Spectrum of clinicoradiological findings in spinal cord infarction: Report of three cases and review of the literature

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Abstract

Spinal cord infarction (SCI) often remains undiagnosed due to infrequent occurrence and lack of established diagnostic procedures. The unique pattern of blood supply explains the heterogeneity of clinical presentation. We present three cases of SCI to highlight the varied spectrum of clinicoradiological findings. The first patient had posterior spinal artery infarction, and spine imaging showed infarction of adjacent vertebral body, which is usually rare. The second patient had anterior spinal artery infarction and the cANCA titers were elevated. The third patient had a pure motor quadriparesis. Initial imaging did not show any cord infarction, but signal changes were noted on serial imaging. Fibrocartilaginous embolism (FCE) seems the most likely etiology in the first and third cases. A high index of clinical suspicion is necessary for prompt diagnosis. Sensitivity of the initial magnetic resonance imaging remains limited, necessitating serial follow-up scans. Infarction of the adjacent vertebral body is a useful confirmatory sign. Fat suppression images can delineate the marrow signal changes better. Elderly patients with vascular risk factors and degenerative discs need to avoid mechanical triggers that predispose to FCE. Younger patients with SCI will need evaluation for cardioembolic source and vasculitis.

Key Words

Anterior spinal artery, fibrocartilagenous embolism, magnetic resonance imaging, posterior spinal artery, spinal cord infarction

Introduction

Spinal cord infarction (SCI) is rare and often remains undiagnosed. Atypical presentations, including delayed onset symptoms, patchy sensory or pure motor presentations, are well described depending on the part of cord involved, presence of collateral circulation and underlying etiology. The varied spectrum of clinicoradiological findings in SCI has been highlighted.

Case Reports

Case 1

A 58-year-old gentleman presented with sudden-onset severe low back pain noted while lifting the shutter of a shop, followed by feeling of heaviness and weakness of both lower limbs. The deficits peaked by 1 h, with urinary retention and decreased sensation below the mid abdomen. Clinical examination revealed asymmetric paraparesis, Medical Research Council (MRC) scale power grade 2 on the right and grade 3 on the left lower limb. There were absent lower limb deep tendon reflexes, bilateral extensor plantars and impaired propioception below the T7 level. Blood pressure and peripheral pulses were normal. Overall features were suggestive of myelopathy at the T7 level. Clinical possibilities considered included intervertebral disc prolapse, SCI due to fibrocartilagenous embolism (FCE), aortic dissection, vascular malformation with bleed and inflammatory or infectious myelitis. Magnetic resonance imaging (MRI) [Figures 1a and b] revealed T2W and STIR hyperintensity extending from the T7 to the T11 level, with relative sparing of the anterior surface of the cord and no contrast enhancement. There was associated narrow edema in the posterior aspect of D10 vertebral body. The cerebrospinal fluid (CSF) analysis was noncontributary. Computerized tomography (CT) angiogram did not reveal any atheromatous plaques or dissection.

Considering the diagnosis of posterior spinal artery (PSA) infarction, conservative treatment was started. Motor power...
improved and he was ambulant by 8 weeks. Follow-up scan done 8 months later [Figure 1c] showed only a subtle posterior column hyperintensity with volume loss; the T10 vertebra was still showing fat replacement along the posterior margin.

Case 2
A 19-year-old girl presented with history of neck pain followed by acute-onset quadriparesis, which peaked within 2 h, of 15 days duration. Pulse dose steroids were administered elsewhere. On examination, power in the upper limbs was grade 1 bilaterally; the lower limbs had grade 3 power. There was dissociated sensory loss with pain and temperature being lost below the C4 level, proprioception was preserved. MRI [Figure 2] showed a long-segment signal abnormality in the anterior aspect of the cervical cord from C3 to C6 level, with mild expansion and patchy enhancement.

Diagnosis of anterior spinal artery (ASA) infarct was considered. Other differentials included myelitis and demyelination. CSF analysis including viral markers was negative. Visual-evoked potentials, echocardiogram, MR angiogram of the aortic arch and cerebral vessels were normal. The cANCA titers were found to be elevated (28.2, normal range less than 15). There was no hematuria or proteinuria to suggest renal involvement. Nerve conduction studies were noncontributory.

Considering the high specificity of cANCA, the possibility of immune-mediated disease with SCI was considered. As steroids were already administered, it was decided to keep her under follow-up considering the fact that she was improving. Further immunomodulation was deferred as she had a urinary tract infection. At 6-week follow-up, there was significant improvement in motor power.

Case 3
A 62-year-old gentleman with a background of hypertension and cervical spondylosis presented with acute onset of quadriparesis. The weakness was preceded by sudden onset of giddiness, following which he had a fall with transient loss of consciousness lasting a few seconds. Quadriparesis evolved within 2 h and bladder distention was subsequently noted. On examination, the sensorium was preserved. Tone was decreased in all four limbs, power was grade 2 in the upper limbs and grade 3 in the lower limbs. Sensory examination was normal. MRI of the cervical spine [Figure 3a] showed features of cervical spondylosis with ossified posterolateral ligament predominantly at the C3–C4 levels causing cord indentation with signal changes. No brainstem infarcts were noted. MR angiogram of the cerebral vessels, neck vessel Doppler, ECG and echocardiogram were normal.

Possibility of an ischemic insult to the cord related to FCE in the setting of a cervical spondylotic myelopathy was considered. The patient was started on supportive therapy, antiplatelets and steroids. The lower limbs improved to grade 4 power and the upper limb power marginally improved to grade 3. Repeat MRI at 3 weeks [Figure 3b and c] showed focal high-signal T2 W and STIR hyperintensity at the C3 level, which was consistent with infarction.

Discussion
Rich anastomotic network of arteries combined with relatively lower atherosclerotic changes could attribute to the relative rarity of SCI. The discussion highlights the following features: (a) importance of clinical history with atypical clinical presentations, (b) MRI changes including vertebral body changes, utility of STIR images and serial imaging for diagnostic confirmation and (c) FCE in the setting of chronic disc disease.
The diagnosis is often clinical based on abrupt onset deficit with associated back pain in nearly 70%. But, presentation can be less abrupt depending on the etiology and presence of collateral circulation. The central part of the ASA territory is supplied by sulcal arteries arising from the ASA; the circumference of the cord is supplied by small penetrating branches of the vasa corona. The paired PSAs feed an anastomotic network on the dorsal aspect of the cord. There is no demarcation of the central and peripheral territories. An intermediate area near the anterolateral part of the anterior horn and central part of the posterior horn is variably supplied by either the sulcal arteries or the penetrating branches of the vasa corona. This unique pattern of blood supply explains the heterogeneity of clinical presentation.

ASA infarcts often involve the central territory. Although dissociative sensory loss is classically described, loss of proprioception can also occur suggesting an extensive infarct with involvement of the inner part of dorsal columns and posterolateral part of lateral columns, mainly in the central territory. Central cord presentations are also described. Pure motor involvement as in case 3 can occur if the SCI mainly involves the grey matter of the motor area. Peripheral white matter could be spared because of presence of pial perforators. PSA syndrome is relatively infrequent. The arterial territories fed by PSA usually encompass the posterior column, posterior horns and posterolateral portion of the lateral column containing crossed corticospinal fibers explaining the loss of vibration sense, arreflexia and weakness (usually transient), respectively. Incomplete Brown-Séquard syndrome due to involvement of sulco-commisural arteries and total transverse SCI involving both ASA and PSA are also described.

The sensitivity of initial MRI of the cord remains limited, with nearly 17–45% of clinically suspected SCI having a normal scan. MRI more importantly rules out compressive myelopathies, vascular malformations, infective myelitis, demyelinating disorders and tumors. MRI patterns identified with ASA infarcts include “owl eye” appearance with involvement of anterior horns of grey matter and central pattern involving both the grey matter and the adjacent white matter. Diffusion-weighted imaging of the spine, although limited by sensitivity to artifacts, can be potentially useful in identifying early infarctions. Fat suppression using STIR images help in better delineation as well as detection of vertebral body signal changes. Contrast enhancement can be seen in subacute infarcts.

A finding of vertebral body infarction (VBI) adjacent to a cord signal abnormality on MRI is a useful confirmatory sign if present, although it is found in only 4–35% of the patients. This may be attributable to the fact that arterial occlusion may be located distal to the anterior or posterior central arteries that supply the vertebral body. The bone marrow abnormality of the vertebra tends to be earlier and more exaggerated compared with the cord. The VBI is usually seen in the thoracolumbar region adjacent to the cord lesion. Concomitant SCI and VBI are usually associated with aortic diseases. MRI finding of VBI has been less described in the PSA syndrome compared with the ASA syndromes. Abnormal bone marrow signal can also be seen with fractures, metastasis and infections. However, absence of epidural lesions with sequential signal changes in vertebral body and cord are usually indicative of an ischemic etiology.

Spinal cord hypoperfusion with peripheral vascular disease or hypotension usually causes extensive central or transverse infarcts. Workup for vasculitis, prothrombotic states and cardioembolic sources is also indicated when the etiology is obscure. Case reports on ANCA-associated vasculitis presenting as SCI as reported in case 2 are scarce.

Ischemia following mechanical triggering movement or falls as mentioned in two of the cases usually cause injury of a radicular artery with unilateral or bilateral SCI. Sudden changes in intervertebral disc pressure, especially in degenerative discs, result in disc rupture and penetration of cartilaginous material into spinal vessels. FCE remains a rarely recognized but important cause of SCI. Most of the available literature in FCE is based on autopsy-proven case reports; hence, the prognosis seems overwhelmingly poor. The lack of definitive formal clinical diagnostic criteria causes severe limitations in the diagnosis and knowledge and prevents accurate assessment of benefits of a specific treatment modality. Features that make the diagnosis most likely include antecedent minor trauma, lifting weights, physical exertion or Valsalva maneuver. There is usually severe pain at the onset and deficits can evolve over 15 min to 48 h, indicating a “spinal stroke in evolution.” There could also be a symptom-free interval between the onset of...
pain and deficit. The spinal cord imaging should be compatible with evolving infarction, cerebrospinal fluid analysis should be normal and other possible etiologies for transverse myelopathy should be excluded. Differentiation from spinal disc prolapse or herniation can be extremely difficult and diagnosis will need clinical correlation. FCE could be an underestimated entity and potential mechanical triggers may be better avoided, especially in the elderly with degenerative discs, considering the limited treatment options.

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