

Chagas Disease: Complementing Supplements

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Resumen

La infección por *Trypanosoma cruzi* esta aún lejos de haberse resuelto en latinoamérica. Investigaciones recientes sobre este germen están fuertemente sesgadas hacia la investigación de ciencias básicas, principalmente a nivel molecular, dejando de lado importantes hallazgos clínicos y epidemiológicos. Presentamos aquí evidencias científicas que demuestran como el *Trypanosoma cruzi* afecta diferentes sistemas neurales incluyendo el autónomo. Las rutas de trasmisión oral y genital relacionadas con este parásito, así como las reacciones cruzadas y falsos positivos descritas durante la realización de diferentes tests serológicos que investigan la posible presencia del *Trypanosoma cruzi*, incluyendo los utilizados para investigar el VIH, deben todas ellas ser tomadas muy en cuenta antes de rotular a los pacientes con el, casi siempre, fatal diagnóstico de enfermedad de Chagas.

Abstract

Trypanosoma cruzi infection is far from being solved in Latin America. Recent research is strongly biased to basic investigations mostly at molecular levels putting aside important clinical involvement and epidemiological findings. Here, we present evidence that *Trypanosoma cruzi* affect neural systems including the autonomous one. Oral and genital routes of transmission of this parasite as well as the cross-reactions and false-positives described with different serological tests including those used to test HIV must be checked out before putting the almost always fatal diagnoses of Chagas disease.

Introduction

Trypanosoma cruzi (T. cruzi) infection is still a challenge to scientists working in basic and/or clinical grounds; some of these problems, mainly the basic ones, have been addressed by different groups in different Latin- American countries and commented in some recent publications as supplements; however, the basic - clinical gap seems to remain in current investigations.^{1,2} In order to help in closing such a gap on this international health public problem we consider timely to go further looking for probable clinical, epidemiological and laboratory advances unaddressed in those previous scientific communications that may influence patient's intervention in clinical practice. We concentrate our efforts in knowing neural structures involvement because the neurotropism of this parasite is clearly established.

Material and Methods

Following methodologies described in previous research published on this topic³ we identified references by searches of Medline/PubMed from 1966 until December 2006 and of Scielo from 1997 until December 2006 with the terms "trypanosoma cruzi" "chagas" "central nervous system" "epidemiology" "spinal cord" "peripheral nervous system" "neuromuscular junction" "muscle" "muscle disorders" "neuromuscular diseases and disorders" "synapticopathies" "pathophysiology" "clinical neurophysiology" "functional neurology" "clinical neurosciences" "transmission route" "laboratory." Articles were also identified through searches of the authors' own files. Papers published in English, Spanish and Portuguese were analyzed. Inclusion and exclusion criteria were similar to those chosen in a previous research; however, we consider at this time publications reporting serological tests using polymerase chain reaction as well.³

Results and Discussion

In clinical grounds, besides T. cruzi infection-associated dysautonomy widely recognized and investigated,⁴ it was noteworthy to find a good number of acute and chronic disorders including cerebrovascular disease (CVD), meningitis, cognitive disorders, peripheral neuropathies, synapticopathies such as myasthenia gravis, myopathies including polymyositis, pupilopathies and impotence.

We highlight here CVD because it is a very common complication of T. cruzi infection with a high morbimortality as well as an increasing trend in social and personal costs.⁵⁻⁷ It should be remarked that T cruzi infection itself, without a clinically established disease including cardiopathy, is more than enough to trigger CVD in humans.^{6,8,9}

Regarding routes of transmission, it was interesting to find that the parasite colonizes human body by means other than bites including oral route thru breast feeding and sugar cane juices.¹⁰⁻¹² Genital route seems to be another way of transmission not well characterized yet in countries where this problem is endemic.¹³

On the other hand, *T. cruzi* generates one of the seventy six cross reactions described so far while looking for HIV antibodies¹⁴⁻¹⁶ making this germ much more dangerous to humans than previously thought. Thus, immunosuppressants given as regular treatments to patients harboring unisolated HIV viruses^{17,18} may increase immunosuppression in patients infected by *T. cruzi* worsening clinical complications produced by this parasite as the ones commented above, and would allow to *T. cruzi* to act more aggressively than usual with devastating consequences to infected people.

These facts call the attention on what might be the true cause of possible complications found in seropositive patients to both *T. cruzi* and HIV including the deterioration of syncopal reactions.¹⁹

All the aforementioned comments support the continued interest in researching and publishing supplementary scientific issues on Chagas disease to complement and up date the knowledge of the complex disorders produced by *T. Cruzii*. However, we want to go further and stress that the “new” routes of infection, the “infrequent” systemic complications that are very far from being included in level I of epidemiological evidence statistically speaking, and the “new” crossed reactions described in the present report can be, all of them, the first evidence of a serious health problem that is still far from being solved in, at least, some Latin-American countries.

Acknowledgments

We dedicate this manuscript to Eugenio Leon, father of one of us (FEL-S). He stated ten years before being diagnosed as *T. cruzi* carrier at the UIS laboratory of parasitology, in Bucaramanga, Colombia, that he acquired the parasite after eating contaminated beef and that never ever he had been bitten by any possible parasite’s reservoir. Unfortunately he already died of chagasic myocardiopathy when he was 77 years-old and he took away with him all of the evidence.

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