Chagas Disease: A Neglected Disease

Mohemmed Faraz Khan¹, Wasim Akhtar¹, Mohammad Shaquiquzzaman¹, Garima Verma¹, Sarwat Tauhid², Syed Rashiduddin* and Mohammad Mumtaz Alam**

¹Department of Pharmaceutical Chemistry, School of Pharmaceutical Education and Research, India
²Department of Chemistry, Oriental College, India

Introduction

Chagas disease, which is also known as American trypanosomiasis is an infection caused by the protozoan, Trypanosoma cruzi (T. cruzi). It is generally transmitted by feces of a triatomine insect, also known as kissing bugs belonging to family Reduviidae [1]. As per WHO report, 2017, about 6 to 7 million people across the world are affected with this infection. This disease presents itself in two phases: acute and chronic, each with its own characteristic features. Various diagnostic techniques are available to confirm this infection. Benznidazole and nifurtimox are clinically used to relieve the infected patients. Both these have 100% efficacy, if given soon after infection at the onset of acute phase. However, both of them are associated with some limitations, thereby necessitating the need for novel and safe agents.

Variable clinical presentation is observed in humans. Just following the parasite infection, there is a short acute phase in which there is abundant parasitemia which is relatively easy to detect by direct blood examination. Very mild or nonspecific symptoms make recognition of the contagion difficult. Majority of the patients of this phase go unrecognized due to scarcity or absence of the clinical manifestations [2]. Following this acute phase, the disease enters chronic phase characterized by long, asymptomatic clinical latency that lasts for 10-30 years or throughout life. In this phase, around 30% of the infected people develop one of the clinical manifestations like cardiomyopathy and/or mega gastrointestinal syndromes [6]. Progressive heart failure and sudden deaths are the main causes of deaths in these patients [7].

Life Cycle of T. cruzi

This disease gets transmitted when an infected triatomine insect vector takes a blood meal and releases trypomastigotes of transmission is through the ingestion of contaminated food or drink. Latrogenic is due to contaminated blood transfusion or organ transplantation [2,4,5].
in its feces near site of wound. These trypomastigotes enter the host through wound or intact mucosal membranes. Upon invasion of cells near the site of inoculation they get differentiated into intracellular amastigotes. Amastigotes multiply by binary fission and differentiate into trypomastigotes and then are released into circulation as bloodstream. These forms then infect cells from a variety of and get transformed into intracellular amastigotes in new infection sites. Clinical manifestations can result from this infective cycle. Kissing bugs become infected by feeding on human or animal blood that contains circulating parasites. These ingested trypomastigotes get transformed into epimastigotes in the vector’s midgut. These parasites multiply and get differentiated into infective metacyclic trypomastigotes in the hindgut \[8,9\]. Life cycle of the parasite is shown in Figure 1.

**Diagnosis and Treatment**

Diagnosis of this fatal disease can be done by both direct and indirect parasitological methods, molecular method. Direct parasitological methods for detection of acute phase include examination of fresh samples, blood smear, micro-strout test and strout concentration method. Xenodiagnosis and blood culture are a part of indirect methods. Polymerase chain reaction can also be employed for determination of acute phase of the disease \[10\]. Chronic phase can be detected by serological methods by enzyme linked immunosorbent assay (ELISA), indirect immunofluorescence, indirect haemagglutination and Western blot \[11,12\].

Since 1960s, the only drugs available for the treatment of this infection have been benznidazole and nifurtimox (Figure 2) \[13\]. These two have been the mainstay of parasiticidal treatment for the past 50 years, despite the fact that their efficacy and safety profile is far from ideal conditions. Nifurtimox was the first drug used. This is administered orally in three to four doses for a period of 60-90 days. Common side effects of its use are hypersensitivity, digestive intolerance, headache and sleeping disorders. Neuropathy and depression of bone marrow are very rare \[16,17\]. Researchers are actively involved in the development of novel agents. However, the major obstacle associated is poor translation of in vivo data to human disease. Animal models available bear a drawback of limited predictive value \[18-23\].

**Conclusion**

Chagas disease is one of the neglected tropical diseases. Benznidazole and nifurtimox have been the mainstay for treatment of this infection since long. However, there are many shortfalls associated with these drugs which necessitate the need for development of newer agents with better safety and efficacy profile.

**References**


This work is licensed under Creative Commons Attribution 4.0 License

Your next submission with Juniper Publishers will reach you the below assets

- Quality Editorial service
- Swift Peer Review
- Reprints availability
- E-prints Service
- Manuscript Podcast for convenient understanding
- Global attainment for your research
- Manuscript accessibility in different formats (Pdf, E-pub, Full Text, Audio)

Unceasing customer service

Track the below URL for one-step submission

https://juniperpublishers.com/online-submission.php