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Case Report

Spinal cord watershed infarction after surgery ☆

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ABSTRACT

Spinal cord watershed infarction is a rare phenomenon due to the rich collateral blood supply to the organ. It often occurs in elderly patients with preexisting atherosclerotic disease in the setting of global hypoperfusion, such as thoracoabdominal surgery, dissection, coagulopathies, or idiopathic. We present a case of spinal cord infarction (SCI) in both longitudinal and transverse watershed areas as a complication of postoperative blood loss. In addition, we demonstrate the imaging evolution of spinal cord infarct in the subacute phase with peripheral enhancement due to the breakdown of the blood-spinal cord barrier.

Introduction

SCI is a rare diagnosis due to the spinal cord's rich arterial supplies, accounting for only 1.2% of all strokes [1]. The extensive anastomoses between the anterior spinal artery, posterior spinal arteries, and multiple segmental arteries make spinal cord watershed infarct exceedingly rare. Symptoms of SCI are highly dependent on the location of the lesion as well as the severity of the infarct. Watershed zone infarct tends to involve the gray matter, which can lead to flaccid paralysis and areflexia in the acute phase [2]. Prognosis is often poor and involves only supportive treatment, including fluid resuscitation and maintaining a mean arterial pressure above 90 mmHg [1].

We present a case of spinal cord watershed infarction in the longitudinal and transverse zones due to postoperative blood loss and prolonged effect of thoracic epidural anesthesia with classic imaging features in the acute phase and subacute phase.

Case report

A 59-year-old female with a history of diabetes, hypertension, and uterine fibroids presented to the hospital for elective hysterectomy, bilateral salpingo-oophorectomies, and ventral hernia repair. There were no reported surgical complications, and the estimated blood loss was 1 liter. The patient was stable in the immediate postoperative period, and PACU admitted her on continuous epidural ropivacaine at T9-T10 for pain control.

Shortly after, she developed acute altered mental status with systolic blood pressure in the 60s and an acute drop in hemoglobin from 12 g/dL at baseline to 7.5 g/dL. Her mentation improved after fluid resuscitation, and she responded

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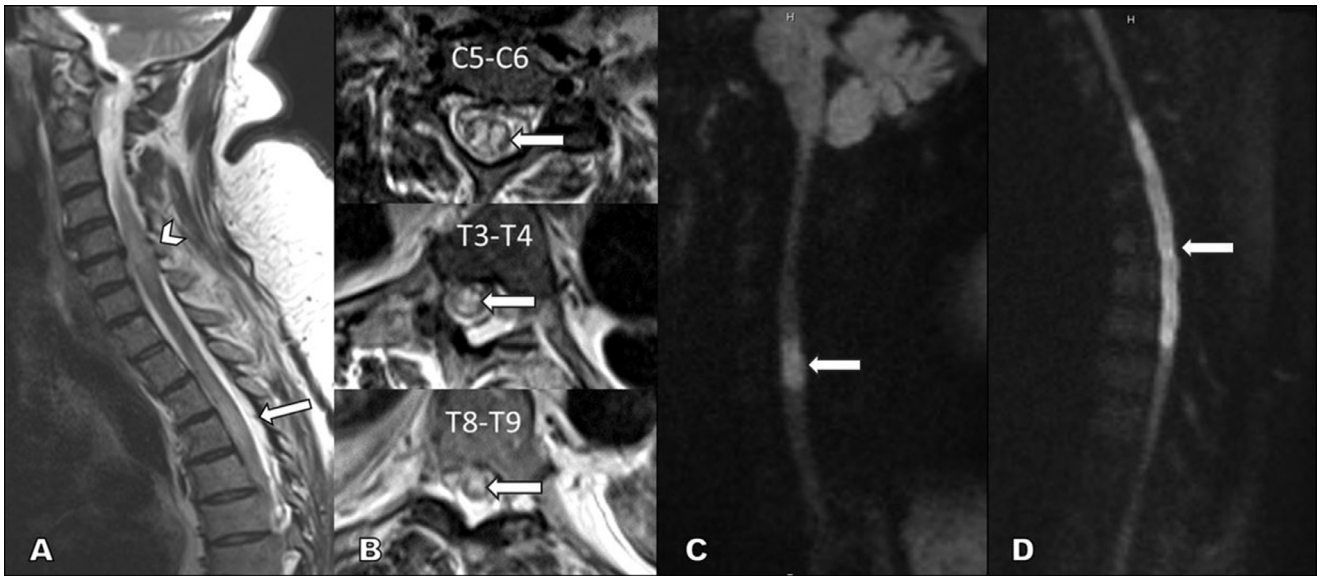

Fig. 1 – MRI of the spine on postoperative day 1 demonstrates 2 separate lesions consistent with acute spinal cord infarct. (A) Sagittal T2WI of the cervical (arrowhead) and thoracic (arrow) spine show 2 lesions of increased signal and caliber of the cord at C5-C6 and at T3-T9, consistent with cord edema. (B) Axial T2WI at multiple levels as labeled demonstrate predominant involvement of the central gray matter with sparing of the peripheral white matter (arrows). Sagittal DWI at the cervical (C) and thoracic (D) spinal cord show hyperintense signal due to cytotoxic edema from acute spinal cord infarcts (arrows). There are corresponding hypointense signals on apparent diffusion coefficient (not shown).

appropriately to packed red blood cell transfusion. On neurological examination, her American Spinal Injury Association (ASIA) score for sensory was 40 with complete sensory deficit at T4 and below. Motor score was 23 with preservation of upper extremities strength bilaterally but complete paralysis of the lower extremities. Her ASIA impairment score was A, with a complete absence of motor and sensory function in the sacral segments at S4–S5.

Magnetic resonance imaging of the spine on postoperative day 1 demonstrated increased signal intensity on T2 weighted images (T2WI) of the central cord gray matter from C5 to C6 and from T3 to T9 (Figs. 1A and B). This finding was associated with hyperintensity on diffusion-weighted imaging (DWI), related to restricted diffusion consistent with acute spinal cord infarcts (Figs. 1C and D). She received supportive care, and her epidural anesthetic catheter was removed due to concern for contributory hypotension and epidural hematoma, which was not confirmed on imaging.

The patient regained some motor strength in her upper extremities, including wrist flexion and extension; ASIA score for motor improved to 26. However, on postoperative day 11, she reports subjective worsening weakness in her upper extremities, prompting repeated spinal imaging. The MRI revealed an increased, now confluent T2 hyperintense signal in the spinal cord, extending from C2 to T9 (Fig. 2A), with focal peripheral contrast enhancement at the margins of previous infarcts at the C5-C6 and at T3-T8 levels (Fig. 2B and C), consistent with evolving subacute infarctions and extensive surrounding vasogenic edema. She continued to receive supportive care without experiencing progression of symptoms and was subsequently discharged to an in-patient rehabilitation facility with a motor ASIA score of 18 (neurological level at C7 for motor bilaterally) for ongoing physical therapy.

Discussion

Three major arteries supply the spinal cord. These include a single anterior spinal artery (ASA) supplying the anterior two-thirds of the spinal cord and 2 posterior spinal arteries (PSA) supplying the remaining posterior one-third. The ASA originates from the foramen magnum as a merged vessel of 2 bilateral branches from the vertebral arteries. The PSAs stem from either the vertebral arteries or the posterior inferior cerebellar arteries. At various spinal levels, segmental arteries divide into anterior and posterior radiculomedullary arteries, supplying the ASA and PSAs, respectively. These segmental arteries are tributaries from the descending aorta, intercostal, and lumbar arteries [3]. The spinal cord depends on these segmental arteries to provide adequate perfusion, with the largest being the artery of Adamkiewicz arising from the left intercostal or lumbar arteries between T8 and L2 [1]. In addition, multiple anastomoses along the pial surface of the cord between the ASA and PSA supply the peripheral cord.

The watershed areas of the spinal cord receive fewer arterial feeders and collaterals, making these regions more vulnerable to hypoperfusion injuries. There are 3 major watershed zones in the spinal cords. The longitudinal watershed zones are generally in the mid-thoracic spinal cord from T4 to T6. Less common are zones from T8 to T9 and in the lumbosacral cord, where there are fewer overlaps between regions of seg-

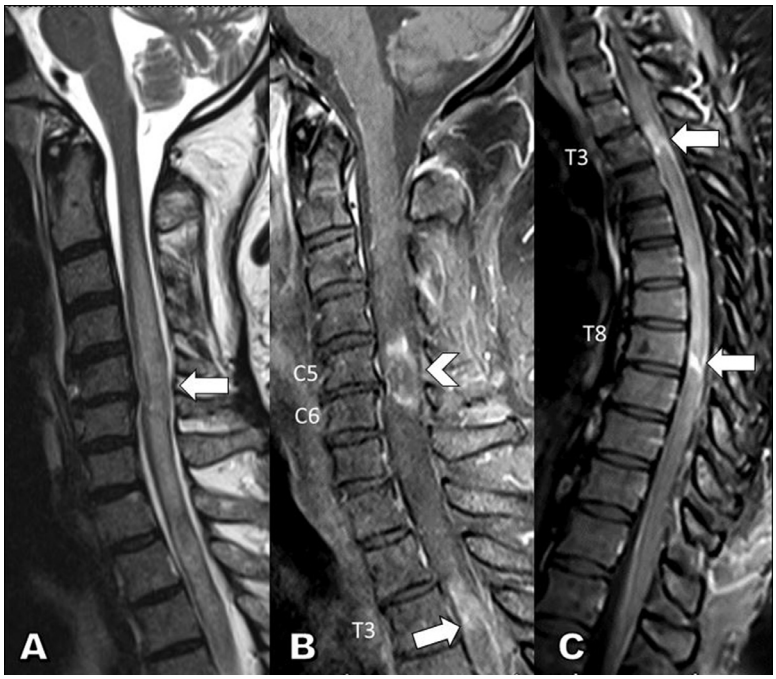

Fig. 2 – MRI of the spine on postoperative day 11 after the patient developed worsening upper extremities weakness. (A) Sagittal T2WI demonstrates increased regional T2 signal with confluency of the 2 previously seen focal areas of ischemia (arrow). Abnormal signal extends rostrally to C2 level and caudally to T9 (not shown). Sagittal T1WI postcontrast with fat saturation of the cervical (B) and thoracic (C) spine demonstrate new peripheral enhancement at C5-C6 (arrowhead) and at T3-T8 (B and C, arrows) corresponding to the sites of previous acute infarcts, consistent with progression to subacute infarction.

mental arterial feeders [4]. The transverse watershed zone is located in the central and peripheral gray matter, where a combination of normally higher metabolic demand and decreased supplies from the central and peripheral arteries can leave these regions vulnerable to ischemia [4]. Additional watershed zones are located along the anterolateral surface of the cord where there is poor anastomosis between the pial branches of the ASA and PSA [5].

Our patient demonstrates infarction in the longitudinal watershed zones at T3-T8 (Figs. 1 and 2C) and also at the transverse watershed zone (Fig. 1B) involving the central and peripheral gray matter with areas of peripheral white matter sparing (Fig. 1B). Despite the more robust vascular supplies, the cervical spinal cord was also involved in this patient at C5-C6, but to a lesser extent. After more than a week, repeated spine imaging demonstrated enhancement at the periphery of these infarcts, consistent with evolving subacute infarcts, a similar phenomenon to subacute infarcts in the brain. This is likely also from a combination of the breakdown of the blood-spinal cord barrier, luxury perfusion, angiogenesis, and granulation formation [6].

The etiology for our patient hypotensive episode was multifactorial from postsurgical anemia and thoracic epidural anesthesia. Studies have shown that the anesthetic blockage along the thoracic sympathetic outflow can lead to vasodilatation and depressive inotropic and chronotropic effects on the heart [7]. A combination of both led to hypovolemic shock, causing her spinal cord watershed infarct.

Conclusion

Acute spinal cord watershed infarction is a rare but devastating diagnosis, often associated with a poor prognosis with limited management options besides supportive care. MRI can assist in confirming the diagnosis and exclude other causes of acute myelopathy. Our case also demonstrates a classic finding of peripheral enhancement in the subacute phase due to the breakdown of the blood-spinal cord barrier [8].

Patient consent

The patient had provided informed written consent for the publication of this Case Report and all relevant data. The consent form is available on record upon request.

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