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Clinical trial of benznidazole and an immunopotentiator against Chagas disease in Chile

Twenty-one asymptomatic Chagas patients with cardiac pathology were studied in an endemic zone of Chile, the Limari river valley (APT *et al.*, 1983). Using the double blind technique they were separated into three different groups which received benznidazole, five patients (Group A), benznidazole plus obioactin, eight cases (Group B) and placebo, eight cases (Group C). Benznidazole was given in doses starting at 2 mg/kg increasing to 5 mg/kg/day by the fifth day, then continued until the 35th day (BARCLAY *et al.*, 1978; CANCADO & BRENER, 1979; APT, 1985). Obioactin is a lymphokine hydrolysate from the spleen and serum of immune animals (SUZUKI *et al.*, 1982).

Before and after treatment all patients were tested for Chagas disease by complement fixation, haemagglutination, indirect inmunofluorescence and xenodiagnosis. Heart function tests were also carried out (ARRIBADA & APT, 1980).

Although the number of treated patients was small, it was clear that there were no differences between the groups in respect of infection or heart function.

Therapy in three patients from Group A was discontinued because of skin rash. There were no haematologically or neurologically based adverse reactions among the patients of Groups A or B. In another trial involving 20 chronic Chagas patients given benznidazole at the same dosage, treatment was stopped because of a high incidence (33%) of skin rashes and neurological symptoms.

Although the exact degree of the antiparasitic effect of benznidazole could not be determined, the presence of a high incidence of side effects has prevented us from continuing studies in Chile with the drug.

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Visceral leishmaniasis complicating acquired immunodeficiency syndrome (AIDS)

We report here a case of visceral leishmaniasis (VL) in a French patient with AIDS, pancytopenia and fever. The patient, a 33-year-old bisexual man, was admitted in February 1985 for Kaposi's sarcoma with numerous skin and mucosal lesions without evidence of opportunistic infections. There was neither anaemia nor thrombocytopenia. WBC count was normal: $4{\cdot}1 \times 10^9/l$ (20% lymphocytes). The T_3 cell count was 710/μl with a decreased T_4 cell count of 170/μl, a T_8 cell count of 470/μl, and an inverted T_4/T_8 ratio of 0·35. The result of a tuberculin test was negative. Serum IgG antibodies to LAV/HTLV III were detected by enzyme-linked immunosorbent assay.

The patient was successively treated with vincristine sulphate and high doses of alpha interferon without improvement. In July 1985, he developed fever (39°C) and severe diarrhoea. Apart from an increase in skin tumour size, physical examination was negative; there was neither lymph node enlargement nor hepatosplenomegaly. *Cryptosporidium* was found in the stool. The patient was treated with spiramycin but diarrhoea persisted. Cytomegalovirus was isolated from blood and urine. Pancytopenia was present: Hb 7·8 g/dl, platelets $80 \times 10^9/l$; WBC $0{\cdot}7 \times 10^9/l$ (65% neutrophils, 32% lymphocytes, 3% monocytes). Gammaglobulin serum level was 17 g/l. Bone marrow aspirate was hypocellular and revealed *Leishmania donovani* organisms. Search for anti-leishmania antibodies was negative (immunofluorescent test). Antimony therapy was started. After 15 days, he showed resolution of fever and pancytopenia improved: Hb 9·8 g/dl, platelets $122 \times 10^9/l$, WBC $1{\cdot}0 \times 10^9/l$. He died abruptly from an acute respiratory failure three weeks after diagnosis of VL. No autopsy was performed.

Visceral leishmaniasis has been known to affect immuno-compromised patients as shown by BADORO (1986). Patients with AIDS are susceptible to infec-