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Letter to the Editor: New Observation

Spinal Intramedullary Abscess of Blastomycosis Etiology in an Immunocompetent 19-Year-Old Male

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Intramedullary spinal abscess of any microbiological etiology is an exceedingly rare but devastating disease.[1] Blastomycosis in particular is an uncommon etiology for abscess of any location of the central nervous system (CNS).[2] Herein, we present the first case documented in the literature of a spinal intramedullary blastomycosis abscess. The rarity of this disease presumably resulted in the delayed diagnosis and illustrates to the clinician the importance of considering pathologies whose acute, appropriate intervention can significantly change clinical outcomes.

A previously healthy 19-year-old male presented to a community hospital with pneumonia refractory to amoxicillin-clavulanic and moxifloxacin and was intubated for deteriorating respiratory status. Subsequent bronchoscopy demonstrated *Blastomyces dermatitidis*, and he was commenced on amphotericin B (650 mg once daily) and voriconazole (200 mg once daily). However, the patient progressed to acute respiratory distress syndrome with hypotension, tachycardia and oxygen desaturation, and he was placed on extracorporeal membrane oxygenation (ECMO). Although the patient was weaned off ECMO, he was persistently ventilator dependent and discovered to have diffuse weakness.

Thus, a neuro-axis MRI was performed that demonstrated T2 hyperintensity from the T1–T2 level down to the conus and across the nerve roots, as demonstrated in Figure 1, with no rim-enhancing fluid collections suggesting abscess formation but with a T2 hyperintense intramedullary component. There were no signs of arachnoiditis intracranially or in the cervical spine. CSF analysis revealed a protein count of 16.5 g/L but without positive microorganisms or white blood cells. His exam revealed MRC 0/5 motor strength in the lower extremities with a sensory level of T10. In the upper extremities, there was pure motor weakness bilaterally below 3/5 with preserved small and large fiber-mediated sensation. Based on the imaging and clinical presentation, the pathology of the longitudinal extensive transverse myelitis was suspected to have originally resulted from blastomycosis leptomeningitis, leading to either an aseptic infectious myelitis, post-infectious autoimmune myelitis or venous congestion of the spinal cord from venous thrombosis. Given the CSF results were aseptic, he was no longer believed to have an active CNS infection – the high protein was suspected to be secondary to CSF stagnation. Thus, medical management continued, and neurosurgery was not consulted.

Given a lack of neurological improvement 2 weeks later with a clear sensory level at T10, a repeat neuro-axis MRI was performed, which demonstrated an enhancing T1 rim and intramedullary diffusion restriction as seen in Figure 2, from T5 down to the conus. As a result, neurosurgery was involved and pursued urgent surgical intervention for source control and diagnosis.

Laminotomy was performed at the T11 and T12 levels, at the thickest part of the rim enhancement corresponding to the sensory level. Durotomy demonstrated the spinal cord to be edematous, with spinal cord herniation through the dura leaflets. Midline myelotomy was performed at T12 to relieve pressure, and purulent viscous material was expressed, at which point an external ventricular drain catheter was passed into the cavity to facilitate drainage. Another myelotomy and catheter passage were then performed at T11. Despite improvement in cord swelling following drainage, there was residual fibrinous solid material in the cavity of both myelotomies, which we opted not to evacuate.

Subsequent neurological examination demonstrated mild improvement in upper limb motor function, which, following discharge from spine rehabilitation 4 months later, unfortunately remained unchanged.

Intramedullary abscess is an uncommon disease with fewer than 250 reported cases,[1] compared to intracranial abscesses with an annual incidence of 1.3 per 100,000 people.[3] Multiple factors have been suggested to explain the discrepancy in frequency between brain and spinal parenchymal abscess, such as the acute angle of origin of spinal arteries and the small volume of the spinal cord that limit microbial transmission and seeding.[4]

A systematic review by Satyadev et al. determined the most common sources of infection for an intramedullary abscess to be contiguous, followed by hematogenous spread and cryptogenic etiology, similar to intracranial abscess.[1] The most common microflora were found to be *Escherichia coli*, *Staphylococcus aureus* and *Mycobacterium tuberculosis*.[1] Of the 200 cases included, only 8 grew fungi, including the species *Coccidioides immitis, Candida albicans, Aspergillus* and *Cryptococcus neoformans*.[1] Fungal presentations tend to be opportunistic. However, most cases of blastomycosis dissemination to the vertebral or epidural space have been in immunocompetent patients, as was our case.[5] Given the azygos vein communicates with Batson's plexus in the thoracic region, and the abscess was primarily localized to the thoracic

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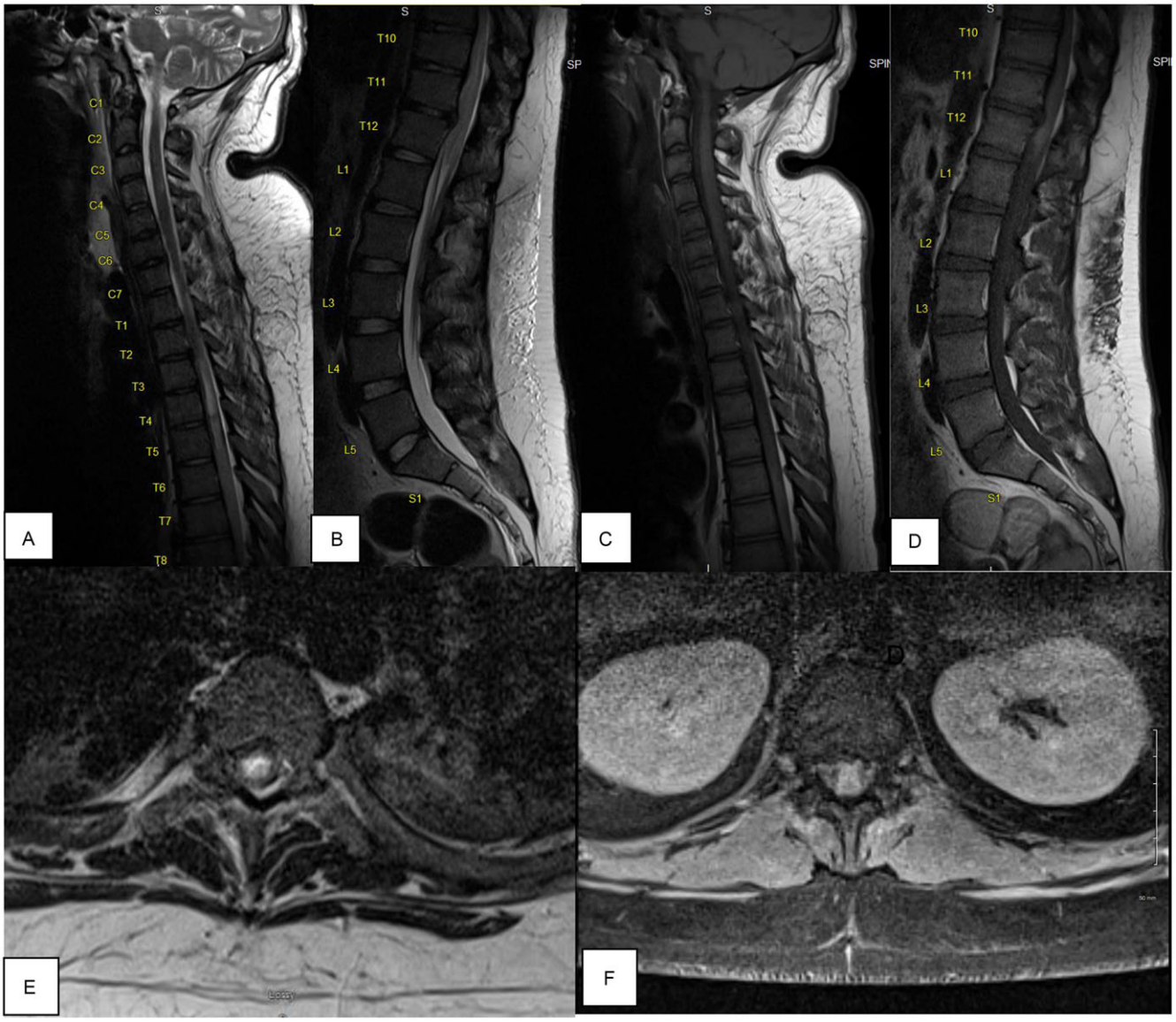

Figure 1. Contrast-enhanced MRI full spine at 4 weeks after presentation. (A) Diffuse T2 hyperintensity extending from T1 to the conus medularis (B), (C and D) patchy contrast enhancement on T1 in keeping with myelitis and arachnoiditis, (E) axial slice at T3–T4 showing intramedullary T2 hyperintensity and (F) axial slice at T12–L1 showing patchy leptomeningeal enhancement.

spine, the mechanism of spread can be presumed to be hematogenous from his unremitting pulmonary blastomycosis.[6]

Blastomycosis is endemic to eastern North America, especially our catchment area of the St Lawrence drainage basin.[7] It is thermally dimorphic, manifesting as budding yeast at body temperature (37°C) and as a mycelial form with spores at room temperature.[7] Disruption of spores near wooded areas and decaying vegetation leads to inhalation, manifesting in 90% of blastomycosis patients as pulmonary localization.[7] Many go on to develop chronic disease, and 25%–40% of patients develop disseminated blastomycosis in the skin, musculoskeletal system and genitourinary system.[2,7] Before the advent of the antifungal therapies such as amphotericin B (0.7–1 mg/kg/d)[8] or fluconazole (minimum, 800 mg/d),[8] the mortality rate for systemic blastomycosis exceeded 90%.[7]

Blastomycosis involvement in any CNS compartment is uncommon, consisting of 5%–10% of disseminated blastomycosis.[2] Bariola et al. published their multicenter institutional experience of blastomycosis cases of the CNS, yielding 22 patients, of which 12 were immunocompromised. Only one of the cases manifested in the spine as an epidural abscess. Of the 11 patients who had lumbar puncture performed, 10 had pleocytosis, and 9 had elevated levels of CSF protein.[2] Although our patient had significantly elevated protein levels that could be suggestive of fungal infection, the WBC count was within normal range. As a result, this led to the conclusion that the infection was resolving or there was an alternative autoimmune etiology until repeat MRI demonstrated the formation of the abscess. A case series published by Bartels et al. investigating intramedullary abscess of any etiology demonstrated that in the surgical group consisting of myelotomy and drainage, the mortality rate was 13.6%, of whom 75% had not received antibiotics, while in the nonsurgical group, the mortality reached 100%, where 97% had not received antibiotics.[9] There are case reports in which physicians elected to continue medical management given the small size of the lesion with rapid response to antibiotic therapy.[10] However, when intramedullary abscesses

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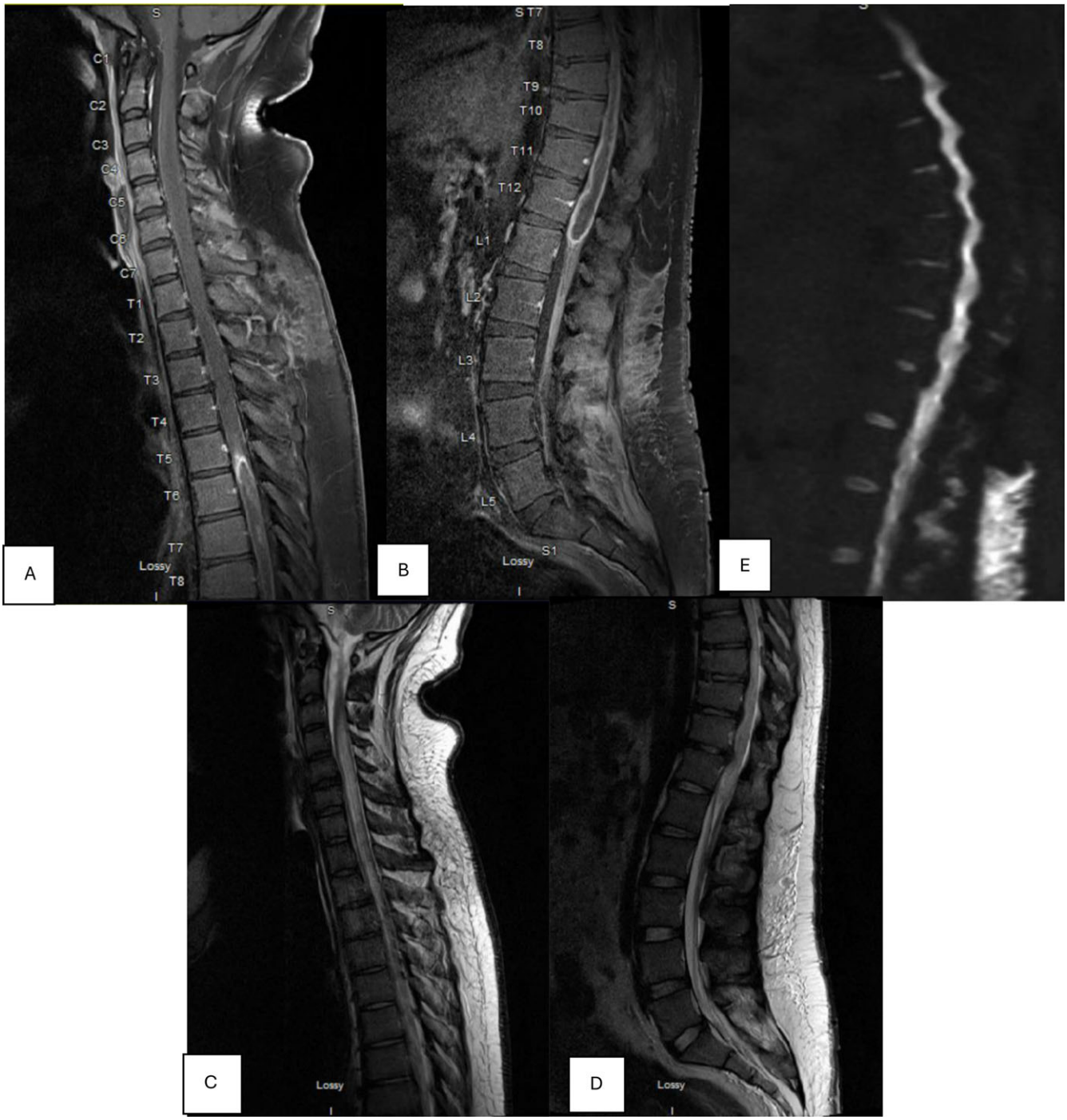

Figure 2. Contrast-enhanced MRI full spine at 6 weeks after presentation. (A and B) T1 ring-enhancing fluid collection at T5 down to the conus, (C and D) progressive T2 hyperintensity compared to previous imaging and (E) intramedullary diffusion restriction from T5 down to the conus.

cause neurological deficits due to mass effect, the preferred management is generally agreed to be surgical debridement.[1] It is reasonable to extrapolate this indication for surgery even when the microbiology is blastomycosis (despite the paucity of evidence owing to the rarity of the disease). The technique of passing a ventricular catheter rostral and caudal to aspirate the full extent of the abscess has been described previously.[11]

Unfortunately for our patient, there was a significant delay (14 days) between neurological decline and decompression. Hence, although there is evidence that patients with intramedullary abscesses improve following decompression,[1] the goal of surgery here was to prevent further secondary spinal cord injury and achieve source control of the blastomycosis, given it was probable that some degree of myelopathic irreversible injury had already occurred. This poor neurological status thus represents an adverse complication that may have been avoided had the diagnosis of intramedullary infection been considered earlier on.

This represents the first case of blastomycosis intramedullary abscess, in a 19-year-old male without immunocompromise, whose only risk factor for disease was living in the St Lawrence

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River valley. Disseminated blastomycosis and intramedullary abscesses are severe diseases that can produce irreversible deficits unless there is prompt surgical decompression through myelotomy, drainage and potentially advancing of an inside catheter to communicate with septated cavities.[11] Imaging may not initially demonstrate frank abscess, and thus, clinicians must maintain an index of suspicion for this diagnosis in endemic regions and interpret test results accordingly, given these diseases are severe yet treatable if addressed promptly.

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CA: Conceptualization, methodology and review of manuscript.
SM: Conceptualization, methodology and revision of manuscript.

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