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Cardiac tamponade in people living with HIV admitted to a tertiary care center: a real-life experience

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Abstract

Introduction: Cardiac tamponade is a critical condition that occurs after a sudden and excessive accumulation of fluid in the pericardial space. It restricts the appropriate filling of the cardiac chambers, disturbing normal hemodynamics, and ultimately causing hypotension and cardiac arrest. Prompt, accurate diagnosis and treatment with percutaneous drainage can minimize patients' mortality.

Material and methods: Fifteen HIV-positive patients with cardiac tamponade were admitted to a tertiary hospital between January 2013 and January 2019. After clinical examination and echocardiographic assessment, all patients were subjected to emergency pericardiocentesis for both therapeutic purposes. Three samples of the aspirate were sent for biochemical, serological, and cytological analysis. Collected data were analyzed.

Results: HIV-infected patients on antiretroviral therapy, including 5 females (33.3%) and 10 males (66.7%), were followed up for 6 years. The mean age was 40.2 years, and the mean CD4+ count was 502/mm^3. Echocardiographic abnormalities were significantly more common in HIV-positive cases with cardiac tamponade with systolic and diastolic dysfunction. The overall mortality rate in the study was 13%.

Conclusions: In general, cardiac abnormalities are more common in HIV-infected people. HIV-positive patients with a CD4+ cell count > 200/mm^3 had no echocardiographic abnormalities. Patients with a more advanced infection and those with a CD4+ cell count < 200/mm^3 had a significantly abnormal left ventricular systolic function and a higher incidence of cardiac tamponade. A careful initial evaluation with clinical, serologic, and echocardiographic assessment is necessary to detect cardiac tamponade in HIV-infected cases.

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Introduction

Cardiac involvement is a recognized complication in HIV-infected individuals, with conditions, such as pericardial effusion and cardiac tamponade posing significant challenges in clinical management [1, 2]. The pathophysiology of cardiac tamponade in HIV patients may vary, including idiopathic or viral pericarditis pericarditis, uremia, and other related factors [3]. Traditional management options for cardiac tamponade include percutaneous catheter drainage and surgical tube placement, each with its associated risks and benefits [4]. However, given the unique considerations in HIV-infected individuals, such as potential complications related to immune dysfunction and medication interactions, a comprehensive evaluation of clinical, etiological, and echocardiographic data is essential for optimal patient care [5].

This study aimed to analyze the specific cardiac manifestations and management strategies in 15 consecutive HIV-infected patients on antiretroviral therapy (ART) with pericardial effusion and tamponade, shedding light on the nuanced approach required for this patient population.

Material and methods

Study design and study period

Between January 2013 and January 2020, a prospective study was carried out in a tertiary care center in South India to determine the prevalence, incidence, and echocardiographic assessment of cardiac tamponade in HIV-infected subjects undergoing ART.

Inclusion and exclusion criteria

HIV-positive patients with cardiac tamponade diagnosed clinically or using echocardiography, undergoing ART were primarily considered for this study. Whereas those who did not consent to pericardiocentesis or with no clinical or echocardiographic features of cardiac tamponade were excluded.

Study protocol

Patient history was collected, and physical examination was performed after obtaining consent for participation. An ECG, chest roentgenography, and laboratory data, including routine chemistry, hematology, and T4 cell counts were performed at baseline. In all 15 HIV-positive ART cases, cardiac tamponade was defined by clinical and echocardiographic criteria, with location and distribution of cardiac tamponade confirmed by 2D echocardiography. All patients were immediately shifted to a catheterization laboratory after diagnosis of cardiac tamponade. Using the subxiphoid approach, pericardiocentesis was performed, and pericardial fluid was fully drained and submitted for culture and cytological analysis. Injecting a small amount of agitated saline readily confirmed the sheath position. As clinically indicated, aspirations were carried out.

Associated diagnosis

Tuberculosis was based on a positive culture, and congestive cardiac failure was considered a sign and symptom of heart failure. If sputum smears were positive, pneumocystis infection was deemed present. Histopathological examination was performed for Kaposi's sarcoma and lymphoma diagnoses.

Echocardiography

Two-dimensional echocardiography was done, and pericardial effusion was diagnosed if an echo-free space between the visceral and parietal pericardium persisted throughout the cardiac cycle. The size of a pericardial effusion was defined as small when the maximum pericardial space at end-diastole was < 10 mm, moderate when the space was ≥ 10 mm but < 20 mm, and large when the pericardial space was ≥ 20 mm between the pericardial layers.

Follow-up

All patients were followed up for 7 days, 1 month after discharge, and every 3 months afterward.

Results

Clinical, etiological, and echocardiographic data of 15 consecutive HIV-infected patients were evaluated. Table 1 shows the patients' demographic and clinical parameters. The mean age of the patients was 40.2 years, the mean CD4+ count value was 502/mm^3 (Tables 2 and 3), and the mean pulse rate was 84.4 bpm. The most common symptoms relevant to the heart were cough (40%), palpitations (7%), and shortness of breath (25%). Fourteen patients had over symptoms of heart failure dyspnea at rest, orthopnea, and paroxysmal nocturnal dyspnea. In all 15 cases, right atrial and right ventricular systolic collapse occurred. The A wave velocity from the mitral flow was statistically different, with a lower E/A ratio in more advanced cases. However, there was no statistical difference between the isovolumetric relaxation time (IVRT) and the deceleration time (DT). In all 15 patients, successful pericardiocentesis was achieved on the first attempt. No patient required surgical intervention due to a failed puncture. Also, no early complication of pericardial punctures, such as cardiac perforation, ventricular fibrillation, or pneumothorax was observed, and no death as a consequence of the procedure was documented. The average volume of pericardial fluid drained was 1,550 ± 280 cc. During follow-up, no fever, infection, or hematoma occurred. The pericardial fluid was sent for cytopathology, and the macroscopic appearance of the drained materials was serous in 3 patients, hemorrhagic in 8, and straw-colored in 7 cases. Two patients died due to opportunistic infections. Biochemical analysis revealed that all specimens were exudates in 5 patients and transudates in 10 patients, according to Light's criteria [6]. The cause of cardiac tamponade in the study patients was HIV infection.

Table 1. Patient demographic and clinical data

Variables	*n* (%)
Gender	
Female	5 (33.3)
Male	10 (66.7)
Age group (years)	
21-30	1 (6.7)
31-40	10 (66.6)
41-50	1 (6.7)
51-60	3 (20.0)
Dyspnea	
Present	14 (93.3)
Absent	1 (6.7)
Clinical presentation	
Dyspnea	14 (93.3)
Fatigue	13 (86.6)
Chest pain	10 (66.7)
Fever	6 (40.0)
Tachycardia (HR > 100)	
Present	5 (33.3)
Absent	10 (66.7)
Hypotension (SBP < 100)	
Present	8 (53.3)
Absent	7 (46.7)
JVP	
Elevated	13 (86.7)
Not elevated	2 (13.3)
Tachypnea	
Present	15 (100.0)
Absent	0 (0)
Anemia	
Present	6 (40.0)
Absent	9 (60.0)
Aspirated fluid quantity (ml)	
< 500	2 (13.3)
500-1,000	10 (66.7)
1,000-1,500	3 (20.0)

HR – heart rate, JVP – jugular venous pressure, SBP – systolic blood pressure

Table 2. Patient HIV parameters (*N* = 15)

Parameters	
Time since HIV diagnosis (years)	4.0 ± 1.5
Patients with viral load < 200 copies/ml	3
Median CD4+ cell count per mm^3	502 (180-600)
Antiretroviral treatment at admission	0
Irregular treatment	1
ART usage	14
Median duration of ART (months)	26 ± 12
Protease inhibitors usage	3 (duration, 12 ± 4 months)

ART – antiretroviral therapy

Table 3. Patient CD4+ count and viral load

CD4+ count (cells/mm^3)	No. of patients	Viral load (copies/ml)	No. of patients
< 100	2	< 400	2
100-199	4	400-3,999	3
200-400	5	4,000-9,999	3
> 400	4	10,000-40,000	4
		> 40,000	3

Discussion

Our study reveals that most patients with HIV infection had echocardiographic abnormalities, which were clinically quiescent. Echocardiography is a relevant tool for diagnosing sub-clinical cardiac abnormalities of tamponade, while pericardial effusion and pericarditis are the most frequently recognized cardiovascular manifestations of HIV. Various causative factors involved in the development of pericardial disease have been described, with tuberculosis seen as the most common cause of pericardial disease in Africa [7]. Other reported causative factors include human immunodeficiency virus, opportunistic infections, such as cytomegalovirus, mycobacterium, cryptococcus, and bacterial infections as well as malignancies, such as Kaposi's sarcoma and non-Hodgkin lymphoma [8]. AIDS patients with pericardial effusions have demonstrated a lower CD4+ count than AIDS patients without effusions; however, the relation of pericardial effusion to mortality has not been described [9]. In a study of Monsuez *et al.* [10] investigating patients with clinical symptoms of heart disease, 22% of them had an evidence of cardiac tamponade and 33% had a significant pericardial effusion. The CD4+ count and serum albumin as survival determinants also demonstrated that the development of pericardial effusion was independently associated with increased mortality.

Several potential explanations exist for this shortened survival after the development of pericardial effusion. First, an effusion may be a marker for undiagnosed opportunistic infections. The development of effusion may also be a function of immune status, as reflected in the CD4+ count. The findings of this study also confirm that HIV infection was associated with left ventricular dysfunction and increased ventricular dimensions, with similar trends noted in other studies [11].

Systolic dysfunction is a significant cause of morbidity and mortality in HIV-infected patients. Also, it is suggested that symptomatic heart failure will occur in approximately 6% of these patients, especially at the end stage of the disease [12]. Asymptomatic pericardial effusion may signal end-stage retroviral disease despite a relatively preserved CD4+ count level, because these effusions are frequently small and progressive. However, large symptomatic pericardial effusions do occur, which may need aggressive evaluation and therapy. Pericardial effusion in the setting of retroviral disease infection can be considered a diagnostic criterion for end-stage retroviral infection. In our study, all 15 patients were receiving adjuvant highly active antiretroviral therapy. The incidence of pericardial effusion in HIV-infected patients with more advanced disease (CD4+ cell count < 200/mm^3) was 20%, a lower rate than previously reported in the literature, where pericardial effusion was described as the most frequent finding.

Conclusions

The current study highlight the significant cardiovascular manifestations observed in HIV-infected patients through echocardiographic evaluations. Contrary to expectations, no significant Doppler's echocardiographic abnormalities were detected in our cases. Notably, individuals with a CD4+ cell count < 300/mm^3 exhibited a higher incidence of pericardial effusion, emphasizing the importance of monitoring CD4+ levels in HIV-positive patients for early detection of cardiac involvement. Our findings support the efficacy and safety of echocardiograpy guided percutaneous pericardial puncture and fluoroscopy-guided pigtail catheter placement as a suitable treatment strategy for cardiac tamponade in HIV-infected individuals. However, further studies with larger sample sizes are warranted to validate our findings and optimize management protocols for cardiac complications in this patient population.

Disclosures

1. Institutional review board statement: The study was approved by the Jayadeva Institute Ethics Committe (approval number SJICR/EC/2015/11).
2. Assistance with the article: None.
3. Financial support and sponsorship: None.
4. Conflicts of interest: None.

References

1. Spodick DH. Pathophysiology of cardiac tamponade. Chest 1998; 113: 1372-1378.
2. Collins D. Aetiology and management of acute cardiac tamponade. Crit Care Resusc 2004; 6: 54-58.
3. Beck CS. Two cardiac compression triads. JAMA 1935; 104: 714-716.
4. Fontanelle LJ, Cuello L, Dooley BN. Subxiphoid pericardial window. A simple and safe method for diagnosing and treating acute and chronic pericardial effusions. Thorac Cardiovasc Surg 1971; 62: 95-97.
5. Salem K, Mulji A, Lonn E. Echocardiographically guided pericardiocentesis – the gold standard for the management of pericardial effusions and cardiac tamponade. Can J Cardiol 1999; 15: 1251-1255.
6. Setty NS, Sadananda KS, Nanjappa MC, Patra S, Basappa H, Krishnappa S. Massive pericardial effusion and cardiac tamponade due to cholesterol pericarditis in a case of subclinical hypothyroidism – a rare event. J Am Coll Cardiol 2014; 63: 1451. DOI: https://doi.org/10.1016/j.jacc.2013.10.093.
7. Currie PF, Boon NA. Human immunodeficiency virus infection and the heart. In: McMurray JJV, Cleland JGF (eds.). Heart Failure in Clinical Practice. London: Martin Dunitz; 1996, p. 85-108.
8. Olusegun-Joseph DA, Ajuluchukwu JNA, Okany CC, Mbakwem AC, Oke DA, Okubadejo NU. Echocardiographic patterns in treatment-naïve, HIV-positive patients in Lagos, south-west Nigeria. Cardiovasc J Afr 2012; 23: e1-e6. DOI: 10.5830/CVJA-2012-048.
9. Cegielski JP, Ramiya K, Lallinger GJ, Mtulia IA, Mbaga IM. Pericardial disease and human immunodeficiency virus in Dar es Salaam, Tanzania. Lancet 1990; 335: 209-212.
10. Monsuez JJ, Kinney EL, Vittecoq D, Kitzis M, Rozenbaum M, D'agay MF. Comparison among acquired immune deficiency syndrome patients with and without clinical evidence of cardiac disease. Am J Cardiol 1988; 62: 1311-1313.
11. Longo-Mbenza B, Seghers KV, Phuati M, Bikangi FN, Mubagwa K. Heart involvement and HIV infection in African patients: determinants of survival. Int J Cardiol 1998; 64: 63-73.
12. Himelman R, Chung W, Chernoff N, Schiller NB, Hollander H. Cardiac manifestations of human immunodeficiency virus infection: a two-dimensional echocardiography study. J Am Coll Cardiol 1989; 13: 1030-1036.