Vertebral Body Infarction – A Useful Imaging Clue in Cord Infarction

Bee Kun Chew[1]*, Chern Yue Glen Ong[1], Alexander Jiawei Yap[2]

[1]*Department of Radiology, Tan Tock Seng Hospital, Singapore*
[2]*Department of Radiology, Woodlands Health, Singapore*

***Correspondence:** Bee Kun Chew, 1Department of Radiology, Tan Tock Seng Hospital, Singapore

b_kun0508@hotmail.com

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ABSTRACT

Spinal cord infarction is a rare and often challenging diagnosis, frequently requiring exclusion of other condi-tions. Its clinical and imaging features overlap with inflammatory and demyelinating disorders, complicating early recognition. Magnetic resonance imaging (MRI) is the preferred modality for diagnosis, though distin-guishing spinal cord infarction from mimics remains difficult. The identification of vertebral body (VB) infarc-tion, attributed to shared vascular supply via segmental arteries, has been recognized as a supportive imaging feature that may aid in diagnosis. This case highlights the role of VB infarction as a useful imaging clue in sus-pected spinal cord infarction, emphasizing its importance in improving diagnostic confidence and guiding ap-propriate management.

CASE REPORT

BACKGROUND

Spinal cord infarction is a rare but devastating condition that can lead to significant neurological deficits. Even more uncommon is its association with vertebral body infarction, a finding that may serve as a critical diagnostic clue. Given their shared vascular supply, particularly from the vertebral and segmental arteries, infarction of the vertebral body may indicate an underlying vascular insult affecting the spinal cord. However, this relationship is not widely recognized, potentially delaying diagnosis and management.

This case highlights the importance of vertebral body infarction as an early imaging marker for spinal cord ischemia, emphasizing the need for clinicians to consider vascular pathology when encountering such findings. By contributing to the limited literature on the co-occurrence of these infarctions, this report underscores the role of prompt recognition in improving patient outcomes.

CASE REPORT

A 66-year-old woman attended the emergency department for acute-onset low back pain associated with right lower limb weakness and reduced sensation. The patient's medical history was significant for hypertension, hyperlipidemia and impaired fasting glucose. Clinical examination further established a sensory level at T10. An emergent MRI of the head was negative for acute stroke.

Imaging Findings

An MRI of the thoracolumbar spine was then requested by the consulting neurologist. This demonstrated hyperintensity spanning the T8 to T9 cord on T2-weighted (T2W), short-T1 inversion recovery (STIR) and diffusion-weighted imaging (DWI) sequences (Figs 1A, 1B and 1C, thin arrows). Corresponding hypointensity was noted on apparent diffusion coefficient (ADC) map (not shown). Axial T2W images showed patchy hyperintensity in the affected cord (Figure 1D, arrowhead). Beyond the cord, focal hyperintensity was also observed in the posterior half of the T10 vertebral body in STIR and DWI, indicating infarction (Figure 1B,1C, thick arrow).

Management & Follow-up

Subsequent autoimmune, infective, nutritional (B12 and folate), and CSF studies were normal. A CT aortogram was also performed to rule out underlying vascular causes. No evidence of aortic pathology or vasculitis was found; however, multiple atherosclerotic plaques were seen.

A final diagnosis of spinal cord infarction was made, and the patient was started on anti-platelet therapy. Good functional recovery was achieved over the course of three months of rehabilitation.

DISCUSSION

Etiology & demographics

Spinal cord infarction is a rare but serious condition, accounting for less than 1% of all strokes. It typically results from vascular compromise due to embolism, thrombosis, or systemic hypoperfusion. The most common causes include atherosclerosis, aortic pathologies (such as dissection or aneurysm), embolic events from cardiac sources, and iatrogenic factors such as aortic surgery or spinal interventions. Less frequently, vasculitis, hypercoagulable states, and systemic inflammatory disorders contribute to spinal cord ischemia. Spinal cord infarction predominantly affects older adults, particularly those over 50 years of age, with risk factors such as hypertension, diabetes mellitus, hyperlipidemia, and smoking playing a significant role. However, younger individuals may also be affected, especially in cases of vasculitis, genetic thrombophilia, or substance abuse. The thoracic spinal cord is most frequently involved due to its vascular watershed areas, particularly between the artery of Adamkiewicz and other segmental arteries, making it more vulnerable to ischemic injury [1].

Clinical & imaging findings

The spinal cord receives its blood supply from a single anterior spinal artery and two posterior spinal arteries. These are further reinforced by segmental branches that anastomose with the spinal arteries, the most prominent being the artery of Adamkiewicz. The anterior spinal artery supplies the majority of the spinal cord, except for the posterior grey columns and white matter, which are perfused by the posterior spinal arteries. Given this vascular distribution, infarcts often affect the anterior spinal cord, leading to syndromes such as anterior spinal artery syndrome. However, patchy or diffuse ischemic involvement may result in variable neurological presentations [2].

Vertebral body infarction has been increasingly recognized as a supportive imaging feature in spinal cord infarction [3-5]. This is attributed to the shared vascular supply between the spinal cord and the vertebral body via segmental arteries. In this case, MRI findings demonstrated hyperintensity in the thoracic spinal cord on T2-weighted and diffusion-weighted imaging (DWI), with corresponding hypointensity on the apparent diffusion coefficient (ADC) map. The presence of vertebral body infarction at the T10 level, one segment caudal to the affected spinal cord, further supported the diagnosis. While the sensitivity of vertebral body infarction as a diagnostic sign has not been well established, its presence serves as an important imaging clue that may improve diagnostic confidence in cases of suspected spinal cord ischemia.

Treatment & prognosis

Spinal cord infarction is a challenging condition with no specific curative treatment. Management is primarily supportive, focusing on optimizing spinal cord perfusion, preventing secondary complications, and addressing underlying risk factors. In this case, the patient was started on antiplatelet therapy, given the presence of atherosclerotic plaque as the likely cause of infarction. Antiplatelet agents such as aspirin or clopidogrel are commonly used to reduce the risk of further thrombotic events, particularly in patients with underlying vascular disease. Hemodynamic support is another crucial aspect of treatment, as maintaining adequate blood pressure can help optimize spinal cord perfusion and prevent further ischemic damage. Although thrombolysis is not routinely used in spinal cord infarction due to the lack of strong evidence, anticoagulation may be considered in select cases where an embolic source is identified. Early rehabilitation and physiotherapy play a vital role in functional recovery, helping to improve mobility, prevent contractures, and enhance the patient's quality of life. The degree of recovery largely depends on the severity and extent of the infarction. In this case, the patient demonstrated good functional recovery over a three-month rehabilitation period, which is an encouraging outcome. Prognosis in spinal cord infarction is variable. Patients with partial cord involvement tend to have better outcomes, while those with complete motor deficits at onset often experience long-term disability. Common complications include chronic pain, spasticity, neurogenic bladder, and bowel dysfunction. Despite these challenges, early diagnosis, risk factor management, and aggressive rehabilitation can significantly improve functional outcomes, as seen in this case [6].

Differential Diagnoses

Spinal cord infarction can present either suddenly or progress over several hours, mimicking other neurological conditions. Its clinical and imaging features often overlap

with inflammatory, demyelinating, and neoplastic disorders. MRI findings typically include cord swelling and increased T2W signal, similar to other pathologies. Key distinguishing factors for spinal cord infarction include its arterial territory distribution, confined anterior or posterior cord involvement, and absence of widespread CNS lesions. DWI plays a critical role, showing marked hyperintensity in ischemic infarction, whereas mild hyperintensity was likely to be seen on DWI of myelitis or tumours, suggesting the possibility of separating ischemic from inflammatory cord lesions and intramedullary tumours by the different range of ADC values [7].

Multiple Sclerosis (MS)

MS is a chronic demyelinating disease affecting the CNS, frequently involving the spinal cord. MS plaques appear as T2W hyperintense and T1W iso- or hypointense lesions. They are well-circumscribed, peripherally located, and usually affect less than two vertebral segments. Unlike infarction, MS predominantly affects the cervical cord and has a relapsing-remitting course. Synchronous brain lesions are common, aiding in differentiation [7].

Idiopathic Transverse Myelitis (ITM)

ITM is immune-mediated and may occur independently or as part of systemic diseases. MRI findings include centrally located T2W hyperintensity extending over multiple vertebral segments, often occupying more than two-thirds of the cord cross-section. Progression over hours, enhancement on gadolinium MRI, and cerebrospinal fluid (CSF) pleocytosis favour ITM over infarction [7].

Spinal Cord Arteriovenous Shunts (SCAVS)

Spinal dural arteriovenous fistulas (SDAVF) and arteriovenous malformations (AVM) can mimic spinal infarction due to venous congestion and cord edema. MRI findings such as T2W hyperintensity, cord swelling, and contrast enhancement overlap with infarction. However, the presence of perimedullary flow voids and a hypointense rim on T2W suggest SDAVF. AVM may also present with haemorrhagic components, further distinguishing them from infarction [7].

Spinal Cord Neoplasms

Primary and metastatic spinal tumours, such as ependymomas, astrocytomas, and hemangioblastomas, can resemble infarction due to intramedullary T2W hyperintensity and cord expansion. Key distinguishing features include a slower clinical onset, diffuse or nodular contrast enhancement, and presence of cystic components. Metastatic lesions often exhibit ring-enhancement and occur in patients with known malignancies.

While spinal cord infarction shares imaging and clinical features with various conditions, factors such as lesion distribution, disease progression, enhancement patterns, and associated systemic findings help differentiate it from MS, ITM, SCAVS, and neoplasms. Advanced imaging, including DWI, contrast-enhanced MRI, and CSF analysis, plays a crucial role in establishing an accurate diagnosis [7].

TEACHING POINT

A combination of clinical assessment, laboratory findings, and MRI is crucial for diagnosing spinal cord infarction. The presence of concomitant vertebral body infarction is a strong supportive imaging clue.

QUESTIONS

Question 1: Which imaging modality is most useful in diagnosing spinal cord infarction?

- Computed Tomography (CT)
- Magnetic Resonance Imaging (MRI) (applies)
- X-ray
- Ultrasound
- Positron Emission Tomography (PET)

Explanation: MRI is the most appropriate imaging modality for diagnosing spinal cord infarction, as it allows for the assessment of diffusion-weighted imaging (DWI) and T2-weighted changes in the spinal cord. DWI hyperintensity and corresponding hypointensity on apparent diffusion coefficient (ADC) maps are key imaging features. ["MR imaging is the most appropriate imaging tool in suspected spinal cord infarction."]

Question 2: Which of the following imaging findings support the diagnosis of spinal cord infarction?

- T2-weighted (T2w) hyperintensity in the spinal cord (applies)
- Diffusion-weighted imaging (DWI) hyperintensity in the spinal cord (applies)
- Apparent diffusion coefficient (ADC) hyperintensity
- DWI hyperintensity in the vertebral body (applies)
- Enhancement of the spinal cord on post-contrast MRI

Explanation: Spinal cord infarction typically presents with hyperintensity on T2-weighted imaging and diffusion-weighted imaging (DWI), with corresponding hypointensity on apparent diffusion coefficient (ADC) mapping. Additionally, vertebral body infarction can be seen as DWI hyperintensity, which serves as a supportive imaging clue. Post-contrast enhancement is more commonly associated with inflammatory or neoplastic conditions rather than acute infarction. ["This demonstrated hyperintensity spanning the T8 to T9 cord on T2-weighted (T2w) and diffusion-weighted imaging (DWI) sequences… Corresponding hypointensity was noted on apparent diffusion coefficient (ADC) map… DWI hyperintensity was also observed in the posterior half of the T10 vertebral body, indicating infarction."]

Question 3: Which of the following clinical features are commonly associated with spinal cord infarction?

- Acute-onset back pain (applies)
- Gradual progression over months
- Lower limb weakness (applies)

- Sensory level deficit (applies)
- Seizures

Explanation: Spinal cord infarction typically presents with acute-onset back pain, motor deficits (e.g., lower limb weakness), and sensory abnormalities such as a defined sensory level. It is usually a sudden event rather than a progressive condition. Seizures are not a typical feature as they are more commonly associated with cerebral infarction. ["Patients present with acute-onset back pain, accompanied by neurologic deficits which range from unilateral limb weakness to catastrophic tetraplegia."]

Question 4: What is the role of vertebral body infarction in diagnosing spinal cord infarction?

- It confirms the diagnosis of spinal cord infarction
- It is an incidental finding with no clinical significance
- It serves as an adjunctive imaging clue (applies)
- It is only seen in traumatic spinal cord injuries
- It suggests an alternative diagnosis such as infection

Explanation: Vertebral body infarction is not a definitive diagnostic feature but serves as a useful adjunctive imaging clue in cases of spinal cord infarction. It is thought to result from shared vascular supply between the vertebral body and spinal cord. ["Vertebral body infarction has been reported as a supportive sign of spinal cord infarction."]

Question 5: Which of the following treatments are commonly used in the management of spinal cord infarction?

- Antiplatelet therapy (applies)
- Thrombolysis as first-line treatment
- Blood pressure optimization (applies)
- Early rehabilitation (applies)
- Immediate surgical decompression in all cases

Explanation: Management of spinal cord infarction is primarily supportive, focusing on optimizing spinal cord perfusion, reducing the risk of further ischemic events with antiplatelet therapy, and promoting functional recovery through rehabilitation. Thrombolysis is not a routine treatment, and surgical decompression is only considered in cases with significant mass effect or compression rather than ischemia. ["A final diagnosis of spinal cord infarction was made, and the patient was started on anti-platelet therapy. Good functional recovery was achieved over the course of three months of rehabilitation."]

REFERENCES

1. Zalewski NL, Rabinstein AA, Krecke KN, et al. Characteristics of Spontaneous Spinal Cord Infarction and Proposed Diagnostic Criteria. *JAMA Neurol.* 2019; 76(1): 56-63. PMID: 30264146.
2. Novy J, Carruzzo A, Maeder P, Bogousslavsky J. Spinal cord ischemia: clinical and imaging patterns, pathogenesis, and outcomes in 27 patients. *Arch Neurol.* 2006; 63(8): 1113-1120. PMID: 16908737.
3. Faig J, Busse O, Salbeck R. Vertebral body infarction as a confirmatory sign of spinal cord ischemic stroke: report of three cases and review of the literature. *Stroke.* 1998; 29(1): 239-243. PMID: 9445357.
4. Börnke C, Schmid G, Szymanski S, Schöls L. Vertebral body infarction indicating midthoracic spinal stroke. *Spinal Cord.* 2002; 40(5): 244-247. PMID: 11987007.
5. Diehn FE, Hunt CH, Lehman VT, et al. Vertebral body infarct and ventral cauda equina enhancement: two confirmatory findings of acute spinal cord infarct. *J Neuroimaging.* 2015; 25(1): 133-135. PMID: 24118402.
6. Ros Castelló V, Sánchez Sánchez A, Natera Villalba E, et al. Spinal cord infarction: aetiology, imaging findings, and prognostic factors in a series of 41 patients. *Neurologia (Engl Ed).* 2021; S0213-4853(21)00002-00005. PMID: 33581950.
7. Boddu, S. R., Cianfoni, A., Kim, K.-W., Banihashemi, M.A., Pravata, E., Goblin, Y.P., Patsalides, A. Spinal Cord Infarction and Differential Diagnosis. *Neurovascular Imaging.* 2015; pg 1-64.

FIGURES

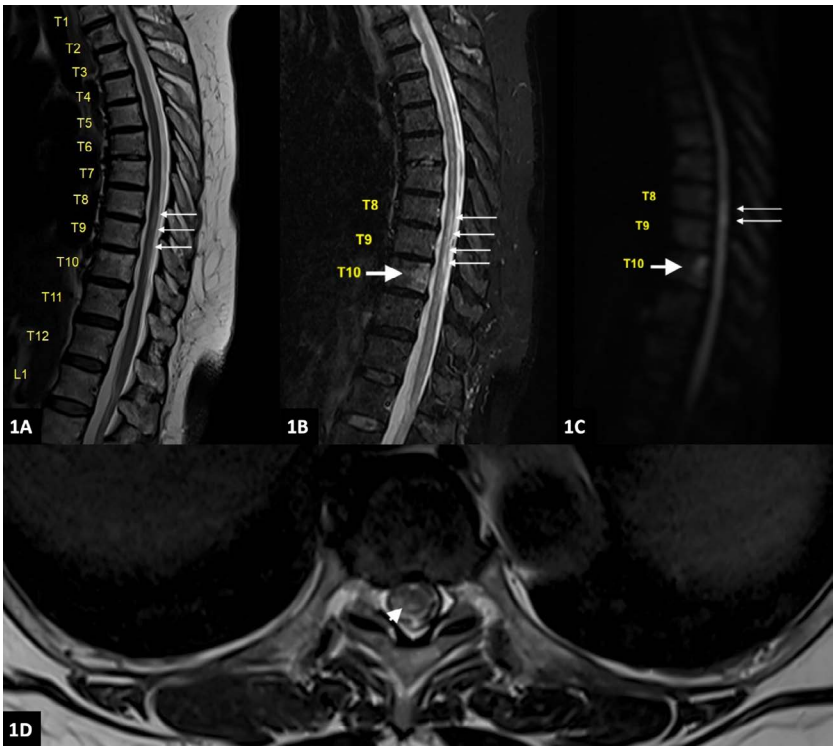

Figure 1: MRI findings. (A) Sagittal T2W, (B) Sagittal STIR, and (C) Sagittal DWI sequences reveal a longitudinal intramedullary hyperintensity spanning the T8–T9 spinal cord levels (thin arrows). (D) Axial T2W image demonstrates patchy hyperintensity within the corresponding segment of the spinal cord (arrowhead). Focal hyperintensity involving the posterior half of the T10 vertebral body is also noted on STIR and DWI sequences (B, C; thick arrows), suggestive of vertebral body infarction.

Table 1: Summary table

Category	Details
Etiology	Vascular compromise due to embolism, thrombosis, systemic hypoperfusion, atherosclerosis, aortic pathology (dissection, aneurysm), embolic events from cardiac sources, vasculitis, hypercoagulable states, systemic inflammatory disorders, iatrogenic causes (aortic surgery, spinal interventions).
Incidence	Rare; accounts for <1% of all strokes.
Gender ratio	No significant gender predilection reported.
Age predilection	More common in adults >50 years, though younger individuals may be affected in cases of vasculitis, genetic thrombophilia, or substance abuse.
Risk factors	Hypertension, diabetes mellitus, hyperlipidemia, smoking, aortic diseases, cardiac embolic sources, spinal surgeries, systemic hypoperfusion.
Treatment	Supportive care, antiplatelet therapy (aspirin, clopidogrel), anticoagulation (if embolic source is identified), hemodynamic support, rehabilitation, and physical therapy.
Prognosis	Variable; better in partial cord infarctions, worse in complete motor deficits. Long-term complications include chronic pain, spasticity, neurogenic bladder/bowel dysfunction. Early diagnosis and rehabilitation improve functional outcomes.
Findings on imaging	T2-weighted MRI shows hyperintensity of affected spinal cord segments; diffusion-weighted imaging (DWI) shows hyperintensity with corresponding hypointensity on apparent diffusion coefficient (ADC) maps. Vertebral body infarction (hyperintensity on STIR and DWI) may serve as a supportive imaging clue.

Table 2: Differential diagnoses

Condition	MRI - T1	MRI - T2	MRI - DWI	Pattern of Contrast Enhancement
Spinal Cord Infarction	Isointense to hypo-	Hyperintense, cord swelling	Hyperintense (significant)	None to minimal, if present, usually localized
Multiple Sclerosis (MS)	Isointense to hypointense	Hyperintense, well-circumscribed lesions	Mild hyperintensity	Homogeneous or heterogeneous enhancement, peripherally located plaques
Idiopathic Transverse Myelitis (ITM)	Isointense to hypointense	Hyperintense, centrally located, spans several segments	Mild hyperintensity	Homogeneous enhancement, often with diffuse involvement
Spinal Cord Arteriovenous Shunts (SCAVS)	Isointense to hypointense	Hyperintense, cord swelling	No significant hyperintensity	Homogeneous or heterogeneous, perimedullary flow voids or rim enhancement in SDAVF
Spinal Cord Neoplasms	Isointense to hypointense	Hyperintense, cord expansion	Mild hyperintensity	Nodular or diffuse enhancement, cystic components, ring enhancement in metastatic lesions

KEYWORDS

Spinal cord infarction; Spinal cord stroke; Vertebral body infarction; Ischemic myelopathy; Vascular myelopathy

ABBREVIATIONS

ADC = APPARENT DIFFUSION COEFFICIENT
AVM = ARTERIOVENOUS MALFORMATIONS
CSF = CEREBROSPINAL FLUID
CT = COMPUTED TOMOGRAPHY
DWI = DIFFUSION-WEIGHTED IMAGING
ITM = IDIOPATHIC TRANSIENT MYELITIS
MRI = MAGNETIC RESONANCE IMAGING
MS = MULTIPLE SCLEROSIS
SCAVS = SPINAL CORD ARTERIOVENOUS SHUNT
SDAVF = SPINAL DURAL ARTERIOVENOUS FISTULA
STIR = SHORT-T1 INVERSION RECOVERY
T1W = T1-WEIGHTED
T2W = T2-WEIGHTED

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