Acute Onset Paraparesis: A Case Study

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Greys Hospital
February 2008
History

- Patient BZ
- 39 old male
- Presented with sudden onset bilateral lower limb paralysis
- Associated numbness and paraesthesia involving both limbs
- Associated urinary retention
- No faecal incontinence, but did complain of constipation
- No other symptoms
PMH: Was admitted 1/52 prior to neurology presentation at Northdale Hospital for a diarrhoeal illness and was subsequently discharged after 3 days.
No other previous medical history

PSH: Laparotomy 2004 aetiology unknown

Medication: No chronic or current medication use.
Social History:
Resides in Mpomeni with his fiancée and ten year old daughter who are both well. Previous smoker with a ten pack year history. Social alcohol intake.

Family History: Non-contributory

Allergies: nil
Physical Examination

- General Examination:
  - Well looking patient with good hydration
  - Tinea capitis and Tinea pedis noted
  - Right posterior triangle and right epitrochlear lymphadenopathy present
  - Melanonychia
  - No other clinical findings
CVS Exam: BP 117/75 PR 87
   JVP not elevated
   AB undisplaced
   Normal heart sounds
   No murmurs
   No carotid bruits

Respiratory Exam: Not distressed
   Lung fields clear
   No adventitious sounds
Abdominal Examination: Laparotomy scar
  Soft
  Non-tender
  Hepatomegaly 3 cm
  No splenomegaly
  No ascites
  Normal bowel sounds
CNS Examination

- Higher function was intact
- Right handed
- No meningism
- All cranial nerves intact
- No anatomical abnormalities of the spine and skull
Motor System Upper Limbs

- Tone, power and reflexes were normal in both upper limbs.
Motor System Lower Limbs

- Decreased tone
- Power was zero
- Absent reflexes
- Downgoing planters
## Sensation

<table>
<thead>
<tr>
<th></th>
<th>Right</th>
<th>Left</th>
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<tbody>
<tr>
<td>Pinprick</td>
<td>L1</td>
<td>T12</td>
</tr>
<tr>
<td>Light touch</td>
<td>L1</td>
<td>T12</td>
</tr>
<tr>
<td>Vibration</td>
<td>Preserved</td>
<td>Preserved</td>
</tr>
<tr>
<td>Proprioception</td>
<td>Preserved</td>
<td>Preserved</td>
</tr>
</tbody>
</table>
- Sphincter tone was decreased
- Decreased peri-anal sensation
- Abdominal Reflexes – absent
- Primitive Reflexes – absent
- No clinical evidence of cerebellar disease
- The patient was not ambulant
Assessment

- 39 year old male
- Recent diarrhoeal illness
- Clinical stigmata of RVD
- Acute onset flaccid paraparesis with bladder and bowel involvement
- Sensory level at L1 on the right and T12 on the left
- Sparing of the dorsal columns
- Features in keeping with spinal shock
Where is the lesion?

- Anterior aspect of the spinal cord between T12 and L1
- Parasagittal lesion
Differential Diagnosis of Acute Onset Paraparesis

1) Extramedullary Lesions (Intra/Extra Dural)
   - Spinal trauma
   - Pathological fractures
   - Epidural abscess
   - Dural AVM
   - Bleeding into a mass lesion

2) Intramedullary lesions

   See flow chart
Differential Flow Chart in Acute Onset Paraparesis: Intramedullary Causes

- Intramedullary
  - Demyelinating
    - MS
    - ADEM
  - Ischaemia
    - Infarction
    - Haemorrhage
  - Myelitis
    - Viral
    - Bacterial
    - Parasitic
    - Parainfection
  - Trauma
  - Vasculitis
  - Global Ischaemia
  - Thromboembolism
Investigations

- Blood investigations
- Structural imaging – MRI spine
- CSF examination
<p>| | | |</p>
<table>
<thead>
<tr>
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<tbody>
<tr>
<td><strong>FBC</strong></td>
<td><strong>WCC</strong></td>
<td>4.8</td>
</tr>
<tr>
<td><strong>Hb</strong></td>
<td>13</td>
<td></td>
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<tr>
<td><strong>MCV</strong></td>
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<tr>
<td><strong>MCH</strong></td>
<td>32.5</td>
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<tr>
<td><strong>HCT</strong></td>
<td>38 %</td>
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<td><strong>PLTS</strong></td>
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<tr>
<td><strong>ESR</strong></td>
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<tr>
<td><strong>CRP</strong></td>
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<tr>
<td><strong>Vit B12</strong></td>
<td>860</td>
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<tr>
<td><strong>Folate</strong></td>
<td>4.81</td>
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# Blood Investigations

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<tbody>
<tr>
<td>U/E</td>
<td>Na</td>
<td>134</td>
</tr>
<tr>
<td>K</td>
<td>4.27</td>
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<tr>
<td>Cl</td>
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<td>HCO3</td>
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<tr>
<td>Urea</td>
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<tr>
<td>Cr</td>
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<tr>
<td>CPM</td>
<td>Ca</td>
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<td>PO4</td>
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<td>Mg</td>
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<td>CK</td>
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## Blood Investigations

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<tr>
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<tr>
<td>Bili</td>
<td>21</td>
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<tr>
<td>DBili</td>
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<tr>
<td>ALP</td>
<td>109</td>
<td></td>
</tr>
<tr>
<td>GGT</td>
<td>99</td>
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</tr>
<tr>
<td>ALT</td>
<td>53</td>
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</tr>
<tr>
<td>Glu</td>
<td>4.6</td>
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<tr>
<td>TSH</td>
<td>0.96</td>
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<tr>
<td>RPR</td>
<td>N/R</td>
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## Blood Investigations

<table>
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<tr>
<th>Connective Tissue Screen</th>
<th>RF</th>
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<tr>
<td></td>
<td>ANF</td>
<td>neg</td>
</tr>
<tr>
<td></td>
<td>SACE</td>
<td>neg</td>
</tr>
<tr>
<td>SPEP</td>
<td></td>
<td>neg</td>
</tr>
<tr>
<td>VCT</td>
<td>RVD</td>
<td>pos</td>
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<tr>
<td></td>
<td>CD4</td>
<td>pending</td>
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</table>
MRI: T1 Without Gadolinium
MRI: T2W clear
MRI: T1W With Gadolinium
MRI Findings

- T1 weighted – hyperintensity at T11 indicating oedema, inflammation and possibly haemorrhage
- T2 weighted – lesion at T11 becomes hypotense, hyperintensity extending longitudinally from T6 to T10
- T1 weighted with Gadolinium- slight enhancement post gadolinium injection at the T11 region.
A final assessment of a longitudinal myelitis extending from T6 – T10 with a possible haemorrhage at T11 localised to the anterior aspect of the spinal cord was made.

Suggestive of an anterior spinal cord infarct.
# CSF Analysis

<table>
<thead>
<tr>
<th>CSF</th>
<th>Polys</th>
<th>Lymphs</th>
<th>RBC</th>
<th>Protein</th>
<th>Glucose</th>
<th>India Ink</th>
<th>CLAT</th>
<th>Cytology</th>
<th>Neurocytercicosis ELISA</th>
<th>Neurotropic Viruses</th>
<th>FTA-Abs</th>
<th>Opening Pressure</th>
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<tbody>
<tr>
<td></td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0.44</td>
<td>2.8</td>
<td>Neg</td>
<td>Neg</td>
<td>Neutrophils, histiocytes</td>
<td>Neg</td>
<td>Neg</td>
<td>Neg</td>
<td>16 cmH20</td>
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Working Diagnosis of Intramedullary Cord Lesion

- Myelitis with or without haemorrhage
- Anterior spinal artery infarct in the region of T11
- Demyelinating disorder
Differential Flow Chart in Acute Onset Paraparesis: Intramedullary Causes

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  - Thromboembolism
Demyelinating Disorders: MS

- Multiple Sclerosis (MS)
  1) Relapsing-remitting or progressive course
  2) Pathological triad of i) CNS inflammation ii) demyelination iii) gliosis
  3) Autoimmune disease
  4) Caucasian population
Demyelinating Disorders: ADEM

- Acute Disseminated Encephalomyelitis
- Antecedent immunisation - post vaccinal encephalomyelitis (rabies, influenza, DPT, hepatitis B etc)
- Antecedent infection - postinfectious encephalomyelitis (measles, rubella, herpesviruses, influenza, mycoplasma)
- Immune response to myelin basic protein (MBP)
ADEM continued

- Clinical features
  1) Rapid onset
  2) Focal or multifocal neurological dysfunction
  3) Recent infection or immunisation
  4) Monophasic course
ADEM continued

- Pathology - perivascular inflammation and demyelination
- Severe form of ADEM also known as acute haemorrhagic leucoencephalitis results in vasculitic and haemorrhagic lesions with a devastating clinical course
ADEM continued

Diagnosis

1) History: recent immunisation or infection
2) Physical exam: neurological deficit of rapid onset
3) CSF: mildly elevated protein, lymphocytic pleocytosis, mixed polymorphonuclear-lymphocytic pattern in initial stages, transient CSF oligoclonal banding (rare)
4) MRI: gadolinium enhancement of white matter in brain and spinal cord
Viral Causes of Myelitis

- **Immunocompetent:** HSV 2, VZV, EBV, rabies virus, polioviruses (now rare because of immunisation)
- **Immunocompromised:** CMV, HTLV1 (Tropical Spastic Paraparesis)
- Chronic viral myelitis can be associated with advanced HIV as well as HTLV1 on the basis of a vacuolar myelopathy
Bacterial Causes Of Myelitis

- Rare
- Almost any pathogenic species
- *Listeria monocytogenes* most frequently isolated
Parasitic Causes of Myelitis

- **Toxoplasmosis**: associated with HIV
- **Schistosomiasis**: intensely inflammatory and granulomatous myelitis secondary to a tissue-digesting enzyme produced by the ova
Parainfectious Myelitis

- Non-specific immune mediated inflammatory reaction related to infection of any aetiology and occurring at any point during the course of the illness.
Spinal Cord Infarction

- Blood supplied to the spinal cord by a single anterior spinal artery and paired posterior spinal arteries.

Causes of spinal cord infarction include:

- Traumatic: fracture dislocation of vertebrae or acute back trauma (embolism of nucleus pulposus material into spinal vessels)

- Global Ischemia: e.g. shock, cardiorespiratory arrest, aortic dissection
Spinal Cord Infarction continued

- Haemorrhagic infarcts – DIC and other platelet abnormalities or bleeding disorders
- Hypercoaguable states
- Endothelial abnormalities – Vasculitis e.g. connective tissue diseases and HIV
Spinal Cord Infarction continued

- Cardiogenic emboli
- Thromboembolism of any cause in arterial feeders
- Surgical clipping of aortic aneurysms
- Pregnancy
Main Considerations

1) Para infectious Myelitis

Supporting the diagnosis:
clinical presentation i.e. recent viral illness and acute onset of symptoms
MRI findings suggestive
Main Considerations

2) Viral Myelitis – HSV / CMV

Supporting the diagnosis:
- CMV – initial presentation with GE
- HSV – commonly associated with HIV

Against the diagnosis
- No suggestive skin lesions
- Neurotropic viral screen negative
3) Anterior Spinal Cord Infarct

Supporting the diagnosis

Injury localised to a vascular territory

Haemorrhagic injury seen on MRI

Acute Onset
Main Considerations

4) ADEM

Supporting the diagnosis:
clinical presentation i.e. recent viral illness and acute onset of symptoms
MRI findings suggestive

Against the diagnosis:
CSF findings
Lesion was localised
Management

- Mr BZ was managed for both ADEM and HSV myelitis
- Acyclovir 750 mg tds ivi 10/7
- Methylprednisolone 500mg od ivi 5/7
- Paracod 1g tds po
- Physiotherapy
- He will be followed up at the clinic in one month to review outstanding results and to assess progress
- At time of discharge there was no improvement in neurological deficit
The End

Thank You