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Review

Clinical Management of Cardiac Hydatid Cysts: A Case Report and Narrative Review of the Literature

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Abstract

Cardiac echinococcosis is a rare but potentially life-threatening manifestation of cystic echinococcosis, representing only 0.5–2% of all hydatid disease cases. Owing to the rarity and nonspecific clinical presentation of cardiac echinococcosis, diagnosis is often delayed, increasing the risk of serious complications such as rupture, embolization, arrhythmias, and anaphylaxis. We report the case of a 46-year-old woman with a 10-year history of an incidentally discovered cardiac cyst who presented with worsening dyspnea and atypical angina. Multimodality imaging, including echocardiography and cardiac magnetic resonance imaging (MRI), revealed a 53 × 52 mm multilocular hydatid cyst at the cardiac apex. The patient underwent successful surgical excision of the cyst under cardiopulmonary bypass, followed by tricuspid valve annuloplasty. The postoperative course was uneventful, and antiparasitic therapy with albendazole was administered. Cardiac hydatid cysts most commonly localize in the left ventricle, likely due to its rich coronary blood supply. Diagnosis relies on multimodal imaging, supported by serological testing. Early surgical intervention combined with antiparasitic therapy remains the cornerstone of management. A comprehensive review of the literature highlights the heterogeneous clinical presentations, diagnostic approaches, and therapeutic strategies for this rare condition. Early recognition and timely intervention in cardiac hydatid disease are critical to prevent life-threatening complications. This case underscores the need for a high index of suspicion in patients from endemic regions and demonstrates the effectiveness of combined surgical and medical management.

Keywords: echinococcosis; hydatid cyst; echinococcus granulosus; heart diseases; infectious diseases

1. Introduction

Cystic echinococcosis (CE) is a parasitic disease resulting from infection by the larval stage of *Echinococcus granulosus*. Cystic hydatidosis is a cyclozoonosis requiring two mammalian hosts to complete its life [1,2,3,4]. Transmission occurs through ingestion of *Echinococcus* eggs, typically via contaminated food, water, or direct contact with infected animals.

The liver and lungs are the most affected organs, serving as primary sites for the development of hydatid cysts (HCs). However, HCs can rarely localize to other organs, including the heart and pericardium [2,3]. HCs affect the liver in 70% of cases and lungs in 20%, with less frequent involvement of muscles, bones, kidneys, spleen and brain [5,6,7,8]. Cardiac echinococcosis is an uncommon manifestation, with an estimated prevalence of 0.5–2% of all HC cases [9,10,11]. Cardiac involvement is primarily influenced by the coronary circulation, which acts as the main route for parasitic larvae to reach the myocardium and may rarely mimic acute coronary syndromes [12].

Based on the rareness of isolated cardiac HCs, specific diagnostic and treatment features of these cases, particularly compared to involvement of the hepatic or pulmonary systems, still need to be addressed. For this reason, we herein present a case of an isolated cardiac HC, notable for the absence of involvement in the hepatic or pulmonary systems and an unusually prolonged 10-year clinically silent course before surgical treatment, followed by a review summarizing the pathophysiology, diagnosis, and treatment of isolated cardiac HC.

2. Case Report

A 46-year-old female patient presented with progressively worsening shortness of breath over the past year. Electrocardiography (ECG) revealed T-wave inversion in leads II, III, and aVF. Notably, ten years earlier, a cardiac cyst had been incidentally detected during routine examination.

Echocardiography demonstrated a cystic lesion at the cardiac apex, accompanied by minimal dilation of the right atrium and right ventricle, mild mitral regurgitation, and severe tricuspid regurgitation. For preoperative evaluation of coronary status and to exclude coronary artery disease as a cause of the patient's angina pectoris, the patient was admitted for coronary angiography. Coronary angiography revealed that the left main coronary artery (LMCA) was normal, the left anterior descending artery (LAD) had 30% stenosis in the ostium, and the right coronary artery (RCA)

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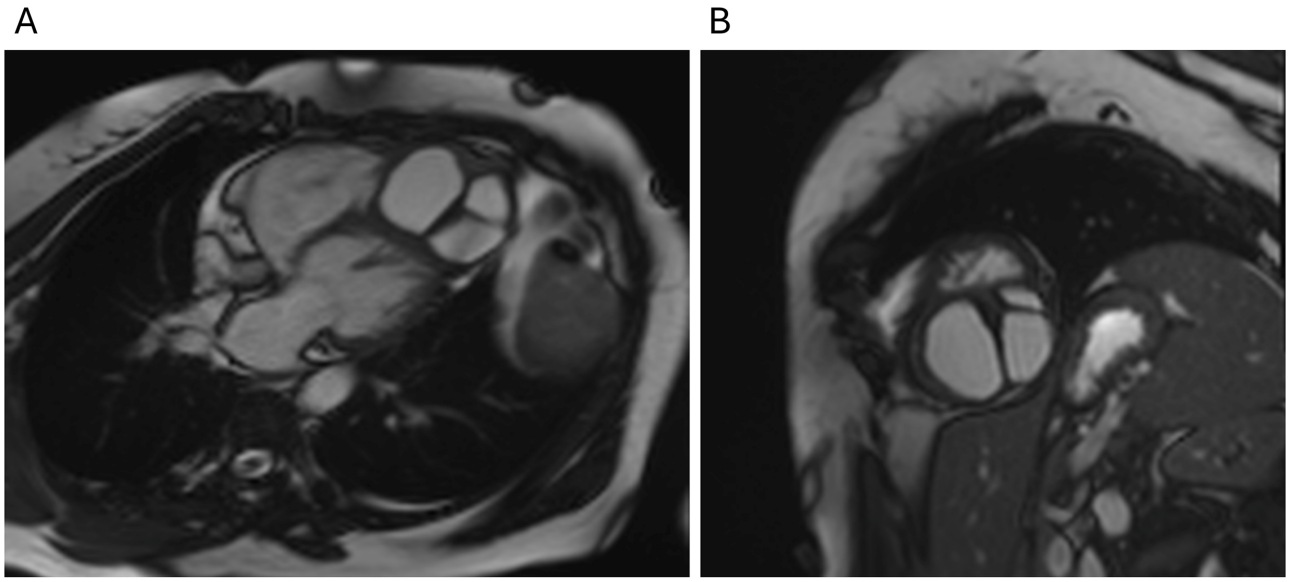


Fig. 1. Cardiac magnetic resonance imaging of a cardiac cyst. (A) Four-chamber view showing a well-defined, multilocular cystic lesion located at the apical region of the heart. The cyst appears to exert a compressive effect on adjacent cardiac chambers, particularly the left ventricle. (B) Short-axis view further illustrating the spatial relationship of the cystic structure with the surrounding myocardial tissue.

was dominant with the presence of some non-obstructive plaque. Cardiac magnetic resonance imaging (MRI) identified a 53 × 52 mm cystic lesion at the apex, compressing both the left and right ventricles and extending from the interventricular septum. The cyst exhibited thick septations, a multilocular structure, smooth borders, and minimal contrast enhancement, and heterogeneous internal components visible on additional imaging planes. These findings were consistent with a WHO Informal Working Group on Echinococcosis (WHO-IWGE) CE3b cyst due to the multilocular appearance with daughter vesicles and associated heterogeneous components (Fig. 1).

Preoperative antiparasitic therapy with albendazole 400 mg twice daily was initiated seven days before surgery. The patient underwent surgical excision of the cyst under general anesthesia. After preparation and draping, a median sternotomy was performed to access the mediastinum, and the pericardium was suspended. Arterial cannulation was established via the ascending aorta, with venous cannulation achieved using bicaval access. A root cannula was placed for cardioplegia, and diastolic arrest was induced with Del Nido cardioplegia solution. Cardiopulmonary bypass was then initiated. Intraoperatively, a 53 × 52 mm cyst was observed near the apex of the heart (Fig. 2A). The cyst was punctured, drained, and injected with 20% saline solution. After a 15-minute wait to ensure sterilization, the cyst was carefully excised (Fig. 2B,C). Histopathological examination confirmed the diagnosis of cystic echinococcosis by demonstrating the characteristic laminated acellular hydatid membrane with surrounding fibrous inflammatory reaction. Following sterilization, the inferior vena cava (IVC) and superior vena cava (SVC) were clamped, and total bypass was initiated. Right atriotomy was performed, and a De Vega annuloplasty was carried out on the tricuspid valve using Prolene sutures. Intraoperative testing confirmed the absence of residual regurgitation.

Postoperatively, the patient was administered Cefazolin, Metoprolol, and Aspirin. Also, Albendazole 400 mg twice daily was continued postoperatively for 6 months. The recovery was uneventful, and the patient was discharged seven days after surgery, including a two-day stay in the intensive care unit. Follow-up consisted of a surgical check-up at six weeks and a cardiological evaluation with ECG and echocardiography after three months. Annual follow-up with echocardiography was subsequently recommended. All follow-up evaluations confirmed successful cyst excision and restoration of tricuspid valve function.

The particular relevance of this case lies not only in the rare cardiac localization but also in the unusually prolonged clinical course, as the intracardiac cystic lesion had been known for approximately ten years before definitive surgical treatment. This highlights that cardiac cystic echinococcosis may remain clinically silent for extended periods due to slow cyst growth, potentially delaying diagnosis and intervention.

3. Echinococcosis: A Complex Pathogenesis

Echinococcosis is a parasitic zoonosis prevalent in endemic regions worldwide, caused by the larval stages of *Echinococcus granulosus* or *E. multilocularis*, with CE and alveolar echinococcosis (AE) representing the two most clinically relevant forms. CE is caused by *Echinococcus*



Fig. 2. Surgical excision and macroscopic features of a cardiac hydatid cyst. (A) Intraoperative visualization of the hydatid cyst at the cardiac apex, demonstrating its intact capsule. (B) Post-excision view of the aspirated apex cavity, showcasing the precise surgical removal of the lesion. (C) Macroscopic appearance of the excised *Echinococcus* cyst, highlighting its multilocular architecture and well-defined morphology.

granulosus and is characterized by the formation of fluid-filled HCs, most commonly affecting the liver and lungs. In contrast, AE is caused by *Echinococcus multilocularis* and exhibits a distinct infiltrative growth pattern resembling a malignant process, with different clinical behavior and therapeutic management [13].

The present review focuses exclusively on CE caused by *E. granulosus*, particularly its rare cardiac manifestation, with the following sections referring specifically to the lifecycle, pathogenesis, diagnosis, and treatment principles of CE.

The disease, commonly referred to as cystic hydatidosis, requires two mammalian hosts to complete its lifecycle [13]. The definitive host for *Echinococcus* is typically a canid, most commonly the domestic dog, which harbors the adult parasite in the small intestine and sheds tapeworm eggs in feces. These eggs contaminate the environment, facilitating infection of intermediate hosts, predominantly ungulates such as sheep, through ingestion of contaminated vegetation [14].

Once ingested by an intermediate host, the parasite's eggs release oncospheres that penetrate the intestinal mucosa and migrate via the bloodstream to various organs. There, they develop into HCs, fluid-filled structures containing protoscolices and daughter cysts. The cycle perpetuates when infected viscera are consumed by canids, a practice common in non-urban areas where sheep organs are fed to dogs, enhancing transmission [15] (Fig. 3).

Humans, as incidental intermediate hosts, acquire infection via the fecal-oral route. This may occur through direct contact with infected dogs or indirectly via ingestion of contaminated food or water. Upon ingestion, the parasite's embryo hatches in the intestine, penetrates the mucosa, and travels through the portal circulation to the liver or other organs. If it evades the host immune system, the larva matures into a HC, characterized by a vesicular structure surrounded by periparasitic host tissue, or pericyst [16]. The lifecycle culminates when a definitive host consumes protoscolex-containing cysts in the intermediate host's viscera, allowing the parasite to mature into its adult form in the small intestine.

Cardiac involvement accounts for 0.5–2% of all hydatid cases [17]. Cardiac cysts grow slowly, at an estimated rate of approximately 1 cm per year, and typically take 1–5 years to fully develop after the embryo has lodged in the myocardium [12,17,18].

The life span of cardiac echinococcosis in humans ranges from 10 to 20 years [19]. While cyst growth and progression vary, their presence in elastic-resistant tissues like the myocardium can lead to slower expansion and distinct clinical challenges compared to other organ sites.

Myocardial cysts may rupture into the pericardial space or cardiac chambers, leading to serious complications, or may degenerate and calcify over time.

Understanding the intricate lifecycle of *Echinococcus* and its pathophysiology is crucial for addressing this significant yet underrecognized zoonotic disease.

4. Distribution and Invasion Pathways of Cardiac Hydatid Cysts

The primary route of myocardial invasion is hematogenous spread via the coronary arteries. A secondary pathway involves pulmonary venous drainage after rupture of a lung cyst, and direct extension from adjacent liver or lung lesions can also seed the heart. Cardiac hydatid cysts predominantly localize in the left ventricle (55–60%), followed by the right ventricle (10–15%), pericardium (7%), pulmonary artery (6–7%), left atrium (5%), right atrium (3–4%), and interventricular septum (4%) [20].

The left ventricle's rich blood supply and greater myocardial mass favor subepicardial cyst growth, with pericardial rupture being rare. In contrast, right ventricular cysts are typically subendocardial and more prone to rup-

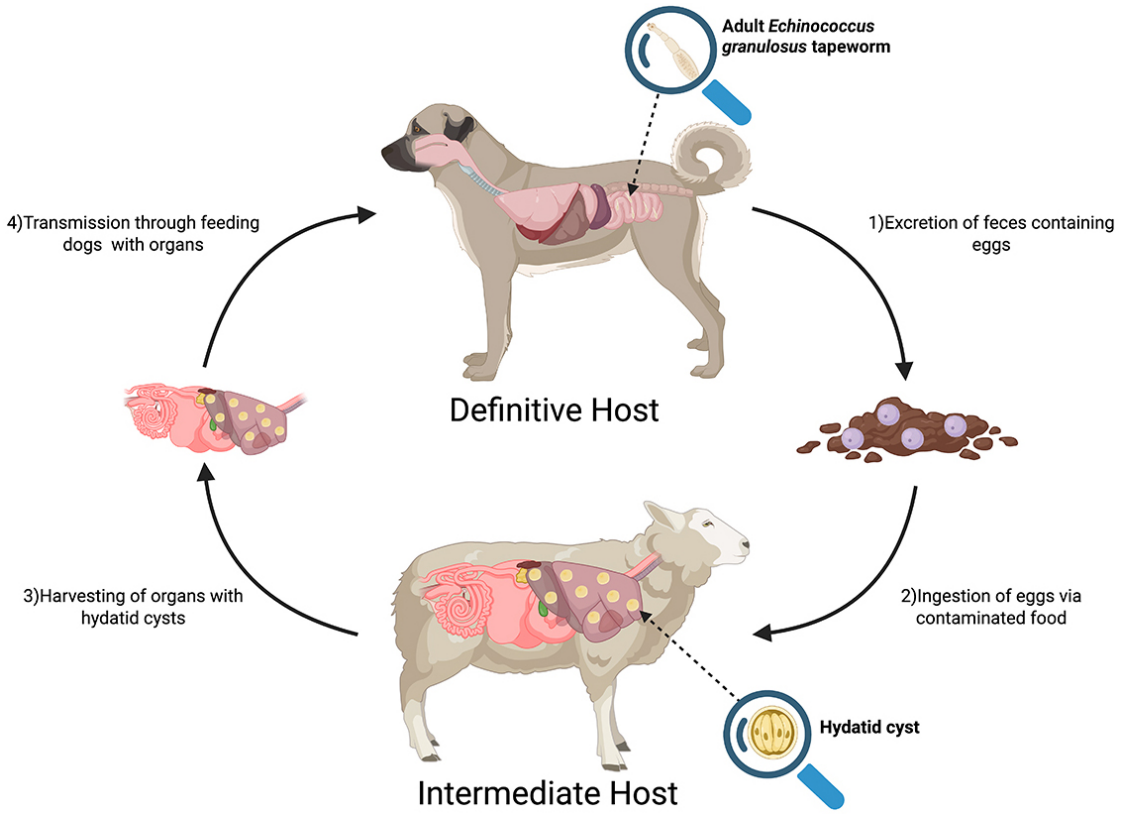


Fig. 3. Life cycle of *Echinococcus granulosus*. The definitive host of *Echinococcus granulosus* is usually a canid—most commonly the domestic dog—which carries the adult tapeworm in its small intestine. (1) The parasite's eggs are excreted with the host's feces, contaminating the environment. (2) Intermediate hosts, primarily ungulates like sheep, become infected by ingesting vegetation contaminated with these eggs. After ingestion, the eggs release oncospheres that penetrate the intestinal lining and spread via the bloodstream to various organs. There, they develop into hydatid cysts - fluid-filled sacs that contain protoscolices and sometimes daughter cysts. (3) Organs with hydatid cysts are harvested. (4) The life cycle continues when canids consume the infected organs of these intermediate hosts. Created in BioRender.

ture, which can lead to pulmonary embolism, anaphylactic reactions, or sudden death [21].

5. Epidemiology of Cardiac Echinococcosis

Hydatid disease, caused by *Echinococcus granulosus*, is endemic in many sheep-farming regions globally, with notable prevalence in Mediterranean countries, the Middle East, South America, and Australia [22]. In a systematic review by Bumann et al. [23], 1210 cases of cardiac echinococcosis reported in 747 publications between 1965 to 2022 were analyzed; 399 of these publications (53.4%) originated from Türkiye and 116 (15.5%) from Tunisia. Though, these reports do not necessarily indicate true regional differences in cardiac localization of echinococcosis infection. Current literature demonstrates that publication patterns do not directly reflect the global distribution of cystic echinococcosis and suggests that differences in reporting activity and medical publishing traditions may substantially influence the apparent geographical distribution of cases [23]. Several other factors may also contribute to potential regional variations. One biological hypothesis involves parasite genotype, as different members of the *E. granulosus* sensu lato complex show distinct geographical distributions, host associations, and transmission patterns. For example, *E. granulosus* sensu stricto (*G1–G3*) represents the dominant cause of human CE in many regions [24]. However, whether these genetic differences translate into organ-specific tropism remains unclear, as available literature about cardiac involvement generally lacks systematic molecular characterization. Non-biological explanations for the observed geographical distribution are currently better supported. Regions with higher levels of pastoral transmission, livestock exposure, close human–dog contact, and limited hygiene infrastructure naturally have a higher overall burden of CE and therefore may report more cardiac cases in absolute numbers without a true increase in the proportion of cardiac involvement. Of note, a recent analysis found isolated cardiac cysts in over half of cases, with the remainder showing extra-cardiac cysts mainly in liver, but also kidney, and lung [25]. Furthermore, differences in healthcare infrastructure, availability of echocardiography, computed tomography, and magnetic

resonance imaging, as well as access to specialized cardiac surgery centers, may influence detection rates. Therefore, the currently observed geographical patterns likely reflect a combination of endemic prevalence, diagnostic capacity, reporting bias, and potentially parasite-related factors rather than proven genotype-specific cardiac tropism.

In the systematic review of Bumann et al. [23], the median age of patients diagnosed with cardiac echinococcosis was 30 years, with no significant difference in age between male and female patients. Of the 1132 cases with available data on sex, 615 (54.3%) were male and 517 (45.7%) female, indicating a slight male predominance. Cardiac HCs are predominantly diagnosed in adulthood; however, regional variations in age and sex distribution may also arise due to differing exposure patterns, healthcare access, and local practices.

The review by Bajdechi et al. [26], based on 47 reported cases, reported a slightly higher prevalence of cardiac echinococcosis in males, with over 70% of cases occurring in men. Additionally, it indicated a median age of 36 years, while highlighting that the condition can affect a broad age range [26].

Although these statistics provide valuable insights into the epidemiology of hydatid disease, the patterns may not be universally applicable due to regional variations in human and animal interactions, agricultural practices, and socioeconomic factors. Understanding these nuances is essential for tailoring public health interventions to local needs.

6. Clinical Manifestations of Cardiac Hydatid Cysts

The signs and symptoms of cardiac HCs are highly variable, depending on the cyst's size and location [27]. HCs typically grow indolently and remain clinically silent in about 90% of cases, often taking five to ten years to enlarge sufficiently to produce cardiovascular symptoms; this results in delayed diagnosis, with overt signs developing only in the minority of patients who have larger lesions [28].

While 90% of cases are asymptomatic, cardiac HCs can exert a mass effect, altering the position of coronary vessels, causing arrhythmias, and mechanically interfering with atrioventricular valves and ventricular function [20,28,29,30,31].

6.1 Symptoms

Precordial pain is the most common symptom, though it is often vague and nonspecific, and may not resemble angina pectoris, often nonspecific or atypical [32], followed by dyspnea, especially during exertion [33], and palpitations [34].

6.1.1 Valvular

When HCs develop near or within cardiac valves, they can lead to valvular dysfunction. Reported cases include tricuspid regurgitation, mitral regurgitation and pulmonary valve dysfunction [31,35,36]. These manifestations may present with symptoms mimicking valvular heart disease, such as dyspnea, fatigue, and signs of right or left heart failure.

6.1.2 Arrhythmogenic

Other possible symptoms include cough and arrhythmias [18,37]. Cyst involvement in the interventricular septum can lead to right or left ventricular outflow tract obstruction, as well as a variety of conduction defects such as Atrioventricular block [28,38,39].

6.1.3 Pericardial and Anginal

In cases of pericardial involvement, patients may present with symptoms resembling acute pericarditis, particularly when a pericardial inflammatory reaction is present; such symptoms are consistent with the general clinical presentation of pericardial inflammation [40].

Compression of coronary arteries and myocardium by cystic masses can lead to ischemia, manifesting as anginal pain that mimics coronary artery disease [41].

6.2 Diagnostic Challenges and the Importance of Early Management

Cardiac HCs often follow silent progression. Larger cysts remain asymptomatic until their space-occupying effects cause symptoms in the organ involved. Given the potentially fatal consequences of cyst rupture, including embolism, anaphylaxis, and tamponade, early diagnosis and prompt management are essential.

Recognizing the varied and insidious clinical presentations of cardiac echinococcosis is critical for initiating timely and effective treatment.

7. Diagnostic Approach to Cardiac Hydatid Disease

The diagnosis of cardiac echinococcosis relies on a comprehensive evaluation that combines clinical findings, imaging techniques, and serological testing. Due to the rarity of cardiac CE, no universally accepted minimum diagnostic criteria have been established. In clinical practice, the threshold to raise suspicion should include the presence of a compatible cardiac cystic lesion on imaging as cornerstone of its diagnosis, preferably detected by echocardiography, together with at least one supportive contextual factor such as residence in or exposure to an endemic region, known extracardiac cystic echinococcosis, relevant animal exposure, or supportive serological findings.

Importantly, clinicians should not wait for highly specific symptoms, positive biomarkers, or histological confirmation before considering the diagnosis. Cardiac CE frequently presents with nonspecific symptoms or may remain asymptomatic for years, while serological tests can yield false-negative results. Therefore, early recognition primar-

ily relies on maintaining clinical suspicion when characteristic imaging findings occur in an appropriate epidemiological or clinical context.

7.1 Imaging as the Cornerstone of Diagnosis

Imaging modalities are paramount in diagnosing CE, with ultrasonography (US), computed tomography (CT), and magnetic resonance imaging (MRI) playing central roles. Echocardiography, both transthoracic echocardiography (TTE) and transesophageal echocardiography (TEE), is the primary diagnostic tool due to its widespread availability, affordability, and ability to visualize cardiac cysts effectively [42,43]. CT and MRI provide detailed information on the cysts' spatial relationships with surrounding structures, aiding in surgical planning and differential diagnosis. The WHO-IWGE classification system [44] categorizes cystic echinococcosis into stages CE1–CE5 based on imaging morphology and cyst activity. CE1 and CE2 represent active cyst stages, with CE1 characterized by a simple unilocular cyst and CE2 by multiple daughter cysts. CE3 lesions represent transitional stages, including CE3a cysts with detached membranes and CE3b cysts with predominantly solid components containing daughter cysts. CE4 and CE5 represent inactive degenerative stages with heterogeneous contents or calcified walls. Although mainly validated for hepatic echinococcosis, this classification provides a useful framework for describing imaging characteristics of cardiac hydatid cysts.

7.1.1 Echocardiography Strengths and Limitations

Echocardiography remains the first-line investigation for CE due to its ability to detect echo-negative components and regular contours of HCs. TEE enhances diagnostic accuracy, particularly in areas obscured by the sternum. However, the technique is operator-dependent and limited in its ability to evaluate extracardiac structures [21].

7.1.2 Role of CT and MRI

CT excels in detecting calcifications and delineating cyst morphology but may miss small lesions. MRI provides superior soft tissue contrast, enabling the identification of multiloculated cysts, daughter cysts, and detached membranes, which are highly specific for hydatid disease [10,45].

7.2 ECG Findings

ECG abnormalities are common but nonspecific in CE. Approximately 75% of cases exhibit abnormal ECG findings, underscoring its utility as a diagnostic adjunct [23]. The nature of changes depends on the cyst's location.

Electrocardiograms frequently reveal T-wave abnormalities, reduced QRS voltage, and the presence of pathological Q waves. Arrhythmias may occasionally occur. T-wave alterations and voltage reductions are useful indicators of left ventricular involvement, while abnormalities in atrioventricular conduction or the presence of a right bundle branch block suggest ventricular septal involvement [46]. When the atria are affected, abnormalities in the P-wave may also be present [47].

7.3 Laboratory Investigations and Molecular Diagnostics

Routine laboratory tests often lack specificity in CE. Serological tests for echinococcosis can be helpful but are not always determinative. Blood eosinophilia, though classically associated with parasitic infections, is neither sensitive nor specific and may be absent even in cases of cyst rupture. Serological tests, including enzyme-linked immunosorbent assay (ELISA) and indirect hemagglutination assay (IHA), offer adjunctive diagnostic support. ELISA demonstrates high sensitivity (94%) and specificity (99%) and is particularly useful in follow-up assessments [48]. However, negative serology does not exclude CE, and positive results must be interpreted in conjunction with imaging findings. While hepatic CE generally shows higher serological sensitivity due to greater antigen exposure, recent data on extrahepatic CE further demonstrate that antibody detection may show substantially reduced sensitivity in certain organ manifestations, particularly in intact cysts with limited antigen exposure [49]. Therefore, negative serological results do not exclude cardiac echinococcosis, especially when characteristic imaging findings and epidemiological risk factors are present.

Molecular diagnostic methods, including PCR-based assays and sequencing, enable species identification and genotype characterization within the *Echinococcus granulosus* sensu lato complex. These techniques are usually applied to cyst fluid or surgically obtained tissue samples and can differentiate clinically relevant species such as *E. granulosus* sensu stricto and *E. canadensis* [50,51]. In rare presentations such as isolated cardiac CE, molecular characterization may provide additional information regarding parasite diversity and potential genotype-associated differences in organ involvement. However, available cardiac cases rarely include molecular data, and no specific genotype has currently been linked to cardiac tropism. Therefore, PCR and genotyping mainly represent confirmatory and epidemiological tools rather than essential components of initial diagnosis.

7.4 Differential Diagnosis

Cardiac echinococcosis cysts must be distinguished from other intracardiac masses, including cardiac tumors such as myxomas, congenital pericardial cysts, aneurysms, and intracardiac thrombi [52]. The cystic nature of CE can mimic cardiac myxomas on echocardiography, leading to diagnostic confusion [53].

7.5 Integrated Diagnostic Workflow

(1) Initial Assessment: Detailed clinical history and symptom evaluation, noting any geographic or occupational risk factors.

(2) Laboratory Testing: Perform blood count (eosinophilia) and serology (ELISA, IHA) for supportive evidence.

(3) Imaging: Begin with echocardiography, followed by CT or MRI for detailed anatomical assessment and surgical planning.

(4) Advanced Techniques: Intraoperative TEE and coronary angiography may guide surgical interventions.

(5) Systemic Screening: Investigate for concomitant hepatic and pulmonary cysts using appropriate imaging modalities.

By integrating these diagnostic modalities, clinicians can achieve accurate diagnosis and effective management of cardiac hydatid disease, mitigating the risk of life-threatening complications.

8. Complications of Cardiac Hydatid Cysts

The rupture of intracardiac HCs represents one of the most severe complications of cardiac echinococcosis, with potentially catastrophic consequences. Sudden rupture can precipitate acute pericarditis, cardiac tamponade, pulmonary or systemic embolism, and anaphylactic shock, all of which pose significant mortality risks [54,55].

The clinical Spectrum of Complications involves:

• Systemic and Pulmonary Embolization

HC rupture into the ventricular or atrial cavities can result in systemic or pulmonary embolization. This can lead to severe complications such as stroke, organ infarction, or pulmonary hypertension due to embolic obstruction [56,57].

• Tamponade and Pericarditis

Pericardial involvement or a rupture of a HC into the pericardial space occurs in 2% to 10% of cardiac hydatid cases and may lead to pericardial effusion, cardiac tamponade, pericarditis and the emergence of secondary cysts [58,59].

• Anaphylaxis

Cyst rupture can trigger a severe anaphylactic reaction due to the sudden release of cystic fluid into the bloodstream. Symptoms may include hypotensive shock, pruritus, and bronchospasm, making anaphylaxis one of the most critical early signs of rupture. In severe cases, this reaction may progress to cardiopulmonary arrest [60,61].

• Arrhythmia

Cardiac HCs are extremely rare but can lead to severe arrhythmias with high fatality rates. The most frequently reported arrhythmia in patients with cardiac echinococcosis is ventricular tachycardia, followed by third-degree atrioventricular block. HCs that cause arrhythmias are most commonly located in the interventricular septum, where they can disrupt the cardiac conduction system, leading to life-threatening rhythm disturbances [62,63].

Cyst rupture, the most hazardous complication of cardiac hydatid disease, underscores the importance of early diagnosis and timely intervention. Left unchecked, these events can cascade into fatal outcomes such as systemic embolization, thromboembolic events, and severe hypersensitivity reactions. Clinicians must maintain a high index of suspicion, particularly in endemic regions, to mitigate the grave consequences of this latent disease.

9. Treatment and Prognosis of Cardiac Echinococcosis

Surgical removal remains the cornerstone of treatment for cardiac CE, providing definitive management of HCs. This approach is preferred due to the high risk of complications associated with untreated cysts, even in asymptomatic patients. The procedure typically involves cardiopulmonary bypass to ensure a controlled surgical field and minimize the risk of rupture and dissemination of cystic fluid [64,65].

The principles of surgery include avoiding manipulation of the heart prior to cross-clamping, complete excision of the cyst's germinal layer, and meticulous prevention of fluid leakage to prevent contamination of adjacent structures. To further reduce risks, many surgeons allow the residual cavity to heal by secondary intention. Surgical complications, though rare, can include anaphylaxis and secondary cyst formation due to spillage of viable cyst contents [66].

9.1 Protoscolicidal Solutions in Surgical Management

To sterilize cyst contents and prevent intraoperative dissemination, various protoscolicidal solutions are used, including:

• Hypertonic Saline (20%): The most commonly used agent, effective for sterilizing cysts but associated with potential risks such as chemical sclerosing cholangitis and hypernatremia [67,68].

• Ethanol (95%): Widely employed in hepatic echinococcosis but avoided in cardiac CE due to its myotoxic properties, which can induce myocardial necrosis [69,70].

• Formalin and Other Agents: Rarely used due to high tissue toxicity. Alternative agents like formic acid and ethacridine lactate have been reported but lack clear efficacy as protoscolicidal agents [23].

For optimal efficacy, the substances have to be left in the cyst cavity for at least 15 min [23].

9.2 Adjunctive Use of Antiparasitic Therapy

Adjunctive therapy with benzimidazole compounds such as albendazole or mebendazole is recommended to complement surgical treatment. Albendazole is particularly effective due to its activity against *Echinococcus* and is typically initiated several days before surgery and continued for weeks to months postoperatively to minimize recurrence risk [23]. There is no evidence regarding the optimal duration of postoperative albendazole therapy in cardiac CE. Extending postoperative treatment to up to 6 months appears reasonable in patients at increased risk of

recurrence or intraoperative seeding. However, in selected cases where complete cyst removal can be achieved safely without opening the cyst, some surgeons may avoid protoscolicidal instillation altogether. Chemotherapy, however, is contraindicated in cases of large, inactive, or calcified cysts due to the risk of rupture [71]. Additionally, certain systemic conditions, such as bone marrow suppression or pregnancy, may preclude pharmacological therapy. Medical treatment with albendazole has a role in reducing the size of cysts and stopping their development and represents the only therapeutic option in inoperable cases [26]. Of note, albendazole should preferably be administered with a fatty meal to improve its bioavailability and therapeutic efficacy.

10. Outcomes and Prognosis

The prognosis for patients undergoing surgical excision of cardiac HCs is not always favorable. In a systematic review, among the cases with reported outcome data, 659 patients (84.9%) survived postoperatively, while 86 (11.1%) succumbed to complications related to CE, and 31 (4.0%) died of postoperative complications unrelated to CE [23].

As a serious perioperative complication, dislodgement of the germinative membrane can result in an acute stroke and may subsequently lead to a distal aortic embolism as a further consequence [72].

In the same systematic review, among 568 cases with available follow-up data (median 9 months), local recurrence of cardiac CE was reported in 9 patients, typically occurring 12 months post-surgery. Additionally, putative secondary CE was diagnosed in 19 cases at a median of 18 months [23].

Late complications like cyst rupture or secondary cyst formation are rare but highlight the need for thorough intraoperative management and postoperative follow-up. Surgical excision is the gold standard for CE, with antiparasitic therapy as key adjunctive. Advances in surgical techniques and perioperative care have improved outcomes, reducing recurrence and enhancing survival. However, due to the risk of delayed complications, long-term follow-up and a multidisciplinary approach are essential for optimal results. Surveillance is primarily based on periodic imaging, with echocardiography representing the preferred first-line modality due to its availability and ability to assess cardiac morphology and function. Advanced imaging may provide additional information in complex cases or when recurrence is suspected.

The optimal duration of follow-up remains uncertain, but prolonged monitoring over several years is generally recommended because recurrence may occur late after treatment. Serial serological testing can be used as an adjunctive tool to monitor antibody dynamics, but persistent antibody positivity after successful therapy and variable sensitivity, particularly in extrahepatic disease, limit its reliability. Therefore, follow-up decisions should rely mainly on clinical assessment and imaging findings rather than serology alone.

11. Conclusion

Overall, cardiac echinococcosis poses significant clinical challenges due to its complex pathogenesis, variable clinical manifestations, and potentially life-threatening complications. As demonstrated by our case, cardiac hydatid cysts may remain clinically silent for many years, even over a decade, emphasizing the importance of considering this diagnosis in persistent cardiac cystic lesions despite an apparently indolent course. However, prolonged clinical stability does not eliminate the risk of unpredictable complications such as rupture, embolization, arrhythmias, or anaphylactic reactions. Early detection and comprehensive management, primarily through surgical removal of cysts and adjunctive antiparasitic therapy, are critical for improving outcomes. While surgical treatment remains the gold standard, the risk of recurrence and secondary cyst formation necessitates vigilant long-term monitoring. Understanding the disease's epidemiology, diagnostic strategies, and treatment options is essential for reducing morbidity and mortality, particularly in endemic regions where this zoonotic disease remains a major public health concern.

Author Contributions

The article has been drafted jointly by AG and KK. KK edited and finalized the manuscript. AG and KK prepared all figures. EM and JG revised the manuscript critically for important intellectual content. AG, KK, EM, and JG contributed to the conception. All authors have read and approved the final version of the manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

Not applicable.

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Conflicts of Interest

The authors declare no conflicts of interest.

Declaration of AI and AI-Assisted Technologies in the Writing Process

During the preparation of this work, including analysis and interpretation, the authors used no AI or AI-assisted

technologies, except for the limited use of ChatGPT for checking spelling and grammar. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

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