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Melanized fungus as an Epidural abscess: A diagnostic and therapeutic challenge

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ABSTRACT

Curvularia a dematiaceous fungus is ubiquitously found in soil around the world. We report an epidural abscess due to *Curvularia lunata* in a 48 years male farmer who underwent decompressive laminectomy as primary modality of treatment followed by isolation, identification and confirmation of the isolate from tissue by ITS sequencing. Antifungal therapy with voriconazole and amphotericin B for 3 and 2 weeks respectively improved patient's condition and is presently on regular follow up with no sequelae since last 7 months.

1. Introduction

CNS infections by melanized fungi have increasingly been reported in recent years. Primary cerebral infections are predominantly caused by *Exophiala dermatitidis, Cladophialophora bantiana, Ramichloridium mackenzie* with occassional *Ochroconis gallopava*. [1] Secondary cerebral infections are usually an extension from chronic sinusitis and are due to grass-inhabiting species under genera the *Bipolaris, Dissitimurus, Exserohilum, Curvularia lunata, Cladosporium cladosporioides, Nodulisporium species*. [1] The infection is encountered in apparently immunocompetent hosts with chronic sinusitis and the agents are commonly airborne saprobes. [1].

Phaeohyphomycosis (PH) are a rare group of heterogenous dematiaceous (phaeoid) brown pigment producing fungi, which cause superficial, cutaneous, and subcutaneous infections in the immunocompetent and systemic illness, especially brain abscesses in the immunosuppressed.[2] A myriad of fungal species causes this unique infection, which includes *Exophiala, Phialophora, Cladosporium, Wangiella, Fonsacaea, Alternaria, Bipolaris,and Curvularia species.* The usual source of infection is usually exogenous, following pricks with thorns or wood splinters. [2].

The pathogenesis of primary CNS phaeohyphomycosis is poorly understood. Melanin, found in the cell walls of dematiaceous fungi, is a known virulence factor and may play an important role in the host immune system evasion of these fungi. Most of the CNS infections are thought to be secondary to extension from paranasal sinuses; however, some infections appear to have resulted from hematogenous dissemination, direct inoculation from penetrating head trauma, and from contaminated wounds. [3].

Curvularia is a filamentous, dematiaceous fungus characterized by melanin pigmentation in the cell walls of its hyphae. It is a mold, ubiquitously found in soil around the world, with preference to the tropical and subtropical regions. It was first documented as a human pathogen in 1959 in Africa, isolated from lung mycetomas. [3] The first central nervous system (CNS) *Curvularia* infection was described in 1977 by Lampert et al. Since then, only eight CNS *Curvularia* infections have been documented in the literature. [3] The ninth case was *Curvularia* infection of brain stem documented by Branko Skovrlj et al. [3].

Curvularia received its current name in 1933 and related to the sexual teleomorph Cochliobolus typified by *C. lunata* and morphologically characterized by the production of sympodial conidiophores with tretic, terminal and intercalary conidiogenous cells and elongate, transversely septate conidia with a dark basal scar [4].

Frequently, diagnosis is understandably delayed in cases of spinal epidural abscess because the initial presentation may be only nonspecific back pain. One half of cases are estimated to be misdiagnosed or have a delayed diagnosis [5]. The classic presentation of spinal epidural abscess involves the triad of spine pain, fever, and neurologic deficit [6].

We present our case, which is the 10th case so far, for documentation and future reference; as melanized fungal infections of CNS are difficult to diagnose and treat.

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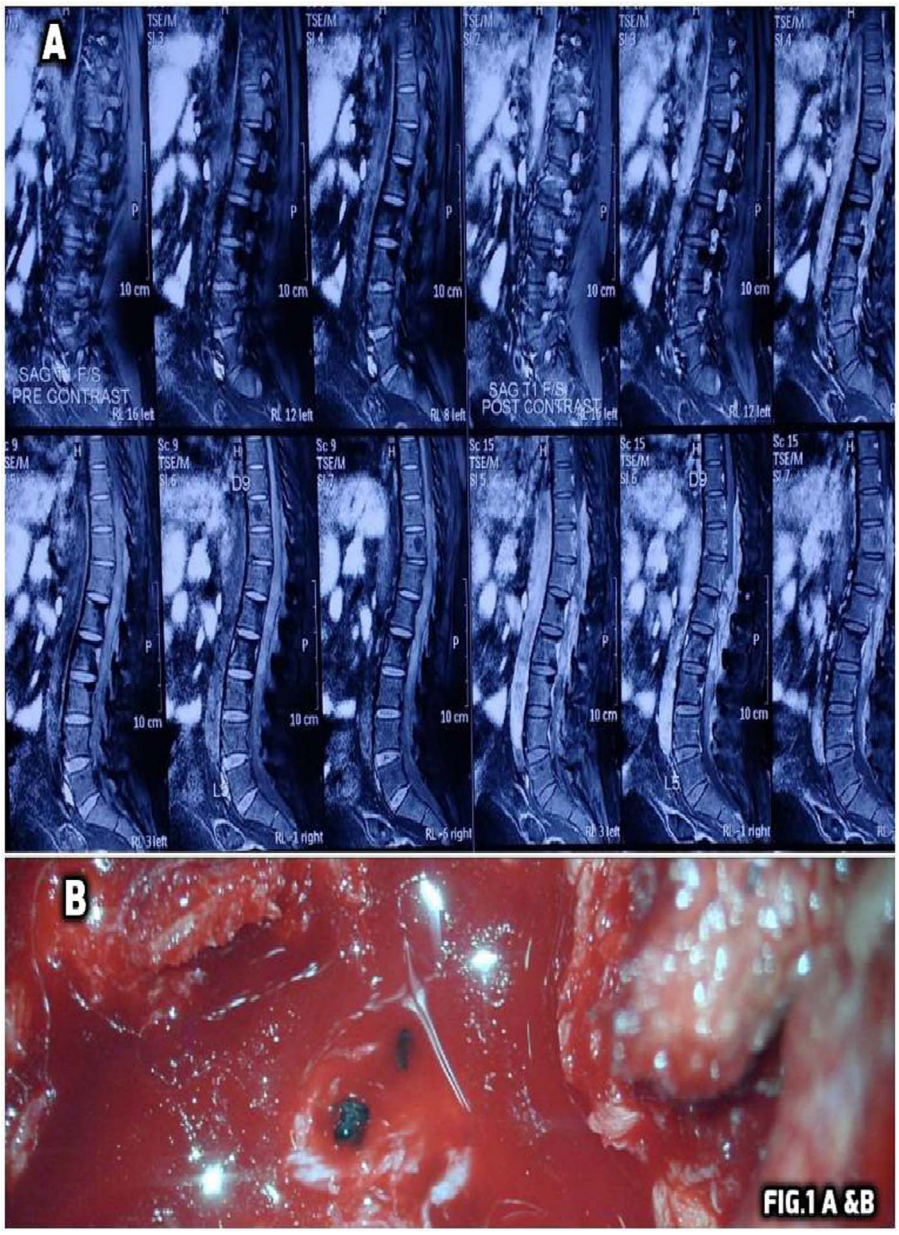

Fig. 1. Shows contrast enhanced MRI of dorsolumbar spine in sagittal plane. Long segment heterogenous intensity lesions are seen in the spinal canal and posterior epidural space extending from D10 to L4 levels appearing hypointense and intense in post contrast enhancement (**A**). Bottom shows black coloured rounded structures with pus pockets in epidural space during decompressive laminectomy (**B**).

2. Case

A 48 years male farmer from central India came to neurosurgery Out Patient Department of AIIMS Bhopal on 4th April 2016 with chief complaint of low backache since 3years which was insidious in onset, gradually progressive, radiating to bilateral lower limb, associated with numbness and paraesthesia, inability to walk since 15 months and decreased urinary sensation. The patient was habituated to tobacco chewing and smoking.

On clinical examination patient was afebrile, oriented, had inability to walk without support. There was loss of sensation bilaterally in L3, L4, L5, S1 region. An atrophic change over palmar muscles due to chronic use of crutches was observed. There was no motor or sensory deficit of upper limb. Rest of the examination was normal.

His routine investigations of blood heamoglobin, total and differential leucocyte count, Erythrocyte sedimentation rate, platelet count, C reactive protein, random blood sugar, serum creatinine kinase, Blood $Na+$, $K+$ and renal function tests were normal. Negative for urine Bence–Jones proteins and normal prostate specific antigen. Electrocardiogram was normal. His HIV status was negative. Magnetic resonance imaging of dorsolumbar spine showed intensely enhancing posterior epidural and intrathecal space occupying lesions extending from D10 to L4 levels causing spinal canal stenosis and compression of lower spinal cord & cauda equina nerve roots with mild erosion of adjoining vertebral bodies (Fig. 1**A**).

Patient was admitted on day of first hospital visit (4th April 2016) which is Day 0 (D0) for our case study.

2.1. Primary treatment

As Primary treatment modality surgery was preferred and a D10- L4 decompressive laminectomy was performed on D8 (12th April 2016). The lesion showed inflammation with fibrosis, pus pockets and several black coloured rounded structures. (Fig. 1**B**).

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