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False-positive Human Immunodeficiency Virus Test and *Trypanosoma cruzi* Infection in Eastern Colombia

To the Editor: Immunodeficiency, either human immunodeficiency virus (HIV)-positive or HIV-negative, is associated with several coinfections, including *Trypanosoma cruzi*.¹ However, several concerns regarding the true cause of some of these illnesses have arisen because many cross-reactions have been found with either enzyme-linked immunosorbent assay (ELISA) or Western blot (WB) testing for HIV infection.² After previous studies performed in eastern Colombia on *T cruzi* infection,³ we found a new cross-reaction for HIV that had never been described before. We wanted to report it because it might make a significant difference between life and death.

From January 1996 to December 2000, 41,059 blood samples from people considered healthy and without risk factors for HIV infection were screened for anti-HIV by HIV-1/2 enzyme immunoassay (EIA) PLUS (Abbott Laboratories, Abbott Park, IL) at the metropolitan blood bank at Bucaramanga, Colombia. Following the guidelines of

the Ministry of Health of Colombia for HIV infection, the samples were tested three times; if positive, they were confirmed by HIV-WB (Genelabs, Singapore Science Park, Singapore) at the National Institutes of Health of Colombia.

When submitted to ELISA, 0.26% of samples were positive; 57.4% of those were considered HIV false-positive after the confirmatory WB tests were performed, for a final prevalence of 0.11% of HIV seropositivity for that population. More interesting was finding that 4.8% of the false-positive results were due to *T cruzi* infection, according to Abbott Chagas Antibody EIA (Abbott Laboratories, Sao Paulo, Brazil); this test has a sensitivity of 99% and a specificity of 98%.³ None of the subjects studied had leishmaniasis.

Cross-reactions of HIV tests by ELISA or WB have been found in systemic lupus erythematosus, pregnancy, hepatitis, malaria, tuberculosis, miscellaneous autoimmune diseases, hematologic malignancies, as well as the amount of viral particles; disease state or some other laboratory conditions seem to play a role for producing such false-positivity.^{2,4} More important were the results obtained recently from 43 blood banks in Colombia. In that study, a lack of accuracy of the serologic tests used for diagnosing transfusion-transmitted infectious disease including HIV and *T cruzi* was observed.⁵ Thus, the HIV false-positivity due to *T cruzi* infection found by us makes sense and confirms those inaccurate results.

The false-positivity found in our study might have been due to the presence of some parasitic proteins such as reverse transcriptase enzyme recently found in this parasitic protozoan.⁶ Therefore, a true increase in Chagas disease reactivation in people infected by HIV reported elsewhere⁷ might, at present, be overestimated. The lack of viral isolation and the incomplete serologic protocols frequently used for HIV testing in many countries⁸ support the contention that *T cruzi* and perhaps

other untested germs might actually be originating a cross-reactivity on HIV testing as shown here. Finally, the small amount of HIV seropositivity found in this Colombian area contrasted with local and more frequent infectious problems present in those communities including not only Chagas disease but also malaria, tuberculosis, cholera, and dengue, among others.

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What Goes Around, Comes Around

To the Editor: In reading the article “Addisonian Crisis Precipitated by Thyroxine Therapy: A Complication of Type 2 Autoimmune Polyglandular Syndrome,”¹ I was reminded of Dr. John Eager Howard’s warning to us some 35 years ago, when I was a resident at Union Memorial Hospital in Baltimore, about the dangers of thyroid replacement in long-standing hypothyroidism. Since thyroxine is needed to hydroxylate cortisone—thereby enabling the kidney to convert it to a glucuronide and excrete it—the serum level of cortisone may fall precipitously with the institution of thyroid

therapy, the “asleep” adrenals failing to respond. We were told to be sure the patient received a physiologic dose of cortisone for the first 6 weeks of thyroid replacement. This was apparently well-known in the “dark ages” of the 1960s and 1970s, before the Internet replaced the library for literature searches.

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