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The Prevalence of Clinical Peripheral Neuropathies in Human Chronic Chagas Disease

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SUMMARY: Twenty-five patients suffering from chronic South American Trypanosomiasis (Chagas disease) were clinically compared with matched controls. Four of the chagasic patients provided histories of mild sensory neuropathies and on clinical examination were found to have impaired light touch sensation, vibration sense and two point discrimination. Other conditions which could have caused this neurological impairment were excluded. This is the first study to provide evidence of a detectable clinical neuropathy in chronic Chagas disease. Although this was only a limited study carried out during a medical school elective, it correlates well with the preliminary, and as yet unpublished, findings of a much larger study being carried out in Buenos Aires by Professor R E P Sica.

Introduction

South American Trypanosomiasis, or Chagas disease, is found widely in both men and animals in Central and South America. Its estimated prevalence is 20 million people of all ages and both sexes from Texas to Southern Chile. The disease is caused by *Trypanosoma cruzi* which is transmitted from mammalian hosts to man by reduviid bugs, known as assassin bugs (or vinchucas in Argentina). The bugs bite sleeping individuals to feed on blood, usually defecating at the same time. The infection is acquired by rubbing the faeces which contain trypanosomes into the wound or eyes. Transmission can also occur congenitally or be caused by blood transfusions.

The disease is normally silent although occasionally periorbital oedema (Romana's sign) or superficial chagomas may be seen followed by a mild parasitaemia and corresponding mild febrile illness. After a latent period of many years complications may occur, usually cardiomegaly with arrhythmias or megaoesophagus and megacolon. Neurological symptoms were first described by Chagas himself in 1911 (1) and peripheral nervous system involvement was demonstrated in 1943 by Couceiro (2) who believed that it corresponded to an interstitial neuritis. Subsequent work has concentrated on experimental Chagas neuropathy in murine models.

However, Sica's (3) study in 1979, using surface and needle recordings of sensory action potentials in 23 chagasic patients, showed 11 with limited action potentials and conductive velocities. Histological samples showed loss of nerve fibres, segmental demyelination and Wallerian degeneration. A further study by Sica (4) investigating ulnar nerve motor conduction velocities of chronic chagasic patients showed that a motor neuropathy could be detected in chagasic patients. He has concluded that some patients in the chronic stage of chagas disease may develop a sensory, a motor or a mixed neuropathy in the peripheral nervous system. However, despite these laboratory investigations there is little evidence of a simple clinical approach to detect the incidence of peripheral neuropathies in chronic human Chagas disease.

Patients and Methods

From 1-22 February 1991, 25 patients from the parasitology clinics at Hospital Rawson, Cordoba, Argentina, were examined. All had serologically proven Chagas disease (Machado: Guerreiro positive) and were between 20 and 60 years old. Coincidental cases of neurological disorders were eliminated by rejecting patients over 60 years of age and those having disorders known to induce nervous damage. Thirty-six patients were rejected (10 age, 14 abnormal LFTs, 3 diabetes, 7 other diseases and 2 neurological diseases). Twenty-five age/sex matched control patients were also selected and examined identically.

A complete clinical history was taken, particular attention being paid to the patients' neurological complaints and Chagas risk factors. These factors were: living in an endemic area, living in adobe houses, living in proximity with domestic carrier animals, having had blood transfusions and having family members suffering from the disease. A comprehensive clinical examination followed concentrating on their neurological systems. A scoring system graded deficits of muscle power and sensation from 1-5. Patients were selected consecutively and prior to their consultant's examination were informed that an English medical student would be carrying out further examinations.

Results

From the 25 (Chagas) patients' histories, 4 indicated diffuse and subtle symptoms of a peripheral neuropathy. Three complained of pins and needles in their hands and the other of heaviness in her hands. On examination, tone, power, reflexes and co-ordination were normal but light touch, sensation, vibration sense and two point discrimination were below normal (average score was 2/25 against 5/5 for the controls). Using Fisher's Exact Test there was no statistically significant difference between them ($p = 0.11$). Three of the patients believed they had contracted Chagas disease following blood transfusions.

Discussion

Until now no study using purely clinical methods to

detect chagasic peripheral neuropathy has been attempted. However, provisional findings from Sica's study, based on the first 200 patients of a total of 1000, suggest that 11% of those with Chagas disease have this diffuse and subtle peripheral neuropathy. These findings are further supported by EMG evidence (Sica, personal communication 1991).

Although this relatively limited medical student elective project lacks the objective support of electromyographic evidence, it can be strongly argued that the cost of EMG investigation prohibits its widespread use in Latin America outside specialist centres. In this study the differences between the chagasic patients and the controls did not reach the level of statistical significance.

However, these findings, and the preliminary results of Sica's study, further support the hypothesis that Chagas disease causes a peripheral neuropathy which can be detected by simple history taking and clinical examination. These are commodities that are readily available in most endemic areas where poverty and soaring inflation mean that expensive investigations are simply not practicable.

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