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CASE REPORT

Transverse myelitis due to *Staphylococcus aureus* may occur without contiguous spread

M Saini[1], K Prasad[2], LM Ling[3] and K Tan[2]

Study design: A case report of staphylococcal transverse myelitis.
Objectives: To illustrate the clinical presentation of acute transverse myelitis due to *Staphylococcus aureus*, without a contiguous source of infection.
Setting: National Neuroscience Institute.
Case report: A 79-year-old female was diagnosed with acute transverse myelitis. Clues to an infectious etiology included fever, raised inflammatory markers and cerebrospinal fluid neutrophilic pleocytosis. Staphylococcal etiology was established based on cerebrospinal fluid and blood cultures. Despite extensive investigations, no contiguous or systemic source of infection could be identified. She was treated with appropriate antibiotics; however, neurological recovery was poor.
Conclusions: Bacterial myelitis may occur in isolation and the diagnosis should not be discounted when evaluation shows an absence of a contiguous or systemic source of infection.
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INTRODUCTION

Acute transverse myelitis (ATM) encompasses a heterogeneous group of autoimmune demyelinating, inflammatory or infectious disorders.[1] Infectious causes of myelitis can be viral, bacterial, fungal and rarely parasitic.[1] Bacterial myelitis or spinal cord abscess has been postulated to occur via hematogenous spread.[2,3] Kastenbauer *et al.*[4] separately described acute spinal cord dysfunction, including myelitis, during the course of bacterial meningitis. We report a rare case of isolated *Staphylococcus aureus* myelitis, wherein differentiation from transverse myelitis is critical for therapeutic decision-making.

CASE REPORT

A 79-year-old immunocompetent female presented with 2 days of fever and acute-onset bilateral lower limb weakness upon awakening. She was last seen ambulating independently the night before. There was no history of back trauma, pain or meningismus. She had well-controlled diabetes mellitus (HbA1c 6.3%), hypertension, hyperlipidemia, cervical spondylosis and ischemic heart disease. She was febrile (temperature 38 °C) and general physical, cardiovascular and respiratory examinations were normal. Neurological examination revealed flaccid paraplegia with acute urinary retention, absent lower limb deep tendon reflexes, flexor plantar responses and normal anal tone and lower limb sensory function.

Magnetic resonance imaging (MRI) of the spine showed a non-enhancing T2-weighted hyperintense intramedullary lesion at T11 (Figure 1). An enhancing L1 vertebral body lesion was noted; this was evident on MRI 1 year previously, with stable characteristics. Brain MRI was unremarkable. Cerebrospinal fluid (CSF) analysis revealed neutrophilic pleocytosis, raised proteins and normal glucose. Systemic inflammatory markers were raised, autoimmune work-up was negative (Table 1) and chest X-ray was normal. HIV serology was not performed. Treatment for transverse myelitis was initiated with intravenous (IV) methylprednisolone, concurrently with IV ceftriaxone (1 g daily).

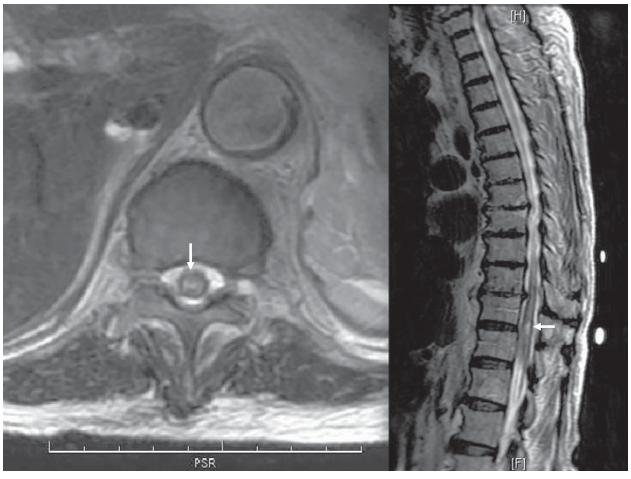

Figure 1 Axial (**a**) and sagittal (**b**) T2-weighted spinal MRI, showing central hyperintense lesion in the spinal cord at T11 (arrows).

Subsequently, blood and CSF culture yielded methicillin-sensitive *S. aureus* (MSSA) 2 days later. Methylprednisolone was stopped; antibiotics were modified to IV crystalline penicillin (4 megaunits 4 hourly) based on culture sensitivity. Further investigations were performed to localize the source of infection. Abdomen computed

[1]Department of General Medicine, Changi General Hospital, Singapore, Republic of Singapore; [2]Department of Neurology, National Neuroscience Institute, Singapore, Republic of Singapore and [3]Department of Infectious Disease, Tan Tock Seng Hospital, Singapore, Republic of Singapore
Correspondence: Dr K Tan, Department of Neurology, National Neuroscience Institute, 11 Jalan Tan Tock Seng, Singapore 308433, Republic of Singapore.
E-mail: Kevin_Tan@nni.com.sg
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Table 1 Selected laboratory results

	Days 1–3	*Days 3–7*	*Week 2*	*Week 3*	*Week 4*	*Reference range*
Laboratory parameters						
WBC Count ($\times 10^9 l^{-1}$)	15 (Neutrophils 85.7%)	7	14.2	10	5.2	4–10
ESR ($mm\,h^{-1}$)	99					
CRP ($mg\,l^{-1}$)	272	32.5	195	47	18	0–5
Procalcitonin ($\mu g\,l^{-1}$)	2	<0.5				<0.5
Antinuclear antibody	Negative					
Anti-double-stranded DNA	Negative					
Anti-cardiolipin IgG/IgM	Negative					
Blood culture	Methicillin-sensitive *Staphylococcus aureus*	No Growth	No growth			
Cerebrospinal fluid analysis						
Microscopy ($cells\,\mu l^{-1}$)	RBC 39, WBC 813 (neutrophils 95%, lymphocytes 5%)					WBC <5
Glucose ($mmol\,l^{-1}$)	4.2 (serum 6)					2.5–5.5
Protein ($g\,l^{-1}$)	2.8					0.1–0.4
Microscopy	Gram Smear Negative					
Oligoclonal bands	Negative					
Tetraplex PCR (CMV; VZV; HSV; *Toxoplasmosis gondii*)	Negative					
Viral culture (measles, mumps, poliovirus, HSV, Coxsackie A and B, echovirus, enterovirus)	Negative					
Bacterial culture	Methicillin-sensitive *S. aureus*					

Abbreviations: CRP, C-reactive protein; CMV, cytomegalovirus; ESR, erythrocyte sedimentation rate; HSV, Herpes simplex virus; Ig, immunoglobulin; RBC, red blood cell; VZV, varicella zoster virus; WBC, white blood cell.

tomography (CT) was normal; chest CT showed lower lobe atelectasis. As she did not have respiratory symptoms or productive cough before presentation, as well as during hospital stay, no other respiratory investigations were performed. Transthoracic echocardiography revealed thickened mitral valve and aortic sclerosis, with no vegetations; the patient declined trans-esophageal echocardiogram. Bone scintigraphy showed focal increased radiotracer uptake at T10/11 vertebrae, indicative of abnormal osteoblastic reaction; spine MRI did not reveal any corresponding changes in this area. Patient did not improve neurologically after 6 weeks of antibiotic treatment and was subsequently lost to follow-up.

DISCUSSION

Myelitis describes inflammation of the spinal cord, and may be secondary to a wide range of conditions. ATM incidence is estimated to be 1.34–4.6 per million.[1] Inflammatory etiologies of ATM are the commonest, with corticosteroids used as first-line treatment. Whereas inflammatory ATM has well-defined diagnostic criteria, it may be difficult to differentiate from infectious myelitis.[1]

Infectious myelitis commonly affects the thoracic cord, and presents with fever and paraparesis. Symptoms evolve over hours to days.[5] Additional clinical features, such as fever, meningismus, rash, concurrent systemic infection, an immunocompromised state, a history of recent travel, recurrent genital infection and radicular burning pain, may suggest specific infectious etiologies.[1] Etiological agents include viruses, bacteria, fungi and parasites. Bacterial myelitis or spinal cord abscess has been postulated to occur from hematogenous spread of a contiguous focus in the spine or secondary to bacteremia arising from a distant source.[2,3] Kastenbauer *et al.*[4] described acute spinal cord dysfunction during the course of bacterial meningitis and postulated venous congestion, ischemic infarction or myelitis as possible mechanisms.

Our patient presented with ATM and initially, it was difficult to distinguish between infectious myelitis and inflammatory ATM. Spine MRI was not helpful in establishing the diagnosis as the features were not diagnostic for either condition.[2,3] Fever, raised inflammatory markers, and systemic and CSF neutrophilic response guided the initial decision to treat with both antibiotics and corticosteroids. Our patient was subsequently diagnosed with MSSA myelitis proven on blood and CSF culture. Extensive investigations did not reveal evidence of cranial meningitis or contiguous MSSA infection in the spine or from a distant source. Bone scintigraphy showed vertebral osteoblastic changes. However, in the absence of corresponding MRI abnormalities, such changes are nonspecific and are unlikely to be indicative of osteomyelitis.

Bacterial myelitis is often not suspected on presentation as inflammatory ATM is relatively more common. Infectious myelitis are most commonly caused by viruses.[1] It is important to identify the etiology of infectious myelitis as they are treatable. In our patient, CSF neutrophilic pleocytosis and raised systemic inflammatory markers were clues to a bacterial myelitis. In addition, our patient demonstrated that bacterial myelitis may occur in isolation and the diagnosis should not be discounted when spinal imaging shows an absence of a contiguous source of infection.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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