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Perspective

# Chagas Disease beyond Endemic Borders: Challenges and Implications

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## DESCRIPTION

*Trypanosoma cruzi*, the parasite that causes Chagas Disease (CD) and only exists in the Americas (mostly in the impoverished rural areas of Latin America), spreads to both animals and humans through insect vectors. American trypanosomiasis is another name for Chagas disease (*T. cruzi* infection). A recent meta-analysis found that migrants from Latin America living in Europe had a pooled frequency of 4.2%, with rates topping 18% in Bolivians. Chagas disease is frequently diagnosed in non-endemic countries as a result of migration. Only 4% of those who have *T. cruzi* infection are really diagnosed, according to estimates that between 68,000 and 123,000 people in Europe are affected. The primary source of *T. cruzi* infection transmission outside of endemic nations is vertical transmission, blood transfusions, or organ transplants. Untreated patients get a chronic infection following infection, and after two to three decades, 10 to 30 percent of them may have cardiomyopathy or digestive system disease. In Argentina and Brazil, co-infection with *T. cruzi* has been observed in 1.3% to 4.2% and 5% of HIV-positive patients, respectively. *T. cruzi* infection behaves as an antagonist in HIV-positive patients.

The chronic nature and complex pathophysiology of CD have prevented the development of efficient medications and vaccines, despite several attempts. The only two Nitro Heterocyclic medications with FDA approval are Benznidazole (BNZ) and Nifurtimox, both of which have significant side effects and have been around for more than 50 years. Finding a specific medication that can eradicate the parasite and, in turn, get rid of the CD symptoms is the goal.

According to CD is a parasite that must live inside of the cells of mammalian hosts and is not at all harmless. Parasites are detectable in the bloodstream during the early acute phase and become broadly distributed in tissues and organs. Later, CD develops into a chronic, asymptomatic phase that is exceedingly low in parasite load and lasts for a very long time. According infected patients will progress to a symptomatic chronic phase, which is marked by organomegaly and cardiomyopathy and has few effective treatment choices. It's fascinating to note that an infection exhibits itself in a certain person is the consequence of a number of intricate connections.

The acute parasitemic phase that precedes the onset of CD is characterised by an excess of *T. cruzi* in the blood, spread to tissue, and intracellular replication. When adaptive immunity is activated, parasite-infected people are able to regulate their parasitemia and progress to an undefinable stage where very low parasite levels are sporadically found in the blood and no to very mild clinical symptoms are seen. A third or so of infected people develop a clinically symptomatic chronic phase and experience significant heart issues. The primary cause of death in CD patients is thought to be cardiomyopathy, which manifests as progressive heart failure, arrhythmias, and cardiac, pulmonary, and brain embolism. Up to 20% of those with the infection may also suffer digestive problems, which can lead to chronic dysphagia, constipation, and malnutrition.

Recent studies have talked about the difficulties in managing the disease and CD. It is commonly acknowledged that low-level parasite persistence produces constant antigens that continue to elicit immune responses, promote pathological tissue damage, and ultimately result in cardiac failure. Our research indicates that the myocardium in CD experiences ongoing oxidative stress. During CD development, the heart has shown a rise in the formation of Reactive Oxygen Species (ROS) by mitochondria and NADPH oxidase as well as superoxide by macrophages.

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