Tree-in-bud Appearance in the Brain: Fungal Granuloma on Contrast Magnetic Resonance Imaging

**Clinical Image**

We describe a case of dural-based homogenously enhancing fungal granuloma in a 29-year-old male who presented with 3 months history of headache. The peculiarity of the case was that there were streaky areas of enhancement around the lesion in the brain parenchyma which resembled tree-in-bud like appearance. The patient underwent surgery and histopathological analysis revealed numerous *Aspergillus* hyphae. To the best of our knowledge, this is the first case report of a fungal granuloma with atypical parenchymal enhancement pattern.

**Keywords:** Fungal granuloma, magnetic resonance imaging, tree-in-bud

**Introduction**

Intracranial fungal granulomas are almost always a clinical surprise because their presentation is subtle, often without any typical diagnostic characteristics, and thus, they are frequently mistaken for tuberculomas, pyogenic abscess, or brain tumor. Difficulty in preoperative diagnosis is further aggravated in immunocompetent patients because of its relative rarity in patients with normal immunity. *Aspergillus fumigatus* is the most common human pathogen in the genus *Aspergillus*, but *Aspergillus flavus*, *Aspergillus Niger*, and *Aspergillus oryzae* are also commonly seen. The primary mode of entry for aspergillosis organisms is the respiratory tract. Infection can reach the brain directly from the nasal sinuses through vascular channels or is blood borne from the lungs and gastrointestinal tract. The pathology depends on the route of spread, host immunity, and type of fungus, hyphae, or yeast.

**Case Report**

A 29-year-old male immunocompetent patient presented with a history of headache for 3 months. There was no history of fever, neck pain. On clinical examination, no neuromotor deficits present. He was referred for magnetic resonance imaging (MRI) brain. MRI revealed a dural-based lesion in the left temporal region, which was isointense on T1-weighted images [Figure 1a], hypointense on T2-weighted images [Figure 1b]. There was also similar signal intensity lesion noted in the nasal cavity involving the posterior septum [Figure 1c]. There was no restricted diffusion [Figure 1g]. Postcontrast study [Figure 1d-f] showed homogenous enhancement of the dural-based lesion and the lesion in the nasal cavity. Surrounding the lesion, tree-in-bud type of parenchymal enhancement (curved arrows) is noted. Due to this type of atypical parenchymal enhancement adjacent to a dural-based lesion and with a similar lesion in the nasal cavity, a radiological diagnosis of fungal granuloma was made. The patient underwent surgery and histopathological examination [Figure 2] confirmed our diagnosis. Patient’s and institutional consent were taken for the purpose of research.

**Discussion**

Brain is significantly resistant to fungal infections owing to the abundant blood supply and also due to the relatively impermeable blood–brain barrier. However, under special conditions and immune system abnormalities, fungal pathogens breach these barriers.[3] Invasive disease is most commonly present in patients who are significantly immunocompromised as in patients with prolonged steroids, hematological malignancies or advanced AIDS, and hematopoietic stem cell
transplant and solid organ transplant. However, *Aspergillus* granulomas are also reported in immunocompetent individuals commonly in countries with temperate climates. Pathophysiologically, intracranial aspergillosis has a predilection for the corticomedullary junction due to the vascular anatomy of this interface and to the hematogeneous route of dissemination of pathogen. In the brain, infection can be found in the cerebral parenchyma, the meninges, or the vascular system. An infectious event in the brain leads to infarction or hemorrhage owing to blood vessel invasion and later leads to cerebritis or abscess formation.

The three imaging patterns of cerebral aspergillosis in immunocompromised patients described by Ashdown *et al.* are (1) multiple cortical and subcortical areas of decreased computed tomography attenuation or T2 lengthening (with or without hemorrhage), (2) multiple ring-enhancing lesions, and (3) dural enhancement with adjacent enhancing lesions of the paranasal sinuses or calvarial, or dural enhancement of the optic sheath with associated enhancement of the optic nerve and orbital fat. The last pattern represents direct extension of sinonasal disease.

Our case partly fits into the last pattern although no dural enhancement was noted. The atypical tree-in-bud type of enhancement noted in our case around the dural lesion could represent the angioinvasive nature of the lesion into the surrounding parenchyma. The treatment for complete cure is total or near-total surgical excision of the lesion with antifungal therapy. The purpose of this case report is to draw attention to the associated atypical parenchymal enhancement which has not been described till date.

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**Conflicts of interest**

There are no conflicts of interest.

**References**