VISCERAL LEISHMANIASIS IN AN ADULT WITH HIV INFECTION.

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SUMMARY

We present the first case of visceral leishmaniasis (VL) in a Spanish patient with HIV infection living in Belgium. After four weeks of stibogluconate and zidovudine treatment, the initially low CD4 count improved, and the splenomegaly regressed. VL is becoming frequently reported in association with HIV infection, especially in countries where leishmaniasis is endemic. The apparent effect of VL on the CD4 count may cause problems in the staging of HIV infections.

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INTRODUCTION.

It is 90 years since Sir William Boog Leishman first noticed heavily stained round and oval bodies in the splenic cells from a British army soldier who had been sent back to Britain from India because of attacks of pyrexia, anaemia and splenomegaly (1). Even short stays in endemic areas can cause leishmaniasis, 80% of cases of the mediterranean form occurring in children (2). The first case of adult visceral leishmaniasis (VL) in a Belgian returning from Spain was reported in 1985 (2).

Several cases of adult VL have been reported in western HIV patients over the last few years (3). We wish to report a case of VL in an HIV positive Spanish adult living in Belgium, and to draw attention to the fact that leishmaniasis may become regularly seen in western countries due to the HIV epidemic.

CASE REPORT.

A 25-year-old woman was brought in coma to our inner-city emergency room, having been found collapsed on the street. The Glasgow coma score was 11/15, needle track marks were found in both arms, and the pupils were pin-point. She had aphtous mouth ulcers with generalized lymphadenopathy and a hard spleen palpable to the umbilicus. Naloxone was given and she recovered quickly.

She had used intravenous drugs for 8 years, and had been told 5 years previously that she was HIV positive. She was of Spanish origin, but had been in Belgium for 7 months. She had lived in Spain and the south of France. She denied sharing needles or sexual contact with people of African origin. There was no past history of other illness, but she had experienced some sweats in the previous weeks.

Blood analysis showed pancytopenia (haemoglobin 75 g/L, white cells 2.5 x 10^9/L, platelets 127 x 10^9/L) with hypochromic red cells. There was marked hypergammaglobulinaemia. Renal and liver function tests were normal. HIV infection was confirmed by ELISA and Western blot. CD4 cells were low (0.18 x 10^9/L), with a

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CD4/CD8 ratio of 0.58. A bone marrow aspirate showed characteristic amastigotes of Leishmania.

She agreed to join a narcotics weaning programme, and was treated with sodium stibogluconate 20 mg/kg/day i.m. for 20 days, and zidovudine 100 mg five times daily. During this treatment the spleen size regressed and CD4 count increased dramatically (Table 1). The mouth ulcers healed spontaneously. A bone marrow examination performed 6 weeks after starting treatment was normal. After 4 months of follow-up, the patient remained well.

DISCUSSION.

This case demonstrates the coexistence of VL and HIV infection. Our patient almost certainly acquired VL in Spain or southern France, both endemic areas for Leishmania donovani infantum. An increasing number of cases of VL are now appearing in HIV positive patients in VL endemic areas (3). HIV patients may develop atypical leishmaniasis, notably of the gastrointestinal tract, and not all cases respond as readily as ours to treatment (4).

Leishmania serology was not tested in our case. Antibodies to L. donovani, which are present in approximately 95% of immunocompetent patients with VL, were absent in 66% of HIV-infected patients with VL (3). Serology appears to have a limited role in the diagnosis of VL in HIV-infected patients.

Another interesting feature is the rapid improvement of the CD4 count with treatment. VL alone, without a co-existing HIV infection, has been reported to lower CD4 counts (5). It is tempting to attribute the rapid CD4 recovery to the treatment of VL. The low absolute CD4 count may be due to hypersplenism because the increase in CD4 cells was concomitant with the regression of the splenomegaly and pancytopenia. In addition, the relative CD4 count, expressed as a percentage of the total lymphocyte count, remained constant (25%). Zidovudine can also improve the CD4 count within four weeks in patients with AIDS and ARC without VL, however the counts appear to drop again after 8 weeks (6). This may make it difficult to draw firm conclusions about the stage of HIV infection until VL has been treated. Some (7) argue that typical VL in association with HIV should be classified as Centers for Disease Control (CDC) group IVE (diseases not classifiable in other CDC groups that may be attributed to HIV infection or a defect in cell-mediated immunity), and that only severe relapsing or disseminating VL should be considered as CDC group IVC-1 (specified AIDS-defining infectious diseases) (8).

HIV infection should be considered in any patient with VL. Conversely, HIV positive patients, living in or having visited a leishmania endemic area, with fever, sweats, anaemia and splenomegaly should have a bone marrow examination for leishmaniasis.

TABLE 1. FOLLOW-UP AFTER STARTING STIBOGLUCONATE TREATMENT.

Week	0	2	4	16
Total white count (x 10^9/L)	2.5	3.3	5.7	3.7
CD4 (% of total lymphocytes)	24.1	24.7	25.7	31.9
CD4 (x 10^9/L)	0.18	0.34	0.59	0.54
Splenomegaly (cm)	18	10	Tip felt	2

SAMENVATTING

Wij stellen het eerste geval voor van viscerale leishmaniase (VL) bij een Spaanse patiente met HIV infectie wonende in Belgie. Na vier weken behandeling met stibogluconate en zidovudine, verbeterde de initiele lage CD4 celtelling, en verminderde de splenomegalie. VL wordt frequent gesignaleerd in patienten met HIV infectie, voornamelijk in landen waar leishmaniase endemisch is. Het effect van VL op de CD4 cellen kan problemen geven bij de «staging» van HIV infecties.

RÉSUMÉ

Nous décrivons le premier cas de leishmaniase viscérale chez une patiente espagnole habitant en Belgique porteuse d'une infection à VIH. Après quatre semaines de thérapie au stibogluconate et à la zidovudine, le taux des lymphocytes s'était amélioré et la splénomégalie avait régressé. La leishmaniase viscérale est fréquemment signalée comme infection opportuniste dans le SIDA, particulièrement dans les pays où la leishmaniase est endémique. L'effet de la leishmaniase viscérale sur le taux des lymphocytes CD4 peut créer des problèmes au niveau de la classification des infections par le VIH.

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