Clinical Reviews

The Patient with Acute Paraplegia: A Problem-Based Review

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Introduction

Acute paraplegia is an emergency requiring immediate assessment by the acute medical team because of the need to rule out compressive lesions of the cord. Early intervention may preserve neurological spinal function and limit persistent disability. In addition, acute paraplegia may be complicated by life-threatening problems. These require prompt recognition and treatment.

The following clinical scenario, based on a real case of acute paraplegia managed by the authors is aimed at providing a problem-based approach to the management of patients presenting with acute paraplegic weakness.

Case History

A previously independent 86-year old woman presented with loss of weakness in both her lower limbs. She said her legs suddenly gave way while she was standing in the kitchen, which then led to her falling down. Since then she had been unable to move either of her legs.

She denied any back pain or fever with no upper limb weakness. She had not passed urine for eight hours with no sensation of bladder fullness and dribbling urinary incontinence. She denied any sensory loss or paraesthesia. She had no headache, visual or speech impairment. She had experienced one similar episode of leg weakness several years previously which had resolved within twenty-four hours. Her past medical history included ischaemic heart disease, hypertension and hypercholesterolemia. There was no known history of atrial fibrillation. She was taking aspirin, simvastatin and bendroflumethiazide.

Examination of both lower limbs showed flaccid tone bilaterally with power graded at 0/5 proximally and distally, bilaterally. Deep tendon reflexes were absent in the lower limbs with absent plantar response bilaterally. Pinprick sensation was absent below the T5 sensory level. Vibration sense was diminished to the hips bilaterally, but position sense and light touch modalities were intact. Abdominal reflexes were absent bilaterally. On digital rectal examination, reduced anal tone was noted and reduced perianal sensation to pin prick was also noted. Cranial nerves and upper limb examination were normal. Cardiovascular, respiratory and abdominal examinations were unremarkable apart from an irregularly irregular pulse of 96 beats per minutes confirmed on 12-lead electrocardiogram as atrial fibrillation (AF).

Where does acute paraplegia localise in the nervous system?

Lesions in a variety of sites in the nervous system may present with acute paraplegia; these include:

- The parasaggital area of the brain, causing predominant lower extremity weakness by involving the motor cortex bilaterally.
- A spinal cord lesion at any level may cause isolated lower extremity weakness and sensory loss, since the lamination pattern of the ascending and descending tracts places the leg fibres peripherally where they are vulnerable to external compressive lesions throughout their course
- The cauda equina, which may be damaged by extrinsic compression or by inflammatory and infiltrative processes.
- Peripheral nerves lesions, such as acute inflammatory polyradiculopathy (e.g. Guillan Barre Syndrome)
- Disorders of muscle or neuromuscular junction such as myasthenia gravis.

Acute paraplegia may also be a manifestation of a non-organic/psychogenic disorder, such as conversion reaction; this diagnosis should usually only be considered after an 'organic' cause has been excluded. A list of common causes of paraplegia is summarised in Table 1.

What key aspects of the history should be sought?

Rate of onset of symptoms is of key importance in determining the cause of acute paraplegia

- Paraplegia of sudden onset is most likely to be of traumatic, vascular or compressive aetiology.
- An inflammatory process of the spinal cord

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would typically have a more subacute, escalating presentation but could also present acutely.[1-2]

A number of other specific historical features may be helpful in localising the site and underlying cause of acute paraplegia:

- Presence of headache, speech or visual impairment in association with bowel and bladder involvement may suggest a parasaggital lesion.
- Back pain in combination with urinary and bowel symptoms would suggest a spinal cord or cauda equinal lesion; additionally presence of saddle anaesthesia would strongly support the latter diagnosis.
- Back pain may also be a feature of Guillian-Barre syndrome (GBS); a history of recent vaccination or infection, nerve root pain or autonomic symptoms may also suggest this diagnosis.
- Fever, backache and spinal tenderness with prodromal symptoms and malaise may point to a diagnosis of epidural abscess.
- Presence of sensory symptoms would exclude primary muscle or neuromuscular pathology, whereas, drooping of eyelids and or double vision and especially fatigable weakness is suggestive of myasthenia gravis.
- Inability to raise the arms up or rise from a low stool would point to a proximal myopathic process.

Careful evaluation of past medical history may also be helpful:

- A prior history of malignancy in a patient with backache and acute paraplegia would suggest a metastatic compressive lesion in the spinal cord.
- Previous cerebrovascular or ischaemic heart disease or vascular risk factors should raise suspicion of an ischaemic vascular cause.
- Past medical history of similar illness could suggest an embolic process or spinal dural arterio-venous fistula (AVF). In the latter the presentation is one of step like progression.
- Use of warfarin could suggest a haemorrhagic process in the spine or brain.

What key physical examination findings could help in localisation and differential diagnosis?

The physical examination is of paramount importance for localisation of the lesion and in differential diagnosis. The following specific features should be considered:

1. Symmetry
- o Symmetical or virtually symmetrical weakness usually is suggestive of a spinal cord or conus medullaris lesion.
- o Markedly asymmetrical weakness could indicate a lesion at the nerve root, in the lumbar plexus, or more distally in a single nerve.
- o A parasaggital lesion may also cause symmetrical or asymmetrical weakness.

2. Reflexes
- o A root lesion, such as a tumour or herniated disc, may cause loss of a single reflex.
- o An acute spinal cord lesion may cause loss of reflexes symmetrically at and below the level of the lesion with normal reflexes above.
- o Symmetrical decrease of reflexes may also be due to peripheral nerve lesions or dorsal root involvement as in acute inflammatory polyneuropathy (GBS). Non-acute lesions of the spinal cord usually cause hyperreflexia below the lesion.
- o Loss of abdominal reflexes occurs in upper motor neuron (UMN) lesions above thoracic levels and are unilateral (loss of upper abdominal reflexes suggests level above T8/9 and lower reflexes loss, lesions above T11/12). In conversion reaction, reflexes would be normal.
- o An extensor plantar response indicates an upper motor neuron lesion i.e. brain or spinal cord.

3. Sensation
- o Sensory examination may be helpful in localisation, but may be misleading as a thoracic level may be associated with cervical lesion.
- o Pain and temperature sensations cross in the spinal cord within several segments of entry, while touch and vibration ascend ipsilaterally in the cord. Therefore loss of pain and temperature on one side and touch and vibration on the other suggest a unilateral lesion (Brown-Sequard syndrome). Loss of all of these on one side suggests a lesion above the cervical cord or a functional disorder.
- o Intrinsic cord lesions (intramedullary) classically spare the perianal area, because the sensory tracts are most lateral in the cord.
- o With extrinsic lesions, however, compression of the outside of the cord involves the sacral areas. Absent anal reflex suggests spinal cord or conus involvement.
- o Peripheral neuropathies may produce a decrease or loss of sensation in the distribution of the nerve or in a ''glove and stocking distribution'' and areflexia.

What investigation should be done, when and why?

Spinal MRI is an essential investigation in the diagnosis of acute paraplegia. Its most important role is to rule out the diagnosis of compressive myelopathy.[3] Even when the deficits are not severe, acute myelopathic signs need urgent evaluation because neurological deterioration can occur abruptly. In addition, the clinical impairment at the time of intervention often dictates the chances of a good neurological outcome. This is particularly the case for compressive myelopathic causes such as epidural spinal

Lesion	Course	Clinical features
Parasaggital lesion: e.g. meningioma	Progressive/ Subacute	Headaches, seizures, spastic paraplegia with bladder and bowel involvement
Cervical spondylotic myelopathy	Progressive or step-wise course	Gait and leg spasticity and amyotrophy of hand or arms in moderate –severe cases
Transverse myelitis	Subacute	Segmental cord syndrome
Guillian-Barre syndrome	Acute-subacute	Ascending weakness, bulbar, respiratory and autonomic involvement
Epidural abscess	Subacute;may worsen abruptly	Segmental cord syndromespinal tenderness,raised inflammatory markers
Spinal cord infarction	Abrupt onset	Anterior cord syndrome(if cord oedema can give impression of complete cord involvemet),spinal shock-flaccidity
Vascular (intradural) arteriovenous malformation(AVM)	Acute to subacute	Age<30 years old, subarachnoid haemorrhage and severe backache('coup de poignard') ,intra-parenchymal haemorrhage, vascular steal phenomenon, or mass effect in spinal cord
Spinal dural arterio-venous fistula(AVF)	Stepwise progression	Age>40 years old, males> females, progressive leg weakness with concomitant bowel and bladder dysfunction
Myopathy	Subacute	Proximal weakness (distal weakness typical in inclusion body myositis)
Neuromuscular junction problem	Usually subacute	Fatigable weakness, diplopia, bulbar and respiratory involvement, proximal weakness
Epidural metastases	Subacute, may worsen abruptly	Segmental cord syndrome
Epidural haematoma	acute	Segmental cord syndrome
Hysteria	Variable	No clear pattern, variable, inconsistent, effort-related give way weakness, normal objective examination

Table 1. Causes of acute paraplegia.

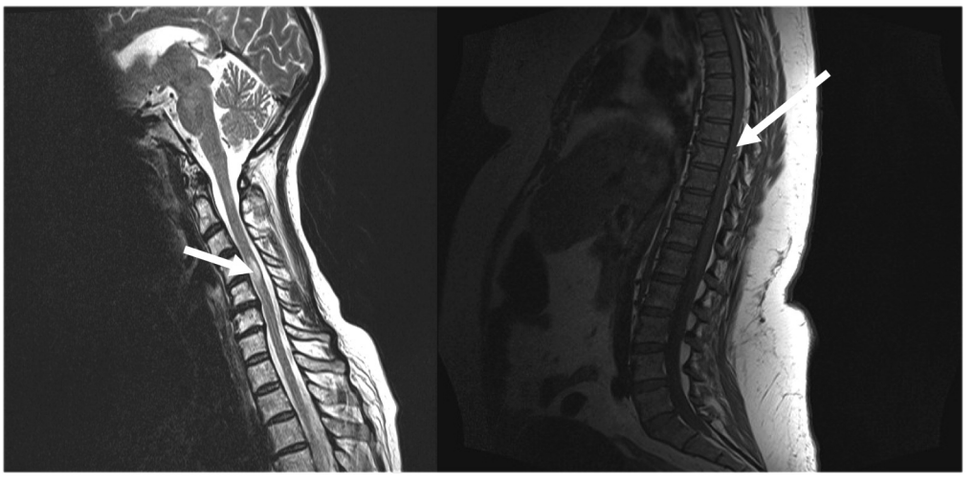

Figure 1. Transverse myelitis (left) and thoracic posterior epidural collection at T8/9 level (right).

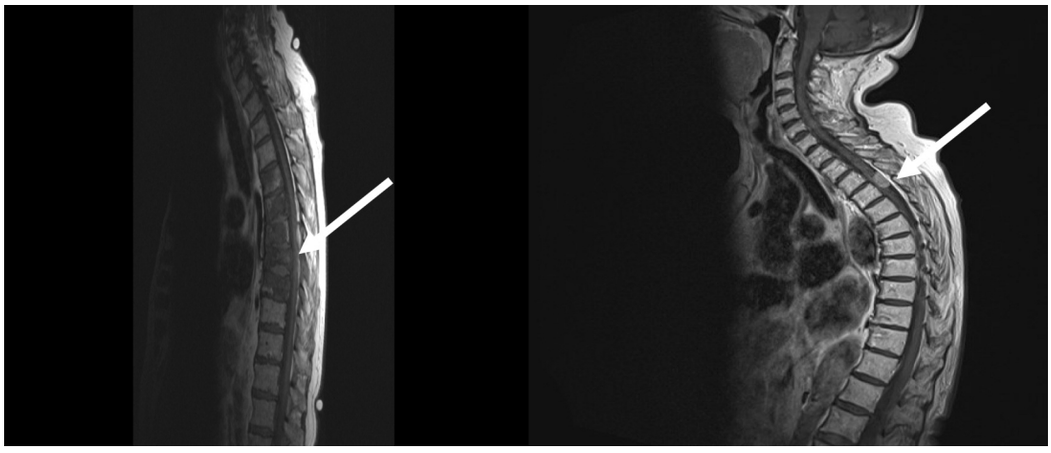

Figure 2. Multiple thoracic metastases with spinal cord compression (left) and thoracic meningioma (right).

haematoma/ abscess and spinal cord metastases. Thorough competent neurological examination can pinpoint the level of the lesion guiding which area to image.

In spinal cord infarction MRI can provide confirmatory evidence and information as to the underlying aetiology.[4] MRI changes associated with spinal cord ischaemia include hyperintense changes within the cord on T2 weighted images.[5] In addition, the presence of vertebral body infarction adjacent to a cord signal abnormality on MRI is a specific indicator of ischaemia and is a useful confirmatory sign.[6] Where a vascular malformation is suspected a dedicated spinal angiogram should be considered for diagnostic and management purposes to find a feeding vessel and suitability for intervention(radiological or surgical).

Other investigations will be determined by the likely cause. Lumbar puncture may be warranted in suspected inflammatory or malignant disease; CSF may also be abnormal in patients with vascular causes of paraplegia.[7-9] Patients with ischaemic vascular lesions may require cardiac imaging with trans-thoracic or trans-oesophageal echocardiography to look for a cardiac source of embolism.[10-12]

What specific treatments are available for acute paraplegia?

Specific treatment in acute paraplegia depends on the underlying aetiology and are summarised in Table 2.

Aetiology	Treatment
Anterior spinal cord infarction	Supportive
Epidural haematoma	Drainage
Epidural metastases	Dexamethasone/ Radiotherapy-if amenable surgical excision
Guillian-Barre syndrome	Intravenous immuno-globulin/plasmapheresis
Parasaggital lesion	Surgery, radiotherapy, dexamethasone, chemotherapy
Transverse myelitis	Steroid if due to demyelination
Vascular malformation	Surgery/ interventional radiology

Table 2. Treatment of common causes of acute paraplegia.

What are the other key management issues in acute paraplegia?

Acute paraplegia from a spinal cord lesion can compromise respiratory function. Lesions that interrupt innervation to the diaphragm via C3-5 segments may compromise respiration predisposing to respiratory complications including weakness of the diaphragm and chest wall muscles. If signs of impending respiratory failure develop, the patient should be urgently assessed for respiratory support.

Low molecular weight heparin should be started to prevent venous thromboembolism assuming a haemorrhagic cause has been excluded. Frequent turning and use of a pressure relieving mattress should be instituted to prevent pressures sores. Prophylaxis against stress ulceration should be commenced particularly in cases with high cervical cord lesions. Patients with a cervical cord lesion may lack vasomotor control and cannot sweat below the lesion making them sensitive to environmental changes in temperature, which should be monitored and maintained. Physiotherapy and occupational therapy should be started as soon as possible. Early psychological support should be provided for patients and their families.

What is the prognosis in acute paraplegia?

Prognosis in acute paraplegia depends on prompt and accurate diagnosis to rule out or treat those lesions requiring surgical intervention. All patients with paraplegia should be transferred to a spinal unit as soon as possible after initial assessment, treatment and stabilisation for further rehabilitation. In the case of spinal cord infarction, poor prognostic factors for recovery include severe impairment at presentation, advanced age, female sex and no sign of improvement in the first 24 hours.[8-9]

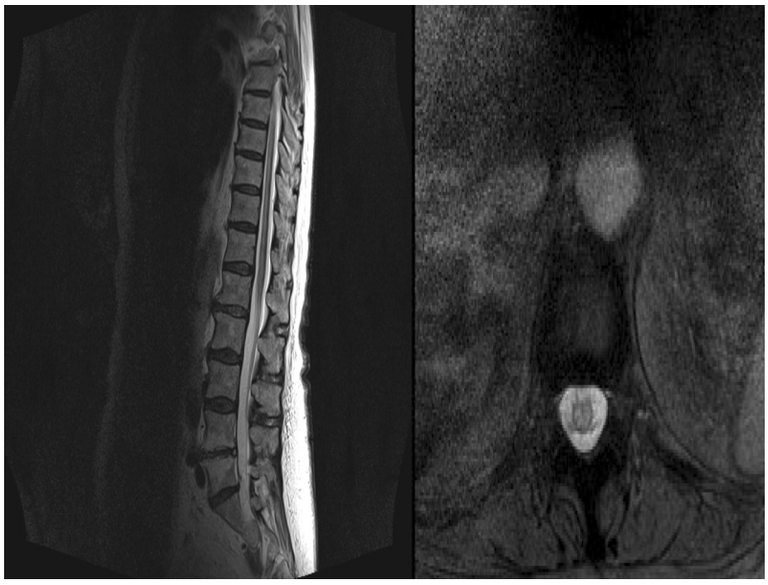

Figure 3. T2-weighted thoracic spine MRI revealing hyper-intensity particularly on axial images (right) suggesting anterior spinal cord infarction.

Aortic disease	**Miscellaneous conditions**
Aortic dissection	Cocaine abuse
Aortic aneurysm	Decompression sickness
Coarctation of the aorta	Spinal arteriovenous malformation
Aortic procedure	Vertebral artery dissection
Aortic surgery	**Spine disease**
Aortography	Spine surgery
Cardiogenic embolism	Cervical spondylosis
Bacterial endocarditis	Fibrocartilaginous embolism
Atrial myxoma	**Systemic low perfusion states**
Mitral valve disease	Cardiac arrest
Patent foramen ovale	Severe bleeding
Haematological conditions	**Trauma**
SCA	**Vasculitic diosrders**
Thrombophilic disorders	Behcet syndrome
Infections	GCA
Bacterial meningitis	PAN
Syphilis	SLE

Table 3. Causes of spinal cord infarction.
SCA=sickle cell anaemia; GCA=giant cell arteritis; PAN=polyarteritis nodosa; SLE=systemic lupus erythematosus

Case progression

MRI revealed increased signal on axial T2 weighted sequences from the thoracic cord region, suggestive of acute spinal cord ischaemia/infarction due to anterior spinal artery occlusion (Figure 3). There was no cord swelling. There was no evidence of bony metastases, vertebral fracture, epidural mass/abscess or vascular malformation. A simultaneous MRI of the head revealed bilateral cerebral hemispheric white matter changes consistent with ischaemia.

The results of full blood count, blood urea, serum electrolytes, creatinine, bone profile, serum protein electrophoresis and inflammatory markers were normal. Chest x-ray was unremarkable.

She was commenced on low molecular weight heparin and warfarin.

After acute stabilisation, she was transferred to a rehabilitation unit. After 2 months of intensive rehabilitation, she had neither regained movement of her lower limbs nor bladder function but was able to use a manual wheel chair. She required hoisting with the assistance of two and needed daily intermittent catherisation. Following home visit, she was discharged home with full care package and 24 hour home care.

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