Case Resolution

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Acute Spinal Cord Infarction after Spinal Surgery

ABSTRACT
Spinal cord infarction is an infrequent neurosurgical complication but is associated with extremely high morbidity. We report the case of a 68-year-old man with multiple cardiovascular and oncological comorbidities (active lung and prostate cancer) who developed rapidly progressive acute paraplegia following percutaneous bone biopsy, percutaneous fixation, and bipedicular kyphoplasty at L1. Although computed tomography ruled out mechanical causes and cement leakage, magnetic resonance imaging confirmed spinal cord ischemia extending from T9 to L4. This report analyzes the multifactorial etiology of the event, highlighting the interaction between paraneoplastic hypercoagulability and the surgical technique as key factors to be considered during preoperative planning.
Keywords: Spinal cord infarction; ischemic stroke; ischemia; kyphoplasty; paraplegia.
Level of Evidence: IV

Infarto medular agudo después de una cirugía espinal

RESUMEN
El infarto medular es una complicación neuroquirúrgica infrecuente, pero con una morbilidad extremadamente alta. Se presenta el caso de un hombre de 68 años con múltiples comorbilidades oncológicas (cánceres de pulmón y próstata en actividad) y cardiovasculares que desarrolló una paraplejía aguda rápidamente progresiva, tras una biopsia ósea por punción, fijación percutánea y cifoplastia bipedicular en L1. A pesar que, con la tomografía computarizada, se descartaron causas mecánicas o fuga de cemento, la resonancia magnética confirmó una isquemia medular desde T9 a L4. Este reporte analiza la etiología multifactorial del evento, destacando la interacción entre el estado de hipercoagulabilidad paraneoplásica y la técnica quirúrgica, como puntos clave por tener en cuenta en la planificación prequirúrgica.
Palabras clave: Infarto medular; accidente cerebrovascular; isquemia; cifoplastia; paraplejía.
Nivel de Evidencia: IV

DIAGNOSIS: Acute spinal cord infarction after spinal surgery.

DISCUSSION

Forty-eight hours after the initial surgery, an emergency spinal magnetic resonance imaging study revealed non-compressive intramedullary hyperintensity consistent with extensive spinal cord ischemia extending from T9 to L4 (Figure 3). The condition was considered a spinal cord injury not amenable to surgical management; therefore, anticoagulation therapy was restarted and the patient was referred to an intensive rehabilitation center.

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How to cite this article: Bazán PL, Pérez Gutiérrez A, Garay AL. Postgraduate Orthopedic Instruction – Imaging. Case Resolution. *Rev Asoc Argent Ortop Traumatol* 2026;91(1):73-76. https://doi.org/10.15417/issn.1852-7434.2026.91.1.2296

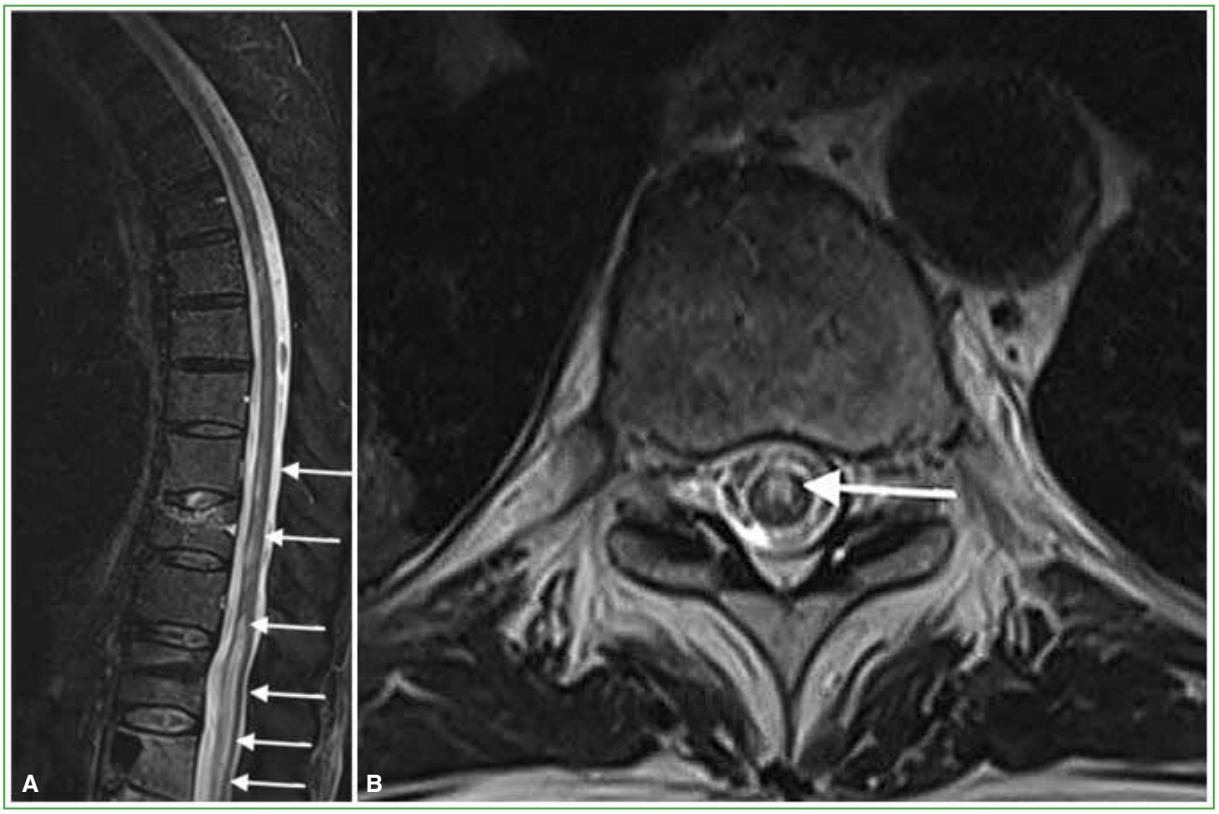

Figure 3. Postoperative magnetic resonance imaging of the spine showing a non-compressive intramedullary hyperintense signal, consistent with vascular injury. **A.** Mid-sagittal T2-weighted sequence. **B.** Axial T2-weighted sequence.

Spinal cord infarction accounts for approximately 0.3%–1% of all ischemic events affecting the central nervous system.[1] Unlike cerebral stroke, the diagnosis of acute spinal cord ischemia represents a significant clinical challenge due to its heterogeneous presentation and the fact that imaging studies may be normal during the hyperacute phase.[2]

In the context of spinal surgery, certain procedures, such as vertebroplasty and kyphoplasty, have been reported as potential iatrogenic causes of spinal cord ischemia, mainly through embolic phenomena or local hemodynamic alterations.

Spinal cord ischemia can be classified as spontaneous or periprocedural.[2,3] In the periprocedural setting, multiple etiopathogenic factors may converge:

Kyphoplasty-related mechanisms: These may be direct or indirect. A direct mechanism includes intracanal cement migration (Figure 4), which may injure neural structures through mass effect or thermal damage. A critical increase in intravertebral pressure has also been described, particularly in bipedicular techniques.[4] Indirect mechanisms include arterial embolism caused by cement microparticles occluding the anterior spinal artery or the artery of Adamkiewicz, congestion of the Batson venous plexus, and thermal injury related to cement polymerization.[5] In our patient, postoperative imaging ruled out cement leakage into the spinal canal (Figure 4).

Figure 4. Computed tomography of a lumbar vertebra, axial view. This is an illustrative case, distinct from the case presented, showing intracanal cement migration (white arrows) with spinal canal involvement.

Oncological prothrombotic state: Patients with active malignancy present a chronic hypercoagulable condition. Discontinuation of rivaroxaban may generate a "rebound effect" with a transient increase in thrombin activity; when combined with the release of tissue thromboplastin during bone manipulation, this facilitates in situ thrombosis of radiculomedullary arteries.[6,7] In this case, the patient was receiving rivaroxaban, whose suspension was managed by the appropriate service, and was under active follow-up for two malignancies.

Hemodynamic compromise: Chronic pericardial effusion limits cardiac reserve.[8] Episodes of perioperative hypotension may lead to infarction in spinal cord "watershed zones," where vascular supply is particularly vulnerable—especially between T4 and T9.[6] The image of intramedullary injury, in our case, began at T9.

Fat and tumor embolism: The pressure exerted by kyphoplasty balloons may force fat or tumor debris into the epidural venous circulation, resulting in venous spinal cord ischemia due to impaired venous outflow.[9]

Postoperative spinal cord ischemia is frequently a diagnosis of exclusion, in which clinical findings outweigh initial imaging results.[4] The absence of abnormalities on computed tomography ruled out direct mechanical compression from cement leakage or bone displacement, but not systemic vascular insufficiency. In oncological patients, the risk is not solely technical but also systemic, as vascular compromise results from the combination of reduced cardiac output and a prothrombotic state exacerbated by anticoagulation withdrawal.[10,11]A thorough evaluation of patients with active cancer or those receiving oral anticoagulants is essential to reduce the risk of this adverse event. Preventive strategies may include the use of a unipedicular technique, modification or bridging of anticoagulation therapy, among others.

Optimal spinal cord perfusion should be prioritized by maintaining a mean arterial pressure greater than 85 mmHg.

In the presence of any sudden neurological deficit following kyphoplasty, urgent magnetic resonance imaging with diffusion-weighted sequences is mandatory, as this is the most sensitive modality for detecting restricted diffusion associated with spinal cord infarction during the hyperacute phase.

Conflict of interest: The authors declare no conflicts of interest.

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