Received: 10 February 2021 | Revised: 21 May 2021 | Accepted: 7 June 2021

DOI: 10.1002/emp2.12493

JACEP OPEN
JOURNAL OF THE AMERICAN COLLEGE OF EMERGENCY PHYSICIANS OPEN
A GLOBAL JOURNAL OF EMERGENCY MEDICINE
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CASE REPORT

Neurology

Compressive epidural fluid collection secondary to varicella zoster transverse myelitis

Vase Bari MD[1] | **Jonathan Frommelt MD**[2,3] | **Vikas Jain MD**[4,5]

[1] Department of Anesthesia, Emory University School of Medicine, Atlanta, GA, USA

[2] Department of Emergency Medicine, Metrohealth Medical Center, Cleveland, Ohio, USA

[3] Assistant Professor Emergency Medicine, Case Western Reserve University School of Medicine, Cleveland, Ohio, USA

[4] Department of Radiology, Metrohealth Medical Center, Cleveland, Ohio, USA

[5] Associate Professor Emergency Medicine, Case Western Reserve University School of Medicine, Cleveland, Ohio, USA

Correspondence
Jonathan Frommelt, MD, Department of Emergency Medicine, Metrohealth Medical Center; Assistant Professor of Emergency Medicine, Case Western Reserve University School of Medicine, Cleveland, OH, USA.
Email: jfrommelt15@gmail.com; jfrommelt@metrohealth.org

Funding and support: By *JACEP Open* policy, all authors are required to disclose any and all commercial, financial, and other relationships in any way related to the subject of this article as per ICMJE conflict of interest guidelines (see www.icmje.org). The authors have stated that no such relationships exist.

Abstract

Transverse myelitis is often clinically indistinguishable from compressive myelopathies that require emergent neurosurgical intervention. Here, we present a case of acute varicella zoster virus transverse myelitis that was associated with a compressive fluid collection on magnetic resonance imaging (MRI) requiring emergent operative intervention. To our knowledge, this is the first reported case of acute transverse myelitis and a compressive cord lesion in the adult population.

KEYWORDS

cord compression, epidural abscess, neurological emergency, Transverse myelitis, varicella

1 | INTRODUCTION

The differential diagnosis for acute myelopathy is extensive and includes degenerative, traumatic, infectious, paraneoplastic, autoimmune, inflammatory, vascular, toxic, and even electrical etiologies.[1–3] The most critical step in the work-up of acute myelopathy is distinguishing compressive from non-compressive sources. Although clinical history and thorough physical examination will narrow this extensive differential diagnosis, imaging (particularly magnetic resonance imaging [MRI]) is required to rule out a compressive lesion.

Transverse myelitis is a rare disorder of the spinal cord usually caused by a para-infectious or autoimmune inflammatory process (although 20% of cases are idiopathic) that causes a wide array of motor, sensory, and autonomic neurologic deficits that develop variably over hours to weeks.[1] This disease is rare with an estimated incidence as high as 3 per 100,000 patients without any real genetic or demographic predilection.[3–5] Despite prompt treatment, significant morbidity persists in roughly two-thirds of all afflicted patients.[1,6] Diagnostic criteria for transverse myelitis include an absence of compressive lesion on imaging.[1,7–9] We present a rare case of transverse myelitis that was also associated with a compressive epidural fluid collection.

2 | NARRATIVE

A 38-year-old male with a past medical history of HIV/AIDS (last CD4 count 9 and viral load 194,000 on highly active antiretroviral therapy (HAART) therapy with poor compliance and trimethoprim/sulfamethoxazole), necrotizing gingivitis, Pneumocystis

Supervising Editor: Juan March, MD

jiroveci pneumonia, Candida esophagitis, and polysubstance abuse presented to the emergency department (ED) with a chief complaint of leg weakness. He reported feeling "weird" the day prior and waking on the day of presentation with ascending numbness starting in both feet up to his mid waist. He endorsed progressive lower extremity weakness and inability to ambulate. He denied any upper extremity weakness or numbness. The patient also noted inability to urinate for the past 24 hours. He denied back pain, recent fall, fever, chills, night sweats, incontinence, recent travel, intravenous drug abuse, or sick contacts. He reported no similar past illnesses and had not attempted any therapy before arrival. Chart review showed a recent admission 1 month prior for suicidal ideation, abdominal pain, and diarrhea. He was also diagnosed with shingles during the admission and was treated with acyclovir. A review of systems otherwise was negative for headache, vision changes, vertigo, neck pain, weight loss, diarrhea, abdominal pain, and vomiting.

Physical examination revealed a temperature of 98°F, a blood pressure of 134/88 mm Hg, a heart rate of 104 bpm, a respiratory rate of 18 breaths/min, and oxygen saturation of 99% on room air. He had no scleral icterus or conjunctival injection. The cardiac exam was notable for regular tachycardia, and the pulmonary exam was unremarkable. His abdomen revealed suprapubic fullness consistent with bladder distention but otherwise was soft, non-tender, and non-distended. Skin examination was significant for scattered erosions with central eschar and circumferential erythema located on his chest, abdomen, and back with some of those lesions exhibiting tense bullae. His neurologic exam revealed paraparesis, diminished lower extremity reflexes, down going Babinski, and no sensation to light touch or temperature below the T6 dermatome. Digital rectal examination revealed diminished tone. He had normal strength, sensation, and coordination in the upper extremities. Cranial nerve examination was normal.

Laboratory test results demonstrated leukopenia (white blood cell count of 4000/uL) without left shift or bandemia, hemoglobin 12.6 g/dL, platelet count of 394,000 cells/uL. The metabolic panel was significant for mild hyponatremia of 127 mmol/L but was otherwise unremarkable. Erythrocyte sedimentation rate of 55 mm/h and C-reactive protein 0.9 mg/L. Lactate was within normal limits. Blood and urine cultures were collected.

A bedside point-of-care bladder ultrasound revealed a bladder volume of 900 mL consistent with urinary retention for which a Foley catheter was placed. MRI of the thoracic and lumbar spine showed extensive signal abnormality of the thoracic cord extending from T3 to T11 with a loculated subdural fluid collection and a compressive epidural abscess located at T9 to T11 (Figure 1).

Neurosurgery was consulted and performed an emergent T9–T12 laminectomy and washout for a suspected thoracic epidural abscess. The operative report revealed "no pus in epidural space with inflammation of the tissue" and the postoperative diagnosis was thoracic epidural phlegmon. The patient was started on intravenous vancomycin, cefepime, and acyclovir empirically per infectious disease recommendations. Intraoperative wound cultures including acid-fast bacilli, fungal, aerobic, and anaerobic cultures all exhibited no growth on final results. Intraoperative viral cultures were not sent. Blood and urine cultures obtained in the emergency department were also negative. Dermatology was consulted for his skin lesions and confirmed varicella zoster virus infection by biopsy and polymerase chain reaction. Due to the patient's lack of clinical improvement, an MRI was obtained on postoperative day 3 and revealed interval worsening of extensive craniocaudal signal abnormality involving the central gray matter from C7–L1 (Figures 2 and 3). At this point, the diagnosis of transverse myelitis was made, and the patient was transferred from the surgical ICU to the medical floor and started on intravenous methylprednisolone 1 g intravenously daily. Postoperative day 8, 5 days after the initiation of high dose intravenous steroids, MRI revealed interval improvement in signal abnormality, which regrettably did not coincide with improvement in neurological function (Figure 4).

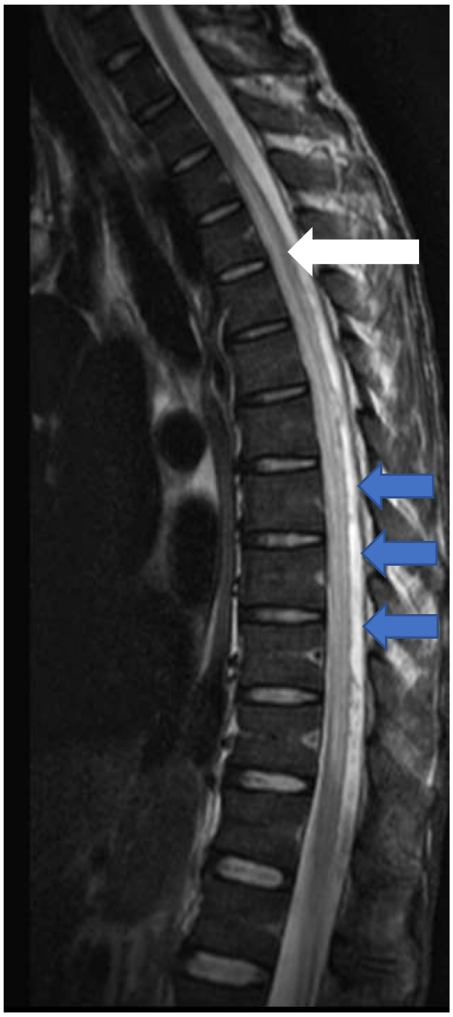

FIGURE 1 Initial MRI. White arrow shows a large area of abnormal hyperintensity in the thoracic spinal cord that extends from T3 level to T11 level. Smaller blue arrows point at the fluid collection posterior to the spinal cord suggestive of an abscess. This collection showed contrast enhancement on the post contrast images (not shown). MRI: magnetic resononance imaging

The patient's final diagnosis was consistent with varicella zoster virus transverse myelitis with an associated compressive epidural and subdural fluid collection. At discharge, the patient unfortunately did

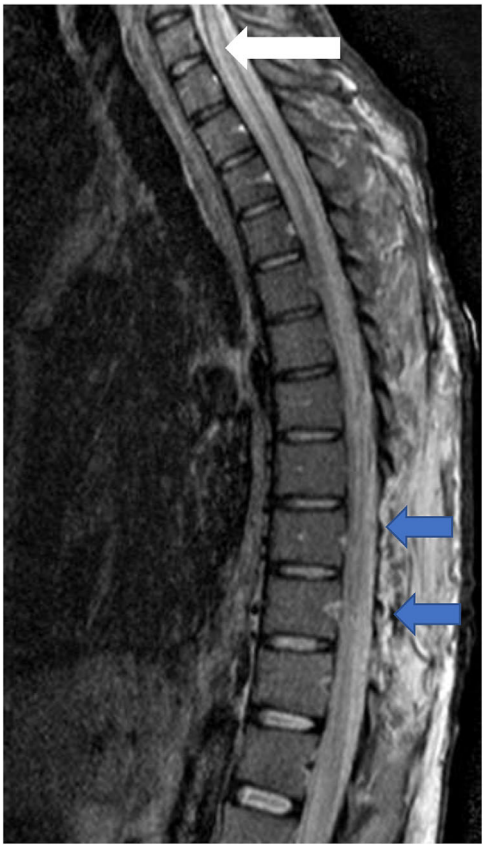

FIGURE 2 Postoperative MRI. Follow-up scan 3 days after surgery and initial scan shows extension of the abnormal hyperintensity in the cervicothoracic spinal cord (white arrow). Post-surgical changes of posterior decompression are seen at T8–T10 level (blue arrows) and the abscess is no longer seen (surgically drained). MRI: magnetic resononance imaging

not regain any neurologic function. He was sent to acute rehabilitation with a peripherally inserted central catheter (PICC) line for completion of an extended course of intravenous antibiotics and acyclovir per consultant recommendations. Chart review later revealed further complications including readmission only 24 hours after discharge for fever and tachycardia where pulmonary embolism was ultimately diagnosed. The patient was started on anticoagulation and left against medical advice 6 days later for unclear reasons. He returned to the ED 2 weeks later for failure to thrive and was eventually placed in a skilled nursing facility. The patient has been compliant with follow-up thereafter.

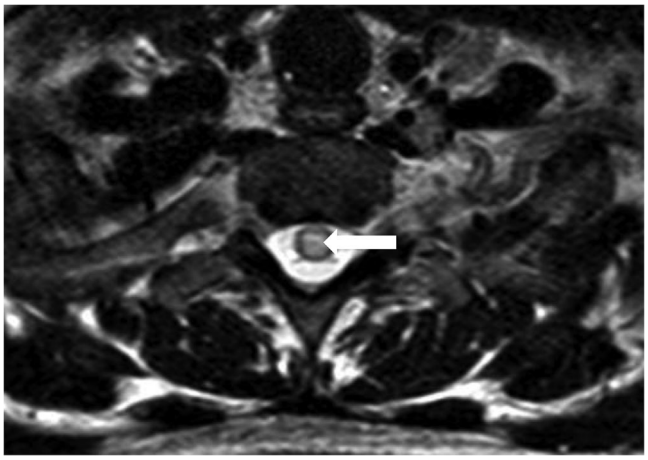

FIGURE 3 Axial T2 on the follow-up scan shows abnormal hyperintensity in the thoracic spinal cord (white arrow)

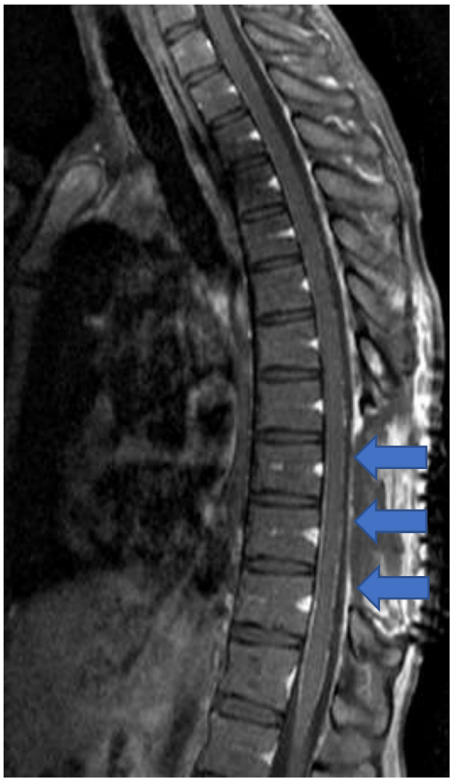

FIGURE 4 No abnormal contrast enhancement in the thoracic spine in follow-up post op scan performed 8 days after the initial scan

3 | DISCUSSION

This case report highlights a case of transverse myelitis that was also associated with a compressive epidural fluid collection. Classically, diagnosis of transverse myelitis is made by the presence of bilateral neurologic deficits that localize to the spinal cord and develop acutely with maximum symptomatology between 4 hours and 21 days in the absence of a compressive lesion on contrast-enhanced MRI of the spine.[1,3] On physical exam, a sensory line, or level at which sensation loss begins, will indicate the location or level of the spinal cord lesion. MRI is the test of choice, but computed tomography (CT) myelography is a reasonable substitute if MRI is contraindicated.[8] The workup for compressive spinal cord lesions requires careful consideration with respect to the extent of imaging and the spinal level of the neurologic deficits as there have been several cases of missed thoracic compressive lesions resulting in paraplegia even after lumbar spinal decompression surgery was performed.[10] Obtaining imaging above and below the level of neurological symptoms is therefore recommended.

Basic cerebrospinal fluid (CSF) studies are sometimes helpful in determining etiology. CSF pleocytosis and elevated immunoglobulin G (IgG) index can be suggestive of transverse myelitis.[9] Specific results and serology, however, will not be immediately available and should not delay treatment. High-dose corticosteroids should be initiated to blunt the inflammatory process.[9] Although no prospectively studied treatment regimen exists, 1 g intravenous methylprednisolone daily for 3–7 days generally is accepted.[11] Current data indicate a good safety profile and benefit to intravenous corticosteroids even in the face of concurrent bacterial or fungal infection.[12] If an infectious etiology is presumed to be a contributing factor to the patient's neurological decline, treatment can be empirically started pending further workup including broad spectrum antibiotics if bacterial myelitis or abscess is suspected. Severe transverse myelitis often does not respond to corticosteroids alone in which case plasma exchange, cyclophosphamide, and immunomodulatory therapy may be used as second-line agents depending on severity and chronicity of disease.[2,13–17] Of these alternatives, plasma exchange may be the most promising. One retrospective study, plasma exchange showed significant reduction in Expanded Disability Status Scale score in 43% of patients, even if the plasma exchange was initiated up to 60 days after the onset of symptoms.[18]

Transverse myelitis has been described as clinically indiscernible from compressive spinal cord lesions and, thus requires emergent MRI for ultimate diagnosis and to determine the need for operative intervention. This case is unique and distinctively highlights an extremely rare clinical association between the primary disease process of acute varicella zoster virus transverse myelitis and a concomitant compressive spinal epidural fluid collection. Varicella zoster is a rare but known cause of transverse myelitis but has only been associated with concurrent bacterial spinal epidural abscess in only a few pediatric case reports.[19,20] Considering the patient's severe immunocompromise (having had 2 prior AIDS defining illnesses), the etiology of the spinal epidural fluid collection could be due to bacterial coinfection that is likely why an extended course of antibiotics was administered. However, despite not receiving preoperative antibiotics, all preoperative and intraoperative bacterial, fungal, and wound cultures were negative in this case. The culprit microorganism for spinal epidural abscess is isolated by intraoperative cultures in 78%-90% of cases leaving little margin for a false-negative and at least some consideration should be given to zoster transverse myelitis as the cause of the epidural phlegmon, although we have found no other case reports of viral central nervous system infections leading to, or concurrent with, spinal canal phlegmon or abscess in the adult population.[21] Unfortunately, intraoperative viral cultures and polymerase chain reaction (PCR) that may have helped elucidate this issue were not sent. Extended course intravenous acyclovir was recommended by infectious disease consultants as well and has traditionally been the treatment of choice for viral central nervous system infections. Oral valacyclovir recently has shown promise and may be used where intravenous acyclovir may not be available. Furthermore, despite being the initial test of choice, MRI was demonstrated to be imperfect in making a definitive diagnosis in this case. Although there was radiographic evidence of an epidural abscess, this was inconsistent with the intraoperative findings. Although obtaining an MRI was important and allowed for early operative intervention, this patient also needed early and simultaneous treatment for transverse myelitis (namely steroids) that was not realized until the post-operative MRI was obtained. Given the severity of his neurologic deficit, plasma exchange should also have been considered. Providers should be aware that overlap can occur between compressive and non-compressive spinal cord lesions, particularly in the setting of acute varicella zoster transverse myelitis. This case clearly demonstrates that there are situations in which treatment for both is warranted.

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How to cite this article: Bari V, Frommelt J, Jain V. Compressive epidural fluid collection secondary to varicella zoster transverse myelitis. *JACEP Open*. 2021;2:e12493. https://doi.org/10.1002/emp2.12493