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Research Paper

Gastrointestinal Infection with Mexican TcI *Trypanosoma cruzi* strains: Different Degrees of Colonization and Diverse Immune Responses

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Abstract

Mexican Ninoa and Queretaro (Qro) TcI strains of *Trypanosoma cruzi* have shown different degrees of virulence, and the two strains produce heterogeneous immune responses in the hearts of infected mice. This work shows that the same strains can invade the intestine by an intraperitoneal route and establish an infection, mainly in the colon. The three segments of the small intestine (duodenum, jejunum and ileum) were infected to a lesser degree than the colon. Despite the fact that parasites were predominantly found in the colon, an obvious inflammatory reaction was observed in the submucosal layer along the entire intestinal tract, with the virulent Qro strain causing significantly more areas of higher immune infiltration. A clear recruitment of CD4+ and CD8+ T lymphocytes to the mesenteric ganglia was observed during infection with the virulent strain. Macrophages were also differentially distributed in the gastrointestinal tract. These later cells infiltrated fewer amastigote nests in the mice infected with the Qro strain than in the mice infected with the Ninoa strain. When IFN-γ, TNF-α, and IL-4 levels were measured, an increase in these cytokines was observed compared with the uninfected mice. The role of these inflammatory reactions in the pathogenesis of Chagas enteropathy is also discussed in this paper.

Key words: Intestinal infection, Mexican *Trypanosome cruzi* strains

INTRODUCTION

Chagas´ disease is caused by the protozoan parasite *Trypanosoma cruzi*, and it affects as many as 10 to 16 million people in North and South America, where it represents a major public health problem [1]. Recently, however, due to the emigration of people from endemic areas, cases of Chagas´disease have been reported in the USA, Canada, Australia, Japan and Europe, extending the range of this parasitic disease throughout the developed world [2, 3]. *T. cruzi* is a parasite with high genetic diversity, and it has been grouped into six discrete typing units (DTUs), *T. cruzi* I to VI [4]. It is known that the TcI DTU is predominant in Central America, the northern region of South America and Mexico [5, 6, 7, 8]. Mexican isolates from humans and from vector insects have been primarily found to be TcI, and these isolates displayed heterogeneous processes of metacyclogenesis and infection *in vitro* [9]. TcI strains are likely to be the strains that cause the clinical manifestations observed in Mexico [10], where it has been shown that cardiac manifestations are the main symptoms presented by patients with Chagas´ disease [11]. The course of infection with *T. cruzi* includes an acute phase, during which the parasite infects a wide variety of tissues, which