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Cardiac imaging in patients with tropical diseases—a scientific statement of the European Association of Cardiovascular Imaging (EACVI) of the European Society of Cardiology and the Cardiovascular Imaging Department of the Brazilian Society of Cardiology (DICSBC)

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Tropical diseases (TDs), or neglected TDs, affect over 1 billion subjects globally, primarily in impoverished regions. Despite their impact, these diseases are often overlooked on the global health agenda, with limited resources allocated to their control. TDs are caused by various pathogens, including viruses, protozoa, helminths, and bacteria. Climate change and migration have led to the spread of TDs beyond tropical regions, highlighting the need for healthcare providers worldwide to address their management. The World Health Organization has released a road map for the prevention, control, and elimination of TDs by 2030. Cardiovascular involvement in TDs, including myocarditis, pericardial disease, pulmonary hypertension, obstructions caused by cysts, myocardial ischaemia, and cardiomyopathies, complicates disease prognosis. However, cardiovascular complications of TDs are often under-recognized and understudied. This scientific statement, prepared by the European Association of Cardiovascular Imaging and the Cardiovascular Imaging Department of the Brazilian Society of Cardiology, reviews the current understanding of the use of cardiovascular imaging in TDs, underscores gaps of knowledge, and proposes potential solutions. Although limited evidence is available, cardiovascular imaging techniques are valuable in diagnosing and managing cardiac manifestations of TDs. The scientific statement addresses some of

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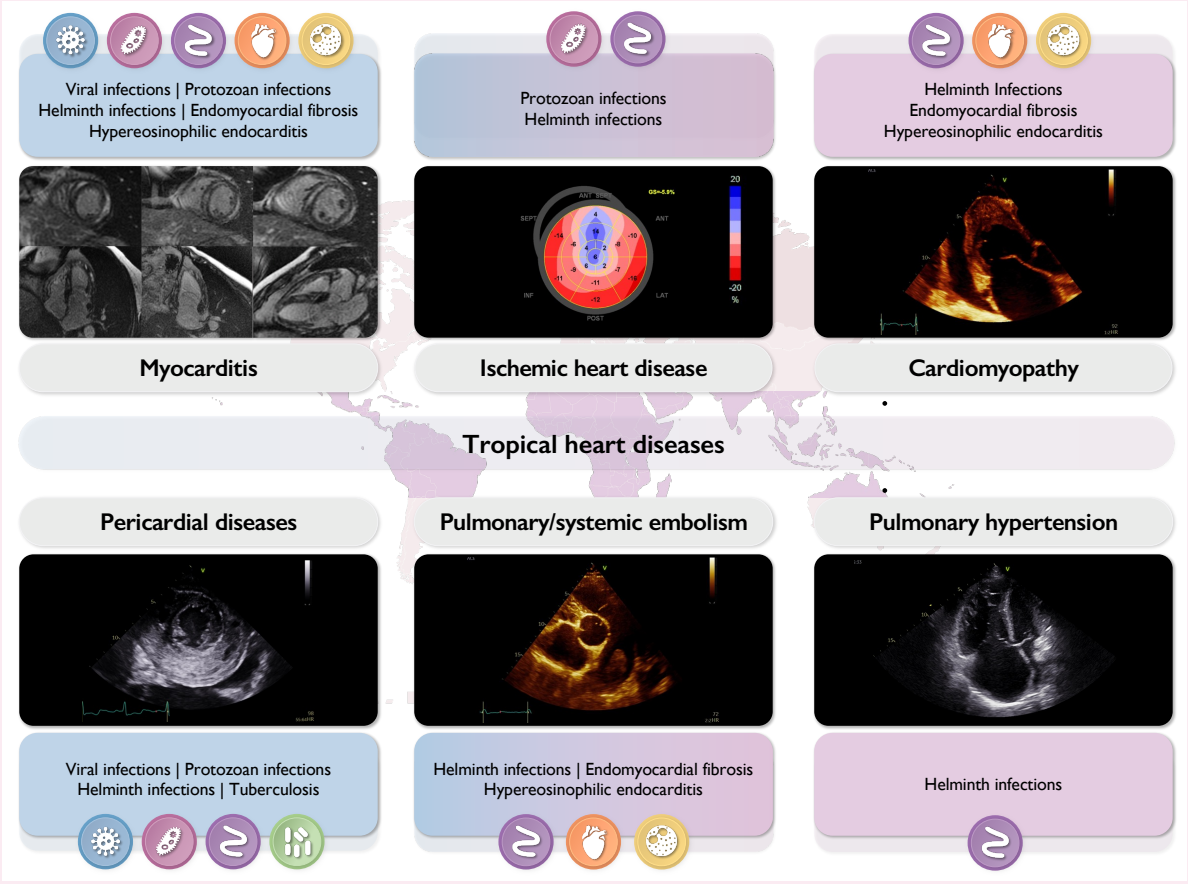
the main tropical infections today, such as dengue, malaria, schistosomiasis, and tuberculosis, but excludes Chagas disease and rheumatic fever, which have been extensively covered in other documents. When used effectively, cardiovascular imaging can potentially aid in early diagnosis, prevention of complications, and management of cardiovascular impairment due to TDs, improving healthcare systems and patient care.

Graphical Abstract



Main cardiovascular manifestations of tropical diseases.

Keywords tropical medicine • cardiovascular imaging • neglected tropical diseases • global health • cardiac care

Introduction

Over 1 billion subjects worldwide are affected by tropical diseases (TDs), commonly known as 'neglected TDs'. These diseases are often disregarded on the global agenda. Funding agencies frequently overlook these diseases, and limited resources are allocated to their prevention, diagnosis, treatment, and control.[1] TDs comprise a varied group of diseases that include infections caused by viruses, protozoans, helminths, and bacteria. Although primarily prevalent in underdeveloped and tropical countries, some of these diseases can also be seen in other geographical regions, for instance, in the context of migration and climate change. Rising temperatures and changing rainfall patterns in southern Europe have expanded the habitat of *Aedes* mosquitoes, resulting in outbreaks of dengue, chikungunya, and Zika, including local dengue transmission in Italy.[2] In the Southern USA, warmer and humid conditions have led to sporadic dengue and Zika outbreaks in Texas and Florida. Meanwhile, migration from endemic regions has increased the burden of diseases like malaria, leishmaniasis, and tuberculosis in northern European countries (Sweden and Germany) and the UK, often linked to refugee and labour migration.[3] Thus, there is also a need to address these diseases in high-income and non-tropical countries, and most physicians must become familiar with diagnostic and management strategies for these diseases.

TDs present a complicated epidemiology, often involving animal and human reservoirs, vector-borne transmission, and complex life cycles, making prevention and elimination challenging. The World Health Organization (WHO) has released a 'road map for neglected TDs 2021–30' that establishes global targets and milestones to prevent, control, eliminate, or eradicate a diverse set of 20 diseases.[4] As outlined in the road map, addressing TDs requires a comprehensive approach that includes strategic interventions to provide innovative disease diagnosis, management, vector control, safe water, sanitation, and public health. This approach is necessary to combat the complexity and depth of this issue. In addition to the WHO, other international societies have raised concerns about TDs and proposed initiatives to tackle the issue. One such initiative is the NET-Heart Project from the Interamerican

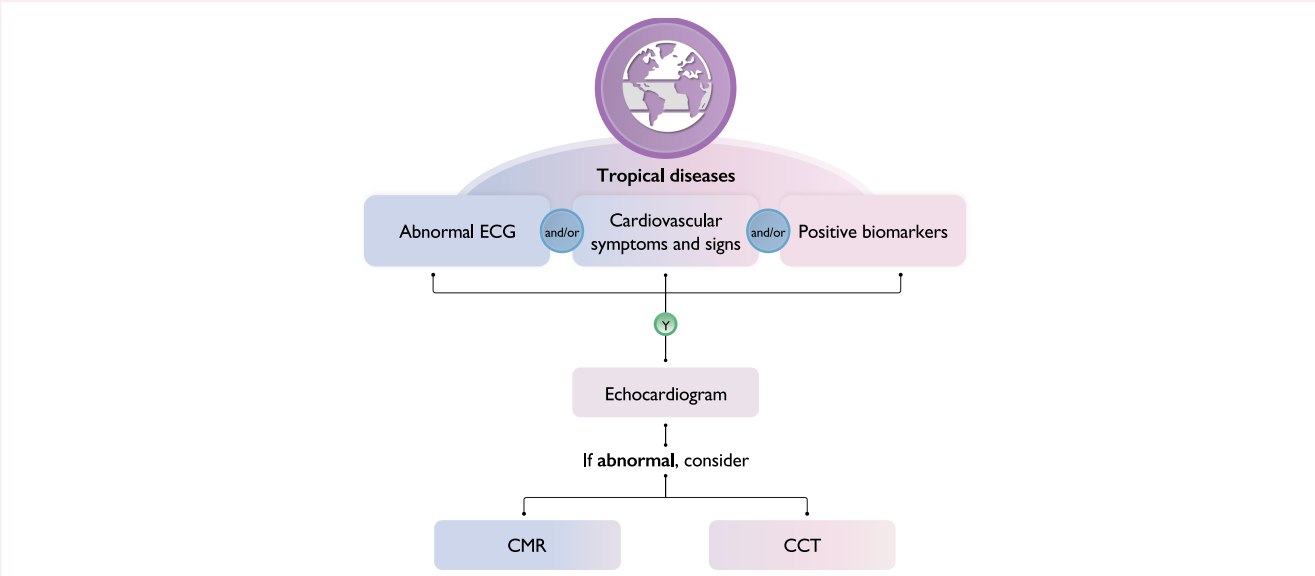


Figure 1 The non-invasive diagnostic work-up of cardiac involvement in tropical diseases. CCT, cardiovascular computed tomography; CMR, cardiac magnetic resonance; ECG, electrocardiogram; ECHO, echocardiography.

Society of Cardiology, which systematically evaluates evidence on TDs and provides recommendations for managing their cardiovascular complications, while recognizing that region-specific barriers may require tailored strategies to improve care access.[5,6]

Why do we need a scientific statement on the use of cardiac imaging in patients with TDs?

TDs have emerged as a growing global public health issue, demanding comprehensive interventions to reach the 2030 elimination goal. This includes the leverage of technologies to improve the diagnosis and management of this group of diseases. Tropical infections can contribute to the burden of cardiovascular disease by themselves (cardiac manifestations of TDs include myocarditis, pericardial disease, myocardial ischaemia, pulmonary hypertension, embolism, cardiomyopathy, and others) (*Graphical Abstract*) and as a consequence of interactions with pre-existing cardiovascular conditions.[7] However, the current knowledge about the cardiovascular complications of TDs is limited, and their impact may be underestimated. Although not often the site of primary involvement, compromise of the cardiovascular system can contribute to a worse prognosis.[8] When used effectively, cardiovascular imaging can aid in early diagnosis, the prevention of complications, and the appropriate management of cardiovascular complications related to TDs, improving patient care and outcomes. However, cardiovascular imaging must be tailored to local resource constraints. This statement aims to (i) explore the cardiovascular phenotypes and morphophysiological findings in neglected TD based on available imaging modalities and (ii) address current knowledge gaps regarding the usefulness of cardiovascular imaging in patients with TDs. It does not advocate for a universal multimodality approach for every patient.

Methodology

A writing group of cardiovascular imaging experts was appointed by the European Association of Cardiovascular Imaging (EACVI) of the European Society of Cardiology (ESC) and by the Cardiovascular Imaging Department of the Brazilian Society of Cardiology (DICSBC) to summarize the current status of imaging in the assessment of cardiovascular involvement in TDs. The writing group acknowledges the relatively limited research on the topic. No evidence-based strategies have been established for the detection and management of cardiac complications associated with most of the TDs discussed here. The use of cardiovascular imaging methods in TDs has not been thoroughly studied due to the unavailability of some of these technologies in remote and underserved regions of low-income countries and because of the lack of comprehensive data collection in the most affected areas. Therefore, all efforts were made to interpret the available scientific data and provide the best information on the topic without giving specific advice for clinical practice. The document was developed through an extensive review of published research papers and reviews. The references included in this document were considered to be the most up-to-date and with the best level of evidence, if any. Each section briefly addresses the epidemiology, diagnosis, and clinical manifestations of the specific disease in question and then focuses on cardiac imaging findings available in the literature.

The document includes the following TDs: mosquito-borne viral infections (dengue, Zika, chikungunya, and yellow fever), protozoal infections (malaria, leishmaniasis, amoebiasis, and toxoplasmosis), helminth infections (echinococcosis, cysticercosis, and schistosomiasis), bacterial infections (tuberculosis), endomyocardial fibrosis (EMF), and hypereosinophilic endocarditis (HE). The selected TDs were chosen based on available literature that reports any level of cardiovascular involvement, regardless of the strength of the evidence. Rheumatic fever[9,10] and Chagas disease[11,12] have been widely addressed in several reviews and guidelines and are not discussed in this document. The final document aligns with the WHO 'road map for neglected TDs 2021–30'.

General considerations

The non-invasive diagnostic work-up of cardiac involvement in TDs may include clinical assessment, laboratory testing (including cardiac biomarkers), an electrocardiogram (ECG), and cardiovascular imaging

(*Figure 1*). Much is still unknown about the pathophysiology of most of these diseases, their impact on the heart, and the appropriate use of imaging methods in the diagnostic pathway and in guiding treatment decisions. However, it is reasonable that patients with a diagnosis of a tropical infection who present with cardiovascular symptoms and signs (such as chest pain, dyspnoea, palpitations, syncope, peripheral oedema, haemodynamic instability) and/or an abnormal ECG and/or positive cardiac biomarkers (troponin, creatinine kinase, N-terminal pro-brain natriuretic peptide) have further evaluation with cardiac imaging.[13]

Echocardiography (ECHO) is the first-line imaging technique, and it is advised in all patients with suspected cardiovascular involvement.[14,15] It is a cost-effective and widely available method for screening patients with clinical suspicion of myocarditis and/or pericarditis.[16,17] Cardiac magnetic resonance (CMR), cardiovascular computed tomography (CCT), and nuclear imaging tests (NIT) are advised if feasible to answer clinical questions not provided by ECHO.[16,17] CMR provides non-invasive myocardial tissue characterization, identifying disease patterns such as fibrosis, inflammation, and myocardial oedema using late gadolinium enhancement (LGE), T1, and T2 mapping.[16,18] CMR should only be considered in specific clinical scenarios and when appropriate infrastructure is available. A subendocardial or transmural LGE pattern suggests myocardial infarction, while mid-wall or subepicardial patterns point to myocarditis.[16,19] Although endomyocardial biopsy remains the gold standard for a definitive diagnosis, it is invasive and has limited sensitivity. In specific cases, CCT can help rule out obstructive epicardial coronary artery disease, providing additional non-invasive diagnostic support. Holter ECG monitoring, event recorders, portable devices, and implantable loop recording may be helpful for arrhythmia assessment.

The writing group acknowledges that areas with limited healthcare access present a challenge for the widespread use of cardiovascular imaging in TDs. In these settings, where imaging is most needed, resources are scarce. Therefore, any advice concerning the use of cardiovascular imaging must take into account the practical realities of its implementation in these settings. This statement does not endorse the uniform use of multimodality cardiovascular imaging in all scenarios, but highlights ECHO as the most accessible tool in certain cases, while other modalities are reserved for more complex situations or referral centres. Furthermore, the acute cardiovascular manifestations of TDs are often challenging to assess with advanced imaging, particularly in neglected communities or in critically ill patients. As such, the utility of multimodality cardiovascular imaging is likely greater during the chronic phase, when patients are clinically stable and techniques like cardiac CMR or CCT can be safely performed. The growing incidence of TDs in high-income countries brings these conditions into healthcare systems where multimodality imaging is available and can support accurate diagnosis and management. Another significant issue is that, while cardiovascular system involvement may result in worse outcomes, many of these diseases exhibit similar changes in multimodality cardiovascular imaging and lack pathognomonic or specific findings. *Table 1* presents the main cardiovascular phenotypes and imaging characteristics of TDs.

Table 1 Main cardiovascular phenotypes and imaging findings of tropical diseases

	Tropical disease[a]	Cardiovascular phenotypes	Key imaging findings	Preferred Imaging modality[b]			
				ECHO	CMR	CCT	NIT
Virus	Dengue	Pericarditis and/or myocarditis, arrhythmias, HF, cardiogenic shock	GLS and GCS reductions in early stages. Pericardial effusion, LV global/regional systolic and diastolic dysfunction, RV dysfunction Myocardial oedema and fibrosis on CMR	+++	++	+	-
	Zika	Pericarditis, arrhythmias, HF, CZS. Rarely, myocarditis	Pericardial effusion (unusual), LV dysfunction (rare). Congenital heart disease in foetuses of infected mothers	+++	+	+	-
	Chikungunya	Frequent cardiac involvement: myocarditis, cardiogenic shock, ACS, pericarditis, arrhythmias, heart failure	LV global/regional systolic and diastolic dysfunction, pericardial effusion Myocardial oedema and fibrosis on CMR	+++	++	+	-
	Yellow fever	Myocarditis, paradoxical bradycardia, conduction abnormalities	LV dysfunction in severe forms Myocardial oedema and fibrosis on CMR	+++	++	-	-
Protozoan	Malaria	Myocarditis, ACS, pericarditis, cardiogenic shock, and rarely PAH	GLS reduction, LV global systolic dysfunction, pericardial effusion, tamponade, increased PASP Myocardial oedema and fibrosis on CMR	+++	++	+	-

Continued

Mosquito-borne viruses

Mosquito-borne viral infections, particularly arboviruses, are increasingly recognized as relevant causes of cardiovascular disease, especially in tropical and subtropical regions,[20] with increasing cases also recently reported in Europe and the USA.[21] While most of these infections are self-limited, a subset of patients may experience cardiovascular involvement, most often in the form of myocarditis, pericarditis, or acute heart failure.[16,22] In chronic stages, patients can also develop arrhythmogenic scars caused by post-myocarditis fibrosis, leading to malignant late ventricular arrhythmias and sudden death.[16,23] The WHO

Table 1 Continued

	Tropical disease[a]	Cardiovascular phenotypes	Key imaging findings	Preferred Imaging modality[b]			
				ECHO	CMR	CCT	NIT
	Amoebiasis	In rare cases, pericarditis, cardiogenic shock, and HF	Pericardial effusion, tamponade, or constrictive pericarditis	+++	+	+	-
	Leishmaniasis	In rare cases, myocarditis and pericarditis	LV systolic dysfunction, pericardial effusion	+++	++	+	-
	Toxoplasmosis	In rare cases, myocarditis and pericarditis	LV systolic dysfunction, pericardial effusion	+++	++	+	-
Helminth	Echinococcosis	Embolic complications, syncope, newly detected heart murmur, and obstructive intracardiac mass	Hydatid cysts predominantly in the LV (but also in other chambers), pericardium, or pulmonary artery. Sometimes, valvular dysfunction, and tamponade (rupture of the cyst). CMR and CT provide tissue characterization and superior cyst visualization	+++	+++	+++	-
	Cysticercosis	Often asymptomatic. Rarely, myocarditis, and obstructive intracardiac mass	Multiple cysts with a characteristic scolex CMR and CT provide tissue characterization and superior cyst visualization	+++	+++	+++	-
	Schistosomiasis	Acute phase: myocarditis, pericarditis, myocardial ischaemia Chronic phase: PAH and *cor pulmonale*	GLS reduction, LV global systolic dysfunction, pericardial effusion, increased PASP, RV dilation, and dysfunction	+++	++	+	-
Bacteria	Tuberculosis	Most commonly, pericarditis Rarely, myocarditis and aortitis	Pericardial thickening and effusion, tamponade, and constrictive pericarditis Multimodality imaging enables assessment of the pericardium (CCT, CMR), myocardial tissue characterization (CMR), and aortic inflammation (18F-FDG PET)	+++	++	+++	+
Restrictive cardiomyopathies	EMF and HE	HF and embolic complications Eosinophilia and systemic signs in HE	Obliteration of the RV and/or LV apex and hypercontractility in the basal segments. Endocardial thickening, thrombus, enlarged atria, restrictive diastolic filling, mitral and tricuspid regurgitation, and pericardial effusion CMR provides a superior assessment of morphological and functional aspects. CT can also help differentiate thrombus from calcification	+++	+++	++	-

18F-FDG PET, 18-fluorodeoxyglucose positron emission tomography; ACS, acute coronary syndrome; CCT, cardiovascular computed tomography; CMR, cardiac magnetic resonance; CZS, congenital Zika syndrome; ECHO, echocardiography; EMF, endomyocardial fibrosis; GCS, global circumferential strain; GLS, global longitudinal strain; HE, hypereosinophilic endocarditis; HF, heart failure; LV, left ventricular; NIT, nuclear imaging tests; PAH, pulmonary arterial hypertension; PASP, pulmonary artery systolic pressure; RV, right ventricle.

[a]The selected tropical diseases were chosen based on existing literature that reports any level of cardiovascular involvement, regardless of evidence strength.

[b]The table highlights preferred cardiovascular imaging modalities for tropical diseases, but practical factors like cost and availability must also be considered. While CMR is excellent for characterizing myocardial tissue, logistical and economic challenges often limit its use, especially in remote, low-income areas.

+++, ++, +, and – indicate the relative usefulness of each imaging modality in the evaluation of cardiovascular manifestations of each disease, based on current literature and expert consensus. +++ = highly useful; ++ = moderately useful; + = occasionally useful; – = not useful or not recommended.

recommends a serologic diagnosis, which includes routine diagnostic testing of whole blood, serum, or plasma for arboviruses and urine analysis with a Zika nucleic acid amplification test.[21] Despite their potential impact, cardiovascular complications are likely underdiagnosed due to non-specific symptoms, limited access to imaging, and the predominant focus on neurologic or haemorrhagic outcomes in these diseases.

Dengue

Dengue (breakbone fever) is an arbovirus disease caused by any of the four serotypes of the dengue virus, transmitted by mosquito species *Aedes aegypti* or *Aedes albopictus*. Subsequent infection with a different serotype can result in a more severe disease, including serious bleeding and shock syndrome.[24] According to the 2009 WHO classification, there are three types of dengue: 'without warning signs,' 'with warning signs,' and 'severe, with or without haemorrhage'.[25] Patients with severe dengue may develop haemorrhagic fever, characterized by thrombocytopenia, plasma leakage, and bleeding.[26] The incidence of cardiac involvement varies significantly from one series to another (between 12 and 50% in hospitalized patients), and the most frequent cardiac manifestations are arrhythmias, pericarditis, myocarditis, and deterioration in myocardial function.[8] Myocarditis may mimic myocardial infarction, and in rare cases, the inflammatory response may also trigger intracoronary thrombosis with acute myocardial infarction, coronary dissection, and intramural haematoma.[27] Some reports suggest a higher incidence of cardiac complications in the paediatric population.[28] Men may face a higher risk of cardiac complications from dengue compared to women, with higher rates of LV systolic dysfunction. In daily practice, only a small percentage of dengue patients have a cardiac evaluation, especially in endemic areas, leading to significant underdiagnosis.[29] The presence of any abnormality on ECHO has been associated with adverse outcomes in patients with dengue infection.[30,31] The main echocardiographic findings are pericardial effusion, left ventricular (LV) regional wall motion abnormalities, and LV global systolic dysfunction (*Table 1* and *Figure 2*; Supplementary data online, *Video S1*).[32] In early stages of the disease, 2D speckle tracking echocardiography (STE) may show subtle subclinical alterations in myocardial deformation, with abnormal global longitudinal strain (GLS) and global circumferential strain (GCS).[33] Severe dengue patients show more advanced systolic and diastolic LV dysfunction and right ventricular (RV) dysfunction,[34] usually transient.[35] As previously mentioned, CMR can support the diagnosis of myocarditis (*Figure 2*; Supplementary data online, *Video S2*) in most cases.[18,19]

Zika

Zika infection is a viral disease from the Flaviviridae family, transmitted by *Aedes* sp. mosquitoes. Most people with Zika virus infection are asymptomatic or develop non-specific symptoms such as low-grade fever, headache, rash, arthralgia, and conjunctivitis.[36] Neurological complications are classically described, with reports of Guillain–Barré syndrome and meningoencephalitis.[37] Cardiac manifestations of Zika infection are rare but can include arrhythmias, pericardial effusion, and heart failure, with potential long-term effects like dilated cardiomyopathy. Congenital Zika syndrome (CZS) may occur in foetuses of mothers infected by Zika virus, leading to congenital heart disease in 10–14% of cases,[37,38] which presents as atrial and ventricular septal defects, anomalous pulmonary venous return, and persistence of ductus arteriosus.[39–41]

Chikungunya

Chikungunya fever, caused by a mosquito-borne alphavirus, is characterized by abrupt onset of fever, often associated with various systemic symptoms.[42] Cardiac involvement is among the most frequent systemic manifestations, in particular myocarditis, often with hypotension and shock, followed by pericarditis, acute myocardial infarction, and arrhythmias, potentially leading to heart failure.[42,43] Since mortality in patients with chikungunya is mainly driven by cardiovascular complications, early detection is crucial to timely treatment.[43]

Yellow fever

Yellow fever is an African mosquito-borne infection of primates, caused by a flavivirus. It is transmitted between monkeys in their natural habitat by forest-dwelling primatophilic *Aedes* mosquitoes.[44] In humans, this disease is a viral haemorrhagic fever, endemic in some parts of South America and Africa. Severe cases often involve myocardial injury and impaired heart function due to various factors, including viral damage and inflammation.[45] Patients may exhibit bradycardia, ECG abnormalities, and LV wall thickening with a hyper-refringent myocardial texture suggesting infiltration.[46]

Protozoan infections

Protozoan infections are endemic in subtropical or tropical regions, particularly in developing countries or those with poor sanitary and hygiene conditions.[5] Excluding Chagas disease, the most prevalent protozoan infections are malaria, leishmaniasis, amoebiasis, and toxoplasmosis. Rarely, these infections can affect the myocardium and pericardium, particularly in severe forms. Cardiac involvement can occur through inflammatory responses or be immune-mediated.[47] However, as neglected diseases, cardiovascular complications may be underdiagnosed or underreported. Imaging modalities can help diagnose and manage the cardiac manifestations associated with protozoan infections (*Table 1*).

Malaria

Malaria is a severe acute febrile illness caused by five different species of the Plasmodium parasite, with two species—*Plasmodium falciparum* and *Plasmodium vivax*—posing the greatest risk. It is spread to humans through the bites of infected female *Anopheles* mosquitoes, but it can also be rarely transmitted congenitally or by blood transfusion and contaminated needles.[48]

Malaria is present in over 100 countries and is responsible for over 400 000 deaths every year, with almost 93% occurring in Africa.[48] Severe malaria, mainly caused by *P. falciparum*, can lead to potentially lethal complications such as lung, kidney, or cerebral injury.[49] Cardiac involvement has not been considered a common cause of morbidity and mortality, possibly due to under-reporting or underdiagnosing.[49,50] In several case reports and series of cases, the main described cardiovascular phenotypes were myocarditis and acute coronary syndrome, but pericarditis, pericardial effusion, and pulmonary congestion have also been reported.[50,51] A meta-analysis including 3117 patients (2403 adults) demonstrated that the estimated prevalence of cardiovascular complications in adults with symptomatic malaria was 7%.[52] The most often observed echocardiographic abnormality was impaired LV function. The significant differences between studies probably result from the lack of standardized recording protocols, variable sample sizes, different parameters, and cut-off points. It is uncertain whether LV dysfunction in patients with acute malaria can lead to heart failure in the long term. A study found that adults with uncomplicated malaria had lower GLS than the general population, which improved after antimalarial treatment.[53] Although the results suggest that malaria may impact LV systolic function, further research is necessary to fully comprehend the cardiac complications of malaria and to develop effective management strategies.

Amoebiasis

Infections with *Entamoeba histolytica* can be asymptomatic (90%) and may cause dysentery, but in 10% of patients, invasive amoebiasis is

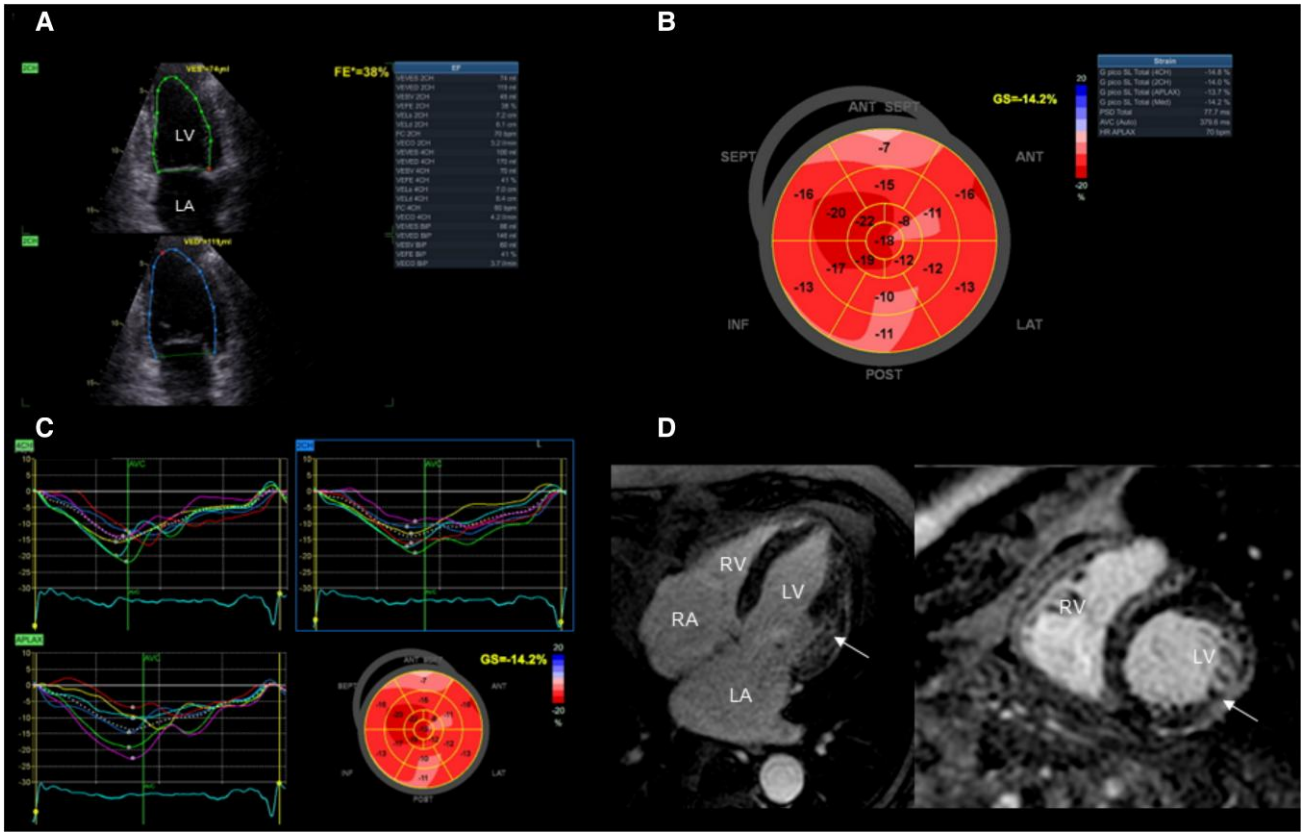


Figure 2 Imaging findings related to cardiovascular involvement in dengue. A 77-year-old male was admitted after dengue acute infection, with fever, cutaneous rash, body aches, atypical chest pain, and mild troponin elevation. Coronary angiography did not demonstrate any obstructive coronary stenoses. Transthoracic echocardiogram showed global LV systolic dysfunction with LVEF = 38% (*A*) (see Supplementary data online, *Video S1*) and diastolic dysfunction with reduced reservoir atrial strain = 18% (see Supplementary data online, *Video S3*). Absolute LV 2D GLS (−14.8%) was reduced with a diffuse pattern, more pronounced in the anterolateral, inferolateral, and inferior walls (*B*), and with post-systolic shortening in the strain curves (*C*), that corresponded to areas of midwall LGE (white arrows) on CMR (*D*). 2D, bidimensional; EF, ejection fraction; LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

present.[47,54] When the heart is involved, the pericardium is the most commonly affected structure, with acute pericarditis, pericardial effusion, cardiac tamponade, or constrictive pericarditis.[54,55]

Leishmaniasis

Leishmaniasis is caused by a specific protozoan parasite transmitted by the bite of infected female phlebotomine sandflies.[56] Leishmaniasis affects people worldwide, especially in tropical and subtropical regions, presenting a broad range of clinical manifestations, from self-resolving skin lesions to disseminated visceral involvement. Cardiac involvement associated with visceral leishmaniasis is not well known and has primarily been described in case reports of myocarditis and pericarditis, especially among immunocompromised individuals.[57,58]

Toxoplasmosis

Toxoplasmosis is caused by *Toxoplasma gondii*, affecting one-third of the world's population. Humans are infected either by eating undercooked infected meat, faecal–oral transmission from feline faeces, organ transplantation, blood transfusion, or transplacental transmission.[59] Toxoplasmosis is usually asymptomatic or has a benign course in healthy individuals, but these microorganisms can spread to various organs in immunocompromised patients or through congenital transmission.[59] While cardiac involvement is rare, myocarditis is the most common manifestation.[60] In patients with acquired immunodeficiency syndrome (AIDS), the heart is the second most frequently affected organ after the brain. Myocarditis associated with *T. gondii* can also arise in transplant patients either due to reactivation or to a new infection from a seropositive donor to a seronegative recipient.[60] Actually, toxoplasmosis is the most commonly reported parasitic disease occurring after heart transplantation.[61] In immunocompromised individuals, toxoplasmosis can lead to severe LV systolic dysfunction requiring haemodynamic support.[61] There have also been reports of pericarditis and pericardial effusion, in isolation or combined with myocarditis.

Helminth infections

Helminths that affect the human heart include a wide range of species, which exert their influence directly or indirectly. They have many forms, including adult worms, larval stages, and eggs, and can cause various cardiac disorders.

Echinococcosis

Echinococcosis, caused by the larval stages of tapeworms belonging to the genus *Echinococcus*, presents in two primary forms: cystic echinococcosis, primarily caused by *Echinococcus granulosus*, and alveolar echinococcosis, mainly attributed to *Echinococcus multilocularis*.[62] *Echinococcus granulosus* is the most widespread species globally, particularly in rural grazing areas where dogs have access to organs from infected animals, perpetuating the transmission cycle.[63] Cystic echinococcosis, characterized by the presence of hydatid cysts, predominantly affects the liver or lungs but can potentially develop in any organ.[47] In cardiac echinococcosis (*Table 1*), cysts are predominantly located in the LV,[64] though they can also occur in the right ventricle,[65] right atrium,[66]left atrium,[67] interventricular septum,[68] pericardium,[69] or pulmonary arteries.[70] Cardiac echinococcosis (*Figure 3*) is an uncommon manifestation of cystic echinococcosis, which affects around 0.5–2% of cases.[62] Patients may remain asymptomatic for long periods before developing symptoms due to the mass effect on surrounding tissues or an acute rupture of a hydatid cyst, which can lead to widespread dissemination or pericardial tamponade (*Figure 4*).[63] Depending on the location, acute cyst rupture can cause arterial or pulmonary embolism and lead to shock due to the release of anaphylactoid substances.[47] The cardiac involvement in echinococcosis can present clinically as chest pain, dyspnoea, recurrent syncope, palpitations, and valvular dysfunction characterized by newly detected heart murmurs. ECG findings may reveal conduction abnormalities and repolarization changes, which can sometimes mimic ischaemic patterns.[63] The diagnosis relies on a comprehensive patient history, specific serologic tests, and confirmation through cardiovascular imaging techniques. Besides transthoracic and transoesophageal ECHO, CMR and CT offer highly reproducible results, which are crucial for precise surgical planning of cyst removal.[65,68,69,71] These imaging techniques assist not just in diagnosis but also in monitoring therapy, assessing prognosis, and tracking disease progression. Screening for cardiac hydatid cysts is advised in patients with liver or lung involvement in cystic echinococcosis. The treatment of cardiac echinococcosis includes surgical excision of hydatid cysts followed by anthelmintic therapy with albendazole or mebendazole.[62,63] Patients with cystic echinococcosis can benefit from interdisciplinary management at tertiary treatment centres and from treatment modalities based on the cyst stage.

Cysticercosis

Cysticercosis, caused by the larval form of the tapeworm *Taenia solium*, affects millions of people worldwide.[72] This parasitic infection thrives in areas with poor sanitation where humans and free-roaming pigs coexist, facilitating transmission.[72] Neurocysticercosis, characterized by cysts in the central nervous system, is the most common clinical presentation with parenchymal and extraparenchymal forms.[63] Cysts can also be detected in various organs, occasionally in the heart. Cardiac involvement, observed in up to 25% of the infected, often presents asymptomatically, with cystic lesions typically discovered during cardiac imaging or surgery.[72] Rarely, it can cause myocarditis[73] or an intracardiac mass causing obstruction. Cysticerci are oval cystic structures with thin, semi-transparent, and serous walls, typically harbouring a characteristic scolex (*Figure 4*).[47] Cardiac cysticerci commonly manifest as multiple lesions, and isolated occurrences of a single cardiac cyst are rare.[72] Symptoms of cardiac cysticercosis depend on cyst number, location, size, and stage of the lesion, as well as host inflammatory response to degenerating cysts.[72] Early manifestations may include chest pain, syncope, arrhythmias, conduction abnormalities, heart failure, and pericardial effusion. Ventricular fibrosis resulting from cyst degeneration can be the substrate for ventricular tachycardia. The occurrence of Takotsubo syndrome or persistent bradycardia caused by acute hydrocephalus in neurocysticercosis is uncommon.[47] Diagnosis relies primarily on serological testing in appropriate epidemiological contexts. Various modalities of cardiovascular imaging can aid in the diagnosis of cardiac cysticercosis and provide insights into the characteristic features of cardiac involvement.[63] ECHO can reveal macroscopic cysts at different stages of evolution. Early cysts typically present as spherical echo-free lesions with smooth walls and a hyperechogenic internal scolex, a distinctive hallmark of cardiac cysticercosis.[72] CMR offers superior tissue characterization, with cysts typically appearing hypointense on T1-weighted images and hyperintense on T2-weighted images, often showing LGE in the cyst wall.[74,75] CCT imaging also plays a valuable role, enabling the visualization of cysts within the heart and occasionally in the surrounding chest wall or lung. Treatment for cardiac cysticercosis usually involves a combination of albendazole or praziquantel with corticosteroids. Surgical resection may be considered for myocardial cysts causing functional complications in select cases.

Schistosomiasis

Schistosomiasis, a tropical helminth infection, is caused by digenetic trematodes of *Schistosoma*.[76] These parasites reside in specific types of freshwater snails, where they release cercariae, the infectious form. Contaminated water enables cercariae to penetrate the skin, causing infection. The larvae migrate through the lungs and settle in splanchnic

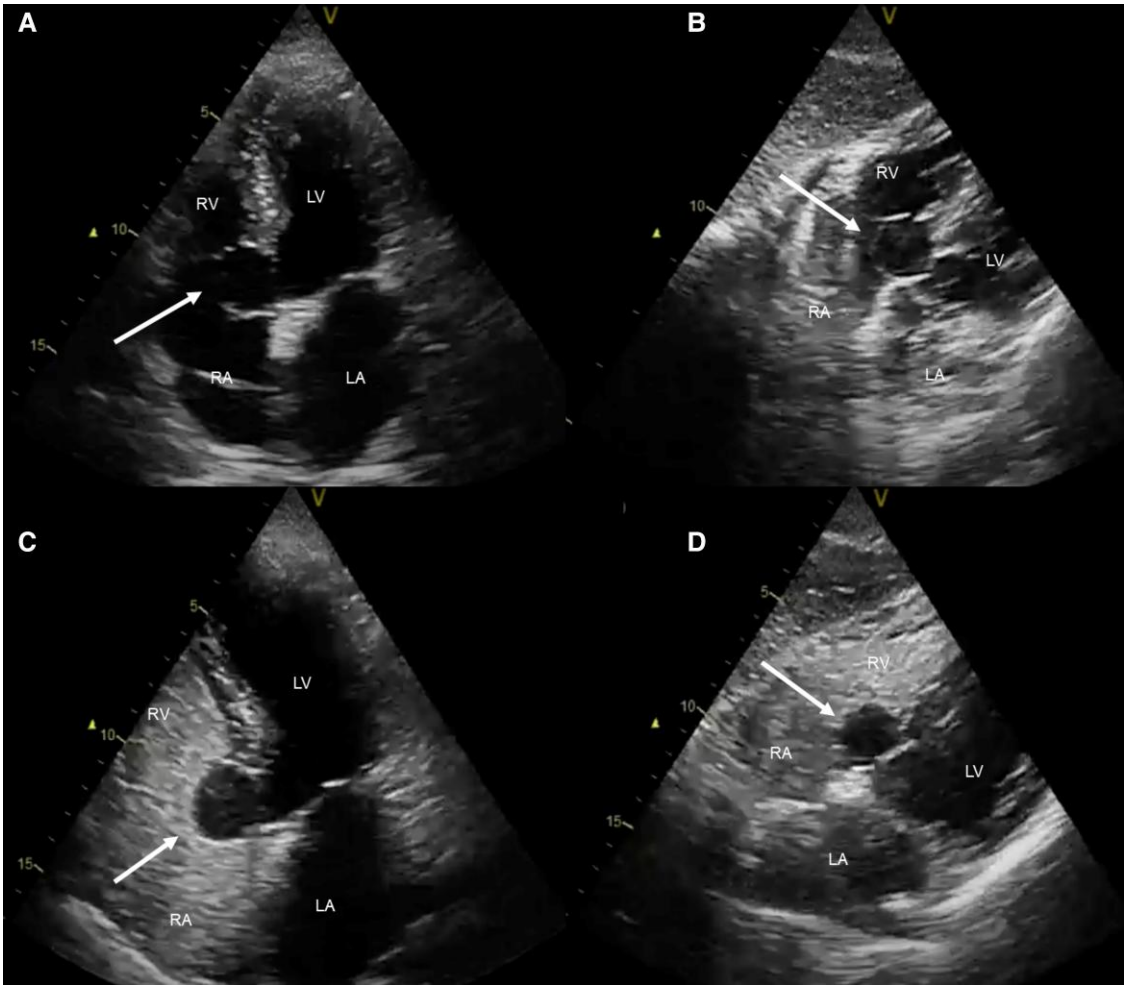


Figure 3 Imaging findings related to cardiovascular involvement in echinococcosis. A 38-year-old man from a rural region endemic to echinococcosis presented with fatigue and syncope, along with a complete atrioventricular block on the electrocardiogram. Transthoracic echocardiogram in the apical four-chamber view (*A*) and subcostal view (*B*) showed an echo-free structure with a cystic appearance located within the right atrium, adherent to the interatrial septum and adjacent to the tricuspid septal leaflet (red arrows). Subsequent injection of agitated saline solution notably improved the visualization of the cyst (*C* and *D*). LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle. Adapted and reprinted with permission.[66]

veins, where they can persist for years. The life cycle completes when eggs are excreted in the host's urine or faeces.[77] Schistosomiasis is prevalent in tropical and subtropical areas, especially in impoverished communities lacking access to clean water and proper sanitation.[77] Although the prevalence of schistosomiasis is highest in sub-Saharan Africa, due to tourism growth, cases among travellers have increased.[78] *Schistosoma mansoni* is the primary species infecting humans and can result in severe cardiovascular complications (*Table 1*). During the acute phase of schistosomiasis, patients may experience myocarditis, pericarditis, and myocardial ischaemia.[76] ECHO may show ventricular dysfunction and pericardial effusion, while CMR can provide additional tissue characterization data.[76] However, the acute phase of schistosomiasis can often go undetected in endemic regions. During the chronic phase of the disease, one of the most serious cardiovascular consequences of schistosomiasis is pulmonary arterial hypertension (PAH).[76,79,80] All patients presenting with PAH in an endemic area should be investigated for schistosomiasis, as it is among the leading causes of PAH worldwide. The pathogenesis of PAH remains elusive, but it is believed that mechanical obstruction of lung vasculature by schistosome eggs triggers a granulomatous response, leading to inflammation and vascular remodelling (*Figure 4*).[47] This process can culminate in fibrosis, PAH, and the eventual development of *cor pulmonale*, characterized by RV dilation and dysfunction, particularly in hepatosplenic schistosomiasis (*Figure 2A*).[81] Moreover, abnormal RV pressure overload may alter LV function in patients with schistosomiasis-associated PAH. A previous study revealed lower GLS in patients with schistosomiasis-associated PAH, regardless of ventricular septal involvement.[82] While ECHO may reveal RV and pulmonary artery dilation, reduced tricuspid annular plane systolic excursion, and diminished s' wave velocity, there is no pathognomonic sign of schistosomiasis-associated PAH.[80] Though the clinical features of schistosomiasis-associated PAH are similar to those of other causes, a considerable dilation of the pulmonary arteries on chest X-ray is more frequently observed in schistosomiasis.[47,80] The diagnosis relies on detecting parasite eggs in stool or urine specimens, while antibodies and/or antigens in blood or urine samples also indicate infection.[76] Praziquantel is the treatment of choice for all *Schistosoma* species. However, the management of schistosomiasis-associated PAH remains controversial, with limited evidence supporting substantial

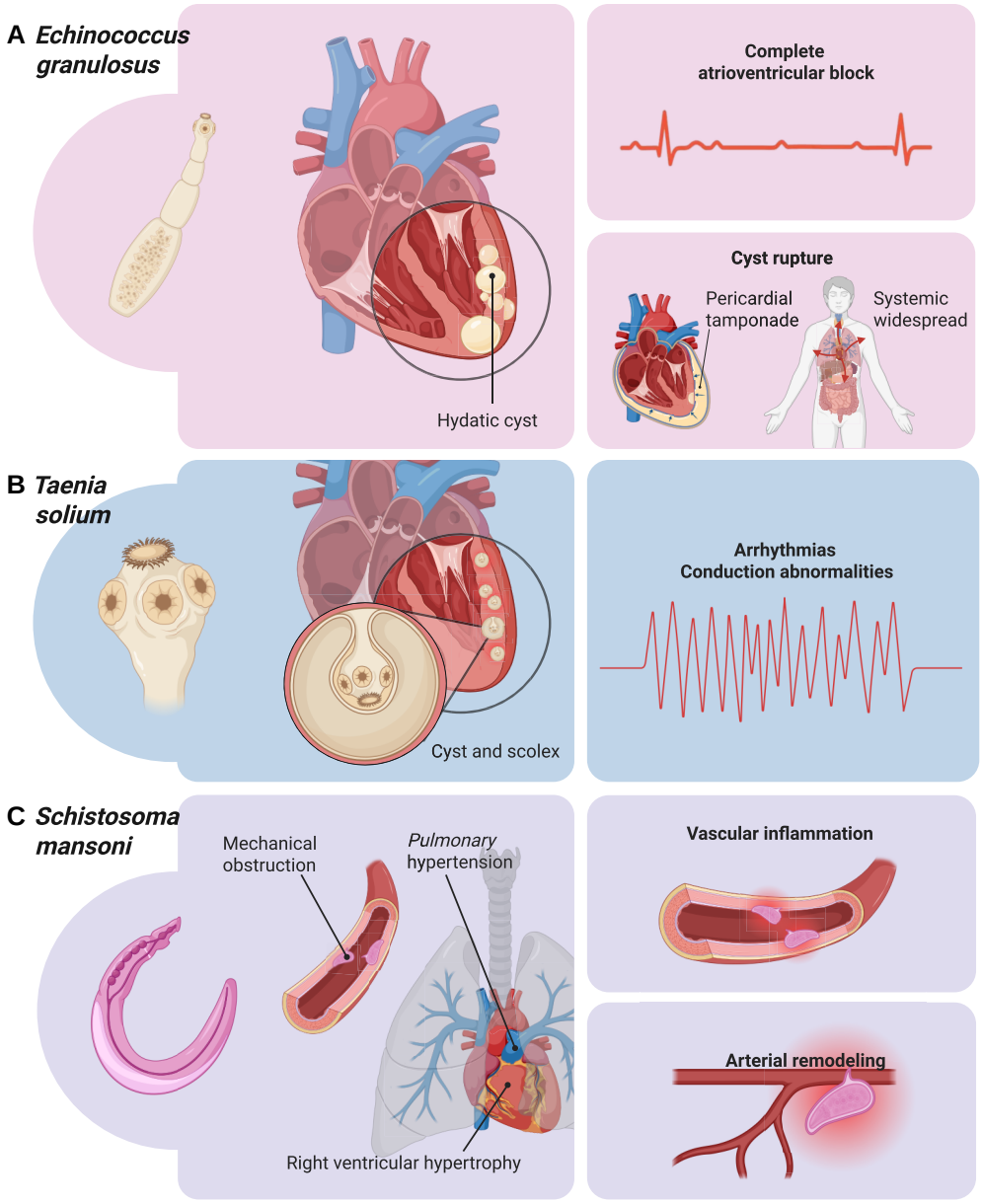


Figure 4 Cardiovascular involvement of helminthic infections. (*A*) Cardiac echinococcosis presenting with cysts located in the left ventricle. Mass compression effect can induce conduction abnormalities. Acute mechanical rupture of a cyst can precipitate pericardial tamponade and widespread dissemination. (*B*) Cysticercosis exhibiting an oval cyst within the myocardium of the left ventricle, distinguished by the presence of a characteristic scolex. Ventricular fibrosis resulting from cyst degeneration can serve as a substrate for arrhythmias. (*C*) Hepatosplenic schistosomiasis with mechanical obstruction of lung vasculature by schistosome eggs, eliciting a granulomatous response leading to inflammation and vascular remodelling. This cascade ultimately culminates in pulmonary arterial hypertension and *cor pulmonale*.

improvement following treatment with anthelminthic agents, especially in cases of advanced disease.[47,63]

Bacterial infections

Tuberculosis

Although primarily a pulmonary disease, tuberculosis can also affect the heart, often in patients with AIDS.[14] Cardiac involvement most commonly affects the pericardium (2–5% of tuberculosis patients), with changes ranging from small effusions to cardiac tamponade and constrictive pericarditis (*Table 1* and *Figure 5*; Supplementary data online, *Videos S4–S8*).[83,84] More rarely, tuberculosis can also result in myocarditis (0.14–2%) and aortitis (0.3%).[85] To diagnose tuberculosis pericarditis, integration of clinical data (chest pain, tachycardia, cough, dyspnoea, pericardial rub, night sweats, weight loss, fever), laboratory findings, chest X-ray abnormalities (cardiomegaly, pleural effusion, parenchymal lung lesion, hilar lymphadenopathy), and echocardiographic findings is essential.[81,82,86] ECHO allows the identification of pericardial effusion, cardiac tamponade, and chronic constrictive pericarditis that should be differentiated from restrictive cardiomyopathy.[17] The diagnosis of myocarditis

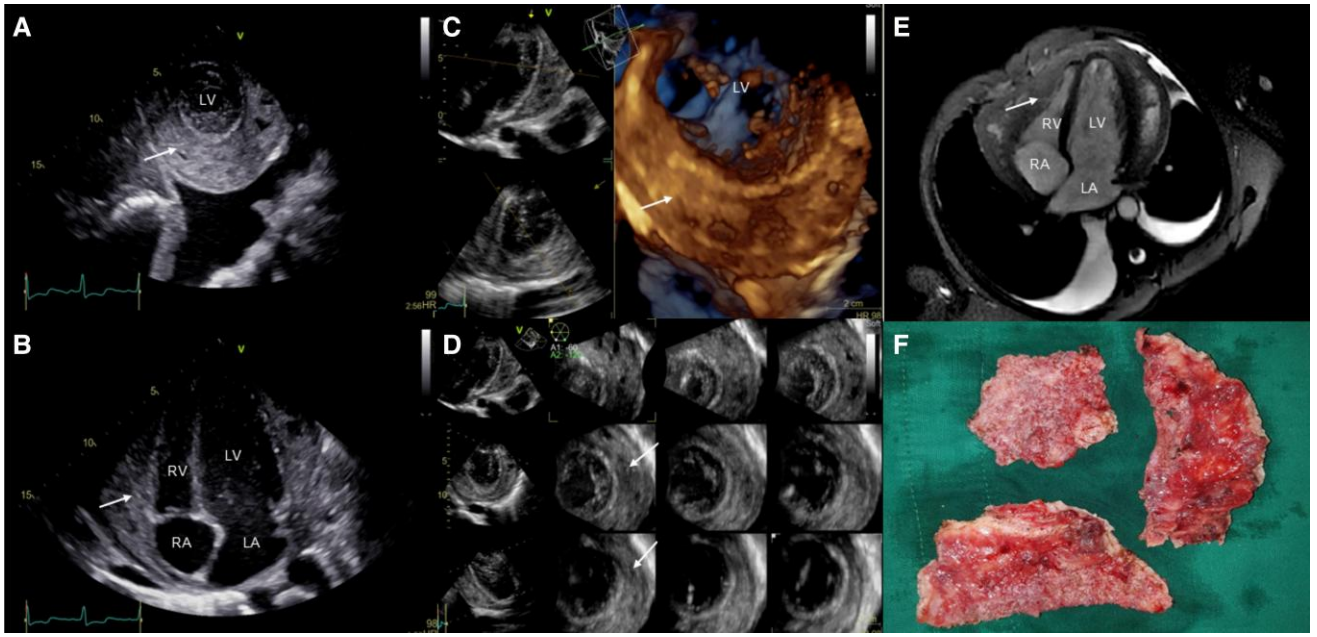


Figure 5 Multimodality cardiac imaging and macroscopic pathology findings in tuberculous pericarditis. A 29-year-old male patient had a 3-month history of fever, weight loss, and progressive dyspnoea. The clinical diagnosis was constrictive tuberculous pericarditis. A transthoracic echocardiogram shows in (*A*), from a short-axis view, a thickened pericardium (white arrow) and pleural effusion (also see parasternal longitudinal view in Supplementary data online, *Video S4*); in (*B*), a four-chamber view shows pericardial thickening (white arrow) and effusion surrounding all cardiac chambers and severe biventricular systolic dysfunction (see Supplementary data online, *Video S5*). In (*C*), 3D rendered reconstruction from a parasternal acquisition is seen (see Supplementary data online, *Videos S6* and *S7*). In (*D*), multiplanar display (slices in short axis) shows the pericardial thickening (white arrows) in multiple levels (see Supplementary data online, *Video S8*). Cardiac magnetic resonance (*E*) shows circumferential pericardial thickening, diffusely enhanced on post-contrast images (white arrow). In (*F*), the anatomical specimen shows three membranous tissue fragments with a coarsely granular aspect. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

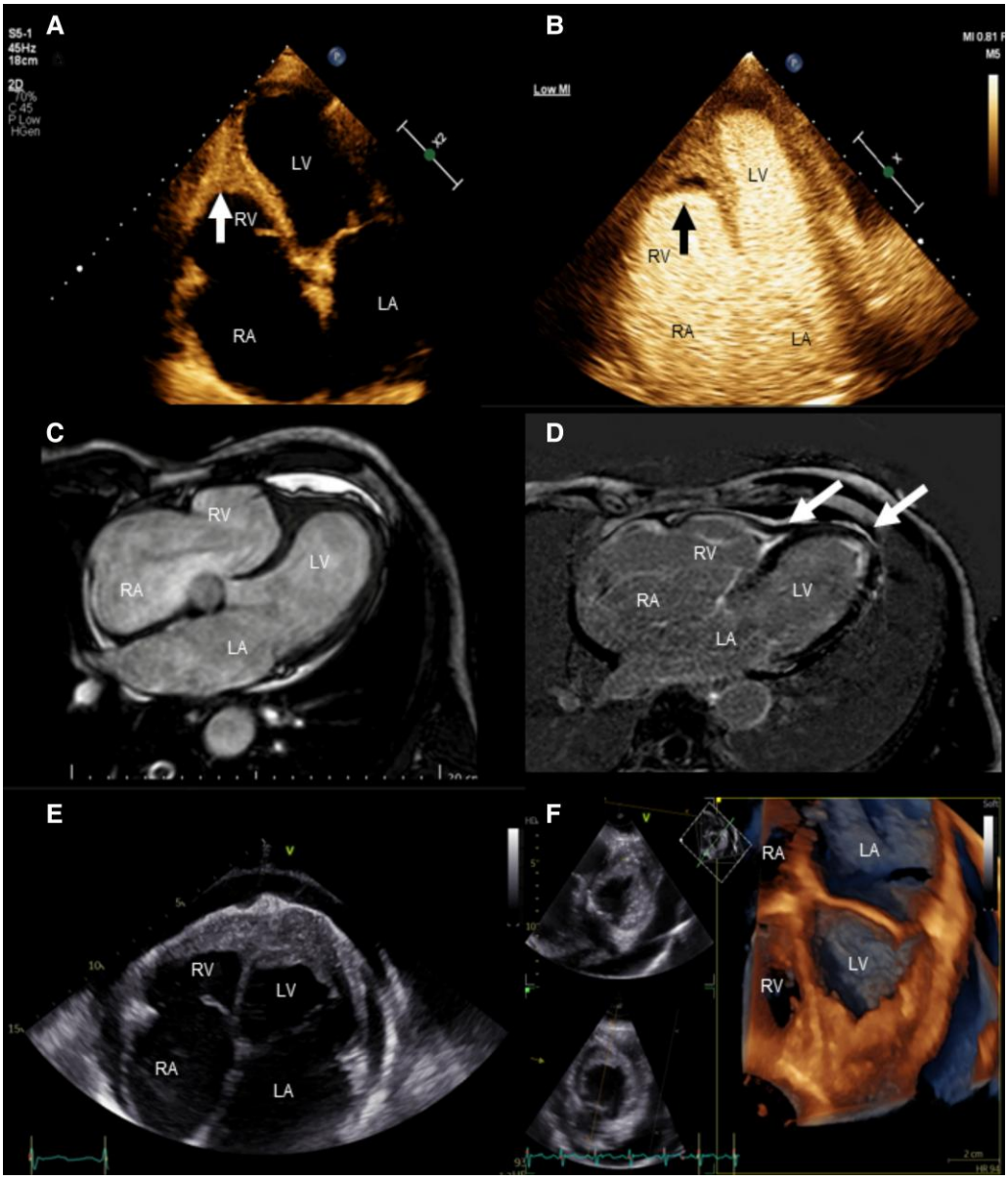


Figure 6 Multimodality imaging approach in a patient with endomyocardial fibrosis. TTE shows obliteration of the RV apex (*A*, white arrow, and Supplementary data online, *Video S9*). TTE with ultrasound enhancing agent shows RV apex subendocardial delayed perfusion associated with a perfusion defect over the RV endocardium (*B*, black arrow, and Supplementary data online, *Video S10*). CMR showing RV apical thickening and obliteration (*C* and Supplementary data online, *Video S11*) and typical 'double V' LGE compatible with subendocardial fibrosis and thrombus, besides the presence of LV LGE in its apex and apical lateral segment (*D*, double white arrows). TTE and 3D TTE images from another patient with evident obliteration of biventricular apex, large atria, and concomitant pericardial effusion (*E* and *F*). 3D, three-dimensional; CMR, cardiac magnetic resonance; LA, left atrium; LGE, late gadolinium enhancement; LV, left ventricle; RA, right atrium; RV, right ventricle; TTE, transthoracic echocardiogram. Adapted and reprinted with permission.[96]

secondary to tuberculosis requires the use of multimodality imaging. ECHO can detect regional LV wall motion abnormalities, impaired systolic function, and heterogeneous strain segmental abnormalities with a non-coronary distribution. CMR can identify pericardial thickening, effusion, and features associated with constriction, additionally enabling myocarditis diagnosis through non-ischaemic fibrosis patterns on LGE and T2 mapping.[87] CCT also allows a detailed assessment of the pericardium by identifying pericardial thickening, calcification, and effusion.[86] It can also detect aortitis based upon circumferential aortic thickening, particularly when combined with 18-fluorodeoxyglucose positron emission tomography (18F-FDG PET) imaging, which depicts increased inflammatory activity in the aorta in a similar circumferential pattern.[88] It is important to mention that 18F-FDG PET/CCT is also a valuable tool for diagnosing systemic tuberculosis and monitoring therapeutic response.[89]

EMF and HE

EMF

EMF is an idiopathic restrictive cardiomyopathy described first in Uganda in 1948.[90] The disease occurs mainly in tropical and subtropical regions of the developing world and affects around 10 million subjects. EMF is endemic in some parts of Uganda, Mozambique, West Africa, and China. In South America, it is mostly reported in Brazil and Colombia. Disease incidence has decreased in India and parts of Nigeria in recent years.[91] The pathogenesis of EMF remains unclear, with multiple contributing factors. Eosinophilia, parasitic infections (such as malaria, schistosomiasis, and filariasis), ethnicity, poverty, malnutrition, autoimmunity, and heredity are frequently implicated in EMF's etiopathogenesis.[91–93] EMF encompasses fibrosis of the ventricular subendocardium and restrictive physiology and is often associated with hypereosinophilic syndrome (HES).[94] Although regarded as distinct pathological processes, the geographic distribution and ubiquity of parasite-induced eosinophilia raise suspicions about the connection between these conditions.[93] The natural history of EMF consists of three phases, only distinctly seen in a few patients.[91–94] The first phase, the active inflammatory phase, is characterized by pancarditis and eosinophilia. The second is a transient phase of progressive thrombosis and fibrosis. The third phase corresponds to a chronic period characterized by RV or LV failure.[92] Mortality is primarily caused by progressive heart failure, pulmonary embolism, and ventricular arrhythmias.[94,95] ECHO is a fundamental component of the EMF investigation process. The most common presentation is biventricular involvement, followed by right-sided and left-sided disease.[93] The typical finding is increased endocardial thickness in the apex with obliteration (*Figure 6A*, *E*, and *D*) and consequent ventricular geometry alteration (mushroom or 'V' sign), with hypercontractility in its basal portion (Merlon's sign).[94] The RV and LV outflow tracts are usually preserved.[97] In the LV, the disease extends from the apex to the inferolateral wall. The mitral posterior leaflet motion is restricted in advanced cases, resulting in eccentric mitral regurgitation. In the RV, the disappearance of the trabecular portion is observed. Tricuspid regurgitation may occur due to the movement restriction of the anterior and septal leaflets, the involvement of the papillary muscles, and the dilatation of the tricuspid annulus. In the advanced stages of the disease, the reduction in RV cavity size is compensated by dilation of the outflow tract. Large atria, restrictive diastolic filling, and pericardial effusion can be observed (*Figure 6E*).[98] The hallmark of advanced disease is ventricular endocardial fibrosis with organized thrombus.[97] Ultrasound contrast-enhancing agents often add information about the extent of myocardial involvement, showing a perfusion defect and the presence of a possible thrombus (*Figure 6B*).[96]

EMF diagnosis criteria were described using ECHO in a Mozambican population.[97] Patients' cardiac abnormalities were categorized as 'major' or 'minor' based on their clinical relevance, uniqueness to the diagnosis, impact on management decisions, and prognostic significance (*Table 2*).[97] A multimodality approach provides complementary information.[95] CMR with LGE imaging is advised after the initial ECHO evaluation, as it is currently the gold standard for imaging this disease.[100] CMR is crucial not only for early and differential diagnosis but also for providing essential information on ventricular morphology and the presence of thrombus or calcifications.[97,98,100] It is also essential for prognosticating EMF by quantifying the endocardial fibrous tissue deposition pattern.[100] The typical LGE pattern in EMF has been described as subendocardial, primarily affecting the apex and eventually extending to the inflow tract.[98] The ventricular apex displays a double V sign, characterized by a three-layered appearance composed of normal myocardium, thickened enhanced endomyocardium, and an overlying thrombus, with or without calcifications (*Figure 6C* and *D*).[96,101] CCT can also accurately depict the morphologic and functional aspects of EMF. This technique can differentiate thrombus from calcification, assisting in clinical and surgical decisions for EMF patients.[102] The differential diagnosis of EMF includes rheumatic fever (in endemic countries), Loeffler's endocarditis, Churg–Strauss syndrome, Ebstein's anomaly, and apical hypertrophic cardiomyopathy.[94]

Table 2 Criteria for diagnosis and assessment of the severity of endomyocardial fibrosis[99]

Criterion[a]	Score
Major criteria	
Endomyocardial plaques > 2 mm in thickness	2
Thin (≤1 mm) endomyocardial patches affecting more than one ventricular wall	3
Obliteration of the right ventricular or left ventricular apex	4
Thrombi or spontaneous contrast without severe ventricular dysfunction	4
Retraction of the right ventricular apex (right ventricular apical notch)	4
Atrioventricular-valve dysfunction due to adhesion of the valvular apparatus to the ventricular wall[b]	1–4
Minor criteria	
Thin endomyocardial patches localized to one ventricular wall	1
Restrictive flow pattern across mitral or tricuspid valves	2
Pulmonary valve diastolic opening	2
Diffuse thickening of the anterior mitral leaflet	1
Enlarged atrium with normal-sized ventricle	2
M-movement of the interventricular septum and flat posterior wall[c]	1
Enhanced density of the moderator or other intraventricular bands	1

A total score of less than 8 indicates mild endomyocardial fibrosis, 8–15 moderate disease, and more than 15 severe disease.
[a]A definite diagnosis of endomyocardial fibrosis was made in the presence of two major criteria or one major criterion associated with two minor criteria.
[b]The score is assigned according to the severity of atrioventricular regurgitation.
[c]M-movement of the interventricular septum refers to a pattern of movement observed on M-mode echocardiography that is thought to be due to obliteration or restriction of the left ventricular apex combined with mitral regurgitation.[99]

HE

HE, also known as Loeffler's endocarditis, is a rare form of restrictive cardiomyopathy resulting from eosinophilic infiltration of the myocardium due to hypersensitivity, helminthic and parasitic infestation, systemic disease, myeloproliferative syndrome, and idiopathic HES.[103] Idiopathic HES is a rare but serious condition characterized by eosinophilia (>1500 cells/mL, persisting for at least 6 months) and signs of organ involvement by eosinophilic infiltration, in the absence of the other causes of eosinophilia.[103,104]

HE encompasses three stages: (i) the necrotic stage, characterized by acute myocardial inflammation, eosinophil infiltration, and necrosis, mediated by eosinophil-released toxic proteins; (ii) the thrombotic stage, with mural thrombosis usually involving the ventricles, but also the atria and atrioventricular valves; and (iii) the fibrotic stage, characterized by EMF, leading to restrictive cardiomyopathy in the final disease stages.[104,105] Although the diagnosis may be challenging (endomyocardial biopsy still represents the gold standard for

Figure 7 Multimodality imaging approach in a patient with hypereosinophilic endocarditis. Thickening of the mitral valve with restricted posterior leaflet motion and a large coaptation defect is noted on apical two-chamber echocardiographic view (*A*). A severe regurgitant mitral regurgitation jet is seen on colour Doppler in the apical four-chamber view (*B*). Subendocardial hypoperfusion of the mid-apical left ventricle (*C*, black arrow) is noted on CCT, along with tethering of the papillary muscles following endocardial fibrosis (*D*). CMR shows increased apical thickness on four-chamber cine sequence (*E*); subendocardial oedema, by means of increased signal intensity, is noted on T2-weighted images (*F*, yellow arrows) with corresponding subendocardial LGE (*G*, white arrows); a stratified apical thrombus is noted as avascular structure on post-contrast four-chamber view (*G*, black arrow). Increased apical thickness is confirmed on cine CMR in the three-chamber view (*H*). CCT, cardiac computed tomography; CMR, cardiac magnetic resonance; LGE, late gadolinium enhancement.

Table 3 Barriers to cardiovascular imaging implementation in the evaluation of tropical diseases and potential strategic solutions

Category	Barriers to implementation	Strategic solutions
Infrastructure and access	Restricted access to advanced imaging technologies in low-income regions	Portable and/or cost-effective tools like cardiac POCUS and rapid CMR protocols Tele-echocardiography programmes
Human resources	Insufficient training of healthcare providers in low-income regions	Provide training programmes to build local expertise Introduce simplified echocardiographic protocols tailored for resource-limited areas Leverage telemedicine and international collaboration to bridge expertise gaps
Knowledge gaps	Lack of awareness and recognition of cardiovascular complications	Raise awareness through scientific documents from cardiovascular and infectious disease societies, education campaigns, and professional training
	Limited research on the pathophysiology of TDs and cardiac imaging findings	Conduct longitudinal studies to assess the predictive value of early imaging findings for long-term outcomes
	Lack of guidelines on when and how to use cardiovascular imaging	Increase multicentre research efforts to develop evidence-based recommendations for cardiovascular imaging in TDs
	Underexplored roles of imaging in treatment monitoring	Encourage studies evaluating the role of imaging in guiding therapeutic decisions and monitoring response to treatment
	Undetected subclinical involvement	Promote early screening in endemic regions to identify asymptomatic cardiac disease
Technology development	Difficulty in automating and streamlining imaging for underserved areas	Integrate artificial intelligence to automate image analysis and provide real-time decision support for cardiac POCUS
Global collaboration	Fragmented efforts in addressing cardiovascular imaging needs in TDs	Align with global health initiatives, such as WHO's road map for TDs, to prioritize funding and innovation for imaging technologies
	Lack of resource sharing between high-income and low-income regions	Strengthen international partnerships to promote resource allocation and share best practices

diagnosis), multimodality imaging plays a pivotal diagnostic role.[100,105] Although there is no pathognomonic sign, ECHO may show biventricular endocardial thickening, most commonly at the apical level, with apical thrombosis, typically without ventricular wall motion abnormalities. Ultrasound contrast-enhancing agents increase the sensitivity of thrombosis detection and help differentiate apical thrombosis from apical hypertrophic cardiomyopathy, which can act as a mimic. Atrio-ventricular valves (aortic and pulmonary valves are rarely involved) may appear thickened (*Figure 7A*), because of fibrosis and/or thrombosis, along with the sub-valvular apparatus; mitral regurgitation, secondary to valve tethering by fibrotic endocardial thickening generally of the posterolateral wall (*Figure 7A* and *B*), is common in advanced disease stages.[103,105] Pericardial effusion can be found in 10–32% of HES patients.[105] ECHO is frequently normal in early disease stages but may be used to monitor treatment response on the reversal of endomyocardial infiltration.[100] CMR offers higher sensitivity and specificity, in all disease stages, even the early one, based on its ability to provide accurate myocardial tissue characterization (*Figure 7E–H*). Subendocardial oedema, as a sign of inflammation noted in early, often asymptomatic, disease stages, can be detected on T2-weighted sequences (*Figure 7F*, yellow arrows). On post-contrast sequences, subendocardial LGE usually involves multiple LV walls (involving both ventricles in up to one-third of patients)[105] extending beyond coronary artery distribution (*Figure 7G*, white arrow). Apical thrombi are more accurately detected on CMR as avascular structures not gaining contrast on post-contrast sequences (*Figure 7G*, black arrow); CMR also helps differentiate apical thrombosis from apical hypertrophic cardiomyopathy.[100,105,106] CCT is indicated when CMR is non-diagnostic or contraindicated and is useful to rule out epicardial coronary stenosis, needed for the differential diagnosis of subendocardial damage (*Figure 7C* and *D*). CCT can detect the presence of thrombosis by showing a morphologically normal ventricle underneath it, and in late disease stages, it is helpful to distinguish restrictive cardiomyopathy from constrictive pericarditis. Further research studies are needed to explore the role of 18F-FDG PET in detecting early disease stages by visualization of active inflammation.[105]

Challenges in the implementation of cardiovascular imaging for TDs

Implementing cardiovascular imaging to evaluate TDs faces significant barriers, particularly in the regions where it is most needed. Lack of awareness of cardiovascular complications, restricted access to advanced technology, and insufficiently trained personnel in low-resource areas are the most prominent challenges that must be dealt with. Addressing these issues requires practical solutions tailored to resource-limited settings. Initiatives to expand the diagnostic capacity and appropriate interventions in healthcare education are vital to improving outcomes. Portable cardiac point-of-care ultrasound (POCUS) shows substantial potential for managing TDs in remote areas with minimal infrastructure due to its portability, affordability, and diagnostic accuracy. Tele-echocardiography may expand access, allowing remote image interpretation and consultation with specialists, thus reducing diagnostic delays.[107] Additionally, emerging technologies like artificial intelligence can enhance POCUS by automating image analysis and providing real-time clinical decision support, improving accessibility and impact.[108] Although ECHO is the first-line diagnostic test when suspecting a wide range of heart diseases,[14] it may not be sufficient in isolation, depending on the clinical scenario. A successful initiative aimed at bringing advanced technology to underserved areas is the development of rapid CMR scanning protocols.[109] These protocols have effectively reduced costs and scan times while maintaining

diagnostic accuracy, making CMR more accessible in low- and middle-income countries. Significant gaps in knowledge also hinder the effective use of imaging in TDs. Few studies have focused on the cardiovascular complications of TDs, resulting in a limited understanding of the connection between pathophysiology and imaging findings. Current data are often limited to small cohorts or case-based studies. There is a lack of longitudinal research linking early imaging alterations with long-term outcomes. Additionally, the roles of imaging in monitoring disease progression or treatment efficacy are underexplored. Finally, subclinical cardiac involvement often goes undiagnosed, and late-stage cardiovascular complications make timely intervention difficult. Filling these gaps is essential to improve patient outcomes in TDs. *Table 3* outlines actionable steps to address these challenges and enhance cardiovascular care in TDs, with focus on accessibility, education, research, and global cooperation.

Conclusions

TDs are common and can seriously affect cardiovascular health. This scientific statement, a joint effort between the EACVI and the DICSBC, aims to provide a concise overview of important clinical knowledge for the various viral, protozoan, helminthic, and bacterial conditions, but notably summarizes the opportunities and value of cardiovascular multimodality in some clinical scenarios. It is important to join efforts not only in the education of physicians treating and imaging patients with TDs but also in an increased research effort to better understand the role of cardiac imaging in diagnosis, treatment, and treatment monitoring.

Supplementary data

Supplementary data are available at *European Heart Journal - Cardiovascular Imaging* online.

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Data availability

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References

1. World Health Organization. Neglected tropical diseases. https://www.who.int/health-topics/neglected-tropical-diseases#tab=tab_1. (September 2024, date last accessed)
2. World Economic Forum. Will climate change affect the spread of tropical diseases? https://www.weforum.org/stories/2015/02/will-climate-change-affect-the-spread-of-tropical-diseases/. (November 2024, date last accessed)
3. Tidman R, Abela-Ridder B, de Castañeda RR. The impact of climate change on neglected tropical diseases: a systematic review. *Trans R Soc Trop Med Hyg* 2021;**115**:147–68.
4. WHO Team Control of Neglected Diseases. Ending the neglect to attain the Sustainable Development Goals: A road map for neglected tropical diseases 2021–2030. https://www.who.int/publications/i/item/9789240010352.
5. Burgos LM, Farina J, Liendro MC, Saldarriaga C, Liprandi AS, Wyss F *et al.* Neglected tropical diseases and other infectious diseases affecting the heart. The NET-heart project: rationale and design. *Glob Heart* 2020;**15**:60.
6. Miranda-Arboleda AF, Zaidel EJ, Marcus R, Pinazo MJ, Echeverría LE, Saldarriaga C *et al.* Roadblocks in Chagas disease care in endemic and nonendemic countries: Argentina, Colombia, Spain, and the United States. *The NET-Heart project. PLoS Negl Trop Dis* 2021;**15**:e0009954.
7. Farina JM, Liblik K, Iomini P, Miranda-Arboleda AF, Saldarriaga C, Mendoza I *et al.* Infections and cardiovascular disease: JACC focus seminar 1/4. *J Am Coll Cardiol* 2023;**81**:71–80.
8. Araiza-Garaygordobil D, García-Martínez CE, Burgos LM, Saldarriaga C, Liblik K, Mendoza I *et al.* Dengue and the heart. *Cardiovasc J Afr* 2021;**32**:276–83.
9. Pandian NG, Kim JK, Arias-Godinez JA, Marx GR, Michelena HI, Chander Mohan J *et al.* Recommendations for the use of echocardiography in the evaluation of rheumatic heart disease: a report from the American Society of Echocardiography. *J Am Soc Echocardiogr* 2023;**36**:3–28.
10. Rwebembera J, Marangou J, Mwita JC, Mocumbi AO, Mota C, Okello E *et al.* 2023 World Heart Federation guidelines for the echocardiographic diagnosis of rheumatic heart disease. *Nat Rev Cardiol* 2024;**21**:250–63.
11. Acquatella H, Asch FM, Barbosa MM, Barros M, Bern C, Cavalcante JL *et al.* Recommendations for multimodality cardiac imaging in patients with Chagas disease: a report from the American Society of Echocardiography in Collaboration With the InterAmerican Association of Echocardiography (ECOSIAC) and the Cardiovascular Imaging Department of the Brazilian Society of Cardiology (DIC-SBC). *J Am Soc Echocardiogr* 2018;**31**:3–25.
12. Ralston K, Zaidel E, Acquatella H, Barbosa MM, Narula J, Nakagama Y *et al.* WHF recommendations for the use of echocardiography in Chagas disease. *Glob Heart* 2023;**18**:27.
13. Liblik K, Florica IT, Baranchuk A. Original algorithms for the detection of cardiovascular involvement of neglected tropical diseases. *Expert Rev Cardiovasc Ther* 2024;**22**:59–74.
14. Barberato SH, Romano MMD, Beck ALS, Rodrigues ACT, Almeida ALC, Assunção BMBL *et al.* Position statement on indications of echocardiography in adults—2019. *Arq Bras Cardiol* 2019;**113**:135–81.
15. Steeds RP, Garbi M, Cardim N, Kasprzak JD, Sade E, Nihoyannopoulos P *et al.* EACVI appropriateness criteria for the use of transthoracic echocardiography in adults: a report of literature and current practice review. *Eur Heart J Cardiovasc Imaging* 2017;**18**:1191–204.
16. Caforio AL, Pankuweit S, Arbustini E, Basso C, Gimeno-Blanes J, Felix SB *et al.* Current state of knowledge on aetiology, diagnosis, management, and therapy of myocarditis: a position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. *Eur Heart J* 2013;**34**:2636–48.
17. Adler Y, Charron P, Imazio M, Badano L, Barón-Esquivias G, Bogaert J *et al.* 2015 ESC guidelines for the diagnosis and management of pericardial diseases: the task force for the diagnosis and management of pericardial diseases of the European Society of Cardiology (ESC)Endorsed by: the European Association for Cardio-Thoracic Surgery (EACTS). *Eur Heart J* 2015;**36**:2921–64.
18. Ferreira VM, Schulz-Menger J, Holmvang G, Kramer CM, Carbone I, Sechtem U *et al.* Cardiovascular magnetic resonance in nonischemic myocardial inflammation: expert recommendations. *J Am Coll Cardiol* 2018;**72**:3158–76.
19. Kotanidis CP, Bazmpani MA, Haidich AB, Karvounis C, Antoniades C, Karamitsos TD. Diagnostic accuracy of cardiovascular magnetic resonance in acute myocarditis: a systematic review and meta-analysis. *JACC Cardiovasc Imaging* 2018;**11**:1583–90.
20. Bhatt S, Gething PW, Brady OJ, Messina JP, Farlow AW, Moyes CL *et al.* The global distribution and burden of dengue. *Nature* 2013;**496**:504–7.
21. World Health Organization. Global arbovirus initiative. https://www.who.int/initiatives/global-arbovirus-initiative (September 2024, date last accessed)
22. Hsu KH, Chi NH, Yu HY, Wang CH, Huang SC, Wang SS *et al.* Extracorporeal membranous oxygenation support for acute fulminant myocarditis: analysis of a single center's experience. *Eur J Cardiothorac Surg* 2011;**40**:682–8.
23. Miranda-Arboleda AF, González-Barrera LG, Liblik K, Farina J, Zaidel EJ, Saldarriaga C *et al.* Neglected tropical diseases and sudden cardiac death: the NET-heart project. *Rev Cardiovasc Med* 2022;**23**:254.
24. Guzman MG, Halstead SB, Artsob H, Buchy P, Farrar J, Gubler DJ *et al.* Dengue: a continuing global threat. *Nat Rev Microbiol* 2010;**8**(12 Suppl):S7–16.
25. Barniol J, Gaczkowski R, Barbato EV, da Cunha RV, Salgado D, Martínez E *et al.* Usefulness and applicability of the revised dengue case classification by disease: multicentre study in 18 countries. *BMC Infect Dis* 2011;**11**:106.
26. Sam SS, Omar SF, Teoh BT, Abd-Jamil J, AbuBakar S. Review of dengue hemorrhagic fever fatal cases seen among adults: a retrospective study. *PLoS Negl Trop Dis* 2013;**7**:e2194.

27. Corré J, Vally S, Boiron P, Bouillaud Y, Travers JY. Spontaneous coronary hematoma concomitant with myocarditis: the first report of double cardiac complication of dengue. *ESC Heart Fail* 2023;**10**:1445–8.
28. Nerella S, Sarkar UK, Namdeo H. Electrocardiographic and echocardiographic findings in children with dengue infection. *J Family Med Prim Care* 2022;**11**:2334–9.
29. Kaagaard MD, Wegener A, Gomes LC, Holm AE, Lima KO, Matos LO *et al.* Potential role of transthoracic echocardiography for screening LV systolic dysfunction in patients with a history of dengue infection. A cross-sectional and cohort study and review of the literature. *PLoS One* 2022;**17**:e0276725.
30. Shah C, Vijayaraghavan G, Kartha CC. Spectrum of cardiac involvement in patients with dengue fever. *Int J Cardiol* 2021;**324**:180–5.
31. Baqi A, Ur Rehman F, Memon PS, Omair SF. Prevalence and outcomes of myocarditis in dengue-infected patients admitted to a tertiary care hospital of low-middle income country. *Glob Heart* 2022;**17**:44.
32. Yacoub S, Wertheim H, Simmons CP, Screaton G, Wills B. Cardiovascular manifestations of the emerging dengue pandemic. *Nat Rev Cardiol* 2014;**11**:335–45.
33. Gupta S, Gupta M, Kashyap JR, Arora SK. Early cardiovascular involvement in dengue fever: a prospective study with two-dimensional speckle tracking echocardiography. *Trop Doct* 2022;**52**:285–92.
34. Yacoub S, Griffiths A, Chau TT, Simmons CP, Wills B, Hien TT *et al.* Cardiac function in Vietnamese patients with different dengue severity grades. *Crit Care Med* 2012;**40**:477–83.
35. Kirawittaya T, Yoon IK, Wichit S, Green S, Ennis FA, Gibbons RV *et al.* Evaluation of cardiac involvement in children with dengue by serial echocardiographic studies. *PLoS Negl Trop Dis* 2015;**9**:e0003943.
36. Duffy MR, Chen TH, Hancock WT, Powers AM, Kool JL, Lanciotti RS *et al.* Zika virus outbreak on Yap Island, Federated States of Micronesia. *N Engl J Med* 2009;**360**:2536–43.
37. Puccioni-Sohler M, Nascimento Soares C, Christo PP, Almeida SM. Review of dengue, zika and chikungunya infections in nervous system in endemic areas. *Arq Neuropsiquiatr* 2023;**81**:1112–24.
38. Scatularo CE, Ballesteros OA, Saldarriaga C, Mendoza I, Wyss F, Liprandi AS *et al.* Zika & heart: a systematic review. *Trends Cardiovasc Med* 2022;**32**:52–8.
39. Cavalcanti DD, Alves LV, Furtado GJ, Santos CC, Feitosa FG, Ribeiro MC *et al.* Echocardiographic findings in infants with presumed congenital Zika syndrome: retrospective case series study. *PLoS One* 2017;**12**:e0175065.
40. Orofino DHG, Passos SRL, de Oliveira RVC, Farias CVB, Leite M, Pone SM *et al.* Cardiac findings in infants with in utero exposure to Zika virus- a cross sectional study. *PLoS Negl Trop Dis* 2018;**12**:e0006362.
41. Barbosa ICQ, de Paula Gomes L, de Almeida Feitosa IN, Botelho LFB, Barbosa BRC, Barbosa A *et al.* Morphological and functional cardiac alterations in children with congenital Zika syndrome and severe neurological deficits. *PLoS Negl Trop Dis* 2023;**17**:e0011762.
42. Cotella JI, Sauce AL, Saldarriaga CI, Perez GE, Farina JM, Wyss F *et al.* Chikungunya and the heart. *Cardiology* 2021;**146**:324–34.
43. Traverse EM, Hopkins HK, Vaidhyanathan V, Barr KL. Cardiomyopathy and death following chikungunya infection: an increasingly common outcome. *Trop Med Infect Dis* 2021;**6**:108.
44. European Center for Disease Prevention and Control. Facts about yellow fever. https://www.ecdc.europa.eu/en/yellow-fever/facts. (September 2024, date last accessed)
45. Giugni FR, Aiello VD, Faria CS, Pour SZ, Cunha MDP, Giugni MV *et al.* Understanding yellow fever-associated myocardial injury: an autopsy study. *EBioMedicine* 2023;**96**:104810.
46. Paixão GMM, Nunes MCP, Beato B, Sable C, Beaton AZ, Oliveira KKB *et al.* Cardiac involvement by yellow fever(from the PROVAR+ study). *Am J Cardiol* 2019;**123**:833–8.
47. Nunes MC, Guimarães Júnior MH, Diamantino AC, Gelape CL, Ferrari TC. Cardiac manifestations of parasitic diseases. *Heart* 2017;**103**:651–8.
48. World Health Organization. Malaria. https://www.who.int/news-room/fact-sheets/detail/malaria (September 2024, date last accessed)
49. Gupta S, Gazendam N, Farina JM, Saldarriaga C, Mendoza I, López-Santi R *et al.* Malaria and the heart: JACC state-of-the-art review. *J Am Coll Cardiol* 2021;**77**:1110–21.
50. Wooldridge G, Nandi D, Chimalizeni Y, O'Brien N. Cardiovascular findings in severe malaria: a review. *Glob Heart* 2020;**15**:75.
51. Wegener A, Holm AE, Gomes LC, Lima KO, Matos LO, Vieira IVM *et al.* Prevalence and dynamic changes in lung ultrasound findings among adults with uncomplicated malaria and controls in the Amazon basin, Brazil. *Am J Trop Med Hyg* 2022;**106**:1637–45.
52. Holm AE, Gomes LC, Marinho CRF, Silvestre OM, Vestergaard LS, Biering-Sørensen T *et al.* Prevalence of cardiovascular complications in malaria: a systematic review and meta-analysis. *Am J Trop Med Hyg* 2021;**104**:1643–50.
53. Brainin P, Gomes LC, Holm AE, Matos LO, Wegener A, Lima KO *et al.* Left ventricular function by strain in uncomplicated malaria: a prospective study from the Brazilian Amazon. *Int J Cardiovasc Imaging* 2023;**39**:595–606.
54. Matsuo T, Saito A, Kawai F, Ishikawa K, Hasegawa R, Suzuki T *et al.* Use of PCR in the diagnosis of pericardial amebiasis: a case report and systematic review of the literature. *BMC Infect Dis* 2021;**21**:960.
55. Keleş Alp E, Alp H, Keçeli M. Myocarditis associated with enteric amebiasis in an adolescent. *World J Pediatr Congenit Heart Surg* 2020;**11**:658–60.
56. World Health Organization. Leishmaniasis. https://www.who.int/news-room/fact-sheets/detail/leishmaniasis (September 2024, date last accessed)
57. Shrivastava R, Sinha PR, Singh VP, Sundar S. Echocardiographic evaluation of cardiac status in Indian visceral leishmaniasis patients. *Trans R Soc Trop Med Hyg* 2007;**101**:429–32.
58. Farina JM, García-Martínez CE, Saldarriaga C, Pérez GE, Melo MB, Wyss F *et al.* Leishmaniasis and heart. *Arch Cardiol Mex* 2022;**92**:85–93.
59. Franco-Paredes C, Rouphael N, Méndez J, Folch E, Rodríguez-Morales AJ, Santos JI *et al.* Cardiac manifestations of parasitic infections. Part 2: parasitic myocardial disease. *Clin Cardiol* 2007;**30**:218–22.
60. Zhou Z, Ortiz Lopez HIA, Pérez GE, Burgos LM, Farina JM, Saldarriaga C *et al.* Toxoplasmosis and the heart. *Curr Probl Cardiol* 2021;**46**:100741.
61. Fabiani S, Fortunato S, Bruschi F. Solid organ transplant and parasitic diseases: a review of the clinical cases in the last two decades. *Pathogens* 2018;**7**:65.
62. Kahlfuß S, Flieger RR, Roepke TK, Yilmaz K. Diagnosis and treatment of cardiac echinococcosis. *Heart* 2016;**102**:1348–53.
63. Mishra A, Ete T, Fanai V, Malviya A. A review on cardiac manifestation of parasitic infection. *Trop Parasitol* 2023;**13**:8–15.
64. Mir H, McClure A, Thampinathan B, Chow C, Cusimano RJ, Bogoch II *et al.* Echocardiographic features of cardiac echinococcal infection. *CASE (Phila)* 2021;**5**:26–32.
65. Berarducci J, Armenta-Moreno JI, Garcia-Cardenas AM, Armendáriz-Ferrari JC, Espinola-Zavaleta N. Cardiac echinococcosis: a multimodality approach. *Eur Heart J Case Rep* 2021;**5**:ytab475.
66. Moraes RP, Brida MS, Reis RM, Silva RS, Farias CB. Cardiac hydatid cyst: an uncommon cause of complete atrioventricular block. *Arq Bras Cardiol* 2023;**120**:e20220597.
67. Ahmad Y, Maree G, Omar S, Alkhalaf H. A rare location of cardio hydatid cyst with multiple organ involvement: a case report. *Ann Med Surg (Lond)* 2023;**85**:1220–2.
68. Wedin JO, Astudillo RM, Kurland S, Grinnemo KH, Astudillo R, Vikholm P *et al.* A rare case of cardiac echinococcosis: the role of multimodality imaging. *CASE (Phila)* 2021;**5**:230–4.
69. Alur N, Kikkeri MS, Marimuthu V, Khamitkar Shankar Rao S, Backer PH. Cardiac echinococcosis associated with severe biventricular dysfunction: diagnosis by multimodality imaging. *Eur Heart J Cardiovasc Imaging* 2022;**23**:e268.
70. Jahanshahi F, Parsaei AH, Naderi D, Davani SZN. Primary isolated extraluminal hydatid cyst of left pulmonary artery. *Int J Surg Case Rep* 2023;**106**:108211.
71. Yılmaz R, Akpınar YE, Bayramoglu Z, Ozyavuz HI, Unal OF, Dursun M. Magnetic resonance imaging characteristics of cardiac hydatid cyst. *Clin Imaging* 2018;**51**:202–8.
72. García-Martínez CE, Scatularo CE, Farina JM, Saldarriaga C, Pérez GE, Wyss F *et al.* Cysticercosis & heart: a systematic review. *Curr Probl Cardiol* 2023;**48**:101195.
73. Littlewood S. A case report of cardiac cysticercosis in a returning traveller: a rare cause of myocarditis. *Eur Heart J Case Rep* 2022;**6**:ytac208.
74. Beyersdorf F, Rylski B. Cysticercosis-rare but important. *J Thorac Cardiovasc Surg* 2018;**155**:e159.
75. Schuuring MJ, van der Bom T, Konings TC, Götte MJW, Koolbergen DR, Boekholdt SM *et al.* Multimodality evaluation of a septal cystic cavity and ventricular septal defect in the setting of neurocysticercosis and endocarditis. *Circ Cardiovasc Imaging* 2021;**14**:e011688.
76. Posada-Martínez EL, Gonzalez-Barrera LG, Liblik K, Gomez-Mesa JE, Saldarriaga C, Farina JM *et al.* Schistosomiasis & heart—on behalf of the Neglected Tropical Diseases and other Infectious Diseases affecting the Heart (the NET-Heart Project). *Arq Bras Cardiol* 2022;**118**:885–93.
77. World Health Organization. Schistosomiasis. https://www.who.int/news-room/fact-sheets/detail/schistosomiasis (September 2024, date last accessed)
78. Meltzer E, Schwartz E. Schistosomiasis: current epidemiology and management in travelers. *Curr Infect Dis Rep* 2013;**15**:211–5.
79. Lapa M, Dias B, Jardim C, Fernandes CJ, Dourado PM, Figueiredo M *et al.* Cardiopulmonary manifestations of hepatosplenic schistosomiasis. *Circulation* 2009;**119**:1518–23.
80. Gavilanes F, Fernandes CJ, Souza R. Pulmonary arterial hypertension in schistosomiasis. *Curr Opin Pulm Med* 2016;**22**:408–14.
81. Carvalho VT, Barbosa MM, Nunes MC, Cardoso YS, de Sá Filho IM, Oliveira FR *et al.* Early right cardiac dysfunction in patients with schistosomiasis mansoni. *Echocardiography* 2011;**28**:261–7.
82. de Amorim Corrêa R, de Oliveira FB, Barbosa MM, Barbosa JA, Carvalho TS, Barreto MC *et al.* Left ventricular function in patients with pulmonary arterial hypertension: the role of two-dimensional speckle tracking strain. *Echocardiography* 2016;**33**:1326–34.
83. Mayosi BM, Burgess LJ, Doubell AF. Tuberculous pericarditis. *Circulation* 2005;**112**:3608–16.

84. Harding E. WHO global progress report on tuberculosis elimination. *Lancet Respir Med* 2020;**8**:19.
85. López-López JP, Posada-Martínez EL, Saldarriaga C, Wyss F, Ponte-Negretti CI, Alexander B *et al.* Tuberculosis and the heart. *J Am Heart Assoc* 2021;**10**:e019435.
86. Klein AL, Abbara S, Agler DA, Appleton CP, Asher CR, Hoit B *et al.* American Society of Echocardiography clinical recommendations for multimodality cardiovascular imaging of patients with pericardial disease: endorsed by the Society for Cardiovascular Magnetic Resonance and Society of Cardiovascular Computed Tomography. *J Am Soc Echocardiogr* 2013;**26**:965–1012.e15.
87. Das KM, Mansoori TA, Alattar YH, Gorkom KV, Shamisi A, Melethil AP *et al.* Tuberculosis of the heart: a diagnostic challenge. *Tomography* 2022;**8**:1649–65.
88. Mazzolai L, Teixido-Tura G, Lanzi S, Boc V, Bossone E, Brodmann M *et al.* 2024 ESC guidelines for the management of peripheral arterial and aortic diseases. *Eur Heart J* 2024;**45**:3538–700
89. Yu WY, Lu PX, Assadi M, Huang XL, Skrahin A, Rosenthal A *et al.* Updates on (18)F-FDG-PET/CT as a clinical tool for tuberculosis evaluation and therapeutic monitoring. *Quant Imaging Med Surg* 2019;**9**:1132–46.
90. Davies JN. Pathology of Central African natives. *East Afr Med J* 1948;**25**:117–22.
91. Sliwa K, Viljoen CA, Hasan B, Ntusi NAB. Nutritional heart disease and cardiomyopathies: JACC focus seminar 4/4. *J Am Coll Cardiol* 2022;**81**:187–202.
92. Duraes AR, de Souza Lima Bitar Y, Roever L, Neto MG. Endomyocardial fibrosis: past, present, and future. *Heart Fail Rev* 2020;**25**:725–30.
93. Grimaldi A, Mocumbi AO, Freers J, Lachaud M, Mirabel M, Ferreira B *et al.* Tropical endomyocardial fibrosis: natural history, challenges, and perspectives. *Circulation* 2016;**133**:2503–15.
94. Scatularo CE, Posada Martínez EL, Saldarriaga C, Ballesteros OA, Baranchuk A, Sosa Liprandi A *et al.* Endomyocardiofibrosis: a systematic review. *Curr Probl Cardiol* 2021; **46**:100784.
95. Scatularo CE, Posada Martínez EL, Alba AC, Renedo MF, N Llober M, Elfman M *et al.* Endomyocardiofibrosis in the americas collaborative study: the EMF-SIAC registry. *Curr Probl Cardiol* 2023;**48**:101995.
96. Rassi DDC, Marçal PC, Cruz C, Masson Silva JB, Hotta VT. Multimodality imaging in endomyocardial fibrosis: diagnosis and assessment of the extent of the disease. *Circ Cardiovasc Imaging* 2021;**14**:e012093.
97. Mocumbi AO, Stothard JR, Correia-de-Sá P, Yacoub M. Endomyocardial fibrosis: an update after 70 years. *Curr Cardiol Rep* 2019;**21**:148.
98. Mocumbi AO. How do I diagnose and classify endomyocardial fibrosis in under-resourced settings. *Arq Bras Cardiol Imagem cardiovasc* 2024;**37**:e20230107.
99. Mocumbi AO, Ferreira MB, Sidi D, Yacoub MH. A population study of endomyocardial fibrosis in a rural area of Mozambique. *N Engl J Med* 2008;**359**:43–9.
100. Habib G, Bucciarelli-Ducci C, Caforio ALP, Cardim N, Charron P, Cosyns B *et al.* Multimodality imaging in restrictive cardiomyopathies: an EACVI expert consensus document in collaboration with the "Working Group on myocardial and pericardial diseases" of the European Society of Cardiology endorsed by the Indian Academy of Echocardiography. *Eur Heart J Cardiovasc Imaging* 2017;**18**:1090–121.
101. de Carvalho FP, Azevedo CF. Comprehensive assessment of endomyocardial fibrosis with cardiac MRI: morphology, function, and tissue characterization. *Radiographics* 2020;**40**:336–53.
102. Senra T, Shiozaki AA, Salemi VM, Rochitte CE. Delayed enhancement by multidetector computed tomography in endomyocardial fibrosis. *Eur Heart J* 2008;**29**:347.
103. Pereira NL, Grogan M, Dec GW. Spectrum of restrictive and infiltrative cardiomyopathies: part 2 of a 2-part series. *J Am Coll Cardiol* 2018;**71**:1149–66.
104. Mankad R, Bonnichsen C, Mankad S. Hypereosinophilic syndrome: cardiac diagnosis and management. *Heart* 2016;**102**:100–6.
105. Polito MV, Hagendorff A, Citro R, Prota C, Silverio A, De Angelis E *et al.* Loeffler's endocarditis: an integrated multimodality approach. *J Am Soc Echocardiogr* 2020;**33**: 1427–41.
106. Gastl M, Behm P, Jacoby C, Kelm M, Bönner F. Multiparametric cardiac magnetic resonance imaging (CMR) for the diagnosis of Loeffler's endocarditis: a case report. *BMC Cardiovasc Disord* 2017;**17**:74.
107. Barberato SH, Lopes MACQ. Echoes of telecardiology guideline. *Arq Bras Cardiol* 2020; **114**:130–2.
108. Kim S, Fischetti C, Guy M, Hsu E, Fox J, Young SD. Artificial intelligence (AI) applications for point of care ultrasound (POCUS) in low-resource settings: a scoping review. *Diagnostics (Basel)* 2024;**14**:1669.
109. Menacho KD, Ramirez S, Perez A, Dragonetti L, Perez de Arenaza D, Katekaru D *et al.* Improving cardiovascular magnetic resonance access in low- and middle-income countries for cardiomyopathy assessment: rapid cardiovascular magnetic resonance. *Eur Heart J* 2022;**43**:2496–507.