

# Neuroimaging Highlight

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## Spinal Cord Infarction from an Unstable Aortic Plaque

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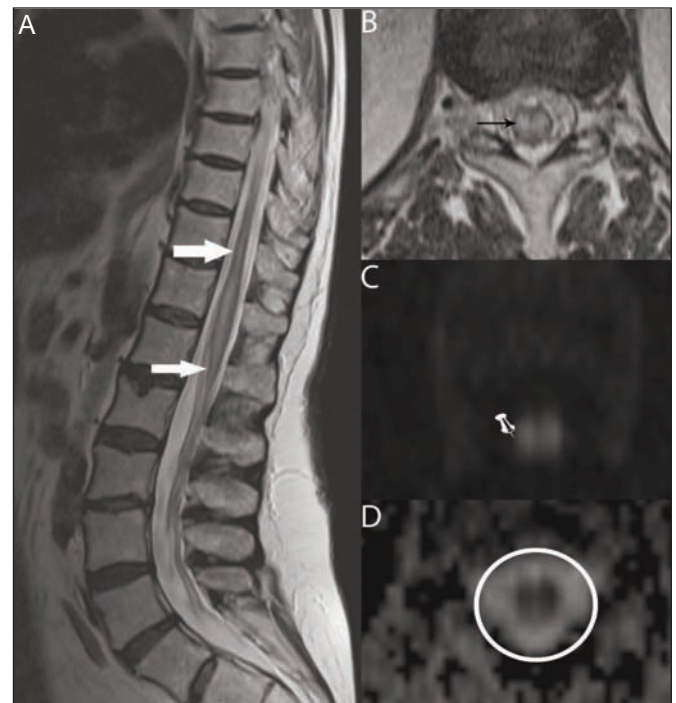
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A 78 year-old woman presented with acute onset low back pain with radiation into the right T12-L2 dermatomes, progressive flaccid paraparesis, urinary incontinence and bilateral lower extremity paresthesias. Examination revealed flaccid paraparesis, and a T12 sensory level to pin with intact vibration and proprioception. Magnetic resonance imaging (MRI) (Figure 1) of the spinal cord demonstrated T2 hyperintensity and restricted diffusion from T12 to the conus medullaris. Computed tomogram (CT) aortogram (Figure 2) highlighted an ulcerated plaque and thrombus at the approximate level of the artery of Adamkiewicz. These findings were consistent with a spinal cord infarct of the cord from T12-L2. The patient was treated with Clopidogrel and IV heparin (x two days) and was transferred for spinal cord rehabilitation.

### DISCUSSION

The spinal column is vascularised from a) radicular arteries b) radiculomedullary arteries which supply the anterior spinal artery and c) radiculopial arteries which supply the posterior spinal arteries. The anterior spinal artery supplies the anterior 2/3 of the spinal cord via the sulcal artery and the anterolateral surface of the cord via the circumferential pial branches. Three major segmental arteries contribute to the anterior spinal artery: the artery of Adamkiewicz, the artery of cervical enlargement and contribution from the distal vertebral arteries<sup>1</sup>. The presented case highlights likely embolic occlusion of the artery of Adamkiewicz from an unstable aortic plaque and resultant anterior spinal cord infarction (SCI) from T12 - conus medullaris and the utility of diffusion weighted imaging (DWI) in the diagnosis of SCI.

Spinal cord infarction is rare, accounting for approximately 1% of strokes<sup>2</sup>. Commonly the infarct is located in the anterior spinal artery territory<sup>3-6</sup> most frequently affecting the thoracolumbar portions of the spinal cord<sup>3,5,6</sup>. In contrast with cerebral ischemia, in SCI motor deficits rarely reach maximal intensity instantaneously, usually evolving over minutes to hours<sup>3</sup>. Additionally pain is frequently reported at symptom onset<sup>3,4</sup>. Moreover, SCI is associated with high mortality rates of 18-22%<sup>5</sup> and prognosis largely depends on the severity of neurologic



**Figure 1:** MRI of the cord demonstrates A) T2 hyperintensity extending from T12 –L2 [white arrows] with B) peripheral rim of sparing [Black arrow], additionally C) demonstrating restricted diffusion [thumb tack] on the axial diffusion-weighted sequence and D) corresponding apparent diffusion coefficient map.

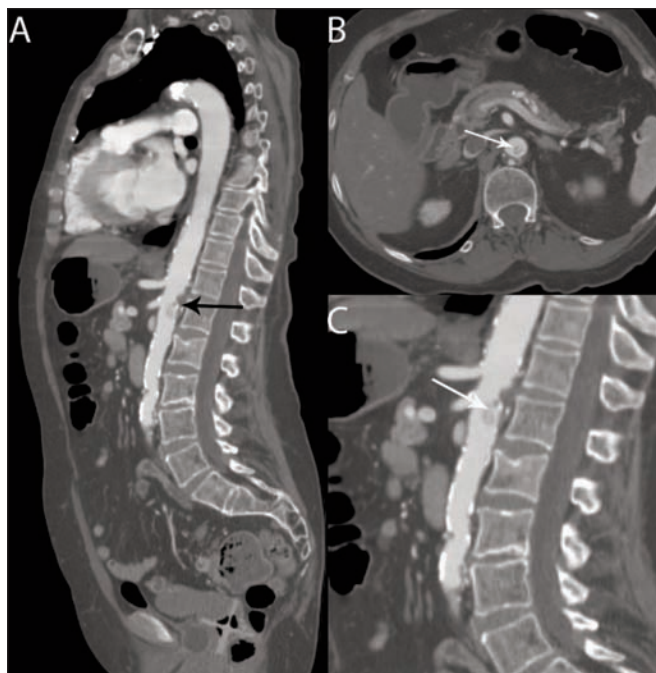
deficit at onset<sup>3,5,7</sup>. A definite etiology for SCI is found in less than 50% of cases<sup>3,4</sup> and most frequently points to aortic pathology<sup>3,5</sup> necessitating imaging of the aorta.

Magnetic resonance imaging findings in SCI can be variable and are influenced by timing of imaging, and pulsation artefacts<sup>8</sup>. T2-weighted hyperintensities accompanied by cord enlargement is usually seen in SCI.<sup>9</sup> However, acute T2-weighted imaging

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**Figure 2:** CT aortogram demonstrated A) posteriorly penetrating atheromatous ulcerated plaque (black arrow) in the descending aorta at T12/L1 and B,C) intraluminal filling defect consistent with thrombus (white arrow).

may be normal secondary to inadequate influx of water into infarcted tissue<sup>8</sup>. Similarly, the earliest DWI positivity in SCI is reported at three hours post symptom onset<sup>9</sup>. In contrast to cerebral infarction DWI of SCI suffers from early (within one week) signal normalization (pseudonormalization) thus providing a shorter window of opportunity for DWI<sup>8</sup>. Nonetheless, DWI of the spinal cord reliably distinguishes inflammatory myelopathies (high apparent diffusion coefficient) from SCI (low apparent diffusion coefficient)<sup>10</sup> and remains a valuable tool in evaluation of SCI.

A clinical picture suggestive of SCI should prompt an MRI of the spinal cord with DWI sequences and a CT-aortogram. However, MRI may be normal for several hours after symptom onset in SCI, and serial imaging may be beneficial in cases with high index of suspicion for SCI. Emerging techniques of diffusion tensor imaging and fiber tractography may allow for precise quantification of diffusion restriction in three dimensions<sup>9</sup> and perhaps allow for better prognostication after SCI.

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