CASE REPORT

Spinal cord infarction: a rare cause of paraplegia

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Accepted 6 June 2014

SUMMARY

Spinal cord infarction is rare and represents a diagnostic challenge for many physicians. There are few reported cases worldwide with a prevalence of 1.2% of all strokes. Circulation to the spinal cord is supplied by a rich anastomosis. The anterior spinal artery supplies the anterior two thirds of the spinal cord and infarction to this area is marked by paralysis, spinothalamic sensory deficit and loss of sphincter control depending on where the lesion is. Treatment of spinal cord infarction focuses on rehabilitation with diverse outcomes. This report presents a case of acute spinal cord infarction with acquisition of MRI to aid diagnosis.

BACKGROUND

Spinal cord infarction is a rarity and can vary in its onset, severity, outcome and recovery resulting in diagnostic challenge for clinicians. Treatment options for this relatively rare condition also remain elusive.

CASE PRESENTATION

An 81-year-old Caucasian man presented to the emergency department of a district general hospital with sudden onset leg weakness. He had a background of ischaemic heart disease, peripheral vascular disease, chronic kidney disease and chronic obstructive pulmonary disease. He described initially feeling a tingling sensation in his lower back which radiated down both legs which subsequently developed into progressive leg weakness. He denied any changes in speech and facial or upper limb weakness. He reported a similar episode of leg weakness which had occurred 3 months previously and spontaneously resolved in a couple of minutes. He lived alone, was independently mobile and had a 40-pack-year history of smoking.

On examination, his blood pressure was 169/79 mm Hg and his heart rate was regular at 70 bpm. Lower limb examination demonstrated a flaccid paraparesis with power of 0/5 on the Medical Research Council (MRC) scale of lower limbs globally with absent deep tendon reflexes.

Sensory modality of pin prick and temperature were impaired below the level of T10 but vibration, touch and proprioception were intact. Cranial nerve and upper limb examination was unremarkable.

No focal spinal tenderness was elicited. A digital rectal examination revealed normal perianal sensation with absent anal tone. He had a palpable bladder with a residual volume of 1000 mL and was subsequently catheterised.

INVESTIGATIONS

Baseline blood tests and urinalysis were unremarkable. ECG showed longstanding right bundle

Figure 1 MRI (sagittal T2-weighted image) of the lumbar spine showing degenerative changes and significant compression to the cauda equina is a possibility at L2/3 and particularly L3/4 (arrowed).

branch block and T wave inversion in the lateral leads and chest radiograph showed hyperinflation of the lungs.

The patient was referred to the orthopaedic team to rule out cauda equina syndrome.

MRI of his lumbar spine (figure 1) was reported as showing possible cauda equina at the level of L2–4. The images were discussed with the neurosurgical team at a tertiary centre who felt the images were more suggestive of an acute vascular event to the spinal cord and advised further imaging. MRI (figures 2 and 3) of the cervical and thoracic spine showed central signal change in the distal thoracic cord with surrounding oedema. The findings were felt to be in keeping with a diagnosis of central cord ischaemia.

DIFFERENTIAL DIAGNOSIS

Some differential diagnosis which could mimic spinal cord infarction resulting in paraplegia include cauda equina syndrome, space occupying lesions, infection and arteriovenous malformations compressing the spinal cord; albeit a vascular lesion presents more acutely.

TREATMENT

The patient was started on aspirin, clopidogrel, simvastatin and low-molecular-weight heparin for thromboprophylaxis and transferred to the stroke unit for rehabilitation. During his admission, he

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To cite: Patel S, Naidoo K, Thomas P. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2013-202793

Figure 2 MRI (sagittal T2-weighted image) of the thoracic spine showing central signal changes in distal thoracic cord with some expansion, consistent with central ischaemia (arrowed).

developed autonomic dysfunction with labile blood pressure which was managed cautiously with intravenous fluids.

OUTCOME AND FOLLOW-UP

Rehabilitation with physiotherapy was started to help build muscle strength and assess balance. On day 6 of admission, some improvement was observed in the power of his lower limbs and he was able to sit up independently. However, the patient voiced that he was reluctant to reach out for objects as he felt that he would fall; he required a lot of patience and reassurance. By 2 months, the patient was able to sit up independently and reach out for objects confidently with some dorsiflexion and plantarflexion of his feet.

Owing to his bedridden status, he developed urosepsis and constipation which were managed with antibiotics and laxatives, respectively. Unfortunately, he had failed his trial without catheter and was discharged home with a catheter and an outpatient urology appointment. After 2 months as an inpatient, he was

Figure 3 MRI (axial T2-weighted image) showing central signal change in the distal thoracic cord (arrowed).

transferred to a spinal rehabilitation centre with neurologist follow-up.

DISCUSSION

A vascular event involving the anterior spinal artery was evident in this case following exclusion of a compressive cause and history of atherosclerosis, together with MRI to confirm the diagnosis and delineate the extent of damage. The previous episode of leg weakness was thought to be due to a transient ischaemic attack. This case report highlights that spinal cord infarction is sudden in onset, dramatic in capacity and frequently disabling.

The clinical picture primarily depends on where the occlusion is, may accompany back pain radiating caudally and loss of sphincter control with hesitancy or inability to void or defecate. The acute stage may last for several days and is marked by initial flaccid paralysis, depression or absent of reflexes with a sensory level distal to the lesion where pain and temperature are not perceived. Over time there is increased muscle tone and reflexes become hyperactive and babinski reflexes positive.¹ Proprioception and vibration are usually intact as they are supplied by the dorsal columns which lie medially whereas the spinothalamic tract lies more laterally. In contrast, sensation to touch is a subjective finding during examination as light touch and well-localised touch is carried by spinothalamic and dorsal columns.²

Diagnostic challenges are attributed by the vast number of differential diagnoses which can mimic spinal cord infarction including Guillain-Barré syndrome, transverse myelopathy, mass lesions and extremes of potassium levels, albeit a vascular lesion presents more acutely.³

MRI is a sensitive and reliable modality for diagnosis of spinal cord infarction. It can show signal changes and swelling in the cord within just a few hours of onset.⁴ MRI may also highlight other causes of circulatory compromise to the spinal cord such as extradural compression, arteriovenous malformations and space occupying lesions.⁵ Spinal cord infarction is often associated with vascular malformations which are not always obvious on MRI. There is suggestion in the literature that young patients with suspected spinal cord infarction should have a spinal angiogram to exclude vascular malformations. Vertebral angiography is risky and technically difficult and often only available at tertiary centres. There is also an associated risk of exacerbating ischaemia.⁶ In this case spinal angiography was not considered clinically justified in light of the patient's advanced age and multiple comorbidities.

Treatment focuses on risk factors and rehabilitation. As the most causative agent is likely to be atherosclerosis, treatment with antiplatelet therapy is fundamental following spinal cord infarction. If the source is judged to be embolic, anticoagulation drugs can be started. Some studies have looked at corticosteroid therapy administered within a short time following spinal cord infarction due to protective effects on cell function and reducing oxidative stress following ischaemic injury.⁷ Another study has looked at treatment with magnesium which is thought to prevent release of certain neurotransmitters and alter calcium levels that occur with ischaemia.⁸ Naloxone has been shown to reduce neurological deficits in animals by increasing blood flow and preventing calcium entry into spinal nerve cells following injury.⁹ Other measures for management include the complications of acute paraplegia such as immobility leading to venous stasis and subsequent thromboembolism disease, decubitus ulcer and infections such as pneumonia and urosepsis.

Recovery following spinal cord infarction is diverse. A prospective study by Masson *et al*¹⁰ showed that impairment of walking or bladder dysfunction at onset was associated with a worse 2-month outcome and frequently led to residual pain irrespective of the extent of the infarct. A study with a 3-year follow-up of patients with stroke infarctions showed that 41% of patients using a wheelchair at hospital discharge were walking and 33% of patients with a long-term catheter at hospital discharge were catheter free,¹¹ highlighting that many patients have the potential to recover and function independently.

Learning points

- ▶ Spinal cord infarction is a rare cause of paraplegia with a wide array of aetiological factors.
- ▶ MRI is a good initial investigation to help rule out cauda equina syndrome and other causes such as extradural compression, arteriovenous malformations and space occupying lesion.
- ▶ MRI is a safe and sensitive modality for the diagnosis of spinal cord infarction.
- ▶ Prognosis following acute spinal cord infarction is wide with many patients having the potential to recover and function independently.

Contributors SP followed up the patient and analysed current reports and data on spinal cord infarction. KN helped edit the report. PT deducted the most appropriate images and reviewed the report.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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