Cureus
Part of SPRINGER NATURE

Open Access Case Report

Anterior Spinal Cord Infarction Following Glucocorticoid Withdrawal-Associated Adrenal Insufficiency: A Case Report

Review began 11/04/2026
Review ended 11/08/2026
Published 11/11/2026

© **Copyright** 2026
Warren et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

DOI: 10.7759/cureus.119495

Caitlyn Warren [1], Shayna Swaine [2], Evgenia Dagiakidi [3], Mohamed Ali [4]

1. Internal Medicine, East Sussex Healthcare NHS Trust, St Leonards-on-Sea, GBR 2. Anaesthesia and Critical Care, Northampton General Hospital NHS Trust, Northampton, GBR 3. Radiology, East Sussex Healthcare NHS Trust, St Leonards-on-Sea, GBR 4. Neurology, East Sussex Healthcare NHS Trust, St Leonards-on-Sea, GBR

Corresponding author: Caitlyn Warren, caitlynjwarren@gmail.com

Abstract

A woman in her early 70s presented with profound hypotension and acute bilateral lower-limb paralysis 10 days after abruptly discontinuing long-term prednisolone prescribed for polymyalgia rheumatica. She had been mobilising independently the previous day. Intravenous hydrocortisone was initiated for suspected adrenal crisis, and subsequent endocrine assessment and short Synacthen testing supported a diagnosis of adrenal insufficiency. Neurological examination revealed complete bilateral lower-limb paralysis, although deconditioning was initially considered in the context of her concurrent acute illness. Initial spinal MRI was incomplete due to patient intolerance. Specialist neurological examination subsequently identified loss of pain sensation below T7 on pinprick testing, preserved fine touch, and extensor plantar responses. Spinal MRI performed 19 days after admission was initially reported as showing no acute spinal cord signal abnormality. However, specialist neuroradiological review of the same examination the following day identified anterior spinal cord signal abnormalities extending from C7 to T7, relative sparing of the posterior columns, and an “owl’s-eye” appearance at T1, supporting a diagnosis of spinal cord infarction in the anterior spinal artery territory. CT aortography excluded aortic dissection. Management included glucocorticoid replacement, antiplatelet therapy, spinal precautions, pressure-area care, and multidisciplinary rehabilitation. Subsequent recovery was characterised by limited toe movement, partial proximal motor recovery, and some improvement in sensation and reflexes. This case highlights the importance of careful neurological reassessment and specialist neuroradiological review when severe neurological deficits remain unexplained by initial clinical assessment or imaging. Profound hypotension associated with adrenal insufficiency was a plausible contributing factor to spinal cord ischaemia.

Categories: Neurology, Internal Medicine, Endocrinology/Diabetes/Metabolism
Keywords: acute paraplegia, anterior spinal artery syndrome, anterior spinal cord infarction, drug-induced adrenal insufficiency, glucocorticoid withdrawal, neurological emergencies, severe hypotension, spinal magnetic resonance imaging

Introduction

Spinal cord infarction is an uncommon but potentially disabling cause of acute myelopathy [1]. Infarction involving the anterior spinal artery territory typically presents with bilateral motor weakness and loss of pain and temperature sensation, with relative preservation of posterior-column function [1,2]. It accounts for approximately 5-8% of acute myelopathies [3]. Recognised aetiologies include aortic pathology, vascular procedures, and systemic hypotension, although the underlying cause may remain unidentified [1,3].

Diagnosis can be challenging because clinical features may overlap with those of other causes of acute myelopathy, while early MRI may not demonstrate characteristic spinal cord abnormalities [4,5]. Subsequent imaging may reveal longitudinal T2-weighted hyperintensity and bilateral anterior horn involvement, producing the characteristic “owl’s-eye” appearance on axial sequences [2,4,5]. These findings should be interpreted in conjunction with a detailed neurological examination [1,2].

Prolonged glucocorticoid therapy can suppress the hypothalamic-pituitary-adrenal axis, increasing the risk of adrenal insufficiency following abrupt withdrawal [6,7]. Current guidelines identify an increased risk of glucocorticoid-induced adrenal insufficiency with treatment lasting three to four weeks or longer at doses exceeding approximately 4-6 mg of prednisolone daily [7]. Adrenal crisis may result in profound hypotension and haemodynamic instability [7,8]. Given that systemic hypotension is a recognised contributor to spinal cord ischaemia [3], adrenal crisis represents a biologically plausible precipitating context for spinal cord infarction, although this specific association remains poorly characterised in the literature.

We report a case of anterior spinal cord infarction identified in a patient presenting with profound hypotension associated with glucocorticoid withdrawal-associated adrenal insufficiency. This case raises the possibility of hypotension-mediated spinal cord ischaemia while highlighting the importance of neurological reassessment, appropriate repeat imaging, and specialist neuroradiological review.

How to cite this article
Warren C, Swaine S, Dagiakidi E, et al. (November 11, 2026) Anterior Spinal Cord Infarction Following Glucocorticoid Withdrawal-Associated Adrenal Insufficiency: A Case Report. Cureus 18(11): e119495. DOI 10.7759/cureus.119495

Case Presentation

A woman in her early 70s presented to the emergency department with breathlessness, generalised weakness, and sudden-onset bilateral lower-limb paralysis. She had reportedly been mobilising normally the previous day but became unable to move her legs or get out of bed on the day of admission. Her medical history included polymyalgia rheumatica and chronic obstructive pulmonary disease (COPD). She had received intermittent glucocorticoid treatment for COPD for more than a decade and regular prednisolone for polymyalgia rheumatica for approximately six years, most recently at a dose of 8.5 mg daily. She had abruptly discontinued prednisolone 10 days before presentation. On admission, she was profoundly hypotensive, with a blood pressure of 76/52 mmHg. Intravenous hydrocortisone was commenced immediately for suspected adrenal crisis. By the end of the day of admission, her blood pressure had improved to 113/69 mmHg.

Initial chest radiography was reported as showing small bilateral pleural effusions (Figure *1*). Non-contrast CT of the head showed no acute intracranial abnormality on axial (Figure *2A*) or coronal (Figure *2B*) images. Spinal MRI performed within 24 hours of admission was incomplete due to patient intolerance; the limited images demonstrated mild cervical degenerative changes without acute cord compression. Admission blood tests showed a serum sodium concentration of 139 mmol/L, a potassium concentration of 4.6 mmol/L, and a blood glucose level of 7.2 mmol/L. The endocrinology team reviewed the patient four days after admission, by which time her clinical condition had improved following intravenous hydrocortisone treatment. A short Synacthen test was performed the following day after a documented 24-hour period without glucocorticoid administration. Baseline serum cortisol was 148 nmol/L, rising to 325 nmol/L at 30 minutes and measuring 288 nmol/L at 60 minutes. The peak cortisol concentration of 325 nmol/L remained below the 420 nmol/L threshold used by the treating endocrine team, who interpreted these findings as supporting adrenal insufficiency secondary to prolonged glucocorticoid use. Oral prednisolone was restarted at 8.5 mg daily, with a tapering plan coordinated with the rheumatology team.

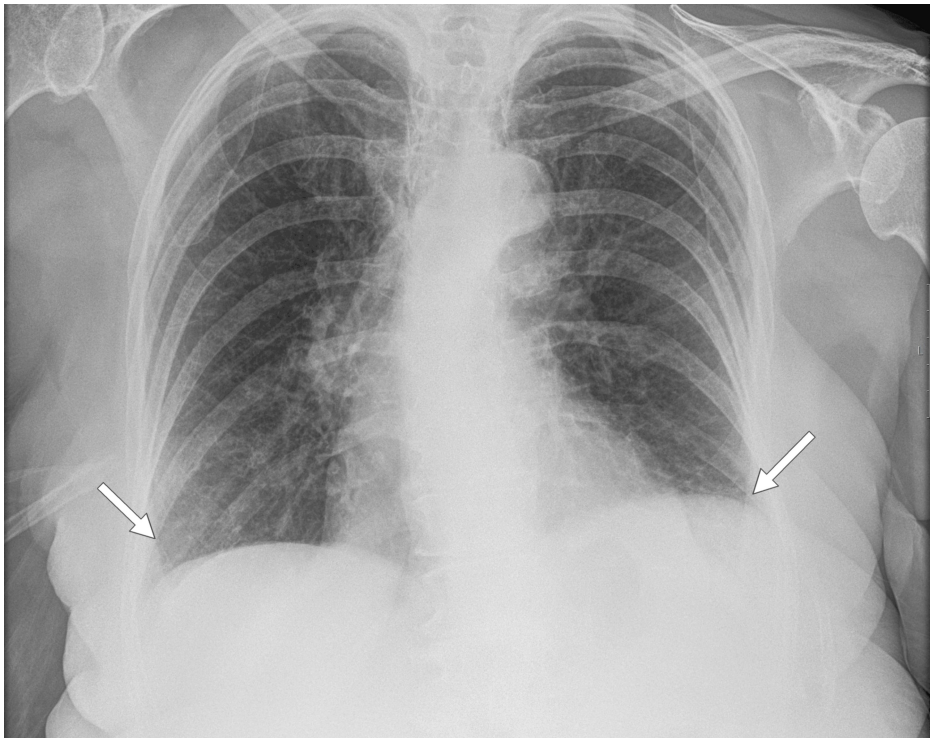


FIGURE 1: Chest radiograph on admission

Anteroposterior chest radiograph demonstrating small bilateral pleural effusions, highlighted by white arrows at the costophrenic angles. No other acute cardiopulmonary abnormalities were reported.

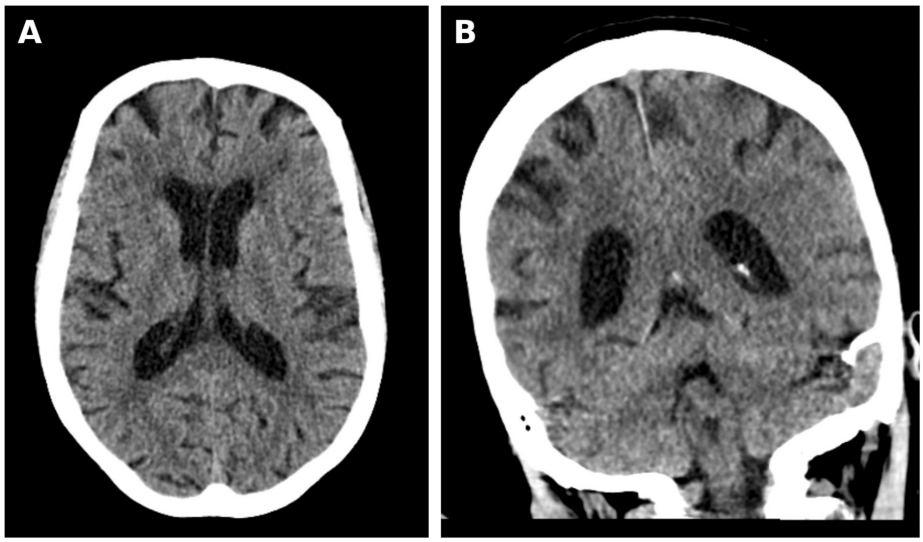


FIGURE 2: CT head on admission

(A) Axial and (B) coronal images from the admission non-contrast CT head. No acute intracranial haemorrhage or large territorial infarction was reported.

CT: computed tomography

Lower-limb power was graded as 0/5 bilaterally according to the Medical Research Council (MRC) scale [9]. In the context of her concurrent acute illness and an incomplete spinal MRI showing no evidence of cord compression, deconditioning was initially considered. However, profound lower-limb weakness persisted despite treatment of her systemic illness, and physiotherapy assessments were unable to elicit any active lower-limb movement. This prompted referral to the neurology team 13 days after admission. A further attempt at spinal MRI with sedation on the same day was also discontinued because the patient was unable to tolerate the examination.

Specialist neurological examination the following day demonstrated normal findings on cranial nerve and upper-limb examination. Both lower limbs were hypotonic, with reduced or absent knee reflexes, brisk ankle reflexes, and extensor plantar responses. Sensory examination revealed loss of pain sensation below approximately T7 on pinprick testing, preserved fine touch, and reduced or absent vibration sense. This combination of profound motor weakness and dissociated sensory loss raised suspicion of spinal cord ischaemia.

Brain MRI demonstrated non-specific small-vessel changes. Spinal MRI performed on day 19 was initially reported as showing no acute spinal cord signal abnormality. However, review of the same MRI at the neuroradiology multidisciplinary meeting the following day resulted in an addendum identifying a long-segment anterior spinal cord signal abnormality extending from C7 to T7, with relative sparing of the posterior columns (Figure *3*).

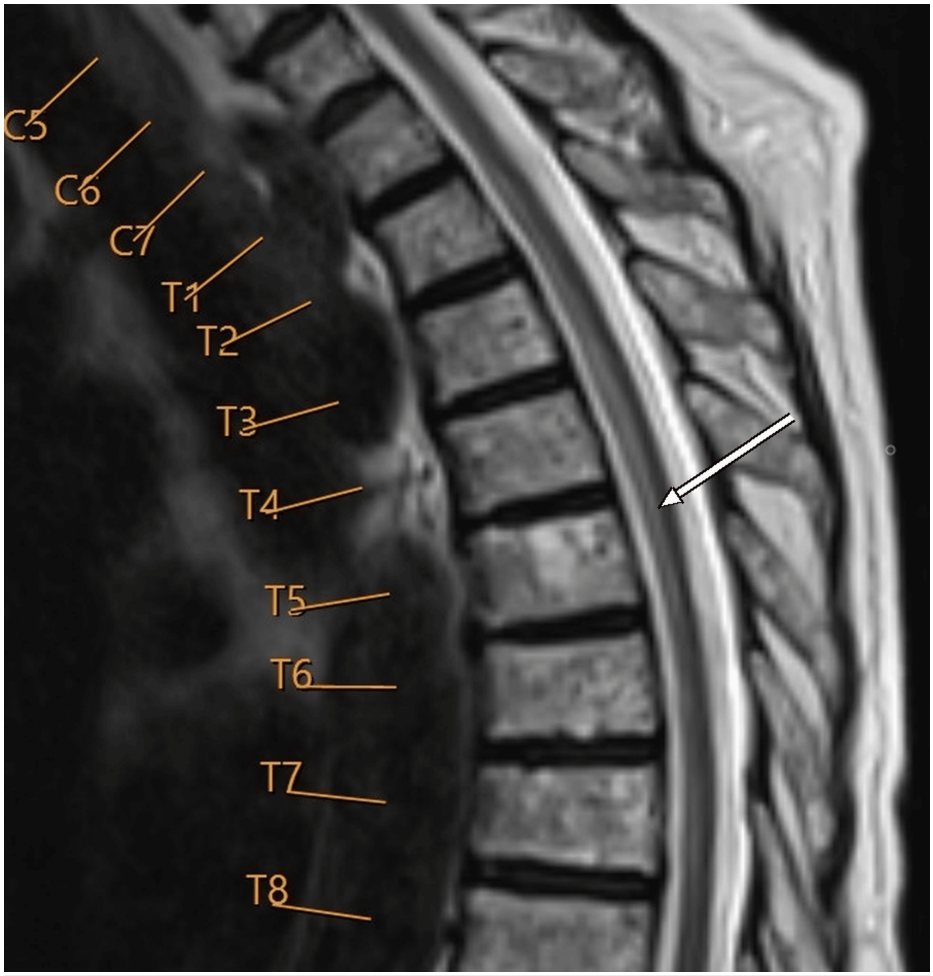


FIGURE 3: Sagittal T2-weighted MRI showing long-segment anterior spinal cord hyperintensity

Sagittal T2-weighted MRI demonstrating the reported long-segment anterior spinal cord signal abnormality extending from C7 to T7. The white arrow indicates a representative region of the anterior cord at approximately T4-T5.

MRI: magnetic resonance imaging

Axial T2-weighted imaging at the T1 level demonstrated bilateral anterior horn hyperintensity, producing the characteristic “owl’s-eye” appearance (Figure *4B*). Diffusion-weighted imaging showed high signal intensity without marked apparent diffusion coefficient reduction (Figure *4C*-*4D*). The combined clinical and radiological findings supported a diagnosis of spinal cord infarction involving the anterior spinal artery territory.

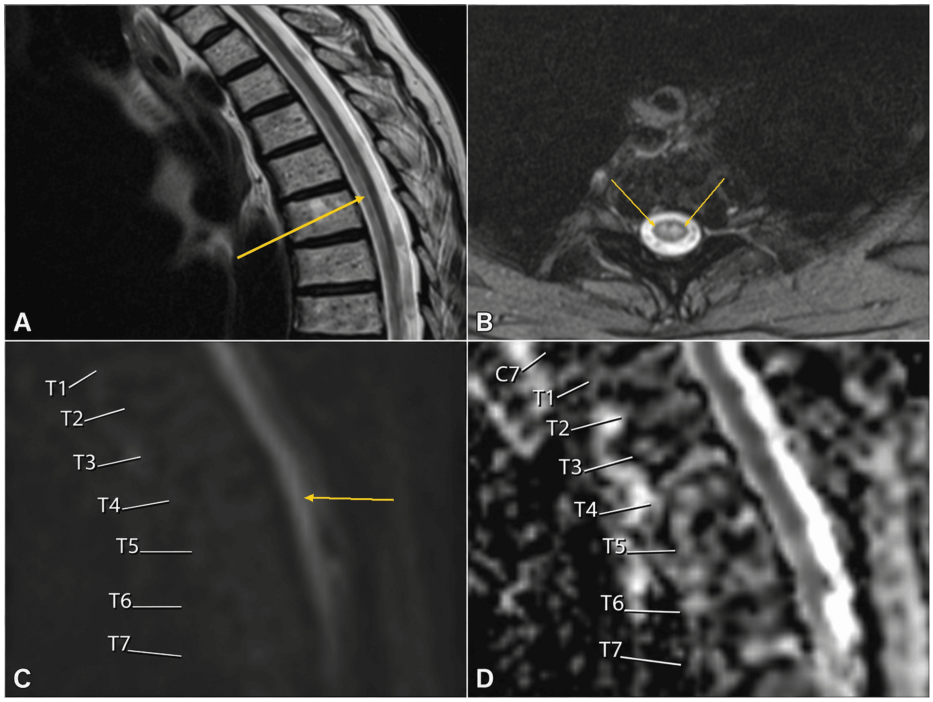


FIGURE 4: MRI findings supporting anterior spinal artery territory infarction

MRI performed on day 19 following admission. (A) Sagittal T2-weighted imaging demonstrates the reported long-segment anterior spinal cord signal abnormality extending from C7 to T7, with relative sparing of the posterior columns (yellow arrow in A). (B) Axial T2-weighted imaging at T1 demonstrates the reported bilateral anterior horn signal abnormalities, producing an “owl’s-eye” appearance (yellow arrows in B). (C) Sagittal DWI demonstrates increased signal along the spinal cord (yellow arrow in C). (D) The corresponding ADC map shows no marked ADC reduction. These findings, interpreted alongside the clinical presentation, supported a diagnosis of anterior spinal artery territory infarction.

MRI: magnetic resonance imaging, ADC: apparent diffusion coefficient, DWI: diffusion-weighted imaging

CT aortography excluded aortic dissection and demonstrated unremarkable intercostal arteries (Figure *5*). Differential diagnoses included inflammatory myelopathies, particularly longitudinally extensive transverse myelitis associated with neuromyelitis optica spectrum disorder or myelin oligodendrocyte glycoprotein antibody-associated disease, infectious myelitis, spinal cord compression, syringomyelia, and, less likely, neoplastic, nutritional, or toxic myelopathies. Investigations to exclude alternative causes of myelopathy included a vasculitis screen, erythrocyte sedimentation rate, vitamin B12 and folate levels, syphilis serology, HIV testing, hepatitis B and C serology, and serum aquaporin-4 (AQP4) and myelin oligodendrocyte glycoprotein antibody testing. All investigation results were negative or within normal limits; complement C3 and C4 levels were also within normal limits. Lumbar puncture was not performed because of the patient's pressure ulcer, lower-back oedema, and additional procedural concerns.

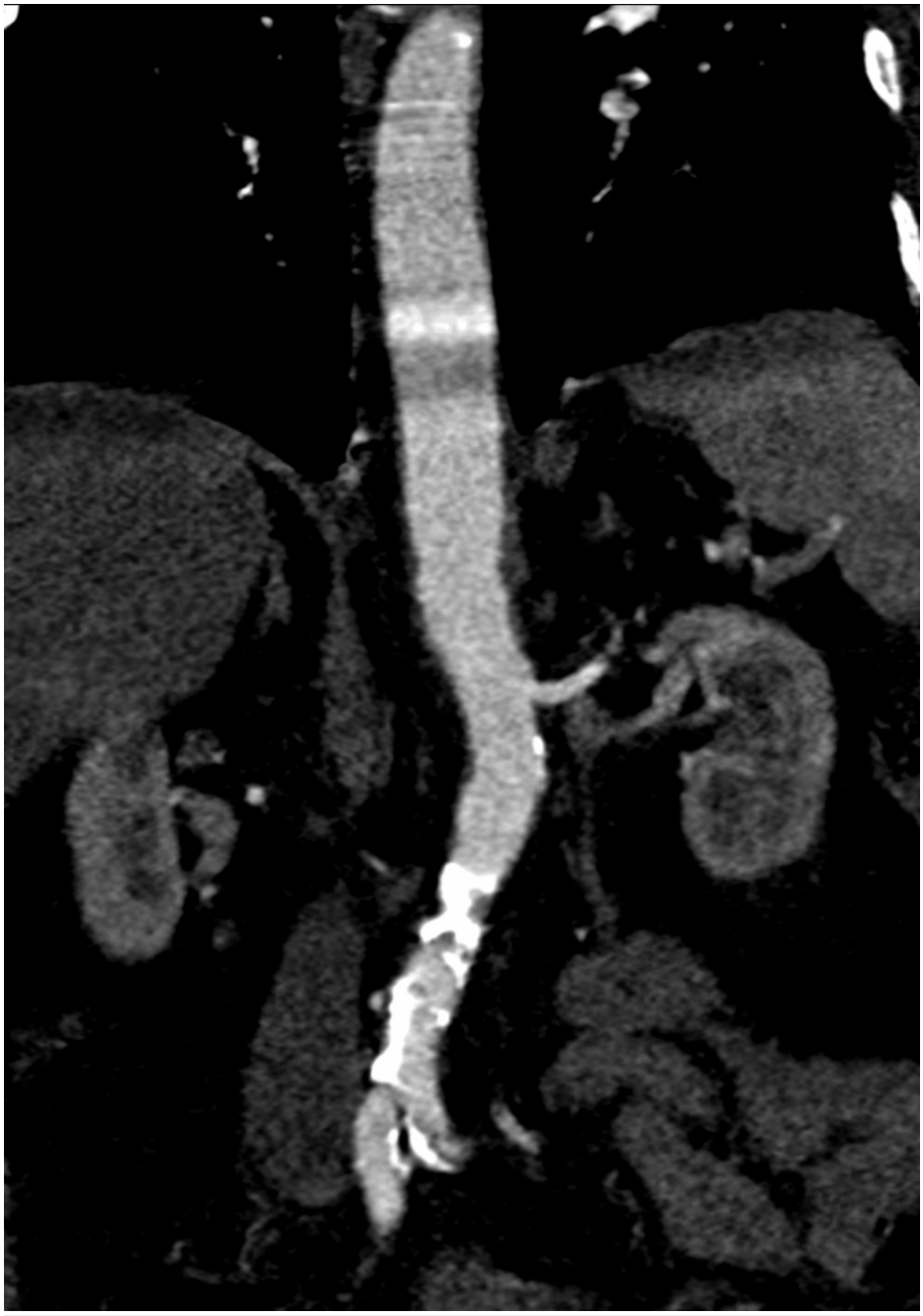


FIGURE 5: CT aortography

Representative coronal image from the CT aortogram. The complete examination was reported to show no aortic dissection, with unremarkable intercostal arteries.

CT: computed tomography

Management included glucocorticoid replacement, clopidogrel, venous thromboembolism prophylaxis, spinal precautions, and multidisciplinary rehabilitation involving physiotherapy, occupational therapy, and dietetics. During admission, the patient developed a stage 4 sacral pressure ulcer requiring surgical debridement, antibiotic treatment, and ongoing wound surveillance.

During neurological reassessment approximately four weeks after admission, the patient had regained limited movement of the right toes, with proximal lower-limb power documented as MRC grade 1-2/5. Pain sensation on pinprick testing had also begun to return, accompanied by recovery of reflex responses. These findings indicated partial neurological improvement, although significant functional impairment persisted. Transfer to a specialist rehabilitation facility was planned, and longer-term neurological and functional outcomes were unavailable at the time of reporting. Key clinical events are summarised in Table *1*.

Time from admission	Key clinical event
Day 0	Acute bilateral lower-limb paralysis and profound hypotension (76/52 mmHg). Intravenous hydrocortisone commenced for suspected adrenal crisis. Blood pressure improved to 113/69 mmHg by the end of the day.
Day 1	Initial spinal MRI incomplete due to patient intolerance.
Day 4	Endocrinology review; clinical improvement following hydrocortisone treatment.
Day 5	Short Synacthen test following 24 hours without glucocorticoid administration; sampled at baseline, 30 and 60 minutes.
Day 13	Neurology referral and second unsuccessful spinal MRI attempt, despite sedation.
Day 14	Specialist neurological examination identified dissociated sensory loss below T7 and extensor plantar responses.
Day 19	Completed spinal MRI initially reported as showing no acute cord signal abnormality.
Day 20	Specialist neuroradiological review of the same MRI identified anterior spinal cord signal abnormalities extending from C7 to T7.
Day 27	Limited right-toe movement, proximal lower-limb power MRC grade 1-2/5, and partial return of pain sensation on pinprick testing documented.

TABLE 1: Timeline of key clinical events and investigations

Day 0 represents the day of hospital admission.

MRI: magnetic resonance imaging, MRC: Medical Research Council

Discussion

This case highlights the potential relationship between glucocorticoid withdrawal-associated adrenal insufficiency, profound hypotension, and anterior spinal cord infarction, as well as the diagnostic challenges of recognising spinal cord ischaemia in a patient with a concurrent endocrine emergency. Systemic hypotension is a recognised contributing factor in spinal cord infarction [1,3], whereas published evidence directly examining its association with adrenal crisis remains limited.

Clinical and anatomical correlation

The anterior spinal artery supplies approximately the anterior two-thirds of the spinal cord, including pathways involved in motor function and pain and temperature sensation (Figure *6*) [2,10]. Infarction in the anterior spinal artery territory therefore commonly produces bilateral weakness and loss of pain and temperature sensation, with relative preservation of posterior-column modalities [1,2,10]. The patient's complete bilateral lower-limb weakness, T7 sensory level, and dissociated loss of pain sensation on pinprick testing were consistent with this distribution. However, impaired vibration sense and mixed reflex findings indicated that her examination did not fully conform to the classical syndrome. The initial consideration of deconditioning illustrates how severe focal neurological pathology may be overlooked in the presence of a plausible systemic diagnosis. Although adrenal insufficiency could account for her constitutional symptoms and hypotension, it could not explain the full pattern of neurological deficits. The mixed reflex findings also precluded reliable determination of the stage of spinal shock.

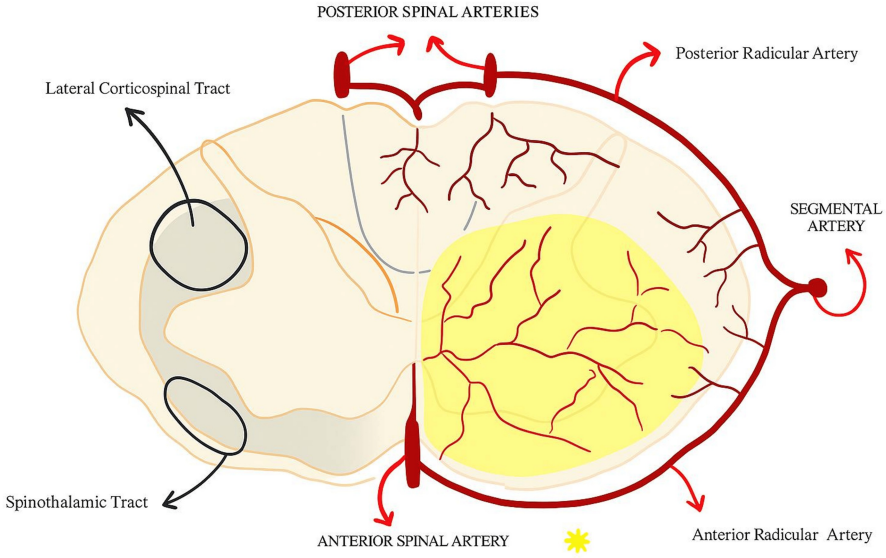


FIGURE 6: Schematic illustration of the anterior spinal artery vascular territory

Schematic cross-section of the spinal cord illustrating the anterior spinal artery territory. Yellow shading represents the approximate supply to the anterior two-thirds of the spinal cord, including regions containing the lateral corticospinal and spinothalamic tracts. The vascular territory is highlighted on one side for illustrative clarity, with relevant pathways indicated on the opposite side. The yellow asterisk is a visual key to the yellow-shaded anterior spinal artery territory, not a separate anatomical structure.

This illustration was drawn by the authors using GoodNotes (Goodnotes Limited, Hong Kong).

Imaging and diagnostic uncertainty

Spinal cord infarction may not demonstrate characteristic abnormalities on early MRI. Studies have shown that follow-up imaging can reveal evolving T2-weighted signal changes, while initially negative scans have also been reported [4,5]. Longitudinal anterior cord hyperintensity and bilateral anterior horn involvement, producing the characteristic “owl’s-eye” appearance on axial imaging, support a lesion in the anterior spinal artery territory [1,2].

Our patient's initial MRI was incomplete due to intolerance. The spinal MRI performed on day 19 was initially reported as showing no acute spinal cord abnormality. However, at the neuroradiology multidisciplinary meeting the following day, specialist review of the same examination led to a reporting addendum identifying anterior spinal cord signal abnormalities extending from C7 to T7, relative sparing of the posterior columns, and the “owl’s-eye” appearance at T1 (Figures *3*-*4*). Diffusion-weighted imaging showed high signal intensity without marked apparent diffusion coefficient reduction. DWI hyperintensity alone can occur in both infarction and inflammatory myelopathy, making apparent diffusion coefficient findings and clinical-radiological correlation important [11]. The “owl’s-eye” appearance is helpful but not specific for infarction [12].

Reports of initially negative MRI findings in hyperacute spinal infarction [4,5] do not, in themselves, explain an examination reported as negative on day 19. In this case, the decisive development was specialist reinterpretation of the existing images, documented in a reporting addendum, rather than the acquisition of new images. The discrepancy between the original report and the multidisciplinary review illustrates the value of specialist neuroradiological reassessment when objective neurological deficits remain unexplained.

Possible endocrine-vascular association

Long-term prednisolone exposure can suppress the hypothalamic-pituitary-adrenal axis, with abrupt withdrawal increasing the risk of adrenal insufficiency and adrenal crisis [6-8]. Severe hypotension and haemodynamic instability are recognised features of adrenal crisis [7,8]. Separately, reduced systemic perfusion is a recognised potential mechanism of spinal cord ischaemia. In a series of 30 cases of spinal cord infarction, Ge et al. attributed three cases to systemic hypotension [3]. Four additional cases of lumbar cord infarction following induced hypotension have also been described, supporting the haemodynamic plausibility of this mechanism in a different clinical context [13].

In this patient, abrupt prednisolone cessation preceded presentation with adrenal insufficiency, profound hypotension (blood pressure of 76/52 mmHg), and severe neurological deficits. A hypotension-mediated reduction in spinal cord perfusion is therefore a biologically plausible explanation for the anterior cord injury. However, the precise onset of infarction relative to the hypotensive episode is unknown, and causation cannot be established solely on the basis of their coexistence. Although CT aortography excluded aortic dissection, it did not establish the underlying vascular mechanism. This case highlights a potentially under-recognised clinical association rather than demonstrating that adrenal crisis directly caused spinal cord infarction.

Management, outcome, and limitations

Treatment combined glucocorticoid replacement with clopidogrel, venous thromboembolism prophylaxis, spinal precautions, and multidisciplinary rehabilitation. As no established therapy reverses completed spinal cord infarction, prevention of complications and functional rehabilitation remain central to management [1,10]. The development of a stage 4 sacral pressure ulcer requiring surgical debridement illustrates the morbidity associated with profound immobility. Limited recovery of toe movement, pain sensation, and reflexes was observed; however, the longer-term neurological outcome remains unknown. Published series describe substantial variability in functional recovery [3].

Limitations of this case include uncertainty regarding the timing and mechanism of ischaemia, incomplete initial imaging, and the absence of cerebrospinal fluid analysis. Because the “owl’s-eye” sign is not pathognomonic [12], inflammatory and other myelopathies remained relevant differential diagnoses. Overall, this case underscores the importance of thorough neurological reassessment and specialist reinterpretation of imaging when severe deficits remain unexplained, while suggesting a plausible relationship between hypotension associated with adrenal insufficiency and spinal cord ischaemia.

Conclusions

This case highlights a potentially under-recognised association between glucocorticoid withdrawal-associated adrenal insufficiency, profound hypotension, and anterior spinal cord infarction. Although a hypotension-mediated mechanism is biologically plausible, the precise timing and cause of the infarction cannot be established. Severe bilateral weakness accompanied by dissociated sensory loss should prompt thorough neurological assessment, even when a concurrent systemic illness offers an alternative explanation for the presentation. An initially negative spinal MRI report should not preclude specialist neuroradiological review when significant neurological deficits remain unexplained. Careful clinical-radiological correlation, reinterpretation of existing imaging, and additional imaging when clinically indicated are essential for establishing the diagnosis. Early multidisciplinary rehabilitation and prevention of complications are equally important in managing the substantial disability associated with spinal cord infarction. Finally, this case reinforces the importance of supervised glucocorticoid tapering and patient education regarding the risks of abrupt glucocorticoid withdrawal and the need for appropriate sick-day management to prevent adrenal insufficiency and adrenal crisis.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Caitlyn Warren, Shayna Swaine, Mohamed Ali

Acquisition, analysis, or interpretation of data: Caitlyn Warren, Shayna Swaine, Evgenia Dagiakidi

Drafting of the manuscript: Caitlyn Warren, Shayna Swaine

Critical review of the manuscript for important intellectual content: Caitlyn Warren, Shayna Swaine, Mohamed Ali, Evgenia Dagiakidi

Supervision: Mohamed Ali

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the

submitted work.

Acknowledgements

Dr Caitlyn Warren and Dr Shayna Swaine contributed equally to the work and should be considered co-first authors. The authors used ChatGPT (OpenAI, San Francisco, CA, USA) to assist with manuscript organisation, language editing, reference formatting and preparation for submission. All clinical information, scientific interpretations, references and final manuscript content were reviewed and verified by the authors, who take full responsibility for the accuracy and integrity of the work.

References

1. Dokponou YC, Ontsi Obame FL, Takoutsing B, et al.: Spinal cord infarction: a systematic review and meta-analysis of patient's characteristics, diagnosis accuracy, management, and outcome. Surg Neurol Int. 2024, 15:325. 10.25259/SNI_477_2024
2. McCarty J, Chung C, Samant R, et al.: Vascular pathologic conditions in and around the spinal cord . Radiographics. 2024, 44:240055. 10.1148/rg.240055
3. Ge L, Arul K, Stoner M, Mesfin A: Etiology and outcomes of spinal cord infarct: a case series from a level 1 trauma center. Global Spine J. 2020, 10:735-40. 10.1177/2192568219877863
4. Kim BR, Park KS, Kim HJ, Kim JY, Kim BR, Lee E, Lee JW: Features of non-traumatic spinal cord infarction on MRI: changes over time. PLoS One. 2022, 17:0274821. 10.1371/journal.pone.0274821
5. Costamagna G, Meneri M, Abati E, et al.: Hyperacute extensive spinal cord infarction and negative spine magnetic resonance imaging: a case report and review of the literature. Medicine (Baltimore). 2020, 99:22900. 10.1097/MD.0000000000022900
6. Broersen LH, Pereira AM, Jørgensen JO, Dekkers OM: Adrenal insufficiency in corticosteroids use: systematic review and meta-analysis. J Clin Endocrinol Metab. 2015, 100:2171-80. 10.1210/jc.2015-1218
7. Beuschlein F, Else T, Bancos I, et al.: European Society of Endocrinology and Endocrine Society joint clinical guideline: diagnosis and therapy of glucocorticoid-induced adrenal insufficiency. J Clin Endocrinol Metab. 2024, 109:1657-83. 10.1210/clinem/dgae250
8. Elshimy G, Chippa V, Kaur J, Jeong JM: Adrenal crisis. StatPearls [Internet]. StatPearls Publishing, Treasure Island (FL); 2025.
9. Medical Research Council: Aids to the Examination of the Peripheral Nervous System. Medical Research Council (ed): Her Majesty's Stationery Office, London, UK; 1943. http://www.ukri.org/publications/aids-to-the-examination-of-the-peripheral-nervous-system/.
10. Sandoval JI, De Jesus O: Anterior spinal artery syndrome. StatPearls [Internet]. StatPearls Publishing, Treasure Island (FL); 2026.
11. Kobayashi M: The utility of diffusion-weighted imaging in patients with spinal cord infarction: difference from the findings of neuromyelitis optica spectrum disorder. BMC Neurol. 2022, 22:382. 10.1186/s12883-022-02903-y
12. Zalewski NL, Rabinstein AA, Krecke KN, et al.: Characteristics of spontaneous spinal cord infarction and proposed diagnostic criteria. JAMA Neurol. 2019, 76:56-63. 10.1001/jamaneurol.2018.2734
13. Singh U, Silver JR, Welply NC: Hypotensive infarction of the spinal cord . Paraplegia. 1994, 32:314-22. 10.1038/sc.1994.54