at that study [8]. Both in-hospital mortality and long-term mortality were significantly higher in hemodynamically unstable at the time of admission. Although there is no study based solely on hemodynamic instability in the literature, patients with cardiac tamponade for any reason and organ malperfusion have been found to have significantly higher in-hospital mortality [12].

In many studies, in-hospital mortality has been reported to be between 14-19%. Some studies have shown an increased in-hospital mortality rate in patient groups with PE secondary to myocardial infarction, coagulopathies/ongoing anticoagulant therapy-related pericardial effusion, and iatrogenic pericardial effusion following percutaneous cardiovascular interventions [9]. However, in our study, the number of such cases is quite low. In our cohort, there were only 7 patients with post-myocardial infarction PE and patients with iatrogenic PE were excluded from the study, and there were no patients with coagulopathy. Therefore, the slightly lower in-hospital mortality observed in our study may be attributed to these factors.

In our study, patients with neoplastic etiology of PE had significantly higher mortality rates during long-term follow-up. However, there was no significant difference in in-hospital mortality. In a recent study conducted in our country, three groups were evaluated for mortality: pa-

tients without malignancy, patients with malignancy and negative pericardial fluid cytology, and patients with malignancy and positive pericardial fluid cytology. Positive pericardial cytology was identified as a poor prognosis indicator [13]. While there may be no difference in mortality during the acute phase, the higher mortality observed in the medium and long term may be associated with the advanced stage of primary malignancy and its prognosis.

This study is a unique report of short-term and long-term results of pericardiocentesis and the results are consistent with the current literature.

Limitations

The study has several limitations. Firstly, it was conducted at a single center and designed retrospectively, which may limit the generalizability of the results and be dependent on the characteristics of a specific population. Secondly, the sample in our study was selected from a single hospital setting over a specific period, potentially limiting the validity of the findings to the general population. Additionally, the sample size may affect the power of specific subgroup analyses. Thirdly, incomplete or missing demographic and clinical data in our study may limit the ability to fully assess all potential effects. Particularly, the lack of clinical evaluations of patients during long-term follow-up could also impact the results. Lastly, the exclusion of certain patients, as example patients with tuberculosis, may limit the generalizability of the results to other patient groups. Considering these limitations is important for interpreting the research findings and assessing their generalizability. Future research could overcome these limitations and achieve more comprehensive results by employing different methodological approaches.

Conclusion

This study was conducted to understand the in-hospital and long-term outcomes of patients undergoing pericardiocentesis at our tertiary cardiovascular center. Our findings demonstrated that complication and mortality rates were consistent with similar studies in the literature. To understand the predictors of short term and long term mortality can enlighten the horizon of clinicians, thus further precautions could be taken in patients with additional risks.

Ethical approval

This study received approval from the Scientific Research Ethics Committee of the University of Health Sciences, Mehmet Akif Ersoy Thoracic and Cardiovascular Surgery Training and Research Hospital (Decision no: 2024.01-10).

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