Extensive Extraaxial Blastomycosis Granuloma at the Skull Base

—Case Report—

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Abstract

A 37-year-old female presented with a rare case of intradural, extraaxial *Blastomyces dermatitidis* granuloma involving a large part of the skull base. She had the principal complaint of worsening vision, but was otherwise healthy. The lesion mimicked an en-plaque meningioma on radiological examination and in gross appearance during surgery. This is a very unusual presentation for a blastomycosis granuloma.

Key words: blastomycosis, granuloma, cranial base, amphotericin B

Introduction

Fungal infections of the brain are rare, and usually occur as opportunistic infections in immunosuppressed individuals.⁷⁾ In contrast, blastomycosis infection has been uniformly recorded not to have a predilection for immunocompromised patients. *Blastomyces dermatitidis*, a dimorphic fungus, is predominantly found in the center of the North American continent.⁶⁾ Sporadic cases from other parts of the world have also been reported. Infection of the central nervous system is frequently a secondary manifestation of the systemic disease, possibly due to hematogenous spread of infection to the brain.²⁾ Chronic meningitis is the more frequent form of presentation,^{2,4,6)} but cerebral and extradural abscesses, intraparenchymal granulomas, and cerebritis have also been reported.^{2,7,10)}

We report a case of intradural-extraaxial blastomycosis granuloma with widespread extensions in the base of the brain.

Case Report

A 37-year-old female was admitted with complaints of progressive diminution of vision in both eyes for

about 7 months. Neurological examination showed her vision was reduced to finger counting at 2 feet in the right eye and 3 feet in the temporal field in the left. There was bilateral primary optic atrophy. There were no other neurological abnormalities. The general health was good and there was no past history which suggested immunocompromise.

Routine hematological examination including total and differential leukocyte counts and erythrocyte sedimentation rate were within the normal ranges. Computed tomography (CT) and magnetic resonance (MR) imaging showed a markedly enhanced tumor. The lesion had spread widely in the base of the skull and had involved the suprasellar region, engulfed both carotid arteries, displaced the pituitary stalk posteriorly, and extended anterior to the pons and the medulla (Fig. 1).

The larger and symptomatic suprasellar part of the tumor was treated surgically through a basal bifrontal craniotomy. The entirely extraaxial, moderately vascular, fleshy, and firm tumor engulfing both optic nerves and the supraclinoid carotid arteries was excised by working from both sides of the falx. There was no visible connection of the suprasellar component of the tumor with the part anterior to the brainstem. The intraoperative appearance of the tumor consistency, vascularity, and proximity to the dura suggested a meningioma.

Histological examination of the specimen showed

Fig. 1 T₁-weighted MR image with enhancement showing the large suprasellar lesion encasing both carotid arteries (left). T₁-weighted MR image showing the isointense retroclival lesion (right).

Fig. 2 Photomicrograph showing broad-based yeast-like bodies amidst inflammatory granulation tissue (upper: HE stain, ×400). Photomicrograph after GMS staining showing the multiple orange staining of *Blastomyces dermatitidis* (arrows) (lower: ×250).

a fungal granuloma. HE staining revealed thick-walled budding yeast-like forms (Fig. 2 upper). Grocott-Gomori methenamine-silver (GMS) staining confirmed that the fungus was *Blastomyces dermatitidis* (Fig. 2 lower).

The postoperative course was complicated by a series of generalized seizures. She had 8 to 10 seizures in a day, each followed by drowsiness. The seizures were controlled with anticonvulsant drugs after 8 days. She was given amphotericin B therapy (1 mg/kg body wt/day for 42 days, total dose 2 g). There was no visual recovery after a follow-up period of 6 months. CT at this time showed no residual tumor in the suprasellar area, but the part anterior to the brainstem had not regressed in size. As this part of the tumor was not symptomatic, she is being clinically observed.

Discussion

A recent review of central nervous system blastomycosis described 21 reports of successful surgical management over the last 50 years.¹²⁾ A significant proportion of the lesions were located in the cerebellum. Intracranial lesions rarely occur in isolation and are usually associated with meningitis.⁴⁾ Unlike tuberculous infection, the inflammatory reaction in response to blastomycosis is rarely complicated by vasculitis and ischemic infarcts. Our patient presented with a chief complaint of worsening vision. The optic nerves were encased and severely compressed by the lesion from both superior and inferior aspects. The visual impairment appeared to be due to the neural compression rather than vasculitis. Postoperatively she suffered a series of seizures. It is not clear whether the surgical manipulation caused a transient dissemination of infection which manifested as irritative foci. The radiological appearance of the blastomycosis granulomas is nonspecific. The diagnosis can be based only by the direct demonstration of the fungus in the infected tissues. Cultures of cerebrospinal fluid have been negative in various reports.^{3,5,8)} Amphotericin B is the drug of choice and is effective in 90% of cases.^{1,9,11)} However, the mortality from central nervous system blastomycosis is high.²⁾

Our patient had a number of unusual clinical features. She was relatively young and otherwise in good health. The widespread lesion was extraaxial, defined, and involved a large part of the base of the brain. There was no clinical or operative evidence of meningeal infection. The radiological appearance, although not characteristic, suggested a meningioma. The lesion had encased both optic nerves and intradural carotid arteries. There was a wide dural attachment and the lesion was fibrovascular in nature, both suggestive of an en-plaque meningioma. The source of infection and the mechanism of intracranial spread were not identified.

Acknowledgment

The authors acknowledge with gratitude the constant encouragement from Dr. Sunil K. Pandya (Professor of Neurosurgery, K.E.M. Hospital and Seth G.S. Medical College).

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