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Spinal artery infarction

SHORT CASE REPORT

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Spinal cord infarctions are rare, and the symptoms vary depending on location and size. One patient presented with severe neck pain and paresis of the left arm. Compression of a cervical nerve root was initially suspected, but the progression of symptoms and MRI findings gradually suggested a different aetiology.

One evening, a man in his fifties who had previously undergone cervical laminectomy of C5/C6 with bone grafting for neck pain radiating to the right arm, experienced acute, moderate lower neck pain. The pain improved after taking paracetamol tablets. The following afternoon, however, he experienced intense neck pain radiating to both shoulders. Upon arrival at the emergency department, he reported pain corresponding to a VAS score of 9. The patient, who was a smoker, was otherwise healthy and did not take any regular medications.

At the emergency department, the weakness in his left arm and shoulder gradually progressed over the course of 1.5 hours. The pain subsided somewhat with the help of painkillers. A cervical disc prolapse was suspected and he was transferred to the neurology department as an emergency admission. Upon arrival, the patient's vital signs were normal and he was afebrile. During a neurological examination, the patient managed left arm abduction of 20 degrees, while flexion of the left elbow and wrist showed grade IV paresis, and finger function was considerably reduced. A Spurling test was negative and blood tests were normal.

Cervical MRI performed on the same day showed nerve root compression for both C5 roots secondary to uncovertebral spurring. This was most pronounced on the left side, where bone marrow oedema was also visible, and interpreted as degenerative. Analgesics in the form of paracetamol, diclofenac and tramadol were given orally, and the pain subsided. Left-sided C5 involvement was assumed to be causing the weakness, and the neurologist discussed the possibility of decompression surgery with the neurosurgeon at the university hospital.

If the C5 nerve root is affected, weakness can be expected during shoulder abduction due to weakened supraspinatus and deltoid muscles. Involvement of these muscles was found in our patient, but his finger strength was also impaired, which could not be explained by C5 involvement alone.

On re-examination of the MR images taken by a neuroradiologist at the university hospital, a subtle high T2 signal was noted centrally in the cervical medulla at levels C2–C4. Consequently, MRI of the neuroaxis (the brain and entire spinal column) and lumbar puncture were recommended. During the night, the patient developed reduced strength in the left hip (grade 4) and was subsequently transferred to the university hospital for further assessment.

Upon arrival at the university hospital three days after the onset of symptoms, the patient still had neck pain, but the painkillers had worked to some extent. A neurological examination found new-onset anisocoria (unequal pupil sizes) with miosis of the left eye but no ptosis or other signs of Horner's syndrome.

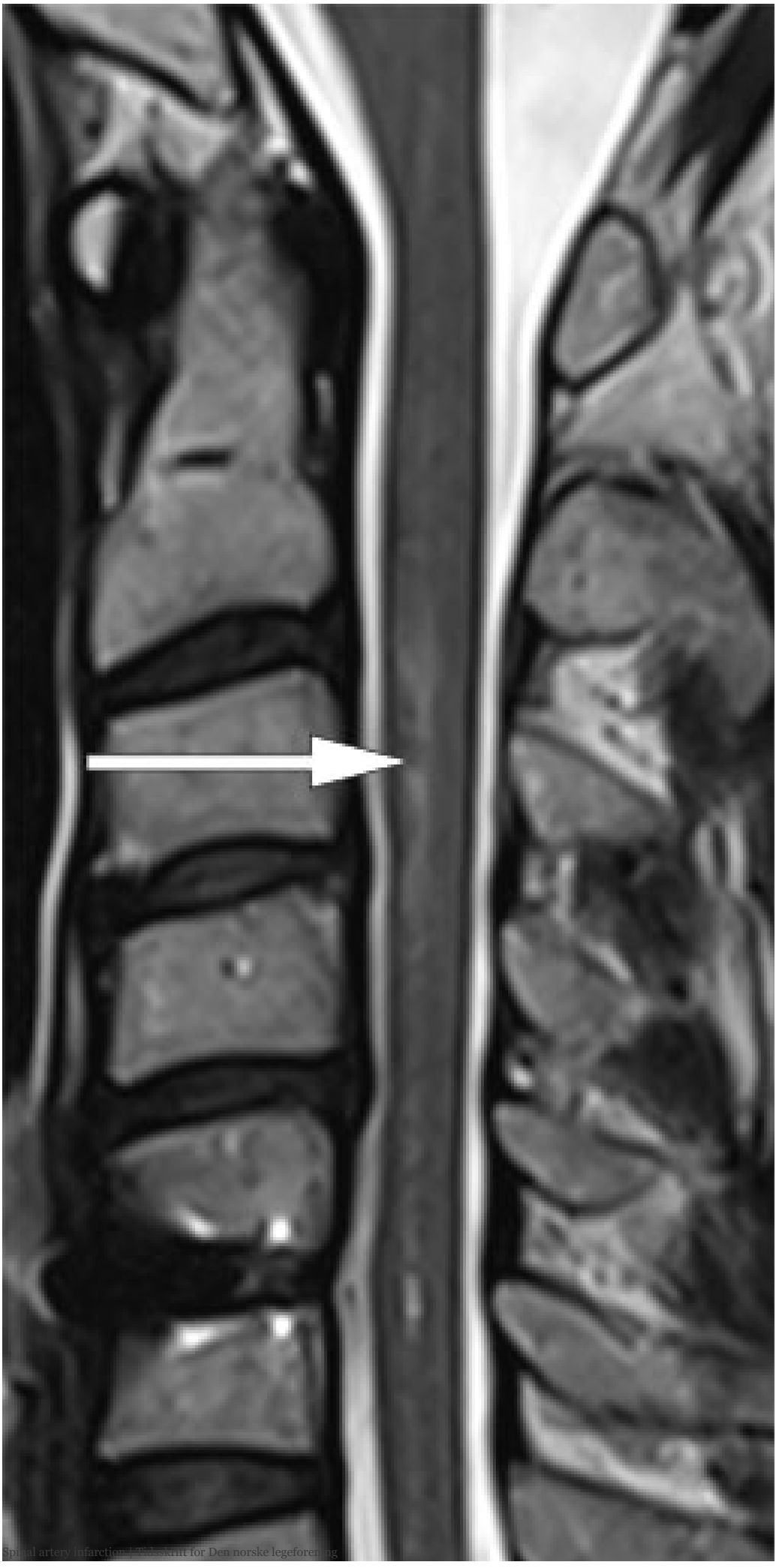
Sensitivity to touch in the face and the upper and lower extremities was normal, while pain and thermal sensation was reduced in the right side of the body below the C6 dermatome. Vibratory sensation was intact and bilaterally equal distally in the extremities. Joint sensation was not tested.

The patient had pronounced flaccid paresis in the upper and lower left extremities, including grade III paresis for abduction of the left shoulder, grade II paresis for flexion of the left elbow, and grade IV paresis for flexion of the left hip. He was unable to walk without support or a walking aid. He was also unable to splay his fingers and had difficulty using his left hand for fine motor activities. His tendon reflexes were normal bilaterally in the upper and lower extremities, with no indication of ataxia.

The patient's blood pressure and glucose levels were normal upon admission, and his lipid profile and HbA1c were within the reference range. Antinuclear antibodies (ANA), antineutrophil cytoplasmic autoantibodies (ANCA) and antiphospholipid antibodies in the blood were normal. An ECG showed no indication of arrhythmia.

The neurological findings were consistent with a classic but incomplete variant of Brown-Séquard syndrome. The patient had paresis without loss of sensation on the left side and reduced pain and thermal sensation on the right side. However, miosis is not part of the syndrome, and further investigation was therefore initiated. On the day of admission to the university hospital, further imaging of the neuroaxis was undertaken with a 1.5T (Tesla) MRI scanner in addition to CT angiography of the pre- and intracerebral arteries.

Brain MRI showed findings consistent with a small, recent infarction in the left cerebellum. There was now a more distinct, increased intramedullary T2 signal intensity at levels C2–C4 (Figure 1), with axial diffusion imaging showing restricted diffusion in areas with an increased T2 signal, predominantly at the left side (Figure 2).



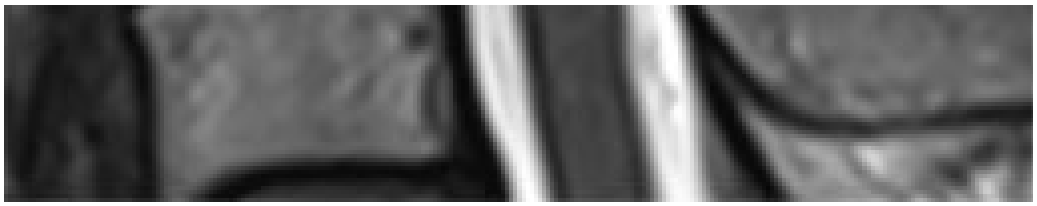


Figure 1 Sagittal T2-weighted MR image of the spinal column shows a pathological intramedullary signal at levels C2–C4 (indicated by arrow), with indication of oedema. Below this is the suggested marking of the central canal of the medulla. At level C5/6, the status after previous disc decompression is seen along with artefacts from the disc implant.

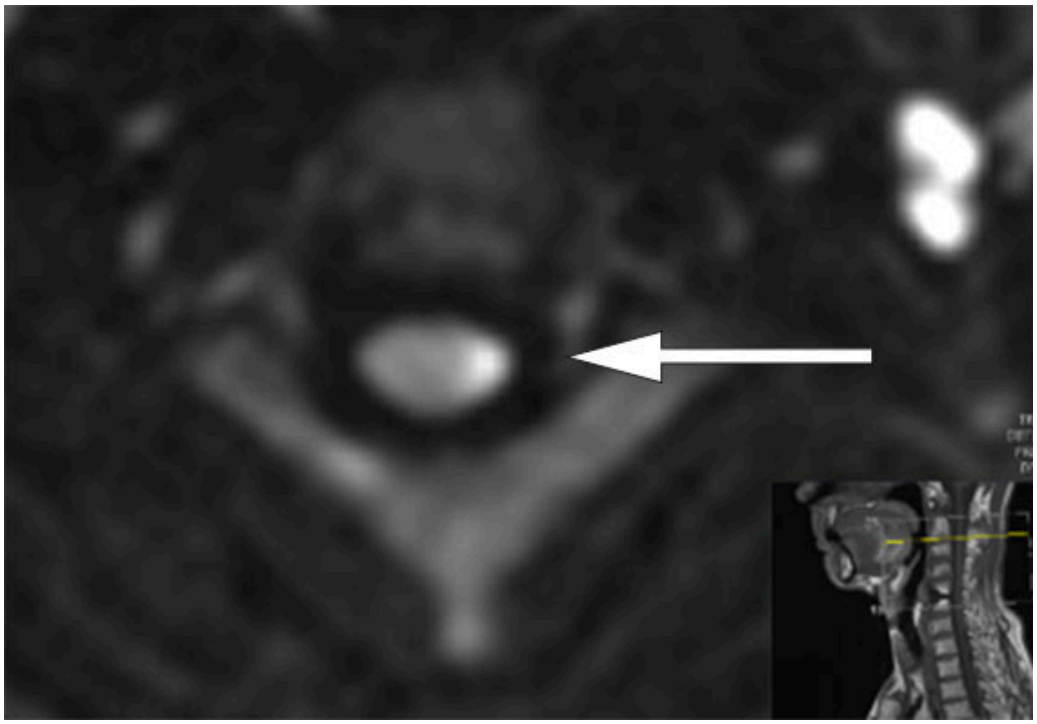


Figure 2 Axial diffusion-weighted MR image (B1000) at level C2/3 showing area with a high signal on the left of the medulla, indicative of cytotoxic oedema. This finding is consistent with arterial medullary infarction (indicated by the arrow).

CT angiography showed no pre- or intracerebral artery dissection or aneurysm, but a small contrast-filled chamber was found in a segment with thickened walls in the left subclavian artery, proximal to the origin of the left vertebral artery. This finding may indicate a recent plaque ulceration and could be a likely source of embolism. The lumbar puncture performed on day four of the clinical course showed normal findings in the cerebrospinal fluid. A transthoracic echocardiogram performed after discharge from the university hospital showed a normal ejection fraction of 55 % and revealed no valvular dysfunction or embolic sources.

Spinal artery infarction was considered the most likely diagnosis based on findings of restricted diffusion in the medulla, recent infarction in the left cerebellum and atherosclerotic plaque in the left subclavian artery. A thrombus may have travelled from here via the vertebral artery and down to the anterior spinal artery, causing the spinal infarction.

There was no indication of internal carotid artery dissection, or brainstem infarction or other structural aetiology of the miosis of the left eye.

The patient was treated with peroral antiplatelets in the form of acetylsalicylic acid 75 mg × 1 and clopidogrel 75 mg × 1 for three weeks, followed by monotherapy with clopidogrel 75 mg × 1 as a lifelong treatment. Six months after the event, the patient had regained almost normal function in the left extremities, but the change in thermal sensation in the right side persisted.

Discussion

Spinal cord infarctions are rare, and the estimated incidence is 0.3–1.0 % of all infarctions in the central nervous system (1). Their clinical presentation differs from that of cerebral infarctions, and diagnosis can be difficult in the early stages. The symptoms can – in contrast to cerebral infarctions, which are hyperacute – develop gradually, as in our patient's case. Motor dysfunction is the most common symptom, followed by sensory deficit, neck and back pain and autonomic dysfunction (2). The symptoms vary depending on the location and size of the infarction.

Brown-Séquard syndrome is characterised by a unilateral spinal lesion, normally at neck level. This leads to motor dysfunction as well as loss of vibratory and joint sensation on the injured side and reduction of pain and thermal sensation on the healthy side. Aetiology of the syndrome can include medullary infarction, spinal cord tumour, trauma (such as stab wounds or bullet wounds to the neck), infections (such as tuberculosis) or inflammatory diseases (such as multiple sclerosis). Our patient presented with incomplete Brown-Séquard syndrome with infarction along the spinothalamic and corticospinal tracts, which explains the motor dysfunction on the injured side and the reduced pain and thermal sensation on the healthy side. Furthermore, the fasciculus cuneatus (tract of Burdach) and the fasciculus gracilis (tract of Goll) were spared, which explains the preserved vibratory sensation on the injured side. In addition, the patient had a unilateral miosis which was probably due to damaged oculosympathetic fibres at cervical level.

Where there is a clinical suspicion of acute medullary infarction, a diffusion-weighted MRI scan should be performed, as T2 signal abnormalities may be subtle or absent early in the acute phase. At levels C3–C5, presentation of the medulla may be affected by various MRI artefacts, which can make it difficult to detect signal abnormalities.

The most common aetiology of medullary infarction is atherosclerosis, abdominal aortic dissection, sources of cardiac embolism and iatrogenic injury (such as aortic surgery, spinal decompression and epidural steroid injections) (2). Risk factors include hypertension, diabetes, hypercholesterolaemia and smoking. Intravenous thrombolytic therapy is not standard for spinal infarction, but individual assessment is possible (3, 4). This is partly because spinal infarction is difficult to diagnose in the acute phase, and the time window for thrombolysis often expires before the diagnosis is made. Treatment for spinal infarction is otherwise similar to that for cerebral infarction, but depends on aetiology. In contrast to the treatment for other types of myelopathy, methylprednisolone is not used.

Patients often have a severe outcome, but respond well to rehabilitation. Up to 50 % of patients have regained the ability to walk upon discharge (2), and they are more likely to return to work than patients with cerebral infarctions, partly because they are generally younger and have fewer cognitive sequelae (1).

The patient has consented to the publication of this article.

The article has been peer-reviewed.

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