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Special Article

Cardiac Manifestations of Parasitic Infections Part 1: Overview and Immunopathogenesis

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Summary

Parasitic infections produce a wide spectrum of cardiac manifestations. They may involve various anatomic structures of the heart and are manifested clinically as myocarditis, cardiomyopathies, pericarditis, or pulmonary hypertension in many resource-constrained settings. However, many parasitic infections involving the heart may also be currently diagnosed in developed countries due to growing worldwide travel, blood transfusions, and increasing numbers of immunosuppression states such as organ transplantation, use of immunosuppressive agents, or HIV/AIDS. Clinicians anywhere in the globe need to be aware of the potential cardiac manifestations of parasitic diseases. This is part one of a three-part series discussing parasites of the heart. In this section, we provide a general overview and immunopathogenesis of parasitic infections of the heart.

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Historically, the epidemiologic pattern of cardiac diseases varies between resource-constrained and resource-rich countries.[1–5] However, cardiac manifestations previously seen only in resource-constrained countries, including certain parasitic infections, can be currently diagnosed anywhere in the globe.[6–24] These epidemiologic transitions have been favored by multiple factors: (i) growing travel and immigration[5,11,12,20,21]; (ii) worldwide spread of the acquired immunodeficiency syndrome (HIV/AIDS) epidemic[8–10]; and (iii) growing number of organ transplantation, increased use of immunosuppressive agents, and blood transfusions.[11,14–18]

Parasitic infections due to protozoa and helminths account for a great burden or morbidity and mortality, particularly, in developing settings. It has been estimated that approximately 30% of the world's population experiences parasitic infections during their lifetime.[25] Many of these parasites are responsible for substantial socioeconomic losses and underdevelopment.[4,6] Most parasites that affect humans go through complex life cycles[11,23,25] (Table 1). During their life cycle certain intestinal, blood, and tissue parasites may directly or indirectly affect various anatomical structures of the heart, such as the myocardium, pericardium, and pulmonary vasculature.[20–24] There is no evidence that any parasite inflict direct damage to the endocardium, heart valves, or coronary arteries.

While the medical literature is replete with articles on the impact of bacterial, viral, and fungal infections on the heart with the resultant endocarditis, myocarditis or pericarditis syndromes, few articles have dealt with the clinical features of parasitic diseases of the heart. Therefore, the purpose of this article is to update the current knowledge of parasitic heart disease. Clinicians

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TABLE 1 Parasites involving the heart according to the causative agent, geographic distribution, and usual mode of transmission

Disease	Causative agent	Geographic distribution	Mode of transmission
Chagas'disease (American Trypanosomiasis)	*Trypanosoma cruzi*	Mexico, Central and South America	Skin or mucosal entry of stools of infected *reduviid* vectors Blood transfusion Organ transplantation Perinatal Oral
African Trypanosomiasis	*Trypanosoma brucei, rhodesiense or gambiense*	Africa (Sudan, Angola, Congo, Uganda, Ivory Coast, Chad)	Bites of *Glossina* (*Tsetse*) flies
Amoebiasis	*Entamoeba histolytica*	Developing countries	Fecal-oral
Toxoplasmosis	*Toxoplasma gondii*	Worldwide	Ingestion of undercooked infected pork meat Fecal-oral Blood transfusion Perinatal Transplantation
Cysticercosis	*Taenia solium*	Rural areas, poor hygiene, more prevalent in developing countries)	Ingestion of contaminated food with *Taenia solium* eggs (green leafs, salads)
Echinococcosis	*Echinococcus granulosus*	Worldwide distribution (rural areas)	Ingestion of eggs from infected canids
Trichinellosis	*Trichinella spiralis*	Worldwide distribution	Ingestion of infected undercooked pork meat

need to become familiar with parasitic diseases that can potentially involve the heart in both immunocompetent and immunodeficient individuals.[11,23]

Methods

We identified studies through a computerized search by reviewing Medline, Current Contents, and EMBASE databases. We used the following text words or their combination: *parasites, myocarditis, pericarditis, pulmonary hypertension, helminth or protozoa*. We limited our search to English and Spanish language papers that were published between 1960 and 2006. We screened all identified articles and cross-referenced studies from retrieved articles.

Immunopathogenesis of Parasitic Diseases of the Heart

Many parasites have evolved within a vertebrate's immune system and therefore have developed survival strategies that lead to chronic infections.[26–28] The persistence of parasites in human hosts leads to innate and specific chronic immune reactions, which are responsible for some of the clinicopathologic consequences of certain parasitic infections.[29–37]

In order to evade the immune system, extracellular protozoa have developed resistance to complement lysis, whereas intracellular protozoa resist killing by avoiding lysosomal toxic molecules.[38–41] Evasion of the complement system attack is achieved by losing surface molecules that act as receptors for complement proteins, or the acquisition of host regulatory proteins that down regulates the system.[38–40] The best described of this mechanisms is displayed by *Trypanosoma cruzi*, the agent of Chagas' disease expressing the molecule gp160, which can bind to C3b and C4b and hence inhibits the complement cascade.[38,41]

Toxoplasma gondii the parasite that causes toxoplasmosis resides intracellularly in phagosomes within macrophages or myocardial cells.[42–44] This parasite is propelled by an actin-myosin-dependent gliding motility mechanism and establishes intracellular vacuoles.[38–41] This remodeling prevents lysosomes fusion, which leads to the intracellular survival of this parasite. In a similar manner *T. cruzi* is transported to the cytoplasm by a parasite-secreted molecule (Tc-TOX) in order to survive the lysosome fusion.[38,39] *T. brucei rhodesiense* and *T. brucei gambiense*, the protozoa responsible for causing African trypanosomiasis, evade the complement system and other soluble mediators. The best characterized of these mediators is the trypanosome lysis factors, which contribute to the resistance of humans to infection with *Trypanosoma brucei*, an important pathogen of

TABLE 2 Treatment of parasitic infections involving the heart

Parasitic infection	Drugs of choice	Alternative
Trichinellosis	Mebendazole Steroids for severe symptoms	Albendazole
Toxoplasmosis	Pyrimethamine and sulfadiazine	Pyrimethamine and Clindamycin or Trimethoprim and Sulfamethoxazole
Schistosomiasis	Praziquantel	Oxamniquine
Amoebiasis (Severe extraintestinal disease)	Metronidazole	Tinidazole
Taenia solium (cysticercosis)	Albendazole (steroids for cysticercosis if viable cysts identified in MRI)	Praziquantel
Echinococcus granulosus	Albendazole and/or praziquantel	PAIR (percutaneous-aspiration-injection-reaspiration)
Echinococcus multilocularis	Surgical excision and albendazole	—
African Trypanosomiasis *T. brucei rhodesiense* (East African trypanosomiasis)	Late Disease-CNS manifestations: Melarsoprol	—
	Hemolymphatic stage (Early disease): Suramin	
African Trypanosomiasis *T. brucei gambiense* (West African trypanosomiasis)	Late disease-CNS manifestations: Melarsoprol	Late disease-CNS manifestations: Eflornithine
	Hemolymphatic stage (Early Disease): Pentamidine isethionate	Hemolymphatic stage (early disease): suramin
Chagas' Disease (American Trypanosomiasis) *T. cruzi*	Benznidazole	Nifurtimox

livestock.[41] *T. b. gambiense* and *T. b rhodesiense* strains are both resistant to the action of these soluble mediators of lysis and therefore can lead to human disease with potential complications to the heart.[39,41]

Various parasites dwell within the myocardial cells eliciting chronic inflammatory responses to the myocardium.[45–55] The best example of this possibility is infection by *Trypanosoma cruzi,* which invades and multiples within the cytoplasm of a variety of mammalian cells including macrophages and cardiac myocytes.[52–55] The recruitment and migration processes of inflammatory cells to the myocardium during *T. cruzi* infection involve proinflammatory cytokines, adhesion molecules, and extracellular matrix interactions.[50,51,56,57] Inflammatory cytokines play a crucial role in the immunopathogenesis of myocardial damage. Several studies have demonstrated that CD8+ lymphocytes are the predominant cell population infiltrating the cardiac tissues during chronic chagasic cardiomyopathy.[56,57] Different chemokines have been shown to play an important role in the control of T-lymphocytes influx, in particular, the chemokine receptor-5 (CCR5).[57] These inflammatory processes may result in myocardial dysfunction, which can manifest clinically as congestive heart failure, arrhythmias, sudden cardiac death, and thromboembolic events.[58–61]

In a similar fashion, toxoplasmosis, trichinellosis, African trypanosomiasis, free-living amoebas, opistorchiasis and many other parasitic diseases may produce damage to the myocardium.[4,11,20,23,25] Other parasites such as amoebiasis, echinococcosis, or cysticercosis may invade the pericardium and may therefore be associated with pericarditis, pericardial effusions, constrictive pericarditis, or cardiac tamponade.[20–25]

The heart may also suffer damage by indirect mechanisms of parasitic infections induced by their presence elsewhere in the human host. Some *Schistosoma* species can cause pulmonary hypertension associated with hepatosplenic schistosomiasis.[62–64] Severe anemia resulting from intestinal parasites such as those caused by hookworm infections (*Ancylostoma duodenale* or *Necator americanus)* infections, malaria, or visceral leishmaniasis may present as high-output heart failure (Table 2.[6,28] Tropical endomyocardial fibrosis, a severe form of restrictive cardiomyopathy, may be an important sequelae of helminth-associated chronic eosinophilia.[65,66]

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