Letters to Dermatology

Dermatology 1997;195:184-185

Chagas’ Disease: A Potential Plague for Europe?

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Key Words

Chagas’ disease
Tripanosomiasis
Trypanosoma cruzi
Tachyarrhythmia

A 52-year-old man, a tour operator, while in central Brazil, developed a flu-like illness. A few weeks later, back to Italy, he had an episode of tachyarrhythmia. A cardiologist diagnosed myocarditis possibly of postviral etiology and prescribed amiodarone. A few days later, an exanthema developed which prompted a dermatological consultation.

The exanthema consisted of several erythematous, nonitching, somewhere annular or targetoid macules, particularly on the trunk. The clinical picture evoked erythema multiforme, drug eruption, Lyme disease and, more closely, African trypanosomiasis [1]. Lyme ELISA and Western blot being negative, the diagnosis remained pending and amiodarone was continued. The eruption faded in a couple of weeks.

In apparently good health, the patient returned to Brazil where soon he had another episode of tachyarrhythmia. Blood tests now included Chagas ELISA which proved strongly positive. A diagnosis of Chagas’ cardiopathy was made and the patient, an occasional blood donor, was instructed to interrupt blood donations.

American trypanosomiasis or Chagas’ disease is endemic in South and Central America where it affects some 20 million people with an annual toll of 50,000 deaths [2]. The causative agent is Trypanosoma cruzi, a flagellate protozoan transmitted by hematophagous Hemiptera of the family Reduviidae, subfamily Triatominae, the most common species being Triatoma infestans, Rhodnius prolixus and Panstrongylus megistus. Adult Triatominae are 2.5 cm long nocturnal feeders and use to defecate close to their bite. When feces contaminate a bite or the conjunctiva, the organisms invade the host multiplying in the cells, especially those of the muscles (intracellular amastigotes). Eventually they rupture out into the blood (trypomastigotes) to be taken up by the insect with the blood meal, to multiply in its gut as epimastigotes and pass into the feces.

184

Letters to Dermatology

Both the causative organism and its vector are largely present throughout the Americas from 40° N (Texas) to 43° S (Argentina). Human contact occurs occasionally in the forest or most commonly in a domiciliary cycle among poor people living in mud huts.
Travelers such as our patient are unlikely to get infected. In the last 20 years, 9 cases of acute Chagas’ disease have been described in the USA, but all of them were imported and none was noted among tourists returning home [2]. Chagas’ disease, therefore, may matter in Europe only because of the capacity of T. cruzí to spread through other modalities. Parasites may pass from mother to fetus, accidental infections may be observed in laboratory workers, transfusion of infected blood and transplantation of infected organs are possible.

In endemic areas, blood transfusion is the second most common route of transmission and is a matter of concern in the USA where millions of immigrants from Central and South America are potential blood donors [2]. In 1990 in Los Angeles, 2.9% of them were found to be seropositive [3]. In fact, the parasite is lifelong present in the blood, irrespective of the clinical conditions of the patient. Most infected people, therefore, are symptomless and bona fide donors. Viable parasites can be observed for 8 months in the blood maintained at room temperature and for 18 days at 4 °C [4] and the risk of transmission is estimated to be 13-23% for each unit of contaminated blood [1]. The seroconversion rate may be as high as 60% [4]. The incubation period lasts up to 114 days.

Considering that in nonendemic vector-free areas of Ecuador and Bolivia about 1/4 of blood donors are positive [5,6] with peaks of 63% [7], the possibility that immigrants from endemic areas may spread the disease is no longer remote. At present, their number in Europe is unknown, but in Italy in 1994 it was officially 79,875 [8]. Considering the clandestines, therefore, there may now be 20,000-40,000 potential infectants in Italy.

Acknowledgment

We are indebted to Maria Teresa Hojyo, MD, who helped us in assembling the literature.

References


Letters to Dermatology

185