ISSN: 2329-891X
Journal of Tropical Diseases & Public Health

OPEN ACCESS Freely available online

Commentary

Mortality and Epidemiology of Chagas Disease

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DESCRIPTION

Trypanosoma cruzi is a pathogen of Chagas disease. Infectious diseases are lifetime without treatment. Therefore, the prevalence may be high despite the low incidence. In the case of disease load on the American parasites disease, the current estimates of 6 million infections and 120 million cardiomyopathy symptoms. *Trypanosoma cruzi* organisms are transcribed when infected vector droplets are generated in mammalian mucosa. Transmittance is also contaminated by vector or vector droplets and may also occur due to blood component blood transfusion, organ transplantation, and food or beverage consumption, contaminated in uterus from mothers to fetus. The typical environment for Chagas disease is in the Latin American countryside, where the presence of mud houses and livestock facilitates the invasion of livestock and livestock mediators. However, the vector control program reduced infections in many rural areas, and infected individuals moved to cities in Latin America, the United States, and Europe [1]. In contrast to Europe, the United States fully characterizes the epidemic infection of *T. cruzi* involving 11 triatomic species and a range of mammalian hosts [2]. Nevertheless, most of Individuals infected with *T. cruzi* in the United States are Hispanic immigrants who have become ill in their country of origin. Infectious metacyclic tripomastigote deposits on the skin of mammalian hosts with fecal droplets extruded by the blood-sucking triatomine bug. Parasites penetrate mucous membranes such as bites, skin abrasions, or conjunctiva. This mechanism occurs through vector feces rather than the mouth area and is known as stercorian transmission. Once internalized, motile tripomastigote enters nucleated cells *via* both lysosome-dependent and independent mechanisms [3, 4]. The parasite is then taken up by the membrane-bound (parasite's) vacuole and then fused with the lysosome. When exposed to a lower pH, the parasite is stimulated to differentiate into an intracellular amastigote, which is simultaneously released into the cytosol in 4-5 days. Here, the amastigote breeds asexually and forms pseudocysts. It can occur in a variety of host tissues, most notably the reticuloendothelial cells of the heart, smooth muscle, skeletal muscle, liver, spleen, and lymphatic system. Within the pseudocyst, the amastigote differentiates into tripomastigote. After cytolysis, trypomastigote infects adjacent tissues and initiates a new replication cycle or spreads through the bloodstream and lymph. Without anti-Trypanosoma treatment, the infection persists for the life of the mammalian host.

Triatomine bugs, which feed on infected hosts, may ingest extracellular tripomastigote and enter the midgut, where it is converted to an intermediate spheromastigote form. Differentiation of spheromastigote into epimastigote occurs in response to decreased glucose levels in the environment when blood meals are being digested [5]. Epimastigote propagates by dichotomy of the hindgut and migrates to the rectum. There, it metamorphoses into flagella and hydrophobically attaches to the waxy intestinal cuticle, transforming into an infectious metacyclic tripomastigote, completing its life cycle. Originally a wild epidemic, Chagas disease invades wild eco-topes by looting, occupying physical space, deforesting, crowding wildlife and homes, and easily containing triatomine. In this way, Triatomine began to use humans as a food source, establishing three communication cycles wild, home environment, and home. Some wildlife, such as Opossum, one of the longest-known reservoirs of *Trypanosoma cruzi*, may play an important role in the epidemiology of Chagas disease. These animals have been shown to not only carry the *Trypanosoma* form of *Trypanosoma cruzi* in the bloodstream, but also house and eliminate all forms of parasites in the scented glands. Conversely, pets such as cats and dogs can enter the wild environment and hunt, thereby verbally infecting and causing infection in and around the house. The adaptation of triatomine to human settlements, along with the circulation between triatomine bugs and wildlife and livestock, is certainly the most important determinant for the establishment of human infection. There are speculations about the genetic transformations that lead to this adaptation, but due to the lack of original sources, it appears that triatomines are primarily the result of searching for new food sources. Forattini (1980) clearly demonstrated this adaptation by placing chicken coops in various eco-topes outside the home environment.

The mechanism by which wild triatomine adapts to their homes and home environment is not fully understood. "Everything that exists in the universe arises from chance or need". In fact, the mechanism of adaptation of the triatomine bug to the human home seems to fit this concept well. When wild triatomine bug is passively brought around the house and around the house with wood for construction and fuel, and palm leaves are attracted to

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Received: 01-Mar-2022, Manuscript No. JTD-22-16113;**Editor assigned:** 17-Mar-2022, Pre QC No. JTD-22-16113 (PQ); **Reviewed:** 21-Mar-2022, QC No. JTD-22-16113; **Revised:** 25-Mar-2022, Manuscript No. JTD-22-16113(R); **Published:** 01-Apr-2022, DOI: 10.35248/2329-891X.22.10.318.

Citation: Ferreira M (2022) Mortality and Epidemiology of Chagas Disease. J Trop Dis. 10:318.

the roof of the house or annex, or to the light. Or, humans invade wild eco-topes, clear forests, shrink homes, flock animals, and triatomines are "artificial niches" in homes and surrounding areas to seek alternative food sources among humans and pets. Some species, such as Triatoma infestans, are completely domesticated except in Bolivia, but Panstrongylus megistus. There are also other species such as *T. brasiliensis* and *T. sordida* and *T. pseudomaculata* are ubiquitous in Brazil. It may be wild, domesticated or semi-domesticated. It shows the habitat of the palms of Opossum and Triatomine infected with *T. cruzi* infested huts. Chagas disease causes heart and/or gastrointestinal illness in 20-30% of people infected with *Trypanosoma cruzi*. The southern half of the United States contains a *T. cruzi* epidemic cycle involving four major triatomine vector species and seven minor triatomine vector species. *T. cruzi* infections have been reported in several mammals, including raccoons, possums, wood rats, and dogs. Over the past decade, locally acquired Chagas disease has become increasingly recognized in the United States, primarily because of the screening of blood donors in Latin America and the screening of infected unexposed blood donors. Nonetheless, chronic *T. cruzi* infections imported among immigrants from Latin America far outnumbered native human cases, and locally acquired infections were rarely recognized in the acute phase. Benznidazole is currently approved by the FDA for treatment and clinical and public health efforts are underway by researchers and some state health agencies to increase access to diagnosis and treatment.

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