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TRYPANOSOMA CRUZI MENINGOENCEPHALITIS IN AIDS MIMICKING CEREBRAL METASTASES

CASE REPORT

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ABSTRACT - A case of Chagas' meningoencephalitis in a 47 year-old patient with AIDS is presented. The diagnosis was established by examination of the cerebrospinal fluid which showed the presence of *Trypanosoma cruzi*. CT-scan revealed multiple cerebral lesions which only became evident by contrast administration. A second CT after 13 days showed a severe evolution with displacement of the midline structures leading to the patient's death. This case, as well as other case reports published, has shown the fulminant evolution of Chagas' meningoencephalitis in AIDS patients and the importance of cerebrospinal fluid examination for the diagnosis.

KEY WORDS: cerebral metastases, encephalitis, Chagas' disease, Trypanosoma cruzi, AIDS.

Meningoencefalite chagásica em AIDS simulando metástases cerebrais: relato de caso

RESUMO - Apresentamos um caso de meningoencefalite chagásica em paciente de 47 anos com AIDS. O diagnóstico foi feito pelo encontro do *Trypanosoma cruzi* no LCR. A tomografia de crânio mostrou múltiplas lesões cerebrais que se tornaram evidentes apenas na fase com contraste. Uma segunda tomografia após 13 dias mostrou evolução tipo tumoral com intenso efeito de massa e acentuado desvio de estruturas da linha média, levando o paciente ao óbito. Este caso e outros da literatura mostram a evolução fulminante da meningoencefalite chagásica em pacientes com AIDS e a importância do exame de LCR para o diagnóstico.

PALAVRAS-CHAVE: metástases cerebrais, encefalite, doença de Chagas, Trypanosoma cruzi, AIDS.

Chagas' disease (CD) is an infection with multisystemic involvement and with an initial acute phase in which fever, node enlargement, hepatoesplenomegaly, cutaneous rash and meningoencephalitis may occur usually without serious consequences. After this initial phase the disease has a slow silent course towards chronicity and only after many years the clinical signs of cardiac and digestive tract disorder start to appear, being the predominant manifestation of the chronic phase of CD. The etiologic agent of this infection is the *Trypanosoma cruzi* (T.cruzi), transmitted to humans and animals via a hematophagus insect of the Reduviidae family (reduviid bug). The T. cruzi has a complex biological cycle with mobile flagellate forms called epimastigotes and trypamastigotes and nonmobile aflagellate forms called amastigotes. The flagellate epimastigote and trypomastigote forms circulate in the blood and the nonmobile amastigotes live inside the tissue cells. When the hematophagus vector sucks an infected person or animal, along with the blood it ingests circulating forms which thus reach its digestive tract. During the passage through the intestine, these circulating forms of the parasite undergo transformations reaching the hindgut as infective forms. At the moment

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the hematophagus vector sucks, penetrating the victim's skin, with the feces it eliminates infective forms which are in the hindgut. These infective forms deposited on the skin penetrate the body through a wound in the skin which may have been caused by the hematophagus insect bite itself or even through the intact mucosa. After penetrating the body, the parasite lodges in cells, replicating untill the cells rupture with release of trypanosomas (trypomastigotes) into circulation and diffusion throughout the body.

The purpose of this report is the presentation of one more fatal case of meningoencephalitis by the opportunistic reactivation of the T. cruzi in an Aids patient with an analysis of the evolving changes

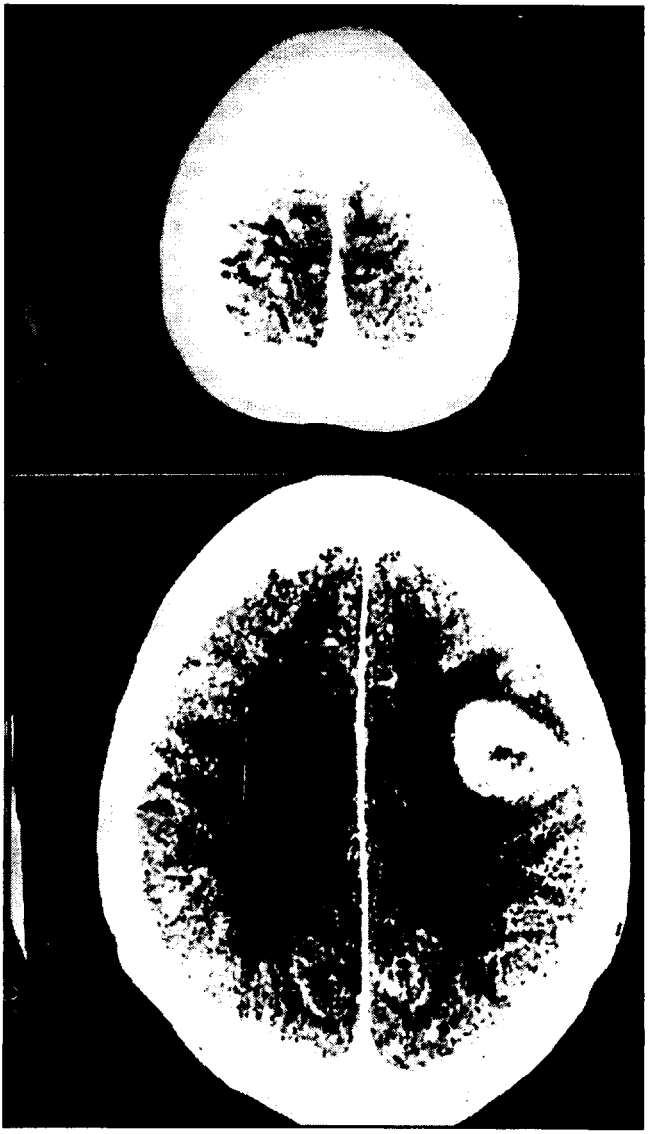

Fig 1. Contrast enhanced axial CT scan shows multiple nodular lesions, the largest one located in the left frontoparietal region.

in two cranial tomographies and also the confirmation of the importance of the cerebrospinal fluid (CSF) for this diagnosis.

CASE REPORT

A 47 years-old man was examined on January 6,1992 presenting some difficulties regarding speech and swallowing and a progressive general weakness. Two months before he had a facial paralysis, diagnosed as being peripheral by a physician. Having received corticoid, he recovered completely from this paralysis in 20 days. Seven months earlier he had suffered a benign gastroduodenal ulcer surgery. Blood transfusion, multiple sexual partners and drug abuse were denied. On neurological examination he was conscious and lucid. There was a slight left distal brachial paresis and on the right a crural and central facial paresis. Muscle stretch reflexes were all absent and the plantar response was extensor on the right side. There was mild dysartric speech. Thus the neurological test suggested a multifocal involvement of the nervous system. A cranial tomography (CT) on January 8 showed multiple nodular lesions in the cerebral hemispheres evidenced only at the contrast phase, except for the largest lesion with 3.5cm in the greatest diameter, located in the left frontoparietal transition, which could be seen in the pre-constrast phase as a slight hypodensity (Fig 1). The patient initially refused to be submitted to CSF examination, and by recommendation of another physician he started receiving dexamethasone 4 mg twice a day. After 4 days, dexamethasone was discontinued and a CSF examination on January 14 showed the presence of the T. cruzi with intense motion (Fig 2). There were 18 cells (lymphocytes 78%, monocytes 17%, plasmocytes 3%, neutrophils 2%; proteins 53 mg/dL, glucose 77 mg/dL. Enzyme linked immunoabsorbent (ELISA) and immunofluorescent assays in CSF and serum were positive for the T. cruzi antibody. Passive hemagglutination titers were 1/6 in CSF and 1/640 in serum. T. cruzi was not found in blood. HIV-1 antibody test was then performed and was positive in both blood and CSF. The patient refused to go to the hospital, but on January 21 he was hospitalized because of his severe clinical condition. At that time he did not react to any verbal stimuli, but he could move his left limbs both spontaneously and on painful stimuli. His pupils, about 3 mm, were photoreactive and corneal reflexes were present.

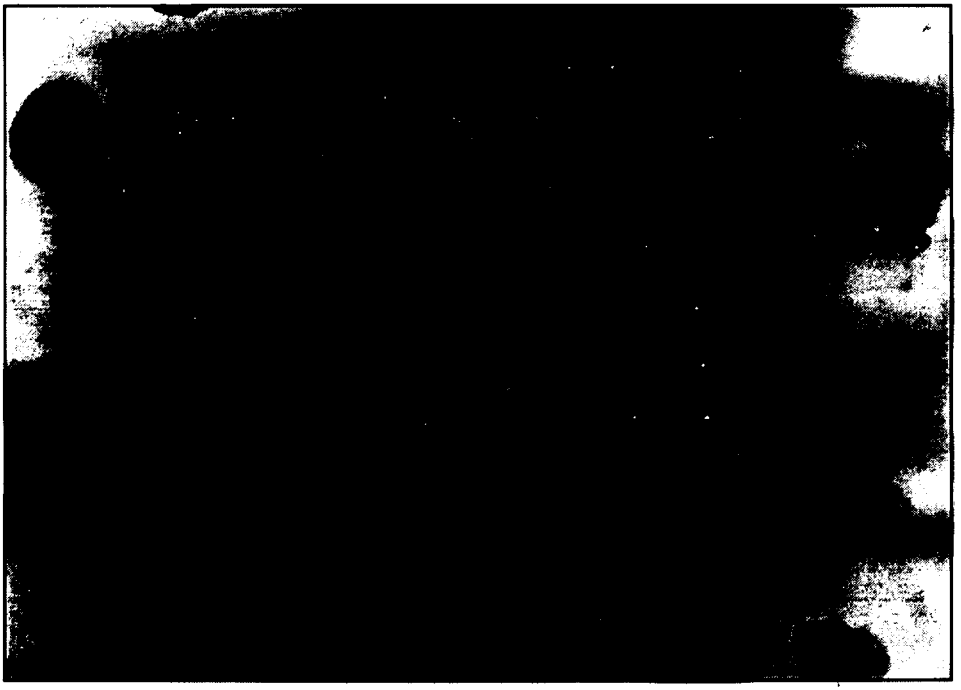

Fig 2. Trypomastigote forms of T. cruzi found in CSF.

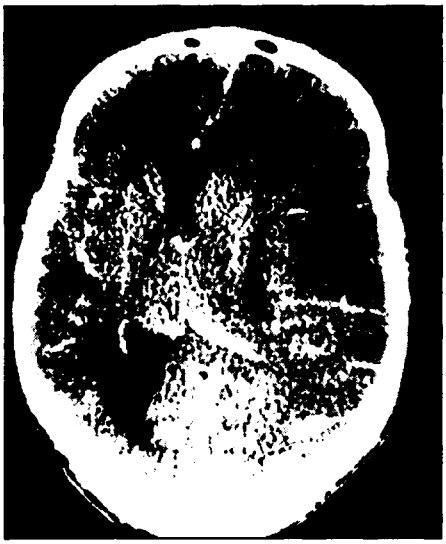

Fig 3. Control CT-scan. Description in the text.

A new CT, on the hospitalization day displayed (Fig 3): irregular enhancement delineating cortical sulci and basal cisterns suggesting leptomeningeal involvement. The nodular lesions seen at the initial CT were less evident even in the late post-contrast images. There was a significant increase of the edema surrounding the fronto parietal lesions causing compression of the left lateral ventricle and third ventricle, with a midline shift to the right and the occupation of the left side of the suprasellar and ambiens cisterns by the adjacent temporal uncus and parahippocampal gyrus (uncal herniation). The patient was treated with benznidazole and manitol in addition to clinical support, but he had a rapid evolution and a few hours later was totally unresponsive. The pupils became fixed and dilated and corneal reflexes were absent. Death occurred two days later caused by cardiorespiratory failure.

DISCUSSION

The presence of the T. cruzi in the CSF in the acute phase of CD in patients without neurological symptoms was proved by Pedreira de Freitas and col.[13] (1953), and Hoff and col.[4] (1978), but what occurs with the parasite in the central nervous system after this initial passage is still unknown. The natural evolution of CD shows that after a silent ten-to-twenty-year long period, the late significant chronicity manifestations are restricted to myocarditis, megaesophagus and megacolon. In the chronic phase of CD the involvement of CNS has occurred in acute reactivations of the T. cruzi in patients suffering from immunodeficiency and under immunosuppressant treatment[5,6,9-11], although in 1973 Queiroz[14] had already reported a tumor-like Chagas' lesion of the brain in a patient without immunodeficiency. Since 1990 there have been publications about acute menigoencephalitis deriving from reactivations of the T. cruzi in patients with HIV acquired immunodeficiency[1-3,12,16,17].

With the increase of the population suffering from immunodeficiency, we believe that this participation of the CNS infestation will increase in edemic areas and in other regions due to immigration[7,8]. It has also been noted through published reports that there has been a delay in diagnoses, which are finally made through biopsies, pathological examination of surgery material or even autopsies[1,3,6,10-12,16]. On the other hand, the early treatment with nifurtimox[10], benznidazol[6] and itraconazol[12] has had a good therapeutical response, making this diagnosis very important and for this the CSF[4,18] is a useful tool.

In our case the CT showed multiple lesions which could be metastases, granulomas or abscesses, and the diagnosis was established by the CSF, with no time for therapeutical response. The second CT, 13 days after the first, showed that the process ceased to be circumscribed with leptomeningeal dissemination, becoming encephalitic pseudo-tumoral in form, with signs of severe intracranial hypertension and subfacial and uncal herniations which led to the patient's death. We think that the steroid taken by this patient may have contributed to this rapid-type evolution.

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