INTRODUCTION

Toxoplasma gondii is an obligate intracellular protozoan of worldwide distribution. It exists in three forms: tachyzoite, tissue cyst (containing bradyzoites), and oocyst (containing sporozoites). The asexual life cycle takes place in all intermediary hosts (including humans) as well as in felines. Ingestion of tissue cysts or oocysts is followed by infection of intestinal epithelial cells by bradyzoites. After transformation into tachyzoites, the organisms disseminate throughout the body via the blood or lymphatics. The parasite forms tissue pseudocysts once it reaches muscle and neural tissue. Oocysts become infectious after they are excreted and sporulation occurs. Transmission to humans occurs primarily by ingestion of undercooked pork or lamb meat that contains tissue cysts, or by exposure to oocysts either through ingestion of contaminated vegetables or direct contact with cat feces. Other modes of transmission include the transplacental route, blood product transfusion, and organ transplantation. Cerebral toxoplasmosis is a major cause of morbidity and mortality in the acquired immunodeficiency syndrome. Intracerebral hemorrhage is a rare complication of AIDS, and there are only a few reported cases of cerebral hemorrhage in toxoplasmosis.

CASE REPORT

We describe the case of a 59 year old female with history of chronic alcoholism who, suddenly, presented incoherent speech, visual hallucinations, generalized weakness and excessive sleepiness. A heat computer tomography scan revealed bilateral intracranial basal ganglia hemorrhages. A reagent tests were positive and neurosyphilis was included in the possible diagnoses. Death occurred on the 6th hospital day and the autopsy was requested to rule-out neurosyphilis. Received post-mortem, the anti-HIV- antibodies and FTA ABS were positive. An autopsy was performed. The brain weighed 1270 g. On coronal sections of the formalin-fixed brain, bilateral hemorrhagic lesions located in the basal ganglia were identified. Both lesions measured 2.5 cm in greatest dimension and had dark red areas of discoloration associated with older hemorrhages. Other smaller lesions were present in the right thalamic nuclei, hippocampi and occipital cortex (fig.1). Histologically, these areas presented hemorrhages, perivascular and vascular wall lymphocytic infiltrate as well as multiple pseudocysts with bradyzoites and tachyzoites around the pseudocysts. Toxoplasma gondii immunohistochemistry confirmed the diagnosis (fig. 2,3,4,5,6,7).

Discussion

Intracerebral hemorrhage represents a rare complication of AIDS. Possible causes are cytomegalovirus infection, cerebral infarction following nonbacterial thrombotic endocarditis, or (in rare cases) vasculitis, mycotic aneurysm, hemophilia, thrombocytopenia, hemorrhagic metastatic disease (Kaposi sarcoma), and cerebral lymphoma. Microhemorrhages and erythrodiapedesis are frequent findings in acute Toxoplasma encephalitis. Direct parasitism of the endothelium with mass destruction of cells or vasculitis with extensive vessel wall necrosis, or concomitant bleeding disorders, provide a possible explanation for hemorrhage in cerebral toxoplasmosis. This case shows bilateral, mirror image, basal ganglia hemorrhages as an extremely rare presentation of Toxoplasma encephalitis. Toxoplasma encephalitis should be in the differential diagnosis of intracerebral hemorrhage in AIDS.

References

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