

CEREBRAL TOXOPLASMOSIS AND AIDS: DIAGNOSTIC CRITERIA (Abstract)*.

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Human immunodeficiency virus (HIV) infection causes the acquired immune deficiency syndrome (AIDS), and 20% to 40% of the patients present involvement of the central or peripheral nervous system that become manifest by several signs and/or symptoms. Cerebral toxoplasmosis is the most common cause of central nervous system (CNS) focal lesions determined by opportunistic agents in AIDS. Therefore since the beginning of HIV infection or in AIDS, patients with antibodies to *Toxoplasma gondii* shall be considered under risk for developing cerebral toxoplasmosis. Although immunologic tests do not distinguish active or latent infection, early specific treatment must be started. The clinical examination shows focal or diffuse signs and/or symptoms in cerebral toxoplasmosis. Neuroimage findings may highly suggest the parenchymal infection by *Toxoplasma*. However, the definitive diagnosis can only be made by the protozoan demonstration in the brain tissue. Patients that do not respond clinically or on neuroimage to toxoplasmosis treatment must be evaluated regarding other causative opportunistic agents.

From April 1990 to July 1995, 95 HIV-1 infected patients engaged at "Hospital Universitário Antonio Pedro, Universidade Federal Fluminense" were studied. The diagnosis of AIDS was established according the diagnostic criteria of the Centers for Disease Control (CDC). In 25 of them, with focal or diffuse neurological signs and/or symptoms, a probable diagnosis of cerebral toxoplasmosis was made on the basis of clinical presentation, blood and cerebrospinal (CSF) immunologic tests for toxoplasmosis, neuroimage findings and treatment response. They are analysed in the present study.

Twenty-three patients were male and 2 female, ranging in age from 21 to 48 years. Twenty-two were infected by sexual transmission: 15 were homosexuals, 3 heterosexuals, 2 bisexuals, 1 parenteral drug abuser and bisexual. One was drug abuser, and in the 2 others the cause of transmission was not identified. First neurological manifestation in the majority of patients was generalized or focal seizure followed by motor deficit.

Serum IgG and IgM anti-toxoplasma antibodies were tested in the serum of 21 patients. IgG test was positive in 19 patients and IgM test was negative in all.

Lumbar puncture was performed in 15 patients. IgG anti-toxoplasma antibodies test was positive in the CSF of 6 patients and was negative in 9.

CT scan was performed in the initial stage of neurological symptoms in 12 patients. It showed focal lesions in 11 patients and was normal in 1. Single lesions predominated. Multiple lesions were predominantly unilateral. Half were with surrounding edema, in 2 cases with mass effect. Contrast retention occurred in the majority of cases. Predominant location of lesions was cortical, corticomedullar and in the basal ganglia.

Seventeen patients received the conservative treatment, a combination of pyrimethamine and sulfadiazine. Eight patients received the alternative treatment, pyrimethamine and clindamycin for 4 weeks.

The response to therapy was evaluated by clinical and neuroimage improvement. Thirteen patients improved clinically with the conservative treatment and 2 with the alternative treatment.

The maintenance treatment was evaluated in 22 patients. Clinical improvement was observed in 6 of the 12 patients who received the conservative treatment, and only in 1 of the 10 who received the alternative treatment.

Comparison of therapeutics and neuroimage findings in the 25 patients showed improvement among patients who received the conservative treatment, while the majority of patients who received the alternative treatment did not present satisfactory outcome, with worsening of images..

In conclusion, the used criteria were effective to presumptive diagnosis of cerebral toxoplasmosis, on the basis of patient's clinical and neuroimage improvement.

KEY WORDS: AIDS, cerebral toxoplasmosis, diagnosis, treatment.

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