longed fever (37°C–40°C), anorexia, weight loss, asthenia, arthralgia, malar rash, and erythema multiform or necrotizing vasculitis-like skin lesions. In addition, one or more autoantibodies are present in the serum of these patients ([3]). Our patient had almost all these features for making a diagnosis of autoaggressive lepromatous leprosy.

The hypergammaglobulinemia and normal serum C3 level seen in our case is in corroboration with other observations ([2, 7]). The hypergammaglobulinemia could be due to polyclonal activation of B lymphocytes.

The lack of an adequate response to systemic corticosteroids in our case suggested that to control this ongoing autoaggressive phenomenon, corticosteroids alone are not as effective as a combination of antileprosy treatment and corticosteroids or thalidomide ([3]).

Although many of the above features are also present in type 2 lepra reactions, the lack of neuritis and iridocyclitis, and the presence of photosensitivity and associated autoantibodies make autoaggressive hanseniasis a distinct clinical and immunological entity.

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Electrocardiographic Alterations in Lepromatous Leprosy Patients with Concomitant *Trypanosoma cruzi* Infection

To the Editor:

Leprosy is endemic in Argentina and prevails mostly in the Littoral area, a region extending from the border with Paraguay in the north to the city of Buenos Aires in the south, where more than 30,000 persons are estimated to be infected with the disease ([11]). Coexisting with the leprosy problem, many people living in this area also are at risk of infection with *Trypanosoma cruzi* (Tc), the causative agent of American trypanosomiasis or Chagas' disease. Chagas' disease, whose acute infection is often asymptomatic and self-resolving, may result in chronic lesions with the heart as the main organ involved. Clinical manifestations of chronic chagasic myocardiopathy (CCM) appear years or decades after initial infection, and consist of cardiac enlargement with disturbances of cardiac rhythm and/or conduction ([9]).

Despite much effort, the pathogenesis of the chronic heart lesions remains incompletely elucidated, and several mechanisms have been put forward to reflect perhaps its multifactorial basis. Some authors state that lesions of intracardiac autonomic ganglia and neurons constitute an important factor in the generation of the heart disease ([7]). Others, instead, invoke autoimmune reac-

tions as mainly responsible for the occurrence of chronic myocardial injury ([13]). More recently, microvascular abnormalities, resulting in local ischemia and focal pathological changes, also have been implicated in the genesis of the heart damage ([15, 17]).

Since lepromatous leprosy (LL) is associated with autoantibody formation ([1, 10]), and may result in autonomic nerve dysfunction along with degenerative changes of striated muscle fibers ([4, 5, 8]), a study was undertaken to investigate whether Tc-infected LL patients show a different pattern of heart involvement.

A sample of 39 individuals was studied. They were distributed as follows: 13 LL patients with no serologic evidence of Tc infection; 10 LL patients with positive Tc serology (LL-Tc); and 15 persons who yielded positive responses for the detection of specific anti-Tc antibodies (Tc). The subjects were matched for age and sex. Patients from both leprosy groups showed no major differences as to the duration of illness, antileprosy treatment, the extent of skin involvement, variety of lesions, and occurrence of type 2 reactional episodes (The Table). Except for two cases (one in group 1 and the other in group 2), all LL patients had active disease. The presence or absence of Tc infection relied on the firm evidence of positive or negative specific serology, respectively. Serology was performed by the direct agglutination, indirect hemagglutination, and indirect immunofluorescence techniques.

The subjects were carefully examined to rule out the existence of additional pathological disorders, particularly concomitant cardiomyopathies, i.e., congenital, rheumatic, and hypertensive. Upon them, a 12-lead resting electrocardiogram (ECG) was taken. The data were recorded and interpreted by the same cardiologist blinded to the study groups.

The subjects did not have any cardiac complaint, and no significant differences in blood pressure levels were registered among the groups (data not shown). While an abnormal ECG was recorded more frequently in people with Tc infection, such a trend did not reach statistical significance when compared with LL patients, since two of them also had an altered ECG (The Table). Although electrocardiographic abnormalities

THE TABLE. *Characteristics of patients on each group.*

Group[a]	No.	Age (mean ± S.E.M.)	Sex M/F	Duration of leprosy (mean ± S.E.M.)	Antileprosy treatment[b]		Active disease	Abnormal ECG	Pathological tracings[c]	
									1 sign	2 or more signs
LL	13	47 ± 3	8/5	6.3 ± 1.4	DDS	6	12/13	2/13 (15%)	1 IRBBB	1 LAH, CRBBB
					DDS + RFP	3				
					MDT	4				
LL-Tc	11	43 ± 3	7/4	5.7 ± 2.2	DDS	3	10/11	5/11 (45%)	2 IRBBB	1 LAH, VRD
					DDS + RFP	4			1 VRD	1 LAH, CRBBB
					MDT	4				
Tc	15	43 ± 2	10/5					6/15 (40%)	1 IRBBB	1 VE, VRD
									2 LAH	1 LAH, CRBBB
									1 VE	

[a] LL = Lepromatous leprosy; LL-Tc = lepromatous leprosy plus *T. cruzi* infection; Tc = *T. cruzi* infection.

[b] DDS = diaminodiphenylsulfone; RFP = rifampin; MDT = multidrug therapy.

[c] IRBBB = incomplete right bundle branch block; CRBBB = complete right bundle branch block; LAH = left anterior hemiblock; VE = ventricular extra systoles; VRD = ventricular repolarization disturbances.

were minimal in one of these cases, the other patient (a man with widespread neural involvement) had disturbances highly compatible with those recorded in CCM.

The prevalence and pattern of pathological ECG tracings in groups 2 and 3 were quite similar. No major differences as to the PR interval, heart rate and corrected QT interval were detected among the groups.

Whether lepromatous leprosy is likely to produce some degree of heart involvement appears worth exploring given the potential implications it may carry for patients living in Latin American countries where Chagas' disease is endemic.

Lepromatous leprosy has long been recognized to be associated with many autoimmune-like serological aberrations, suggestive of faulty immunoregulation, and some clinical manifestations of anti-self reactions, such as those seen during erythema nodosum leprosum episodes ([6, 10]). In this regard, and as observed in chagasic individuals ([2]), a pattern of EVI antibody (an antibody reacting against myocardial structures, endothelium, vessels and interstitium) was recently detected in some lepromatous sera (unpublished observations).

From a different standpoint, several reports of altered autonomic nerve function have been described in lepromatous leprosy, i.e., impaired responses to Valsalva's maneuver among others ([3]). Such abnormality is also present among chagasic persons, and is strongly indicative of a disturbance of the autonomic control of the heart which may contribute to the generation of arrhythmias and myocardial contractile dysfunction ([7, 12, 16]).

The evidence recorded in this preliminary sample does not seem to support the possibility of lepromatous leprosy as an aggravating element in the outcome of chronic Chagas' disease. However, when considering that the lepromatous leprosy patient with serious neural compromise showed pathological ECG tracings quite similar to the ones registered in Chagas' disease ([14]), the question arises whether lepromatous leprosy in some instances might itself affect the heart tissue.

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Histoid Leprosy in Early Macular Lepromatous Leprosy; Incidental Finding or Sign of Augmented Local Immunity?

TO THE EDITOR:

Wade first coined the term "histoid leprosy" by describing a variant of leprosy clinically characterized by cutaneous and subcutaneous nodules over an apparently normal skin, and histologically by bacillary-rich lepromas primarily exhibiting single tissue elements such as fibromas and the like ([4]). The origin of histoid leprosy is yet to be elucidated, and ever since the first description by Wade, this type of leprosy has aroused interest.

We report here an untreated case of leprosy with histoid nodules occurring along with macular lesions of active lepromatous leprosy and a macular lesion with features of regressive lepromatous leprosy.

A 29-year-old black man originating from Ghana, who had been living in Italy for 2 years, presented with a history of the appearance 3 months earlier of a small nodule on his right arm which subsequently was followed by several other nodules on his limbs and face. The nodules were of various sizes (ranging up to 0.5 cm in diameter), were skin-colored, and had a shiny surface, well-defined edges, and a hard consistency.

In addition to the nodular lesions, careful examination of the skin disclosed coppery, ill-defined macules of which the patient was unaware. The macules were present on the flanks and spared the extremities. A solitary, round, coppery macule of about 2–5 cm diameter with hyperpigmented and ill-defined margins was present on his back. His hair, eyelashes and eyebrows were preserved. General examination disclosed a subjective sensory impairment of pain over the ulnar side of the right forearm.

Skin biopsies were done on one of the nodules and on the macula on his back. Hematoxylin-eosin staining of the nodular lesion showed a pseudoencapsulated mass made up of fusiform histiocytes arranged in crisscross fashion along with polygonal and irregularly shaped histiocytes. The nodule was deep seated with borders pushing down the subcutis and pushing aside hair follicles. Fite-Faraco staining revealed numerous, well-preserved, solid-stained acid-fast bacilli (AFB) scattered either singly or in small bundles throughout the nodule. Globi were occasionally present.

Histopathology of the macular lesion on the back showed an accumulation of vacuolar histiocytes in the superficial dermis, with rare lymphocytes and scattered eosinophils. Fite-Faraco staining showed many intracellular AFB, mostly granular. A nasal swab was positive for AFB.

The diagnosis of subpolar lepromatous leprosy featuring both macular and histoid