Enteroviral Transverse Myelitis
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Citation

Abstract
We report a case of acute transverse myelitis due to enteroviral infection. Diagnosis was made following positive PCR of cerebrospinal fluid. She was treated with pleconaril, an antienteroviral agent. Enteroviral infection should be considered in the differential diagnosis of acute transverse myelitis.

INTRODUCTION
Transverse myelitis is an acute monophasic inflammatory disorder affecting the spinal cord. Patients may be of any age and present with a subacute paraparesis. CSF examination shows a cellular pleocytosis, often with polymorphs at the outset. Enterovirus infection is an uncommon cause of central nervous system infection, usually occurring in the immunosuppressed. We report a case of an 81-year-old female presenting with enteroviral induced transverse myelitis. We aim to overview the literature on enteroviral myelitis and discuss the possible role for pleconaril in its treatment.

CASE
An 81-year-old woman with a history of breast cancer ten years previously was admitted to hospital in August 2001 with an ascending paraplegia. Three weeks prior to admission she suffered an episode of shingles affecting her right side T8 to T10 dermatomes that resolved spontaneously. Seven days before admission she noticed tingling in the toes and soles of both feet, which subsequently ascended in association with weakness in both legs. Within five days she was unable to walk and subsequently developed painless urinary retention requiring catheterisation. On examination her cognitive function, speech, cranial nerves and optic fundi were normal. She had a flaccid paraparesis and a sensory level at T5. Initial blood tests revealed a low serum sodium (124 mmol/L), an elevated C-reactive protein (66 mg/L) with a normal ESR (12 mm/hr). An urgent MRI scan was performed which revealed high signal within the thoracic and lumbar spinal cord consistent with an acute transverse myelitis (ATM, figure1).
Figure 1
Figure 1: Sagittal (a) and axial (b) T2 images of the thoracic cord were obtained on a 1.5T MR scanner (Philips Gyroscan Intera 1.5T, Eindhoven, The Netherlands). Both images show a swollen thoracic cord with high T2 signal extending from T4 level to the conus. There was neither abnormal T1 signal, nor abnormal enhancement on T1 images post gadolinium. All imaging features are consistent with a transverse myelitis.

Brain MRI was normal. Subsequent CSF analysis revealed a polymorphonuclear leukocytosis (215 white cells / mm$^3$ of which 50% were polymorphs and 50% were degenerative cells), and a high CSF protein (1.21 g/L) but a normal CSF glucose. CSF from the initial tap was negative for Varicella Zoster (VZV) and Herpes simplex virus PCR, but serum VZV IgM was positive with a titre of 1:32 consistent with recent infection. There was no clinical or radiological evidence of recurrence of breast cancer. She was commenced on intravenous methylprednisolone (500 mg daily for five days) and acyclovir (10mg/Kg five times daily for 10 days).

Repeat CSF analysis performed five days later revealed a persistently raised CSF protein (1.70 g/L) but a reduction in the number of leukocytes (72 / mm$^3$ of which 10% were lymphocytes, 80% were polymorphs and 10% degenerative cells). PCR analysis of the CSF was again negative for VZV but was positive for enterovirus (EV) group RNA. A further CSF sample taken five days later was again VZV PCR negative, but positive for EV RNA. CSF analysis for oligoclonal bands was negative on each occasion. Pleconaril (200mg t.i.d.) was obtained from the manufacturer on a named-patient basis, and commenced one month after the onset of symptoms. She completed a week's course without improvement in her neurological status. There was no evidence of neurological improvement when she was reassessed 3 months after initial presentation.

DISCUSSION
EV infection is common in the general population, but most cases are mild and are associated with an isolated fever or a specific syndrome such as hand-foot and mouth disease. Rarely EV may cause more severe infections including meningo-encephalitis or myocarditis, often in the immunocompromised patient. In a significant proportion of cases the aetiology of ATM remains obscure, although a viral cause may be implicated. Enterovirus has been identified in some children with ATM, and EV may be an under-recognised cause of ATM in adults. Rapid diagnosis is now possible with polymerase chain reaction (PCR) analysis for EV RNA, which is sensitive and specific for EV infection. Pleconaril interferes with EV attachment by binding to the virus protein capsid, and has an antiviral activity of over 90% on commonly circulating serotypes. It is effective in treating children with EV infection, and preliminary evidence supports its use in adults. ATM leads to permanent disability in 40% to 60% of patients, and our patient showed no signs of recovery three months after initial presentation. It is possible that the early diagnosis of EV myelitis and prompt treatment with Pleconaril may improve the clinical outcome.

References
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