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## Neurocysticercosis as an important differential of paradoxical response during antituberculosis therapy in HIV–negative patient

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### ABSTRACT

Neurocysticercosis can simulate a paradoxical response during antituberculosis therapy with neurological ailments. We report the case of a 31 year–old–man, treated for tuberculous meningitis who developed neurological deficit after nine weeks of early antituberculous therapy. The diagnosis of neurocysticercosis was confirmed by CT scan and cerebrospinal fluid analysis. Neurocysticercosis should be sought as an important differential of paradoxical response during antituberculosis therapy.

## 1. Introduction

Neurocysticercosis is the presence of the larvae of *Taenia solium* in the central nervous system<sup>[1,2]</sup>. A paradoxical response (PR) during antituberculosis therapy in non–HIV–infected patients is defined as the clinical or radiological worsening of pre–existing tuberculosis lesions or the development of new lesions in a patient who initially improves<sup>[3,4]</sup>. It usually occurs at least 2 weeks after the initiation of antituberculosis therapy. Cerebral involvement in these two pathologies gives similar neurological symptoms<sup>[1,3,5,6]</sup>. We report a case of neurocysticercosis mimicking a paradoxical response in antituberculosis therapy in non–HIV–infected patients.

## 2. Case report

A 31 year–old–man was admitted to the hospital with

confusion, headache and vomiting developed for one week. He had a temperature of 39 °C, and a Glasgow coma score of 9/15. He presented nuchal rigidity, Kernig's sign, and Brudzinski's sign, without neurological deficits. HIV status was negative. Analysis of a cerebrospinal fluid (CSF) sample obtained by lumbar puncture was consistent with tuberculous meningitis. He received corticosteroids, and 2 months of induction of antituberculosis therapy with rifampicin, isoniazid, pyrazinamid, and ethambutol, followed by maintenance therapy with isoniazid and ethambutol. By the end of 2 weeks he was asymptomatic and was discharged from the hospital.

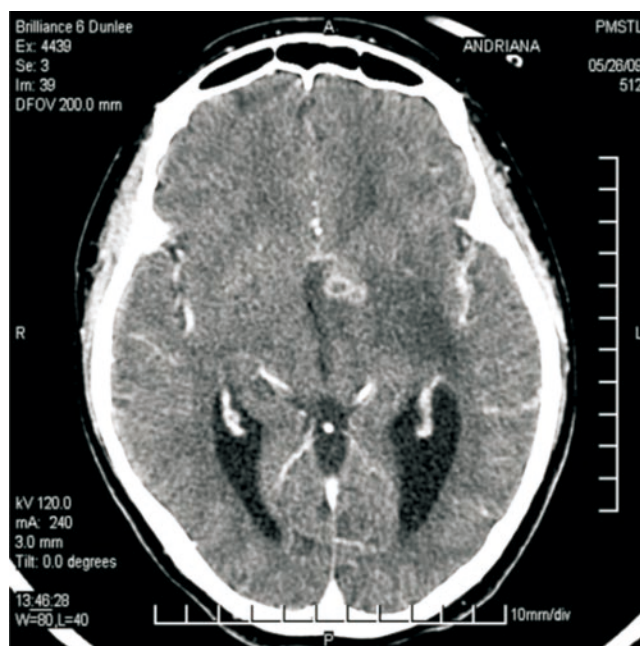
Seven weeks later, he was readmitted to the hospital with blurred vision and dysarthria, without fever. He presented monoparesis of the right upper limb and a right homonymous hemianopia. The rest of his physical examination was unremarkable. HIV test, serological syphilis, cysticercosis and toxoplasmosis were negative. Laboratory testing of CSF samples obtained by lumbar puncture revealed 54 WBCs/ $\mu$ L with 95% lymphocytes, a protein level of 87 mg/dL, and a glucose level of 258 mg/dL. Direct examination and CSF cultures for bacteria were sterile. CSF ELISA for detection of anticysticercal antibodies was positive with the presence of 13 and 14 kD antigens

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in Western blot. CT scan of cranium with contrast showed two ring-enhancing lesions 6 and 8 mm in diameter in the left capsulo-lenticular (Figure 1), with surrounding large perilesional oedema causing mass effect on the anterior horn of the lateral ventricle. Antituberculosis therapy was not changed. Neurocysticercosis was treated by albendazole (15 mg/kg/day) for 10 days and corticosteroids (1 mg/kg/day) for 7 days. The patient was asymptomatic after 4 weeks.



**Figure 1.** CT scan of cranium with contrast. Two ring-enhancing lesions 6 and 8 mm in diameter in left capsulo-lenticular with oedema.

### 3. Discussion

PR is identified in 6%–30% of non-HIV-infected patients receiving antituberculosis therapy[2]. It affects the central nervous system in 49% of cases and 73% of those with intracranial tuberculomas[2]. The tuberculomas are secondary to tuberculous meningitis in 39%–94% of cases[5,6] and occur within 2 to 3 months[2,7]. For our patient, although the onset of neurological symptoms 9 weeks after the beginning of tuberculous meningitis, treatment was evocative of PR[7–9]. The diagnosis of PR cannot be accepted without investigating the inefficiency of antituberculosis therapy and the presence of another concurrent infection[2,10]. Neurocysticercosis was raised here. Its diagnosis is based on the criteria proposed by Garcia *et al*[8]. Cysticercosis is endemic to Madagascar[6]. The ring-enhancing lesion in CT scan may show at the same time intracranial tuberculoma[7,11–13] and neurocysticercosis[1,6]. Finally, neurocysticercosis was selected as a definitive diagnosis[14] to the presence of a major criterion (lesions highly suggestive of neurocysticercosis), two minor criteria (clinical manifestations suggestive of neurocysticercosis and positive CSF ELISA for detection of anticysticercal antibodies) and an epidemiological criterion (individuals living in an area where cysticercosis is endemic).

PR during antituberculosis therapy in HIV-negative patients is a diagnosis of exclusion. In endemic countries, cysticercosis should be ruled out if there are neurological symptoms.

### Conflict of interest statement

We declare that we have no conflict of interest.

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