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CASE REPORT

Acute anterior spinal cord syndrome following cardiac arrest during coronary angiography

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ABSTRACT

Spinal cord infarction is an uncommon neurological emergency and accounts for approximately 1–2% of all vascular neurological disorders. Anterior spinal cord syndrome (ASCS) is the commonest clinical presentation and is characterised by acute motor weakness and dissociated sensory loss with preservation of posterior column functions. I report the case of a 70-year-old man who developed acute spastic paraparesis with loss of pain and crude touch sensation up to the xiphisternum following recovery from a cardiac arrest that occurred during coronary angiography. Neurological examination revealed spastic paraparesis, bilateral extensor plantar responses, a sensory level at approximately T6, preservation of vibration and joint position senses and urinary retention requiring catheterisation. The clinical findings were highly suggestive of anterior spinal cord syndrome secondary to spinal cord ischaemia resulting from global hypoperfusion during cardiac arrest. This case highlights the importance of recognising spinal cord ischaemia in patients presenting with acute myelopathy following cardiac arrest or invasive cardiac procedures.

Keywords*: Acute anterior spinal cord syndrome, cardiac arrest, coronary angiography*

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BACKGROUND

Spinal cord infarction is a rare but devastating neurological condition, representing approximately 1–2% of all ischaemic strokes and 5–8% of acute myelopathies [1]. The anterior spinal artery supplies the anterior two-thirds of the spinal cord, including the corticospinal and spinothalamic tracts. Interruption of blood flow within this vascular territory results in anterior spinal cord syndrome (ASCS), characterised by bilateral motor weakness and impairment of pain and temperature sensation with preservation of proprioception and vibration sensation due to sparing of the dorsal columns[2].

Cardiac arrest is an uncommon but recognised cause of spinal cord ischaemia. Prolonged systemic hypotension and global hypoperfusion during cardiac arrest may particularly affect the vulnerable thoracic watershed zones of the spinal cord[3]. Although neurological sequelae involving the brain are well recognised after cardiac arrest, spinal cord infarction remains under-recognised. I report a probable case of ASCS developing after cardiac arrest during coronary angiography.

CASE PRESENTATION

A 70-year-old man underwent elective coronary angiography for evaluation of suspected coronary artery disease. During the procedure, he developed sudden cardiac arrest. Immediate cardiopulmonary resuscitation was instituted and return of spontaneous circulation was successfully achieved.

Three days following the event, after regaining full consciousness and orientation, he noticed sudden weakness of both lower limbs associated with loss of sensation below the upper abdomen. He denied any history of trauma, back pain, fever, loose motion, preceding respiratory or gastrointestinal illness, recent vaccination or constitutional symptoms.

On examination, the patient was conscious, alert and fully oriented. Higher mental functions were normal. Cranial nerve examination was unremarkable.

Motor examination demonstrated spastic paraparesis with Medical Research Council grade 2/5 power in both lower limbs. Tone was increased bilaterally and deep tendon reflexes at the knees and ankles were exaggerated. Plantar responses were extensor on both sides.

Sensory examination revealed loss of pain and crude touch sensation below the xiphisternum, corresponding approximately to the T6 dermatome. In contrast, vibration and joint position senses were preserved in both lower limbs. Upper limb neurological examination was normal. Bladder involvement was evident as the patient required an indwelling urinary catheter because of urinary retention.

INVESTIGATIONS

Routine haematological and biochemical investigations, including complete blood count, serum electrolytes, renal and liver function tests, were within normal limits.

MRI of the thoracic spine was planned to exclude compressive myelopathy and confirm spinal cord ischaemia. Typical MRI findings expected in ASCS include T2-weighted hyperintensity involving the anterior two-thirds of the spinal cord and the characteristic axial ‘owl's eyes’ appearance[1].

Additional investigations that may be considered include:

- MRI of the whole spine with diffusion-weighted imaging.
- CSF examination to exclude inflammatory myelopathies.
- Echocardiography and vascular imaging to identify potential embolic sources.
- Screening for vasculitic and prothrombotic disorders where clinically indicated.

DIFFERENTIAL DIAGNOSIS

The differential diagnoses considered included:

- Acute anterior spinal cord syndrome secondary to spinal cord infarction.
- Acute transverse myelitis.
- Compressive thoracic myelopathy.
- Spinal epidural haematoma.
- Guillain–Barré syndrome.
- Functional neurological disorder.

The presence of dissociated sensory loss with preservation of posterior column modalities strongly favoured ASCS.

TREATMENT

The patient was managed conservatively with supportive measures, including:

- Maintenance of adequate haemodynamic status and spinal cord perfusion.
- Prevention of secondary complications such as pressure sores and deep vein thrombosis.
- Bladder care with an indwelling catheter.
- Intensive physiotherapy and rehabilitation.
- Passive and active-assisted limb mobilisation.

Although corticosteroids are frequently administered empirically in acute myelopathy, evidence supporting their use in spinal cord infarction remains limited[4].

OUTCOME AND FOLLOW-UP

The patient underwent multidisciplinary rehabilitation with physiotherapy, bladder care and prevention of complications associated with immobility. Neurological recovery following spinal cord infarction is variable and depends largely on the extent of ischaemic injury and timely rehabilitation. At follow-up, continued assessment of motor recovery, bladder function and functional independence was planned.

DISCUSSION

Anterior spinal cord syndrome is the most common manifestation of spinal cord infarction. The syndrome typically presents with abrupt onset of bilateral motor weakness, impairment of pain and temperature sensation and autonomic dysfunction, with preservation of vibration and proprioception[2].

Global hypoperfusion resulting from cardiac arrest is an uncommon but recognised mechanism of spinal cord infarction. The thoracic spinal cord, especially the watershed region between T4 and T8, is particularly susceptible to ischaemic injury because of its relatively limited collateral blood supply[3].

The clinical features in my patient strongly suggested involvement of the anterior spinal artery territory. The abrupt onset following a documented cardiac arrest, absence of antecedent infection or trauma, presence of dissociated sensory loss and urinary dysfunction all support an ischaemic mechanism.

MRI remains the investigation of choice for confirming spinal cord infarction and excluding alternative causes such as compressive myelopathy or inflammatory lesions. However, MRI may be normal in the early stages, and repeat imaging may occasionally be required[1].

Management is largely supportive and focuses on optimisation of haemodynamic parameters, prevention of complications and early rehabilitation. Long-term outcomes vary considerably, with some patients achieving substantial functional recovery while others remain severely disabled[5].

PATIENT'S PERSPECTIVE

The patient stated that regaining consciousness after the cardiac event but subsequently discovering inability to move his legs was distressing. He expressed hope that rehabilitation would improve his mobility and independence.

LEARNING POINTS

- Acute spinal cord infarction should be considered in patients developing sudden paraparesis following cardiac arrest or invasive cardiac procedures.
- Dissociated sensory loss with preserved vibration and joint position sense is highly suggestive of anterior spinal cord syndrome.
- MRI is the investigation of choice for confirming spinal cord ischaemia and excluding alternative diagnoses.
- Early supportive management and multidisciplinary rehabilitation are essential to optimise outcomes.
- Cardiac arrest-induced global hypoperfusion may selectively affect vulnerable watershed regions of the thoracic spinal cord.

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