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COMMENTARY

Devastating yet treatable complication of tuberculous meningitis: the resistant TB abscess

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Neurotuberculosis (NTB) is a persistent disease in developing countries and its morbidity and mortality remain high in children, especially when there is a delay in diagnosis. NTB may be present mainly as tuberculous meningitis (TBM) or tuberculomas. TBM is the most common form of the disease in children and characteristically takes the form of a basal exudate seen in CT scan as a hyperdense lesion as shown by the authors. In this form, hydrocephalus is a common and often troublesome complication. Tuberculomas are less frequently seen and appear as isolated or multiple granulomatous lesions containing caseous material. Tuberculous abscesses (TBA) are encapsulated collections of pus containing viable acid-fast bacilli [3], are a rare form of NTB, and must be differentiated from tubercular granuloma with central caseation or necrosis [7]. The case of a patient with TBM who initially responded to a standard four-drug regimen plus corticosteroids, but subsequently deteriorated, is presented.

This unusual evolution can be seen in two situations: paradoxical progression or multidrug-resistant tuberculosis (MDR-TBM). In the first case, lesions increase in size and number despite the drug susceptibility and adequate adherence to treatment [1, 6]. Late deterioration is ascribed to enlargement of preexisting lesions, development of new lesions, or hydrocephalus. These lesions usually resolve themselves over time, without changing the drugs regimen, although in some cases surgical excision is recommended [4, 7]. These phenomena can develop for even up to 1 year [1, 4]. Multidrug-resistant tuberculosis has become a worldwide problem and is often associated with a grave outcome. It is defined as resistance to at least isoniazid and rifampicin. It can be due to an "acquired" resistance or to reinfection with MDR strains, especially if in the household there is an adult source with drug-resistant tuberculosis [8]. More information regarding the bacteriological and epidemiological data would be useful in this case.

There is no mention about HIV screening of this patient. It is known that AIDS-related tuberculosis is more prevalent and the incidence of MDR-TBM in HIV infection is very high. Additionally, in the AIDS era, paradoxical reactions have been related to concurrent administration of highly active antiretroviral therapy with antituberculous therapy [5]. Moreover, in the case of associated HIV infection, a differential diagnosis with other ring-enhanced lesions is mandatory, especially when dealing with refractory cases.

Concerning the brain atrophy and the diffuse ischemic lesion, it is known that basal exudates seen in NTB can entrap and even occlude arteries of the circle of Willis and inflammatory vasculitis can induce focal and diffuse brain changes as well [2]. It is not usual for patients with status epilepticus present with such a severe anoxic lesion and in the present case one must consider TBM-induced vascular changes at least as an important adjuvant factor.

We agree with the authors that ring-enhancing lesions that do not respond to specific therapy should be explored in order to reduce complications, improve the prognosis, and eventually make evident other unexpected intracranial lesions.

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