cured. Multidrug-resistant MG strains are rapidly becoming a reality, and a global coordinated effort is urgently needed to examine the efficacy of novel treatment regimens using currently available drugs.

Note

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Acute Chagas Disease and Risk of Congenital Infection

To the Editor—Acute Chagas infection can be asymptomatic or present with nonspecific symptoms, such as fevers, myalgia, and the presence of lymphadenopathy and hepatosplenomegaly. Occasionally, the Romaña sign is present (unilateral painless periorbital swelling) [1]. This phase is characterized by a high parasitemia detectable by means of peripheral blood smear with a Giemsa stain and/or by polymerase chain reaction (PCR). Detection of IgG antibodies can be seen during acute infections [2,3]. In contrast, chronic Chagas disease is characterized by structural and/or conduction cardiac abnormalities and very low levels of parasitemia in immunocompetent hosts [4]. PCR can miss up to 40% of the chronic infections [5]; its sensitivity to such infection is considered highly variable, and it is not recommended for diagnosis [6]. Likewise, parasitemia levels in the mother’s blood are associated with the likelihood of congenital Chagas infection [7], which is also seen in other infectious diseases [8].

The study by Kaplinski et al [9] showed that sustained domestic vector exposure was associated with cardiomyopathy but inversely associated with risk of congenital transmission. Conversely, positive PCR results and higher parasitemia levels were correlated with an increased risk of congenital transmission. Although the association was very interesting, the study did not show a distinction between acute and chronic infections or control for other potential factors favoring higher parasitemia levels or reactivations during pregnancy (eg, coinfections, such as human immunodeficiency virus infection; comorbid conditions; and nutrition status). Thus, acute or subacute infections and, to a lesser degree, reactivations or re-infections with higher parasitemia levels may account for the majority of congenital infection cases seen by Kaplinski et al [9]. They also observed lower levels of parasitemia, an increase in cardiac abnormalities, and a decreased risk of congenital transmission in the group with prolonged residence in an infested house. Hence, sustained domestic vector exposure is a surrogate for chronic Chagas infection.

To decrease congenital transmission, asymptomatic acute or subacute infections should be better recognized and treated. These patients often do not display epidemiological characteristics as clear as those observed by Kaplinski et al [9] in chronically infected women. Their findings call for better screening and testing for women of childbearing age in areas of moderate or high endemicity. These approaches not only may prove useful in reducing congenital transmission rates, but they may also have an impact on vector-associated transmission.

Note

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Reply to Henao-Martínez, et al

To the Editor—Because of the natural history of Trypanosoma cruzi infection as well as their vector exposure history, it is highly unlikely that the women in our study [1] were in the acute phase of infection, as suggested by Henao-Martínez [2]. The acute phase begins 1–2 weeks after vector-borne parasite exposure and lasts 4–8 weeks. In contrast, the chronic phase is lifelong in the absence of successful treatment, resulting in high infection prevalence despite low incidence [3]. Even for women with ongoing vector exposure, the probability of being tested during the brief acute phase is extremely low.

Moreover, the prevalence of reported domestic infestation at the time of study was even lower for women who transmitted infection to their infants than for those who did not (12.9% vs 23.4%, respectively), the reverse of the relationship expected if acute phase infection were responsible for the higher parasitemia levels in transmitting mothers [1]. The parasite loads in women who transmitted infection, while higher than in nonpregnant adults with chronic T. cruzi infection, were substantially lower than those seen in the acute phase (eg, in congenitally infected infants) or in patients with human immunodeficiency virus (HIV) coinfection and T. cruzi reactivation [4, 5]. HIV coinfection is also very unlikely to explain our findings. The reported prevalence of HIV infection in women screened during prenatal care in Bolivia in 2014 was 0.12% [6], corresponding to <1 case of HIV–T. cruzi coinfection among our 456 seropositive participants. There is no published evidence to suggest that nutritional status or comorbid conditions other than immunosuppression affect T. cruzi parasitemia levels, nor is a “subacute” phase of T. cruzi infection described in the literature.

I agree with Henao-Martínez [1] that to decrease congenital transmission of T. cruzi infection we need better programs to screen pregnant women and their infants. We also need screening and treatment programs for children and young adults. Antitrypanosomal drugs are contraindicated during pregnancy, but women treated earlier in life have much lower transmission risk than untreated women [7]. Because T. cruzi infection is zoonotic, human screening and treatment programs are unlikely to have a significant impact on vector-borne transmission; control of this transmission route will require improvements in housing and appropriate insecticide use.

Note

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