

# Disseminated *Fusarium oxysporum* neurospinal infection

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## ABSTRACT

We report a case of disseminated meningospondylodiscitis in an elderly diabetic patient caused by *Fusarium oxysporum*. As the clinical presentation was nonspecific, the diagnosis of the condition could only be arrived at after laboratory and imaging studies. The diagnosis of the condition requires a high index of suspicion. Patient underwent thorough surgical debridement along with a short course of voriconazole and remained asymptomatic after 36 months of diagnosis. *Fusarium* is a large genus of filamentous fungi widely distributed in soil and in association with plants. It is known to cause local infections (nail, cornea) in healthy humans and disseminated infection only in the immunocompromised.

**Key words:** Disseminated fungal meningospondylodiscitis, *Fusarium oxysporum*, voriconazole

## INTRODUCTION

The diagnosis of neurospinal infections particularly fungal infection, is often difficult and delayed.<sup>1</sup> The clinical presentation may be very nonspecific. We describe a very unusual clinical presentation a case of disseminated meningospondylodiscitis caused by *Fusarium oxysporum*. To the best of our knowledge, this is the first reported case of neurospinal infection with *Fusarium*.

## CASE REPORT

A 60-year-old female patient was admitted with altered sensorium and fever for 2 weeks. The patient was drowsy with severe neck stiffness. She was a known case of type II diabetes mellitus and systemic hypertension.

Routine blood examination revealed polymorphonuclear leucocytosis (TC 12400, DC P97, L02, M01) and normal

erythrocyte sedimentation rate (ESR). Lumbar puncture was performed, cerebrospinal fluid (CSF) study showed lymphocytic pleocytosis with elevated protein and normal glucose. Computed tomography (CT) brain showed small ring enhancing lesions in bilateral frontal lobes and features suggestive of meningoencephalitis. Magnetic resonance imaging (MRI) brain [Figure 1a] showed multiple well-defined enhancing lesions in both cerebral hemispheres and left cerebellum with pial enhancement. Treatment was started presuming bacterial/tuberculous meningitis with a broad spectrum antibiotic and anti tuberculous therapy. CSF culture was negative for any organisms. She was discharged after 3 weeks with the diagnosis of chronic meningitis with cerebral granuloma, type II diabetes mellitus and systemic hypertension.

One month later, she came with hypoglycemia, hypotension and fever. She was diagnosed with urinary tract infection with extended-spectrum beta-lactamase *Klebsiella* and was appropriately treated. Repeat MRI brain was done which showed no new lesions; anti TB treatment was continued.

She was readmitted after 5 months with vomiting, poor intake, fatigue and generalized body ache. She had developed right parieto-occipital infarct, so low- molecular- weight heparin and antiplatelets were started. MRI brain now showed persistence of lesions with only mild resolution of edema surrounding the lesions. She showed mild symptomatic improvement. Hence, she was further evaluated with tumor markers and CT thorax and abdomen. This showed a collapse at D8-D9 vertebral level. MRI spine with contrast showed spondylodiscitis at the D8-D9 level [Figure 1b and c]. Patient underwent laminectomy,

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debridement of discovertebral infection and fusion. The lesion was approached through the posterior midline. Pedicle screws were applied at D6, D7 and D10, D11 levels. D8, D9 laminectomy was done as an approach to the infected site and D8/9 disc space was curetted [Figure 2a]. Pus and granulation tissue were sent for histopathology and microbiological studies. The space was packed with grafts taken from the transverse process and stabilized with rods. Histopathology examination revealed chronic nonspecific granulomatous inflammation. Microbiology tests showed fungal hyphae on culture and the fungus was identified as *F. oxysporum*.

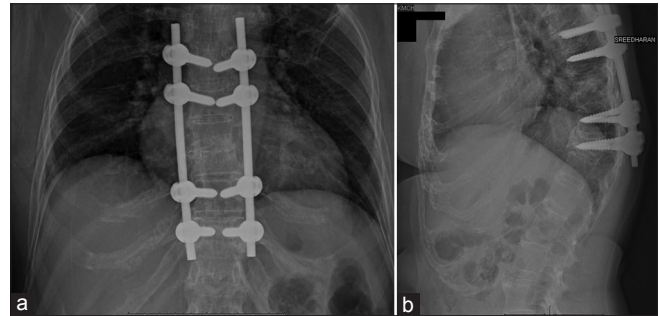
She was diagnosed as having meningospondylodiscitis due to *F. oxysporum* and was started on intravenous voriconazole 200 mg 12 hourly for 3 weeks, then changed to oral voriconazole 200 mg twice daily for a period of 7 weeks. In the meantime, tuberculosis (TB) polymerase chain reaction from the spinal tissue was found to be negative. Hence, ATT was stopped. At the time of discharge, 2 months later, she was asymptomatic and ambulant with the support of a brace. She was followed up regularly in the outpatient department with X rays and laboratory tests (ESR 13, TLC 9000), which showed reduction in infection and a progressive spine fusion [Figure 2b] X ray shows bony trabeculae crossing the fusion site and clinically there is no tenderness at the site. At her last followup, 36 months after the surgery, she was doing well, living an independent life [Figure 3].

## DISCUSSION

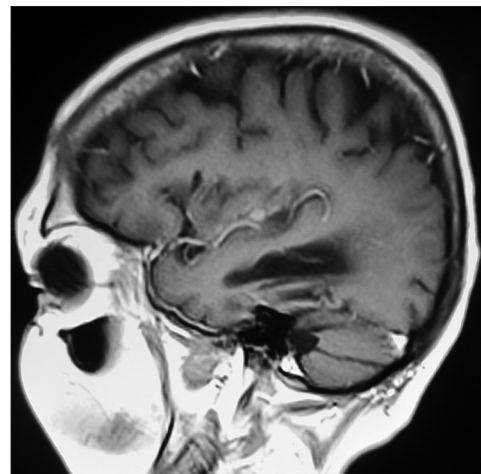
The incidence of spondylodiscitis is very low and estimated to be in the range of 4-24/million/year as per literature.<sup>2</sup> Spondylodiscitis usually occurs in association with other infections, malignancies and immunocompromised conditions.<sup>3</sup> It can occur as a complication of long term glucocorticoid use and thecal sac puncture.<sup>3</sup> Rarely, it

presents as an isolated disease. Detection of the causative organism may be usually unsuccessful, in view of the fact that routine culture techniques only detect bacteria. Fungal culture should be done in suspected longstanding cases of neurospinal infection with non classical presentation.

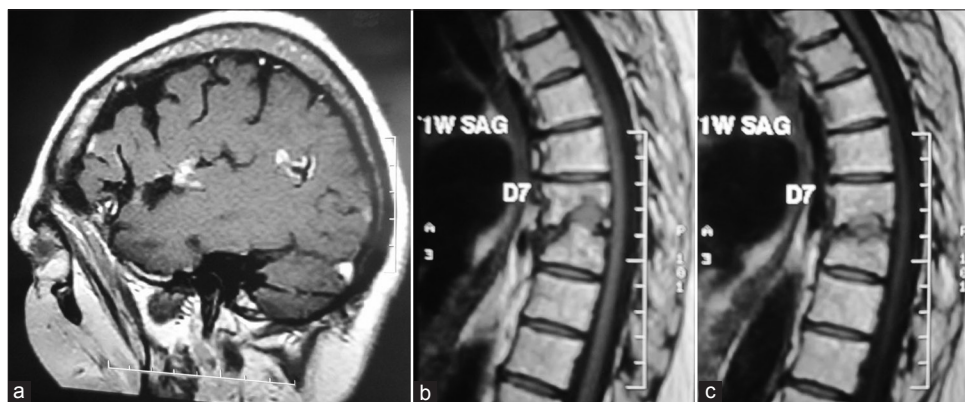
Reported causative organisms in spondylodiscitis include *Staphylococcus aureus* (40-60%), *Mycobacterium*



**Figure 2:** (a) X-ray Thoracic spine anteroposterior view showing decompression of D8, D9, fusion and posterior instrumentation (b) X-ray Thoracolumbar spine lateral view 30 months postoperative film showing good fusion



**Figure 3:** Followup magnetic resonance imaging brain showing resolution of lesions



**Figure 1:** (a) Magnetic resonance imaging brain contrast enhanced T1 sagittal section showing multiple ring enhancing lesions in the brain parenchyma (b-c) Preoperative T1W sagittal magnetic resonance imaging showing spondylodiscitis D8-D9

TB (20%), *Streptococci*, *Brucella* and normal flora of the gastrointestinal tract.<sup>3</sup> Fungal species such as *Candida*, *Aspergillus*, *Scedosporium* and *Trichosporon* cause about 5% of spondylodiscitis.<sup>3</sup> The clinical presentation of spondylodiscitis is commonly dull aching pain with no specific clinical signs in the early stages.<sup>2</sup>

The radiographic changes in vertebral osteomyelitis are seen 2-8 weeks after the onset of symptoms. MRI is most sensitive (96%), specific (93%) and accurate (94%) for early recognition and localization of infection and is considered as the imaging modality of choice for the radiological diagnosis of Spondylodiscitis.<sup>4,5</sup> MRI is also useful in demonstrating the presence of epidural or paraspinal extension of the infection.<sup>4</sup> In our case MRI showed ring enhancing lesions in the brain parenchyma. This picture can be seen in fungal meningoencephalitis in immunocompetent patients; though, it is commonly seen in India in cases of bacterial/tuberculous meningitis. In immunosuppressed patients, due to a lack of the inflammatory response, neuroradiological appearance can be nonspecific in fungal meningoencephalitis. So a high index of suspicion is needed to identify fungal etiology in such cases.<sup>6,7</sup>

Short course antifungal therapy with adequate surgical debridement is effective in the management of fungal spondylodiscitis instead of prolonged medical management alone.<sup>8</sup> Our patient underwent debridement and fusion in view of the MRI findings. Fusion was performed to prevent post laminectomy kyphosis and chronic back pain. Cultures from the spine were positive for fungi, which grew *F. oxysporum* species. She was treated with intravenous voriconazole preparation. Voriconazole is effective against many fungal pathogens, but it is a hepatotoxic drug.<sup>9</sup> Although, long term therapy with voriconazole is advised in the available literature, we optimized our treatment in view of thorough surgical debridement, excellent clinical response after 10 weeks and previous history of elevated liver enzymes with ATT. After 36 months, she remains healthy with no recurrence. Followup MRI brain shows resolution of the lesions.

*Fusarium* is a large genus of filamentous fungi widely distributed in soil and in association with plants. In humans with normal immune systems, fusarial infections may occur in nails (onychomycosis) and cornea (keratomycosis or

mycotic keratitis). Aggressive disseminated infections are caused by members of *Fusarium solani* complex, *F. oxysporum*, *Fusarium verticillioides*, *Fusarium proliferatum* and rarely, other species in immunocompromised patients.<sup>10</sup>

To conclude, fungal neurospinal infections are rare. Physical findings may be minimal and a high index of suspicion helps to order the appropriate tests for fungi, which are otherwise missed in the routine tests, to hasten the diagnosis.

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