

SYSTEMATIC REVIEW

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Cardiac tamponade in people living with HIV: a systematic review of case reports and case series

Amirreza Keyvanfar^{1*}, Hanieh Najafiarab², Sepehr Ramezani³ and Shabnam Tehrani¹

Abstract

Background Cardiac tamponade is a life-threatening condition requiring prompt diagnosis and therapeutic intervention. Diagnosis and management of cardiac tamponade in patients with human immunodeficiency virus (HIV) infection pose a major challenge for clinicians. This study aimed to investigate clinical characteristics, paraclinical findings, therapeutic options, patient outcomes, and etiologies of cardiac tamponade in people living with HIV.

Methods Pubmed, Embase, Scopus, and Web of Science databases were systematically searched for case reports or case series reporting HIV-infected patients with cardiac tamponade up to February 29, 2024. Baseline characteristics, clinical manifestations, paraclinical findings, therapeutic options, patient outcomes, and etiologies of cardiac tamponade were independently extracted by two reviewers.

Results A total of 37 articles reporting 40 HIV-positive patients with cardiac tamponade were included. These patients mainly experienced dyspnea, fever, chest pain, and cough. They were mostly presented with abnormal vital signs, such as tachypnea, tachycardia, fever, and hypotension. Physical examination predominantly revealed elevated Jugular venous pressure (JVP), muffled heart sounds, and pulsus paradoxus. Echocardiography mostly indicated pericardial effusion, right ventricular collapse, and right atrial collapse. Most patients underwent pericardiocentesis, while others underwent thoracotomy, pericardiotomy, and pericardiostomy. Furthermore, infections and malignancies were the most common etiologies of cardiac tamponade in HIV-positive patients, respectively. Eventually, 80.55% of the patients survived, while the rest expired.

Conclusion Infections and malignancies are the most common causes of cardiac tamponade in HIV-positive patients. If these patients demonstrate clinical manifestations of cardiac tamponade, clinicians should conduct echocardiography to diagnose it promptly. They should also undergo pericardial fluid drainage and receive additional therapy, depending on the etiology, to reduce the mortality rate.

Keywords AIDS, Cardiac Tamponade, HIV, Pericardial Effusion

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Introduction

Human immunodeficiency virus (HIV) infection is a significant global health concern, impacting millions of individuals worldwide [1]. According to the last report from the Joint United Nations Program on HIV/AIDS (UNAIDS) in 2022, 85.6 million individuals have become infected with HIV, leading to 40.4 million deaths over the past four decades [2]. The introduction of highly active antiretroviral therapy (HAART) has transformed HIV infection from a terminal illness to a chronic condition, resulting in improved life expectancy for many patients [3]. However, many HIV-positive patients experience HIV-related complications, such as opportunistic infections, metabolic disorders, malignancies, or organ dysfunctions [4, 5]. Cardiac complications have emerged as a prominent concern, contributing to increased morbidity and mortality in people living with HIV [6]. Cardiac involvement in these patients can present as ischemic heart disease, endocarditis, HIV-related cardiac malignancies, pulmonary artery hypertension, cardiomyopathy, myocarditis, pericarditis, constrictive pericarditis, and cardiac tamponade [7].

Cardiac tamponade is a life-threatening emergency characterized by dyspnea, tachycardia, hypotension, and pulsus paradoxus. It is essential to promptly diagnose and treat cardiac tamponade to prevent cardiac shock and potential mortality [8, 9]. Performing pericardiocentesis as early as possible to relieve pressure on the heart chambers and prevent hemodynamic compromise is crucial, which can improve patient outcomes and reduce the risk of complications associated with delayed treatment [10]. However, when performing therapeutic procedures for cardiac tamponade in HIV-positive patients, there is a risk of transmitting pathogens through blood or body fluids to healthcare workers [11]. This emphasizes the importance of stringent infection control and precautionary measures when managing cardiac tamponade in people living with HIV among healthcare workers. The intersection of HIV infection and cardiac tamponade presents unique challenges in clinical practice, emphasizing the need for evidence-based guidelines to ensure optimal patient care and healthcare worker safety [12].

Therefore, this study aimed to investigate clinical characteristics, paraclinical findings, therapeutic options, patient outcomes, and etiologies of cardiac tamponade in people living with HIV. This information can help clinicians promptly diagnose similar cases and make appropriate therapeutic decisions.

Methods

This study conforms to the “Preferred Reporting Items for Systematic Reviews and Meta-Analyses” (PRISMA) statement [13]. The study protocol was registered in the

International Prospective Register of Systematic Reviews as PROSPERO (CRD42024517212).

Eligibility criteria

In our systematic review, full-text English-language case reports/case series reporting HIV-infected patients with cardiac tamponade were included. Original articles, review articles, randomized controlled trials, animal studies, commentaries, editorials, guidelines, and conference papers were excluded.

Search strategy and information sources

PubMed/Medline, Embase, Scopus, and Web of Science were systematically searched for eligible articles published between January 1994 and February 29, 2024 using the following search strategy: (((HIV) OR (human immunodeficiency virus)) OR (AIDS)) OR (acquired immunodeficiency virus) AND (((cardiac tamponade) OR (pericardial tamponade)) OR (heart tamponade)). Further studies were identified by manually searching all the references in the selected publications.

Study selection

Searching databases yielded records that were merged and duplicate records were removed using EndNote X6 software (Thomson Reuters, New York, NY, USA). Initially, records were independently screened by two reviewers regarding the title and abstract (HN and SR). Subsequently, the full texts of those that passed the initial screening were independently assessed for eligibility by the same reviewers (HN and SR). Disagreements were resolved by the principal investigator (AK).

Data extraction

The following variables were independently extracted from the included publications by two reviewers (HN and SR): first author name, publication year, country where the cases lived, age, sex, underlying diseases, treatment for HIV infection, clinical characteristics (signs and symptoms), imaging findings, laboratory results, reports of cardiac paraclinical investigations (electrocardiography and echocardiography), etiologies of cardiac tamponade and how to treat them, and patients outcome. Disagreements were resolved by the principal investigator (AK).

Quality assessment

The checklist provided by the Joanna Briggs Institute (JBI) was used to perform the quality assessment of case reports and case series [14].

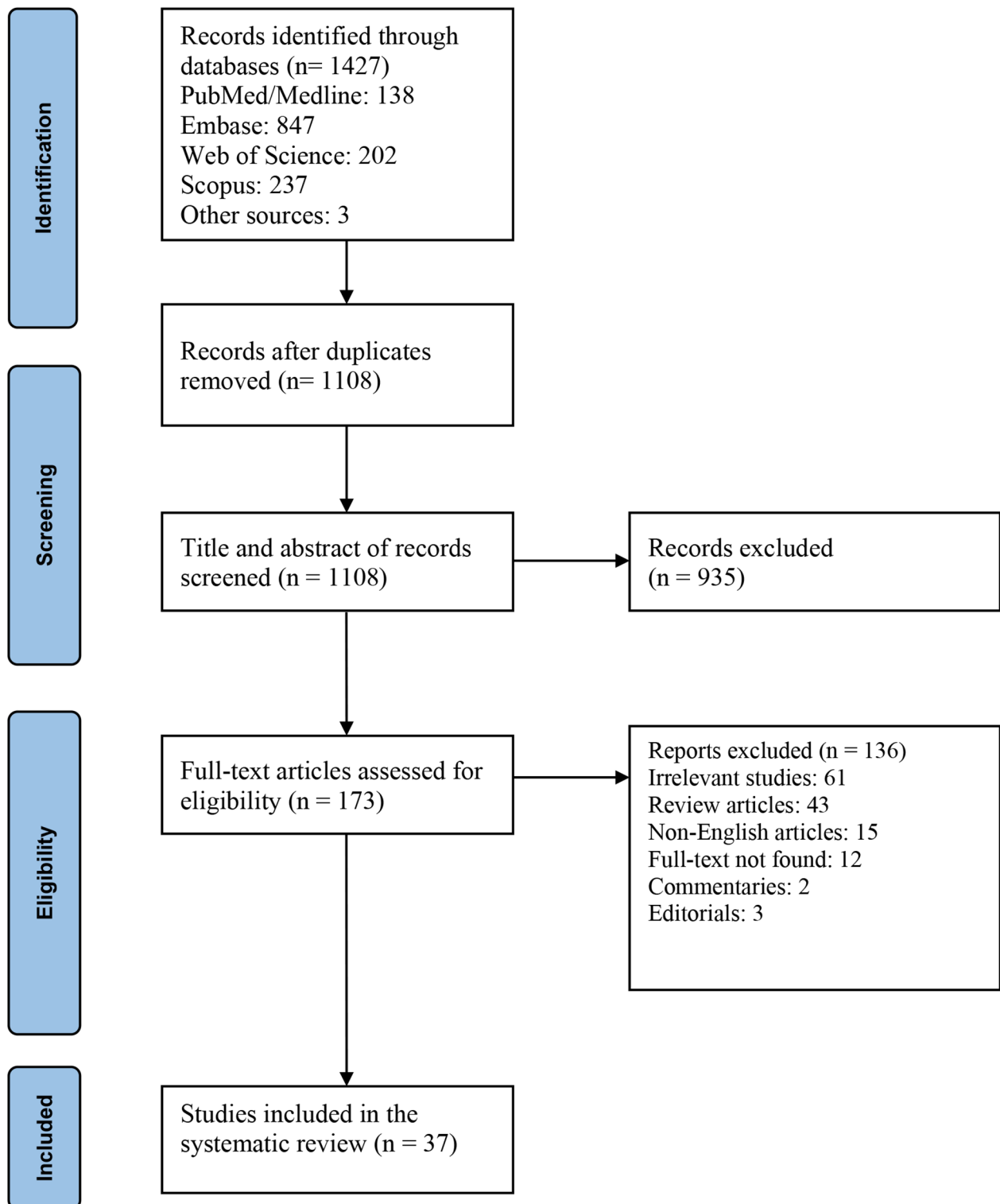


Fig. 1 Flow chart of study selection for inclusion in the systematic review

Results

Study selection

After removing duplicates from 1427 records obtained

from the database search, 1108 of them screened regarding title and abstract. Subsequently, full texts of the remaining records were assessed for eligibility, and 37

Table 1 Characteristics of the included studies

First author	Country	Year of publication	Type of study	Number of cases	Age (year)	Sex	Quality of studies
Aboulafia et al. [27]	USA	1994	Case report	1	42	Male	High
Legras et al. [28]	France	1994	Case report	1	29	Male	High
Scerpella et al. [29]	USA	1995	Case report	1	34	Male	High
Serrano-heranz et al. [30]	Spain	1995	Case series	3	31	Male	High
					27	Male	
					28	Male	
Vooren et al. [31]	Belgium	1995	Case report	1	49	Male	High
Bennis et al. [32]	Morocco	1996	Case report	1	45	Male	High
Vijay et al. [33]	USA	1996	Case report	1	32	Male	High
Currie et al. [34]	UK	1997	Case report	1	22	Male	Moderate
Azrak et al. [35]	USA	1998	Case report	1	52	Male	High
Chyu et al. [36]	USA	1998	Case report	1	32	Male	High
Rivero et al. [37]	Spain	2000	Case report	1	34	Male	High
Theodossiadis et al. [38]	Greece	2000	Case report	1	18	Male	High
Goldberg et al. [39]	South Africa	2003	Case report	1	16	Female	High
Leang et al. [40]	Belgium	2004	Case report	1	24	Female	High
Louw et al. [41]	South Africa	2007	Case report	1	33	Male	High
Russell et al. [42]	South Africa	2008	Case report	1	31	Male	High
Chandrashekar et al. [43]	India	2009	Case report	1	35	Female	High
Heller et al. [44]	South Africa	2010	Case report	1	34	Male	High
Park et al. [45]	South Africa	2010	Case report	1	29	Male	High
Kabangila et al. [46]	Tanzania	2011	Case report	1	22	Female	High
Aisenberg et al. [47]	USA	2013	Case report	1	37	Male	High
Tsao et al. [48]	Taiwan	2015	Case report	1	36	Female	High
Laksananun et al. [49]	Thailand	2017	Case report	1	32	Male	High
Lopezluis et al. [50]	Mexico	2018	Case report	1	29	Male	High
Qureshi et al. [51]	USA	2018	Case report	1	33	Male	High
Lamas et al. [52]	Brazil	2019	Case report	1	39	Male	High
Ali et al. [53]	USA	2020	Case report	1	46	Male	High
Farouji et al. [8]	USA	2021	Case report	1	35	Female	High
Khaba et al. [54]	South Africa	2021	Case series	2	15	Male	High
					30	Female*	
Sinit et al. [55]	USA	2021	Case report	1	52	Male	High
Chlilek et al. [56]	France	2021	Case report	1	47	Male	High
Jacobs et al. [57]	South Africa	2022	Case report	1	40	Male	High
Yanes et al. [58]	Philippines	2022	Case report	1	20s	Male	High
Chang et al. [59]	Malaysia	2022	Case report	1	36	Male	Moderate
Rathore et al. [60]	USA	2023	Case report	1	32	Male	High
Sufryd et al. [61]	Poland	2023	Case report	1	42	Male	Moderate
Yan et al. [62]	USA	2023	Case report	1	62	Female	High

*pregnant woman

studies were included for systematic review. Figure 1 depicts the flow chart of study selection for inclusion in the systematic review.

The detailed characteristics of the included studies are illustrated in Table 1. In this systematic review, 35 case reports and 2 case series reporting 40 HIV-positive patients with cardiac tamponade (31 men and 9 women) were examined.

Clinical characteristics of the patients

Table 2 presents the clinical characteristics of the patients. The most common underlying diseases were Kaposi's sarcoma (4/18, 22.22%), hepatitis B (3/18, 16.67%), tuberculosis (2/18, 11.11%), syphilis (2/18, 11.11%), and hypertension (2/18, 11.11%). They mainly experienced dyspnea (32/38, 84.21%), fever (19/38, 50.00%), chest pain (17/38, 44.73%), and cough (15/38, 39.47%). They were mostly presented with abnormal vital signs, such as tachypnea (20/21, 95.23%), tachycardia (25/33, 75.75%), fever (13/26, 50.00%), and hypotension

Table 2 Clinical characteristics of the patients

Variables	n/N	Percentage
Past medical history		
Kaposi's sarcoma	4/18	22.22
Hepatitis B	3/18	16.67
Tuberculosis	2/18	11.11
Syphilis	2/18	11.11
Hypertension	2/18	11.11
Hepatitis C	1/18	5.56
Hepatitis D	1/18	5.56
Hypothyroidism	1/18	5.56
Diabetes mellitus	1/18	5.56
Under retroviral therapy for HIV infection	11/13	84.61
Interval from symptom onset		
< 1 week	10/24	41.67
1–2 weeks	7/24	29.16
2–4 weeks	4/24	16.67
> 4 weeks	3/24	12.50
Symptoms		
Dyspnea	32/38	84.21
Fever	19/38	50.00
Chest pain	17/38	44.73
Cough	15/38	39.47
Lower limb edema	6/38	15.78
Night sweat	5/38	13.15
Weight loss	5/38	13.15
Malaise	4/38	10.52
Nausea and vomiting	3/38	7.89
Diarrhea	3/38	7.89
Orthopnea	3/38	7.89
Anorexia	2/38	5.26
Chills	2/38	5.26
Dizziness	2/38	5.26
Abdominal pain	2/38	5.26
Violaceous spot	2/38	5.26
Others*	5/38	13.15
Abnormal vital signs		
Tachypnea	20/21	95.23
Tachycardia	25/33	75.75
Fever	13/26	50.00
Hypotension	10/31	32.25
Hypertension	6/31	19.35
Physical examination		
Elevated jugular vein pressure (JVP)	27/30	90.00
Pulsus paradoxus	15/31	48.38
Cardiac auscultation		
Muffled heart sound	19/29	65.51
Friction rub	7/29	24.13
Chest auscultation		
Basilar crackle	8/23	34.78
Decreased pulmonary sounds	7/23	30.43
Lower limb edema	9/40	22.50
Hepatomegaly	8/40	20.00

Table 2 (continued)

Variables	n/N	Percentage
Lymphadenopathy	6/40	15.00
Ascites	3/40	7.50

n: the number of patients with each variable, N: the total number of studied patients

*others included myalgia, puffy face, arthralgia, paroxysmal nocturnal dyspnea (PND), and lymphadenopathy, which presented in different patients

(10/31, 32.25%). Physical examination predominantly revealed elevated Jugular venous pressure (JVP) (27/30, 90.00%), muffled heart sounds (19/29, 65.51%), and pulsus paradoxus (15/31, 48.38%).

Paraclinical findings of the patients

Table 3 shows the paraclinical findings of the patients. The majority of patients had a $CD4 < 200$ cell/mm³ (16/27, 59.25%) than $CD4 > 200$ cell/mm³ (11/27, 40.75%), and more patients had detectable viral loads (9/13, 69.23%) than undetectable ones (4/13, 30.77%). Leukocytosis (8/21, 38.10%) and normal white blood cell (WBC) count (8/21, 38.10%) were more common than leukopenia (5/21, 23.80%). Normal platelet count (7/11, 63.63%) was more common than thrombocytopenia (4/11, 36.37%). Furthermore, most patients exhibited elevated C-reactive protein (CRP) (9/10, 90.00%) and erythrocyte sedimentation rate (ESR) (6/7, 85.71%).

The most common electrocardiographic finding was sinus tachycardia (8/16, 50.00%), followed by low voltage QRS complex (7/16, 43.75%), ST elevation in inferior leads (3/16, 18.75%), diffuse ST elevation and PR depression (3/16, 18.75%), inverted T in inferior leads (3/16, 18.75%). Echocardiography mostly revealed pericardial effusion (38/38, 100%), right ventricular collapse (20/38, 52.63%), and right atrial collapse (12/38, 31.57%). In addition, the most evident findings on chest imaging were pleural effusion (20/39, 51.28%), enlarged cardiac silhouette sign (15/39, 38.46%), cardiomegaly (14/39, 35.89%), pulmonary consolidation (11/39, 28.20%), and pericardial effusion (11/39, 28.20%).

Therapeutic options and outcomes of the patients

Table 4 demonstrates the therapeutic options and outcomes of the patients. Most patients underwent pericardiocentesis (34/39, 87.17%), while others underwent thoracotomy (4/39, 10.25%), pericardiotomy (3/39, 7.69%), and pericardiostomy (2/39, 5.12%). Table S1 displays results of pericardial fluid analysis of the patients. Furthermore, most patients survived (29/36, 80.55%), while the rest expired (7/39, 19.45%).

Etiologies of cardiac tamponade in HIV-positive patients

Table 5 summarizes the etiologies of cardiac tamponade. According to our systematic review, infections (25/40, 62.50%), malignancies (12/40, 30.0%), and systemic diseases (2/40, 5.00%) were the most common

etiologies of cardiac tamponade in HIV-positive patients, respectively. *Mycobacterium tuberculosis* (6/25, 24.00%), *Nocardia* species (5/25, 20.00%), multidrug-resistant *Staphylococcus aureus* (3/25, 12.00%), and *Streptococcus pneumoniae* (2/25, 8.00%) were the most common infectious etiologies, respectively. Moreover, Kaposi's sarcoma (4/12, 33.34%), Burkitt lymphoma (3/12, 25.00%), and diffuse large B cell lymphoma (2/12, 16.67%) were the most common malignant etiologies, respectively. Additionally, HIV-associated immune complex kidney disease (1/40, 2.50%) and Hashimoto's thyroiditis (1/40, 2.50%) were two systemic diseases that caused cardiac tamponade in people living with HIV.

Discussion

This study was conducted to provide more information regarding cardiac tamponade in patients with HIV infection. The annual incidence of cardiac tamponade in people living with HIV is estimated to be about 1%. However, it can increase up to 9% in cases with pericardial effusion [15]. Patients with pericardial effusion have lower CD4 counts compared to those without pericardial effusion, indicating advanced HIV infection [7]. The findings of our systematic review demonstrated that cardiac tamponade predominantly occurred in men aged 20 to 40 years, possibly due to the demographic distribution of HIV infection, which was more prevalent in young males.

The diagnosis of cardiac tamponade is established by clinical suspicion and confirmation by echocardiography [10]. HIV-infected patients with cardiac tamponade may experience dyspnea, chest pain, fever, or other symptoms, depending on the disease etiology [16]. In most cases with cardiac tamponade, physical examination reveals evidence of increased systemic venous pressure, tachycardia, tachypnea, and pulsus paradoxus, which is consistent with our results. Nevertheless, systemic blood pressure may be normal, decreased, or even elevated [10]. Moreover, evidence of friction rub or muffled heart sounds may be found [16]. Upon echocardiography, sinus tachycardia, low voltage QRS complex, ST or T changes, and electrical alternans may be found [17], which is in line with our findings. Our results indicated that pericardial effusion, right ventricular collapse, and right atrial collapse were frequently observed in the echocardiography of cardiac tamponade cases. Based on a review article by Fowler, massive peripheral effusion, diastolic compression of the right ventricle or right

Table 3 Paraclinical findings of the patients

Variables	n/N	Percentage
Laboratory findings		
CD4 (cell/mm ³)		
< 200	16/27	59.25
> 200	11/27	40.75
Viral load		
Detectable	9/13	69.23
Undetectable	4/13	30.77
Leukopenia	5/21	23.80
Normal WBC count	8/21	38.10
Leukocytosis	8/21	38.10
Anemia	13/15	86.67
Normal Hb concentration	2/15	13.33
Thrombocytopenia	4/11	36.37
Normal PLT count	7/11	63.63
Elevated CRP	9/10	90.00
Elevated ESR	6/7	85.71
Electrocardiographic findings		
Sinus tachycardia	8/16	50.00
Low voltage QRS complex	7/16	43.75
ST elevation in inferior leads	3/16	18.75
Diffuse ST elevation and PR depression	3/16	18.75
Inverted T in inferior leads	3/16	18.75
Electrical alternans	2/16	12.50
RV hypertrophy	2/16	12.50
RA dilatation	2/16	12.50
LA dilatation	1/16	6.25
Echocardiographic findings		
Pericardial effusion (mostly massive)	38/38	100.0
RV collapse*	20/38	52.63
RA collapse*	12/38	31.57
Mass in RA	3/38	7.89
Pericardial mass	2/38	5.26
Others#	6/38	15.78
Chest CT scan or radiography findings		
Pleural effusion	20/39	51.28
Enlarged cardiac silhouette sign	15/39	38.46
Cardiomegaly	14/39	35.89
Pulmonary consolidation	11/39	28.20
Pericardial effusion	11/39	28.20
Globus heart	5/39	12.82
Lymphadenopathy	3/39	7.69
Centrilobular nodules	2/39	5.12
Pulmonary venous congestion	2/39	5.12
Interstitial pulmonary edema	2/39	5.12
Patchy infiltration	1/39	2.56
Small mycetoma	1/39	2.56

Table 3 (continued)

Variables	n/N	Percentage
Milliary pattern	1/39	2.56
Pulmonary cavity	1/39	2.56

n: the number of patients with each variable, N: the total number of studied patients

CRP: c-reactive protein, CT scan: computed tomography scan, ESR: erythrocyte sedimentation rate, IVC: inferior vena cava, Hb: hemoglobin, LA: left atrium, LV: left ventricle, PLT: platelet, RA: right atrium, RV: right ventricle, WBC: white blood cell

*Most right ventricular and atrial collapses were diastolic

#others included septal shift, decreased ejection fraction, diastolic compression of the LV, enlarged aortic root, congestion of IVC & hepatic vessels, and pulmonary hypertension, which presented in different patients

atrium, abnormal respiratory alterations in ventricular dimensions, flow velocities of the atrioventricular valves, and the inferior vena cava (IVC) plethora, are indicators of cardiac tamponade in echocardiography [18].

Laboratory results and imaging findings play a crucial role in diagnosing the etiology of cardiac tamponade in people living with HIV. For instance, elevated inflammatory markers (e.g., leukocytosis, and elevated ESR & CRP) may indicate the infectious etiology of cardiac tamponade [19]. Additionally, certain imaging findings such as mediastinal and hilar lymphadenopathy, parenchymal consolidations, cavitory lesions, pleural effusion, miliary pattern, and centrilobular nodules point towards pulmonary tuberculosis as the potential etiology. It is important to note that in patients with pulmonary tuberculosis, cardiac tamponade may result from mycobacterial invasion into the cardiac tissue [20].

According to our findings, the most common etiologies of cardiac tamponade were infectious (*Mycobacterium tuberculosis*, *Nocardia* species, *Staphylococcus aureus*) and malignant (Kaposi's sarcoma, Burkitt lymphoma, and diffuse large B cell lymphoma) conditions, which align with existing literature [17, 21]. According to a systematic review by Gowda et al., the majority of cardiac tamponades in HIV-positive patients were attributed to *Mycobacterium* species (42.1%). Malignancies were the second leading cause of HIV-related cardiac tamponade (16.1%), including lymphoma, Kaposi's sarcoma, and adenocarcinoma. Bacterial causes accounted for 10.6% of cases, with *Staphylococcus aureus* being the most common agent, followed by *Streptococcus* species, *Klebsiella pneumoniae*, *Rhodococcus equi*, *Pseudomonas aeruginosa*, and *Listeria monocytogenes*. Furthermore, fungal (*Cryptococcus neoformans*, *Nocardia asteroides*, and *Aspergillus* species) and viral (Cytomegalovirus and Herpes simplex virus) etiologies were also identified [17].

Overall, the evaluation of pericardial effusion/cardiac tamponade in people living with HIV involves the following steps. Firstly, clinicians should suspect pericardial effusion/cardiac tamponade based on symptoms and signs. Secondly, they should carry out comprehensive

Table 4 Therapeutic options and outcomes of the patients

Variables	n/N	Percentage
Therapeutic options		
Pericardiocentesis	34/39	87.17
Thoracotomy	4/39	10.25
Pericardiectomy	3/39	7.69
Pericardiostomy	2/39	5.12
Antibiotic therapy	12/39	30.76
Antituberculosis therapy	9/39	23.07
Glucocorticoids	6/39	15.38
Chemotherapy	5/39	12.82
Outcomes		
Survived	29/36	80.55
Expired	7/36	19.45

n: the number of patients with each variable, N: the total number of studied patients

Table 5 Etiologies of cardiac tamponade

Variables	n/N	Percentage
Infectious etiologies		
<i>Mycobacterium tuberculosis</i>	6/25	24.00
<i>Nocardia spp.</i>	5/25	20.00
Multidrug resistant <i>Staphylococcus aureus</i>	3/25	12.00
<i>Streptococcus pneumoniae</i>	2/25	8.00
<i>Salmonella typhi</i>	1/25	4.00
<i>Rhodococcus equi</i>	1/25	4.00
<i>Arcobacter spp.</i>	1/25	4.00
<i>Klebsiella pneumoniae</i>	1/25	4.00
<i>Haemophilus influenzae</i>	1/25	4.00
<i>Neisseria gonorrhoea</i>	1/25	4.00
Gram-negative bacteria	1/25	4.00
Mixed Herpes simplex virus & cytomegalovirus infection	1/25	4.00
HIV-associated hemorrhagic pericarditis	1/25	4.00
Malignant etiologies		
Kaposi's sarcoma	4/12	33.34
Burkitt lymphoma	3/12	25.00
Diffuse large B cell lymphoma	2/12	16.67
Large cell immunoblastic lymphoma	1/12	8.33
Primary effusion lymphoma	1/12	8.33
B cell malignancy	1/12	8.33
Cardiac tamponade secondary to systemic diseases		
Membranoproliferative glomerulonephritis secondary to HIV-associated ICK	1/2	50.00
Hashimoto's thyroiditis	1/2	50.00
Miscellaneous		
Non-specific inflammation	1/1	100.0

HIV-associated ICK: HIV-associated immune complex kidney disease, n: the number of patients with each variable, N: the total number of studied patients

Etiologies of cardiac tamponade were evaluated based on pericardial fluid analysis, culture, cytology, and pathology results of tissue biopsies

diagnostic measures to evaluate the extent of pericardial effusion and subsequent hemodynamic compromise. Thirdly, they should make therapeutic decisions based on the benefits and risks of pericardiocentesis and closely monitor the patient's response [10, 22, 23].

Therefore, unexplained dyspnea, Beck's triad (hypotension, elevated JVP, and muffled heart sounds), or cardiomegaly on chest imaging in people living with HIV should rapidly be evaluated using echocardiography. If cardiac tamponade is confirmed, it is necessary to perform pericardiocentesis to drain effusions and stabilize vital signs of the patient [24]. In case of massive effusion, reaccumulation of pericardial fluid, or the need for tissue biopsy, more aggressive interventions such as pericardiostomy or thoracotomy may be required [21]. It is important to be alert that during therapeutic procedures for cardiac tamponade in HIV-positive individuals, there is a risk of transmitting the virus from the patient to healthcare workers. Thus, clinicians should take appropriate precautions to prevent the transmission of HIV when managing cardiac tamponade [11]. Our systematic review revealed that the majority of HIV-positive patients with cardiac tamponade underwent pericardiocentesis as the primary therapeutic option. However, a few patients underwent secondary procedures for diagnostic or therapeutic purposes. For instance, some patients had a thoracotomy to obtain biopsies. Pericardiostomy and pericardiectomy were performed in cases where the primary pericardiocentesis did not have the desired effect.

A study by Kwan et al. reported that 54% of HIV-positive patients with cardiac tamponade expired on discharge [25]. However, our systematic review indicated that 80.55% of cases survived after receiving appropriate therapy. This discrepancy in survival rate may be influenced by changes in healthcare facilities over time. In recent years, improved access to medical facilities and advancements in medical care have led to earlier diagnosis and timely treatment of cardiac tamponade, resulting in a lower mortality rate. There is ongoing debate regarding whether the type of therapeutic procedure is linked to the mortality rate. A study by Flum et al. reported that pericardiostomy performed under general anesthesia was associated with high mortality rates [26], while another study reported that pericardiostomy under local anesthesia was safer. Additionally, the mortality rate may be influenced by the progression of HIV infection and its complications rather than solely by the therapeutic procedures used to treat cardiac tamponade [21].

Our findings should be considered in light of some limitations. Although reviewing multiple databases with appropriate queries, some relevant articles might be unintentionally missed. We included publications written in English, which could lead to a language bias. Additionally, the included records reported patient outcomes

as either survived or expired, which means that no prolonged follow-ups were available to assess the recurrence rate of cardiac tamponade. Given the rarity of cardiac tamponade in people living with HIV, we had to include only case reports/case series in the systematic review. Therefore, caution should be exercised when generalizing the findings from these limited cases.

Conclusion

Infections and malignancies are the most common causes of cardiac tamponade in HIV-positive patients. If these patients demonstrate clinical manifestations of cardiac tamponade, clinicians should conduct echocardiography to diagnose it promptly. They should also undergo pericardial fluid drainage and receive additional therapy, depending on the etiology, to reduce the mortality rate.

Supplementary Information

The online version contains supplementary material available at <https://doi.org/10.1186/s12879-024-09773-4>.

Supplementary Material 1

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Author contributions

AK conceptualized the study, analyzed the data, interpreted the data, and critically edited the manuscript. HN collected the data, extracted the data, and wrote the primary draft of the manuscript. SR collected the data, extracted the data, and wrote the primary draft of the manuscript. ST supervised the study, interpreted the data, and critically edited the manuscript. All authors read and approved the final manuscript.

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Data availability

The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Declarations

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

Ethical approval and consent to participate

Not applicable.

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