CEREBELLAR CRYPTOCOCCOMA SIMULATING METASTATIC NEOPLASM

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Neurocryptococcosis is a common mycotic central nervous system infection caused by the neurotropic fungus *Cryptococcus neoformans*[1]. It is most frequently found as the pathological presentation of cryptococcal meningoencephalitis[1]. Other rare forms of central nervous system cryptococcal infections are their solid-granulomatous appearance and abscesses, both being commonly named neurocryptococcomas, because of the mass effect they cause in surrounding tissue and their "tumoral aspect", many times simulating a brain tumor. Alternatively, Nucci et al.[2] described the unique report of a pseudocystic form of neurocryptococcosis. These medical conditions were classically described in immunocompromised hosts, mainly in HIV/AIDS infected patients.

However, more recently, such disease has also been found in HIV-negative, immunocompetent individuals[1,3,4]. HIV-negative patients who were diagnosed with neurocryptococcoma had frequently other predisposing conditions to immunologic dysfunction such as sarcoidosis, diabetes, recent use of glucocorticoid or other immunossupresive therapy, as pointed by Gologorsky et al.[4].

CASE

A 46 year-old male patient was admitted to the emergency room complaining of about two months of occipital headache, associated with nausea and vomiting in the last two days. Altered mental status had already been detected by his family within the last twelve hours before admittance. On neurological examination there was mild confusion and Glasgow coma scale score was 13. The patient was submitted to a contrast enhanced head computed tomography (CT) scan, which revealed a left cerebellar hemispheric tumor-like lesion with ring-like contrast enhancement, measuring about 2.8 cm on its major diameter. The lesion presented with surrounding edema, which caused fourth

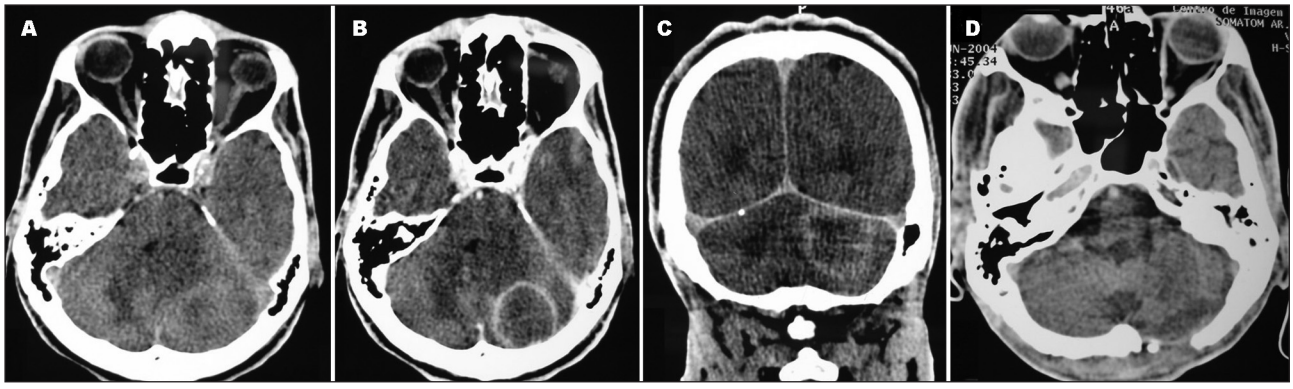

Fig 1. Head CT scans showed a contrast-enhanced mass effect lesion on the left cerebellar hemisphere superiorly limited by the tentorium and remembering a metastatic neoplasm [A, B and C]. Complete resection of the lesion by a suboccipital craniectomy is shown in Fig 1D.

CRIPTOCOCOMA CEREBELAR SIMULANDO NEOPLASIA METASTÁTICA.

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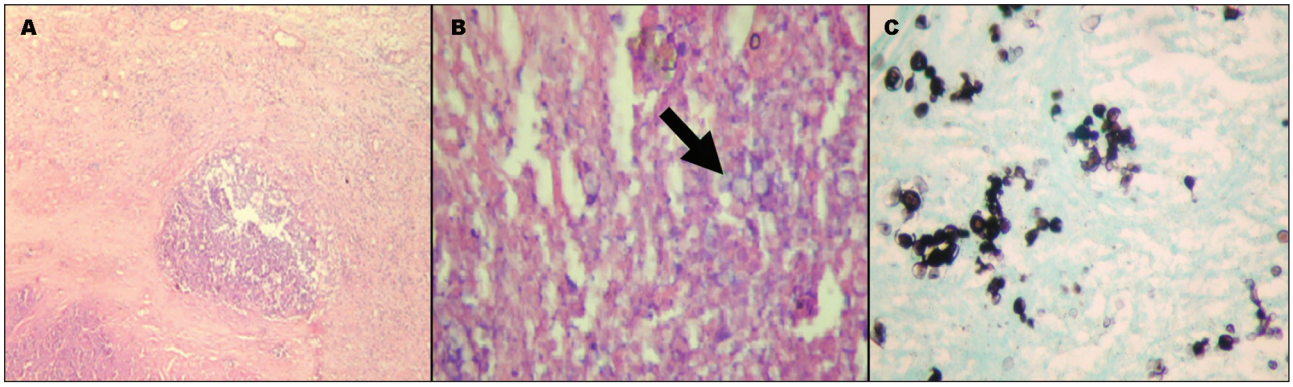

Fig 2. Histopathological analysis of the mass ressected with the haematoxilin and eosin staining [A and B] and Grocott staining [C]. They revealed the presence of Criptoccocus neoformans as indicated by the arrow [B] and confirmed as dark-colored yeasts on the Grocott staining. Magnification: [A=50x], [B and C=400x].

ventricle displacement (Figs 1A, 1B and 1C, coronal reconstruction) and consequent obstructive hydrocephalus (not shown). Previous medical history revealed the diagnosis of pulmonary sarcoidosis and tuberculosis (the last one completely treated ten years before). Tests for HIV infection were consistently negative. Our initial diagnostic hypothesis pointed to a cerebellar metastatic lesion or even a piogenic cerebellar abscess. Because of the fast evolution of the symptoms, and the risk of worsening his neurologic status, he was submitted to urgent surgical evacuation of the mass lesion by a suboccipital craniectomy with dural grafting of galea and peroperative external ventricular draining. After a brief cerebellar corticectomy, a fibroelastic brownish capsulated solid mass lesion could be reached and completely resected. Patient obtained immediate neurological improvement few hours after surgery, as well as good relief of all the symptoms. Figure 1D shows the enhanced head CT scan performed on the 60th postoperative day, revealing complete mass resection, no ventricle displacement and no signs of residual disease. Pathological examination of mass revealed the presence of yeasts on haematoxylin and eosin staining (Figs 2A and 2B), later confirmed with Grocott staining as *Criptococcus neoformans* (Fig 2C). Therapy was complemented with intravenous dexametasone and amphotericin B plus oral flucytosine during four weeks, followed by a course of fluconazole for 6 months. No signs of residual disease were detected so far.

Approval of this study was granted by the Ethics Committee of Hospital Municipal Souza Aguiar and patient written informed consent was obtained.

DISCUSSION

Neurocryptococcomas are definitely rare lesions. Dubey et al.[5] have reported 40 cases of granulomatous mycotic brain lesions collected in 23 year-time span. In this series, the authors referenced only 3 cases (7 %) caused by *C. neoformans*. Consequently, posterior fossa cryptococcoma must be an even more uncommon lesion. Isolated cases of posterior fossa cryptococcomas have been reported in both immunosupressed[6] and immunocompetetent individuals[2,4,6]. Gologorsky et al.[4] reported a case of a cryptococcal abscess successfully operated in an imunocompetent child. Kanaly et al.[7] also carried out surgical treatment of a patient with cerebellar pseudotumoral cryptococcosis masquerading as a metastatic brain tumor. On the other hand, Nucci et al.[2] reported in Brazil a pseudocystic form of cryptococcosis affecting brainstem and cerebellum and ultimately causing the death of a pregnant HIV-negative patient.

In this case, we have reported a solid cerebellar cryptococcoma of nearly 3 cm on its major diameter and significant surrounding edema, enough to cause fourth ventricle and brainstem displacements, acute hydrocephalus and rapid neurological deterioration in a patient with previous history of chronic infeccious-inflamatory pulmonary disease (sarcoidosis and tuberculosis). This was the first case of a solid cryptococcal mass lesion reported in Brazil, thus confirming the rarity of this pathology. Aside the presence of the solid component of such lesions, the inflammatory and edematous component may be a significant factor contributing to mass effect and neural structures displacement in the narrow and limited space of posterior fossa.

Setting a preoperative diagnosis of such fungal infection may be very difficult in the emergency room because of the absence of specific radiological landmarks of neurocryptococcoma, the possible radiological similarity with neoplastic (metastatic) disease (as in the case reported) and mainly the difficulties in obtaining cerebrospinal fluid samples in a patient with posterior fossa mass lesion, because of the risk of downward transtentorial herniation. Therefore, such fungal lesions placed in posterior fossa, in our opinion, may be considered as "neurooncological emergencies" and should be approached like this. Even if the diagnosis of an infectious disease could be obtained preoperatively, we strongly suggest urgent surgical evacuation of any symptomatic mass effect pseudotumoral my-

cosis (as cryptococcoma) of posterior fossa, considering the following: the imminent risk of death by tonsilar herniation; slow response to antimicrobial therapy alone and the rapid neurological improvement obtained by the patient in the case presented. Antimicrobial therapy should be considered as an adjunctive to surgery, and we believe it should be initiated in the early postoperative period if there is surgical suspicion on any infection disease or after pathological examination, if indicated.

In conclusion, posterior fossa cryptococcomas are rare lesions and its mass effect and edematous/inflammatory component may contribute to a rapid neurological decline. In spite of that, urgent surgical approach may result in good outcomes, as observed in the case reported.

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