Gac Méd Caracas 2026;134(2):624-631
DOI: 10.47307/GMC.2026.134.2.24

Spinal cord infarction due to Adamkiewicz artery involvement: When thinking the worst leads to the right diagnosis

Infarto espinal por compromiso de la arteria de Adamkiewicz: cuando pensar mal te lleva al diagnóstico correcto

Santiago Andrés Vintimilla-Pesántez[1a*], Fernando Patricio Villa Infante[2c], Karla Elizabeth Escobar Bustamante[3c], Estefani Franchesca Villa Orozco[4c], Rita Teresa Mera Jarrín[5c], Marco Alexander Vásquez Poveda[6c], Luis Alfredo Masabanda Campaña[7cd], José Rodolfo Molina Melendres[8bce]

SUMMARY

Background: *Anterior spinal artery syndrome is an infrequent cause of acute ischemic myelopathy, often caused by aortic pathology involving the artery of Adamkiewicz. This case is presented due to its rarity and the complex decision-making required for treatment in a patient with multiple comorbidities.* **Case Presentation**: *We present a 79-year-old male with hypertension and ischemic heart disease admitted for progressive lower limb weakness and sphincter dysfunction. MRI revealed anterior spinal cord infarction (T9–L1). CT angiography showed a Stanford type B (DeBakey IIIb) aortic dissection extending to the infrarenal segment with a fusiform aneurysm and mural thrombus, likely compromising the artery of Adamkiewicz. Due to high surgical risk and unfavorable vascular anatomy, a multidisciplinary team opted for conservative management.* **Conclusions**: *Vascular involvement of the artery of Adamkiewicz must be considered in patients with acute myelopathy and cardiovascular risk. Conservative management combined with rehabilitation can be a reasonable alternative in patients with complex anatomy and high surgical risk, although the functional prognosis remains reserved.*

Keywords: *Spinal cord infarction, Adamkiewicz artery, aortic dissection, paraparesis, anterior spinal artery syndrome, conservative management*

ORCID:
[1] 0000-0003-1450-6128
[2] 0009-0006-6009-3722
[3] 0000-0001-5871-4100
[4] 0009-0006-9701-4717
[5] 0009-0000-9676-7457
[6] 0000-0002-5559-0525
[7] 0009-0009-0121-7387
[8] 0009-0008-9081-648X

[*]**Autor de correspondencia:** Santiago Andrés Vintimilla-Pesántez. Address: Neurology Department, Hospital de las Fuerzas Armadas HE1. Quito - Ecuador. Av. Gran Colombia S/N y Queseras del Medio. Telephone number: (+593) 994147561. E-mail: santiago.vintimilla.pesantez@udla.edu.ec

Recibido: 23 de marzo 2026
Aceptado: 26 de abril 2026

RESUMEN

Antecedentes: *El síndrome de la arteria espinal anterior es una causa poco frecuente de mielopatía isquémica aguda, a menudo provocada por una patología aórtica que compromete la arteria de Adamkiewicz. Se presenta este caso por su rareza y la complejidad de la toma de decisiones que conlleva el tratamiento de un paciente con múltiples comorbilidades.* **Presentación**

a. Universidad Politécnica Salesiana. Quito - Ecuador.
b. Posgrado de Neurología. Universidad de las Américas. Quito - Ecuador.
c. Hospital de Especialidades de las Fuerzas Armadas. Quito- Ecuador.
d. Pontificia Universidad Católica del Ecuador. Quito- Ecuador.
e. Universidad de las Fuerzas Armadas – ESPE. Quito- Ecuador.

del caso: *Presentamos el caso de un varón de 79 años, con hipertensión y cardiopatía isquémica, ingresado por debilidad progresiva de las extremidades inferiores y disfunción esfínteriana. La resonancia magnética reveló un infarto de la médula espinal anterior (T9–L1). La angiografía por tomografía computarizada mostró una disección aórtica tipo B de Stanford (DeBakey IIIb) con extensión al segmento infrarrenal, con un aneurisma fusiforme y un trombo mural que probablemente comprometía la arteria de Adamkiewicz. Debido al alto riesgo quirúrgico y a la anatomía vascular desfavorable, un equipo multidisciplinario optó por un manejo conservador.* **Conclusiones**: *Se debe considerar la afectación vascular de la arteria de Adamkiewicz en pacientes con mielopatía aguda y riesgo cardiovascular. El tratamiento conservador, combinado con rehabilitación, puede ser una alternativa razonable en pacientes con anatomía compleja y alto riesgo quirúrgico, aunque el pronóstico funcional sigue siendo reservado.*

Palabras clave: *Infarto de la médula espinal, arteria de Adamkiewicz, disección aórtica, paraparesia, síndrome de la arteria espinal anterior, tratamiento conservador*

Background

The blood supply to the spinal cord depends on a complex vascular network, consisting mainly of three longitudinal arteries: one anterior spinal artery, which supplies the anterior two-thirds of the spinal cord, and two posterolateral spinal arteries, responsible for the posterior third. The anterior spinal artery, which originates from the vertebral arteries, receives segmental contributions along the aorta; notable among these is the arteria radicularis magna, or artery of Adamkiewicz. This artery, with a caliber of 0.6-1.8 mm, most frequently originates between T8 and L1 on the left side and is primarily responsible for irrigating the thoracolumbar region of the spinal cord (1–3).

Anterior spinal artery syndrome is an infrequent entity, responsible for 5% to 8% of acute vascular myelopathies, representing up to 87.2% of spinal cord infarction cases. The average age of presentation is between 50 and 70 years, with no predilection for sex, although some studies report a worse prognosis in women (4).

Pathophysiologically, it may be due to direct mechanisms such as spinal cord compression by hematomas, or more frequent indirect mechanisms, such as ischemia secondary to arterial occlusion or hypoperfusion. Common causes include aortic surgical procedures, aortic dissection, atherosclerosis, cardiac emboli, vasculitis, spinal trauma, fibrocartilaginous embolic myelopathy, hypercoagulable states, cocaine use, and anemias such as sickle cell disease (5–7).

Clinically, it presents with acute radicular pain, loss of motor strength, hyporeflexia or areflexia, and sensory dissociation with loss of thermoalgesic sensitivity and relative preservation of proprioception and vibratory sensitivity (8,9). Given its infrequent nature and the initial nonspecificity of the clinical picture, diagnosis is often delayed. The objective of this case report is to describe a patient with spinal cord infarction secondary to compromise of the artery of Adamkiewicz in the context of an aortic dissection, and to discuss the rationale for conservative management in a high-surgical-risk patient with complex vascular anatomy.

Case Report

A 79-year-old male patient with a personal history of long-standing arterial hypertension with irregular treatment, ischemic heart disease with coronary stent placement in 2011, stage 3A chronic kidney disease, hyperuricemia, and known allergy to penicillin, presented to the emergency department. He reported a clinical picture with approximately 3 days of evolution, characterized by progressive weakness in the lower limbs, profuse diaphoresis, difficulty walking, postural instability, and progressive alteration in sphincter control. The onset occurred following a change in position when going to the bathroom, without evident trauma or falls.

He was initially evaluated by the orthopedics service, which ruled out musculoskeletal lesions or spinal compression due to degenerative pathology and indicated outpatient management. However, due to the persistence and progression of neurological symptoms, he was readmitted to the emergency department two days later with

flaccid paralysis of the lower limbs. On admission, the patient was hemodynamically stable but with mild hypoxemia: blood pressure 146/80 mmHg, heart rate 87 bpm, respiratory rate 19 breaths/min, oxygen saturation 92% on room air, and temperature 36.7 °C.

Upon admission, the neurological physical examination revealed bilateral flaccid paraplegia, areflexia in the lower limbs, anal sphincter atonia, thermoalgesic anesthesia with a sensory level located at T11-T12, and preservation of vibration and joint position sense, suggesting selective involvement of the anterolateral spinal system with preservation of the posterior columns. Motor strength in the upper limbs was normal, with preserved deep tendon reflexes and no signs of cervical myelopathy. Based on these findings, the patient's neurological status at admission was consistent with ASIA Impairment Scale (AIS) grade A, with a neurological level of injury at T10–T11.

Laboratory tests showed elevated creatinine (2.1 mg/dL), urea (89 mg/dL), and a slight elevation in D-dimer, without thrombocytopenia or signs of disseminated intravascular coagulation.

Magnetic resonance imaging (MRI) of the thoracic and lumbar spine was performed in both simple and contrast-enhanced sequences, showing a hyperintense signal on T2 from the T9 to L1 levels, predominantly in the anterior region of the spinal cord. Restriction to diffusion was evident (Figure 1), along with an absence of enhancement on T1 post-contrast sequences, resulting in the characteristic "owl's eye sign" in the axial view, highly suggestive of anterior spinal cord infarction (Figure 2). Likewise, signs of spinal cord edema and subacute ischemic changes were identified, with signal alterations in adjacent vertebral bodies, especially in the right margins of T11 and T12; additionally, a double lumen was evident in the abdominal aorta (Figure 3).

A contrast-enhanced thoracoabdomino-pelvic computed tomography angiography (CTA) evidenced a Stanford type B (DeBakey IIIb) aortic dissection extending to the infrarenal segment, with a fusiform aneurysm of 5 cm in maximum diameter, an intimal flap (Figure 4) extending from the level of the renal arteries to the common iliac bifurcation (Figure 5), partially organized mural thrombus, and complete occlusion of the left renal artery. The right renal artery presented significant stenosis.

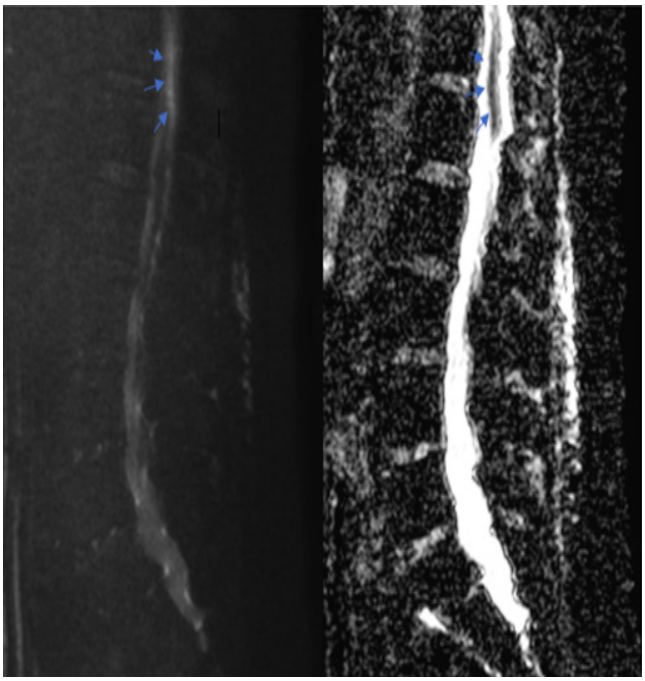

Figure 1. Lunbosacral magnetic resonance imaging: sagittal DWI and ADC sequences showing hyperintensity in the anterior portion of the spinal cord in DWI correlating with hypointensity in ADC (blue arrows) at segments T12 and L5, evidencing spinal cord infarction.

Due to a sustained hypertensive crisis of 190/110 mmHg, the patient was admitted to the intensive care unit for three days for intravenous antihypertensive management, with a good response. He was evaluated by multiple specialties: Cardiology (adjustment of antihypertensive treatment and comorbidity control), Nephrology (monitoring of renal function), Vascular Surgery (ruled out surgical intervention due to unfavorable vascular anatomy and high operative risk), Urology (management of urinary retention with bladder catheterization), and Physiatry (initiated rehabilitation plan).

Evolution showed limited recovery, with minimal recovery of strength in the bilateral psoas (2/5) and right foot extensors (2/5), and flaccid

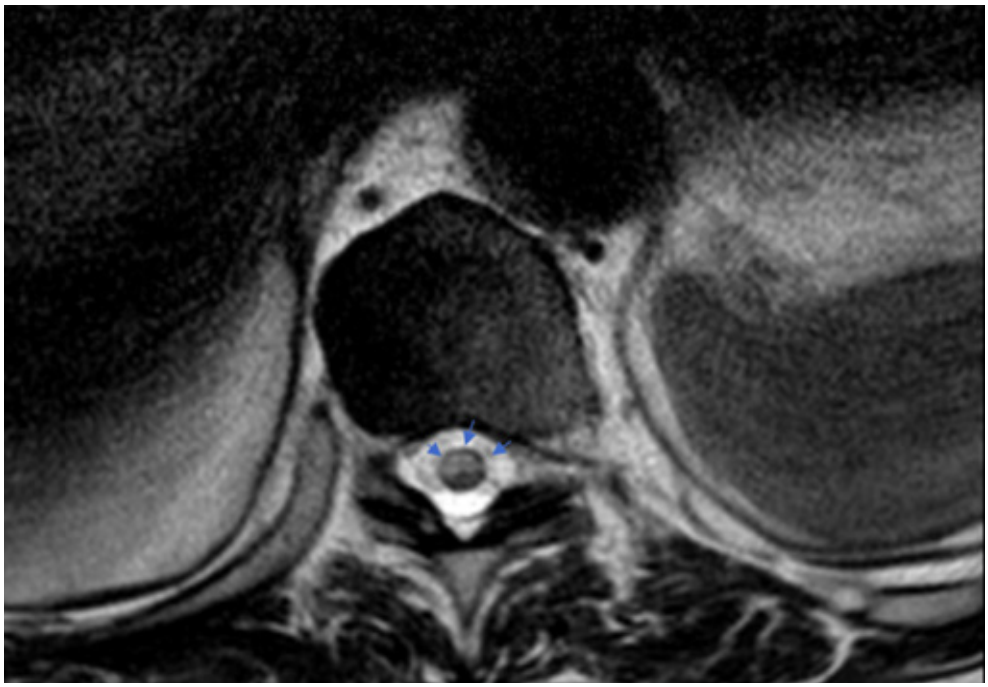

Figure 2. Thoracic magnetic resonance imaging: T2-weighted axial sequences showing hyperintensity in the anterior portion of the spinal cord (blue arrows) at the T11 segment, consistent with spinal cord infarction.

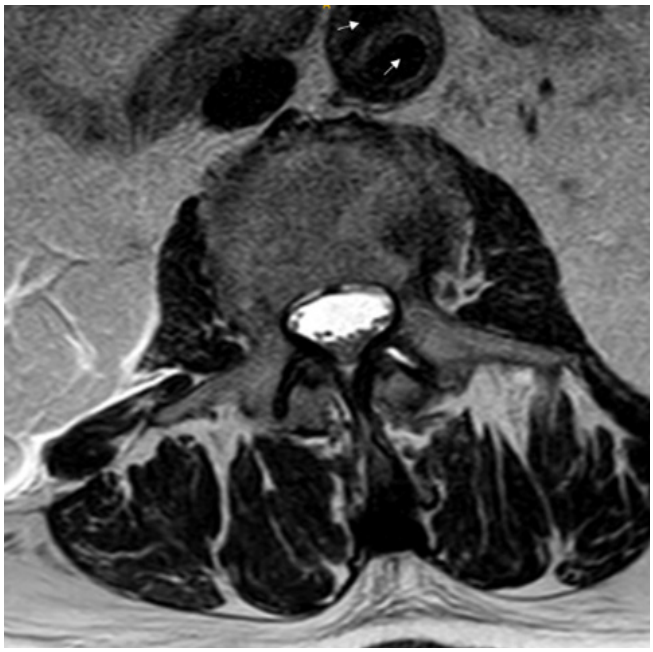

Figure 3. Thoracic magnetic resonance imaging: T2-weighted axial sequences showing hyperintensity in the anterior portion of the spinal cord (blue arrows) at the T11 segment, consistent with spinal cord infarction.

paraparesis remained a severe functional deficit. Thermoalgesic sensitivity remained altered below T10, with bladder dysautonomia (urinary retention) and compromise of anal sphincter control.

Due to the persistence of neurological deterioration and the presence of multiple comorbidities limiting the possibility of a surgical intervention with an adequate risk-benefit ratio, the multidisciplinary team decided to continue with conservative management using acetylsalicylic acid 100 mg daily and atorvastatin 20 mg daily, in addition to motor physical rehabilitation and nutritional support.

The patient was discharged after 18 days of hospitalization, with outpatient follow-up by Neurology, Vascular Surgery, and Physiatry. At discharge, he presented flaccid paraparesis with strength 1-2/5 in the right lower limb and 0-1/5 in the left lower limb, without sphincter control and with functional dependence for basic activities of daily living (Barthel Index of 40).

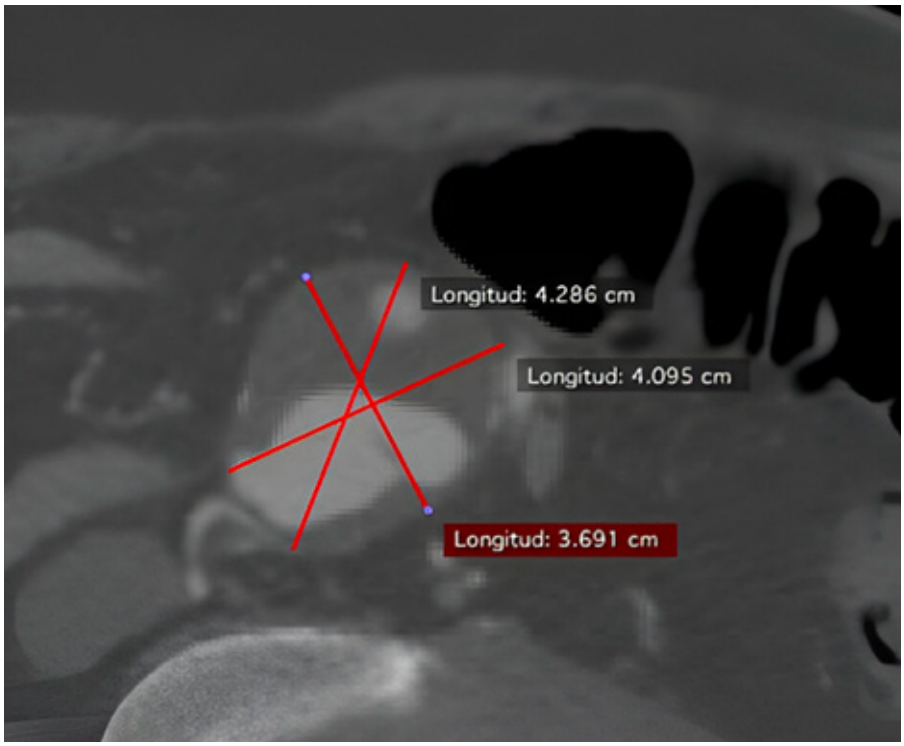

Figure 4. Abdominal computed tomography angiography: Axial view showing aneurysmal dilatation (red lines) with an intimal flap indicative of aortic dissection in the abdominal aorta.

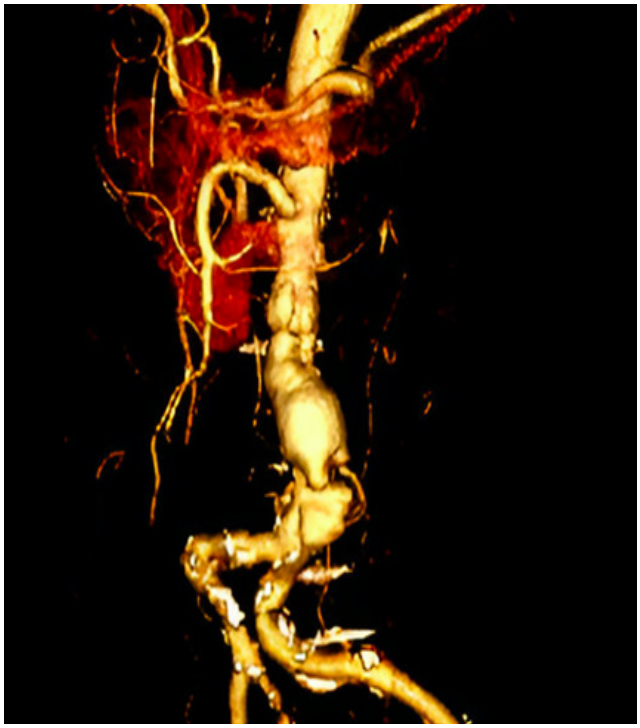

Figure 5. Computed tomography angiography, 3D reconstruction of the aorta: Anterior view showing the start of aneurysmal dilatation below the origin of the renal arteries, measuring up to 37 mm in caliber x 76 mm in length.

DISCUSSION

The artery of Adamkiewicz, also known as the great anterior radiculomedullary artery, represents the main segmental supply to the anterior spinal artery in the thoracolumbar region (1–3). Its origin is highly variable, usually between levels T8 and L1, and it originates more frequently from the left side, from intercostal or lumbar branches (10,11). This artery plays a critical role in spinal cord irrigation; therefore, its obstruction can trigger extensive spinal cord infarction with severe functional consequences.

In the present case, the patient presented a sudden onset of neurological symptoms which, together with imaging findings, were compatible with spinal cord infarction in the territory dependent on the artery of Adamkiewicz. Causes described in the literature for this type of event include aortic dissection, atherosclerosis, fibrocartilaginous embolism, vasculitis, severe hypotension, and vertebral artery dissection (12). Clinically, it usually presents flaccid or spastic

paralysis, loss of thermoalgesic sensitivity with preservation of deep sensitivity, and autonomic dysfunction (12).

This patient had several relevant risk factors, such as uncontrolled arterial hypertension, ischemic heart disease, and chronic kidney disease. The onset of symptoms following a sudden change in position suggests a hemodynamic trigger in the context of a previously undiagnosed aortic dissection. This hypothesis is confirmed by tomographic findings, which evidenced a Stanford type B (DeBakey IIIb) dissection accompanied by a fusiform infrarenal aneurysm with mural thrombus and an intimal flap extending from the renal arteries to the iliac bifurcation.

The association between aortic dissections and Adamkiewicz artery involvement has been previously described by Altuwaijri et al. (13) and MacGillivray et al. (14), who reported intermittent flow obstruction from a false lumen as a cause of spinal cord ischemia. Similarly, in the context of aortic aneurysms, studies such as those by Yogendranathan et al. (15) and Lee et al. (16) have documented spinal cord infarctions due to thrombotic mural compromise in non-dissecting thoracic and non-ruptured abdominal aneurysms, respectively.

From a diagnostic standpoint, evaluating the involvement of the artery of Adamkiewicz remains a challenge. Computed tomography angiography (CTA), although widely used, may not accurately distinguish between the artery and the anterior radiculomedullary vein due to their morphological similarities. Magnetic resonance angiography (MRA) offers a non-invasive alternative, although its applicability may be limited by factors such as claustrophobia or the presence of metallic devices. Digital subtraction angiography (DSA) is considered the gold standard, but its use is limited by its invasive nature and associated risks (17). Despite these limitations, MRI remains fundamental for identifying the compromised vascular territory and estimating the extent of the infarction (18).

The prognosis of anterior spinal cord infarction depends largely on diagnostic precocity and the early institution of therapeutic measures. The initial severity of the motor deficit and the presence of autonomic dysfunction are associated with worse functional evolution. Follow-up imaging findings, such as persistent T2 hyperintensity and spinal cord atrophy, also correlate with a guarded prognosis (18,19). In our case, clinical evolution showed mild improvement during hospitalization. At the time of discharge, the patient presented with flaccid paraparesis and persistence of sphincter dysfunction, reflecting a severe spinal cord lesion without significant functional recovery.

Regarding the therapeutic approach, conservative treatment is generally preferred in patients with high surgical risk or complex vascular anatomies. Aguilar-Salinas et al. (20) state that close observation, neuromonitoring, and the use of steroids can lead to progressive neurological improvement. The 2024 joint guidelines of the European Association for Cardio Thoracic Surgery (EACTS) and the Society of Thoracic Surgeons (STS) recommend conservative management for uncomplicated type B aortic dissections, based on strict control of systolic blood pressure below 120 mmHg. Surgical or endovascular approaches should be reserved for cases with signs of malperfusion, spinal cord ischemia, aneurysmal rupture, or accelerated aneurysm expansion (21).

In accordance with these recommendations, conservative management with acetylsalicylic acid and moderate-potency statins (atorvastatin) was decided for this patient due to multiple comorbidities, deterioration of renal function, and the presence of a “shaggy aorta” with extensive abdominal compromise. This strategy sought to avoid additional complications associated with an invasive intervention that would likely not have represented a net benefit to the patient.

From a pathophysiological standpoint, spinal cord perfusion follows principles analogous to

cerebral perfusion: spinal cord perfusion pressure (SCPP) is defined as the difference between mean arterial pressure (MAP) and intrathecal pressure, mirroring the relationship between MAP and intracranial pressure that determines cerebral perfusion pressure (22). Unlike the brain, however, the spinal cord has a sparse collateral network and relies heavily on a small number of segmental feeders—chiefly the artery of Adamkiewicz—which renders it particularly vulnerable to even brief episodes of hypotension. Loss of autoregulation at the injury site further amplifies this susceptibility, so that minor drops in MAP can propagate secondary ischemic injury (22,23). Current guidelines on acute spinal cord injury recommend avoiding systolic blood pressure below 90 mmHg and maintaining MAP within a target range of 75–80 to 90–95 mmHg for the first 3–7 days (22). In the context of aortic dissection involving the artery of Adamkiewicz, this creates therapeutic tension: aggressive blood-pressure lowering to control aortic wall stress may further compromise already marginal spinal cord perfusion, whereas permissive hypertension risks dissection propagation (23,24). In our patient, this dilemma was managed in the intensive care unit with individualized hemodynamic targets, prioritizing avoidance of hypotension while controlling sustained systolic peaks; this strategy parallels the multimodal perfusion-oriented approach advocated in recent reviews of spinal cord protection during aortic surgery (23,24).

CONCLUSION

Vascular involvement of the artery of Adamkiewicz, although infrequent, must be considered a cause of spinal cord infarction in patients with cardiovascular risk factors and acute neurological symptoms; conservative management may be the most appropriate option in the presence of multiple comorbidities. Spinal cord infarction secondary to aortic dissection with involvement of the artery of Adamkiewicz represents a diagnostic and therapeutic challenge due to its low frequency and high morbidity. Timely identification via MRI and CTA is fundamental; however, in patients with complex vascular anatomy and high surgical risk, conservative treatment combined with rehabilitation offers a reasonable alternative, albeit with a guarded functional prognosis.

Institutional Review Board Statement: Ethical review and approval were waived for this study due to the retrospective nature of the case report, which poses no risk to the participant.

Informed Consent Statement: Written informed consent has been obtained from the patient and his son to publish this paper.

Data Availability Statement: Data sharing is not applicable to this article as no new datasets were generated or analyzed during the current study.

Funding Declaration: There is no funding for the publication of this case report.

Consent to Publish declaration: All authors consent to the publication of this case report.

Ethics and Consent to Participate declarations: We have the informed consent for publishing this case report, signed by the patient and his son.

REFERENCES

1. Lindeire S, Hauser J. Anatomy, Back, Artery of Adamkiewicz. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2025. Available from: https://www.ncbi.nlm.nih.gov/books/NBK532971/
2. N'da HA, Chenin L, Capel C, Havet E, Le Gars D, Peltier J. Microsurgical anatomy of the Adamkiewicz artery–anterior spinal artery junction. Surg Radiol Anat. 2016;38:563–7.
3. Guziński M, Bryl M, Ziemińska K, Wolny K, Sąsiadek M, Garcarek JS. Detection of the Adamkiewicz artery in computed tomography of the thorax & abdomen. Adv Clin Exp Med. 2017;26:31–7.
4. Sandoval J, De Jesús O. Anterior Spinal Artery Syndrome. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2025. Available from: https://www.ncbi.nlm.nih.gov/books/NBK560731/

5. Rahman M, Rahman S, Siddik AB, Chowdhury FH, Hossain SS, Rahman MM. Review on the pathophysiology and management of anterior spinal artery syndrome. J Spine Res Surg. 2020;2:1–5.
6. Pearl N, Weisbrod M, Dubensky L. Anterior Cord Syndrome. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2025. Available from: https://www.ncbi.nlm.nih.gov/books/NBK559117/
7. Gupta M, Chhabra HS. Nucleus pulposus embolism causing anterior spinal artery occlusion: A rare but possible cause of fibrocartilaginous embolic myelopathy. Int J Spine Surg. 2020;14:391–6.
8. Santana J, Dalal K. Ventral Cord Syndrome. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2025. Available from: https://www.ncbi.nlm.nih.gov/books/NBK541011/
9. Weidauer S, Nichtweiß M, Hattingen E, Berkefeld J. Spinal cord ischemia: aetiology, clinical syndromes and imaging features. Neuroradiology. 2015;57:241–57.
10. Taterra D, Skinningsrud B, Pękala PA, Henry BM, Vikse J, Hsieh WC, et al. Artery of Adamkiewicz: a meta-analysis of anatomical characteristics. Neuroradiology. 2019;61:869–80.
11. Alvernia JE, Simon E, Khandelwal K, Lanzino G, Rabinstein AA, Brinjikji W, et al. Anatomical study of the thoracolumbar radiculomedullary arteries, including the Adamkiewicz artery and supporting radiculomedullary arteries. J Neurosurg Spine. 2022;38:233–41.
12. Yadav N, Pendharkar H, Kulkarni GB. Spinal cord infarction: clinical and radiological features. J Stroke Cerebrovasc Dis. 2018;27:2810–21.
13. MacGillivray TE, Gleason TG, Patel HJ, Aldea GS, Bavaria JE, Beaver TM, et al. The Society of Thoracic Surgeons/American Association for Thoracic Surgery clinical practice guidelines on the management of type B aortic dissection. Ann Thorac Surg. 2022;113:1073–92.
14. Altuwaijri M, Delis KT, Vrtiska TJ, Gloviczki P, Bower TC, Noel AA, et al. Aortic fenestration for chronic aortic dissection type B complicated by transient ischemic attacks of spinal cord. J Vasc Surg. 2006;44:186–93.
15. Yogendranathan N, Herath HMMTB, Jayamali WD, Kulatunga A, Kulatunga P, Ranasinghe P, et al. A case of anterior spinal cord syndrome in a patient with unruptured thoracic aortic aneurysm with a mural thrombus. BMC Cardiovasc Disord. 2018;18:48.
16. Lee H, Papanagnou D, Berman M, Zhang XC. Man with sudden paralysis: insidious spinal cord infarction due to a non-ruptured abdominal aortic aneurysm. J Emerg Med. 2019;56:413–6.
17. Bonnet B, Kobeiter H, Pescatori L, Maurel B, Ben Abdallah I, Becquemin JP, et al. Preoperative spinal arterial supply mapping using non-selective cone beam computed tomography before complex aortic repair. J Clin Med. 2024;13:796.
18. Yasuda N, Kuroda Y, Ito T, Matsumoto H, Takahashi T, Kato M, et al. Postoperative spinal cord ischaemia: magnetic resonance imaging and clinical features. Eur J Cardiothorac Surg. 2021;60:164–74.
19. Furukawa K, Kamohara K, Nojiri J, Okada K, Inoue T, Totsugawa T, et al. Operative strategy for descending and thoracoabdominal aneurysm repair with preoperative demonstration of the Adamkiewicz artery. Ann Thorac Surg. 2010;90:1840–6.
20. Aguilar-Salinas P, Lima J, Brasiliense LBC, Hanel RA, Sauvageau E, Binning MJ, et al. Ruptured aneurysm of the artery of Adamkiewicz: is conservative management the standard of treatment in the current era? J Neurointerv Surg. 2018;10:e22.
21. Czerny M, Grabenwöger M, Grimm M, Etz CD, Kari FA, Kondov S, et al. EACTS/STS guidelines for diagnosing and treating acute and chronic syndromes of the aortic organ. Ann Thorac Surg. 2024;118:5–115.
22. Kwon BK, Tetreault LA, Martin AR, Arnold PM, Marco RAW, Newcombe VFJ, et al. A clinical practice guideline for the management of patients with acute spinal cord injury: recommendations on hemodynamic management. Global Spine J. 2024;14(3_suppl):187S–211S.
23. Torre DE, Pirri C. Spinal cord protection in open and endovascular aortic surgery: current strategies, controversies and future directions. Front Cardiovasc Med. 2025;12:1671350.
24. Brotis AG, Kalogeras A, Bareka M, Arnaoutoglou E, Spanos K, Matsagkas M, et al. Prevention and management of spinal cord ischemia after aortic surgery: an umbrella review. Brain Sci. 2025;15(4):409.