

## EPISODES OF MALARIA IN A PATIENT WITH ACQUIRED IMMUNODEFICIENCY SYNDROME

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It has been recognized that either malaria or HIV infection may cause dysfunctions of the immune system (M. Ho et al., 1986, *J. Infect. Dis.*, 153: 763-771; A. S. Fauci et al., 1988, *Science*, 239: 617-622), although HIV infection leads to immunodeficiency with higher impact in clinical feature than malaria infection. Thus, it can be anticipated that the association of both infections would further deteriorate the immune function and modify the natural history of these infections. We show in this report a patient who presented both infections but had some immune functions surprisingly preserved.

**Case Report** — A 29 years-old man bisexual drug-addict, was admitted to the Tropical Diseases Hospital of Goiânia, in Brazil, on March 1987 complaining of diarrhoea and fever for the last 30 days, and loss of 15 kg in weight. The clinical examination was essentially normal, except for the presence of bilateral inguinal lymphadenopathy and a few lesions in the glans attributable to *Herpes simplex*. T lymphocytes were moderately decreased in number (52%), and intradermal tests with recall antigens gave negative results. Anti-HIV antibodies were detected by ELISA (Nucleonics) and immunofluorescence (Prolab Merrieux). After 15 days he was stable and so he was discharged. On January 1988 he returned to hospital with the same manifestations excluding genital herpes. *Cryptosporidium* sp. was detected in his faeces. Three months later he came back from an endemic area of malaria, and presented fever, headache and abdominal pain. Physical examination was normal. Liver and spleen showed no increase in size, and GOT and GPT were 100 and 150 U/ml, respectively. *Plasmodium falciparum* was detected by thick smear (210 trophozoites/100 leukocytes) and, although he

presented lymphopenic ( $740/\text{mm}^3$ ), both spontaneous (1860 cpm) and phytohaemagglutinin-induced (58,400 cpm) lymphocyte proliferation were normal, as detected by H-thymidine. Interestingly, NK cell cytotoxicity towards the K 562 tumour cell line, as measured by the single cell assay of B. Wahlin et al. (1984, *Scand. J. Immunol.*, 19: 529-539) showed an increased activity (6.02% as compared with 0.51% in the paired control). Quinine cleared the parasitaemia in 3 days and the patient was discharged. The patient remained in a non endemic area of malaria and one month later (May 1988) *P. vivax* infection was diagnosed during an episode of high fever, chills and headache. The low parasitaemia (25 trophozoites/100 leukocytes) was quickly cleared after the third day of therapy with chloroquine, and, though he had taken a 14-day schedule of primaquine he relapsed twice, 4 and 5 months later.

**Comments** — Although no change in the clinical course of malaria has been reported in areas of high prevalence of HIV infection (R. J. Biggar et al., 1986, *Br. Med. J.*, 293: 1453-1454), a fatal case of *P. vivax* infection with high parasitaemia has recently been published (E. Katongole-Mbidde et al., 1988, *Brit. Med. J.*, 296: 827). In the case herein described no apparent interference between HIV infection and *P. falciparum* malaria occurred. However, although other causes should be considered, the possibility that *P. vivax* relapses in a patient properly treated with chloroquine and primaquine, could have been influenced by HIV infection cannot be ruled out. Malaria, in its turn, does not seem to alter the clinical features of AIDS, at least during the course of the *Plasmodium* infection. However, since the immunological follow-up of the patient was not done during and after each malaria infection it remains to be elucidated if malaria infection can temporarily

alter the immune functions of the HIV infected organism.

Mostly surprisingly was the finding that either spontaneous and mitogen-induced lymphocyte proliferation, or NK-cell activity, reported to be depressed in AIDS (A. S. Fauci

et al., 1988, *loc. cit.*; J. D. Katz et al., 1987, *J. Immunol.*, 139: 55-60) and malaria (M. Troye-Blomberg et al., 1983, *Clin. Exp. Immunol.*, 53: 335-344) were normal in the presence of both infections. Further longitudinal studies in patients with both AIDS and malaria are needed to understand the real interaction between these infections.