CASE REPORT

Anterior spinal cord infarct: a rare yet disabling stroke

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Accepted 17 April 2018

SUMMARY

We present a case which demonstrates the classical clinical symptoms and signs evident in an anterior spinal cord syndrome and explains the anatomical basis for the features seen with respect to the ascending and descending tracts in the spinal cord. It also demonstrates the clinical importance in conducting a detailed sensory examination to look for dissociated sensory loss and ascertaining the level of the pathology within the cord. The patient had made improvements following over a month of extensive rehabilitation on a specialist stroke unit.

BACKGROUND

Spinal cord ischaemia accounts for less than 1% of all strokes.¹ Given its rarity, clinicians are less likely to come across such cases; thus, it may pose a diagnostic challenge in the acute presentation. Diagnosis requires a detailed history and a thorough neurological examination with attention to detail in the sensory examination, as they often present with a dissociative sensory loss, sparing the dorsal column tracts. It is therefore imperative for physicians to have an understanding of how spinal cord ischaemia can present in order to prevent delays in diagnosis and management. Spinal cord ischaemia has a number of aetiologies and clinical syndromes which vary significantly from each other, further complicating accurate diagnosis and management.² The vast majority of spinal cord ischaemia cases involve a compromise in flow to the anterior spinal artery, which make up the majority of presentations.² We present this case in the hope that trainees will gain an understanding of how an anterior spinal cord infarct can present. It also serves as a beautiful anatomical demonstration of the vascular territories in the spinal cord and how this anatomy can manifest clinically.

CASE PRESENTATION

We present an 82-year-old caucasian man who attended the emergency department with bilateral upper limb weakness. His symptoms began shortly following a long nap on his sofa. He woke up and walked to the kitchen to make a cup of tea and then developed sudden onset bilateral proximal upper limb weakness. He had normal function in his lower limbs and there was no visual, speech, swallow, bladder or bowel disturbance at the onset.

He has a history of bladder carcinoma and malignant melanoma 20 years previously, both of which have been in remission, with no history of recurrence. He also has a history of non-insulin-dependent

diabetes mellitus and asthma. His regular medications included metformin, ferrous fumarate, salbutamol and seretide inhalers.

He is a smoker of 20 pack years and drinks 6 units of alcohol per week. He has no significant family history.

On examination in the emergency department, he had flaccid tone in both upper limbs with a Medical Research Council (MRC) grade 0/5 power in the left arm and an MRC grade 4 in the right arm (muscle groups tested included shoulder abduction, elbow flexion, elbow extension, wrist extension, finger abduction). Reflexes in the upper limbs (biceps, triceps and supinator) were present and symmetrical. In his lower limbs, he had full power bilaterally in all muscle groups (hip flexion, hip extension, knee flexion, knee extension, ankle dorsiflexion) with bilaterally brisk reflexes (knee and ankle) and extensor plantars. Lower limb sensation was assessed at presentation in the emergency department as being normal.

He complained of mild cervical pain and had normal observations, blood tests, ECG and chest X-ray. A cervical spine X-ray revealed possible spinous fragments but this was followed up with a CT brain and cervical spine which ruled out spinal cord compression and any acute intracranial pathology.

The following morning, in addition to his existing left arm weakness, he had developed worsening right arm weakness to only antigravity strength (3/5 power in all muscle groups) and acute flaccid left leg weakness (0/5 power in all muscle groups). His right leg remained unaffected.

A neurological review demonstrated significant dissociated sensory loss with spinothalamic loss below the level of C4 bilaterally which had not been identified at presentation. Proprioception remained unaffected. This dissociated spinothalamic sensory level with intact proprioception in the presence of bilateral limb weakness suggested an anterior spinal cord syndrome.

Given his level of cord injury, a respiratory assessment was made to ensure he was safe with no respiratory compromise. His forced vital capacity was 2.5 L and he was kept under close observation.

INVESTIGATIONS

An MRI of the whole spine was undertaken.³ On the sagittal T2, there appeared to be an intramedullary abnormal signal intensity from C2 to C5 with central cord swelling cord (figure 1A). This was restricted to grey matter as seen on axial (figure 1B). For further confirmation, we performed diffusion-weighted imaging (figure 1C)

To cite: Davda N, Osman C.
BMJ Case Rep Published
Online First: [please
include Day Month Year].
doi:10.1136/bcr-2017-
221655

Figure 1 (A) (top left) Sagittal T2 cervical spine showing high signal C2–C5. (B) (top right) Axial T2 confirming high signal centrally in the cord. (C) (bottom left) Axial diffusion-weighted imaging (DWI) showing restricted diffusion within the grey matter. (D) (bottom right) Axial apparent diffusion coefficient (ADC) showing signal loss within the the corresponding area of the DWI. AC, Alternating Current; FFS, Fast Fourier Sequence; FRFSE, fast recovery fast spin echo; TE, echo time; TR, repetition time.

(DWI) with apparent diffusion coefficient to look for restricted diffusion (figure 1D) which can be seen in patients with acute ischaemia. The MRI DWI showed restricted diffusion within the grey matter with signal intensity suggestive of cord ischaemia in the spinal cord.

An MR angiogram demonstrated irregular appearances of the intraluminal flow in the right vertebral artery; however, a CT angiogram of the neck demonstrated no convincing evidence of vertebral artery dissection or thrombus.

DIFFERENTIAL DIAGNOSIS

This case raises some potential differentials. The most important is to exclude a cord compression although it would be unusual to

cause such selective anterior cord involvement. Given the speed of progression, one must consider an inflammation of the spinal cord giving rise to his symptoms. The MRI³ was very helpful in demonstrating the area of the cord that was involved including restricted diffusion on the MRI DWI images which would fit with a vascular event.

TREATMENT

This patient was given aspirin 300 mg daily for 2 weeks followed by clopidogrel 75 mg daily. He was given atorvastatin 80 mg daily. He began specialist stroke physiotherapy involving passive limb movements and stretches along with reflex-induced training and active therapy. He was also given gait training using

Figure 2 Anatomy of the arterial and venous distribution to the spinal cord.⁴

a treadmill and observed for respiratory compromise and reflex dysautonomia which he did not develop.

His bladder and bowel care was managed as well as his nutrition.

OUTCOME AND FOLLOW-UP

Over the subsequent weeks, he continued inpatient rehabilitation on the stroke rehabilitation unit. Follow-up after 1 year demonstrated full resolution of the right arm weakness and his left arm weakness had regained some movements in his shoulder abduction, elbow flexion and wrist extension but not against gravity. His left leg had improved significantly from 0/5 power to being able to flex his hips and with knee extension 4+ and knee flexion 4– and dorsiflexion 4– requiring bilateral foot splints. He walked with a walking stick and was able to be discharged home.

DISCUSSION

The presentation of this patients' symptoms shows the classic clinical features of an anterior spinal cord infarction, namely bilaterally paretic limbs, extensor plantar reflexes and loss of spinothalamic sensory function with intact dorsal column sensation.

Let us consider the anatomical basis for why this is the case. Figure 2⁴ shows the arteries that supply the spinal cord. The arterial supply to the spinal cord is provided by the anterior spinal artery and two posterior spinal arteries. The anterior spinal artery arises from the two vertebral arteries and descends along the midline of the spinal cord anteriorly. The posterior arteries arise from either the vertebral arteries or the posterior inferior cerebellar arteries. All of these arteries then enter the spinal cord to supply their respective territories.

Figure 3 Anatomy of the tracts in the spinal cord.⁵

Learning points

- ▶ In patients presenting with sudden onset bilateral arm and/or leg weakness, extensor plantar reflexes associated with spinothalamic but not dorsal column sensory loss, consider an anterior spinal cord infarction in your top list of differentials.
- ▶ A thorough neurological sensory examination should be undertaken in such presentations as this will help to localise the pathology.
- ▶ MRI is a useful imaging modality to investigate a possible spinal cord infarction to look for distribution of the signal change but also consider MRI diffusion-weighted imaging to look for restricted diffusion to help further confirm the suspicion of an anterior spinal cord infarct.

The anterior spinal artery supplies the anterior and central parts of the spinal cord. Thus, pathology of this artery will mainly affect the tracts in this part of the spinal cord. [Figure 3⁵](#) shows the anatomy of the tracts within the cord.

The tracts in the anterior portion of the spinal cord are the ventral corticospinal tracts and the spinothalamic tracts. These are responsible for motor innervation to the limbs and pain and temperature sensation from the limbs, respectively. Thus, we can expect disruption in these modalities in patients with an anterior spinal artery infarct, as is the case with our patient who had reduced pinprick sensation below the level of C4 and weakness of his arms and one of his legs.

It must be noted that some motor innervation to the limbs is carried by the lateral corticospinal tract which is located more posteriorly. Thus, motor innervation may not be entirely lost, again, as is the case in our patient.

The dorsal columns (consisting of fasciculus gracilis and fasciculus cuneatus) are situated posteriorly and are therefore unaffected by an anterior spinal artery lesion, as in our patient.

In patients presenting with bilateral weakness, extensor plantar reflexes with disruption in spinothalamic tract but intact dorsal column sensation, anterior spinal artery ischaemia should therefore be considered in the top list of differentials. Care should be taken before making this diagnosis to rule out other more common pathologies such as inflammation or cord compression.⁶ The history and detailed sensory examination are key with appropriate imaging.

Thus, we hope that our case serves as a useful aide memoir when such a patient presents through the front door.

Contributors ND is the SHO on the team looking after the patient. His contribution included collecting the clinical information about the case and writing up the case report and discussion. He sourced the diagrams used in the discussion of the case report. CO was the neurology registrar who was involved in the case and provided advice to the ND's team looking after the patient. After ND's initial draft, CO reviewed and edited and rewrote several paragraphs throughout the case report. CO also collected the appropriate radiological images and discussed the images with the neuroradiologist in order to ensure that author's description of them was accurate.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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