

Spinal Cord Infarction following Abdominal Surgery and Postoperative Epidural Analgesia

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احتشاء الحبل الشوكي بعد إجراء عملية جراحية في البطن وتسكين حول الجافية

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الملخص: الاحتشاء الإفقاري سبب نادر للاعتلال النخاعي الحاد. ندرج هنا حالة امرأة شابة رقدت في مستشفى جامعة السلطان قابوس بسلطنة عمان، أصيبت باحتشاء واسع النطاق في الحبل الشوكي بعد إجراء استئصال جراحي لورم دموي في الكبد وتعرض المريضة لتسكين حول الجافية، ولم يكن لديها عوامل اختطار للسكتة الدماغية الوعائية. سيتم مناقشة آلية الأوعية الدموية الدماغية الكامنة وراء اعتلال النخاع الشوكي الإفقاري وعلاقة ذلك بعملية البطن الجراحية والتسكين حول الجافية.

مفتاح الكلمات: الاعتلال النخاعي، احتشاء الحبل الشوكي، جراحة البطن، التسكين حول الجافية، التصوير بالرنين المغناطيسي، ورم دموي في الكبد، تقرير حالة، عمان.

ABSTRACT: Ischemic infarction is a rare cause of acute myelopathy. We report the case of a young woman admitted to Sultan Qaboos University Hospital, Oman, who developed extensive spinal cord infarction in the setting of surgical evacuation and packing of liver haematoma and post-operative epidural analgesia. She had no vascular risk factors for stroke. The vascular mechanism underlying ischemic myelopathy and the relationship to abdominal surgery and epidural analgesia are discussed.

Keywords: Myelopathy; Spinal cord infarction; Abdominal surgery; Epidural analgesia; Magnetic resonance imaging; Liver hematoma; Liver packing; Radiculomedullary artery; Case report; Oman

ISCHEMIC INFARCTION IS A RARE CAUSE of acute myelopathy.¹ The unique vascular supply predisposes the spinal cord to potential ischaemic insults in the perioperative setting of complicated abdominal surgery. Epidural analgesia is a common and relatively safe procedure to alleviate pain and to speed up recovery after abdominal surgery. The incidence of acute myelopathy due to central neural blockade after epidural analgesia is extremely rare. In view of the rarity of neurological complications following epidural analgesia alone, careful consideration should be paid to other medical or surgical causes (other than post-operative epidural injection as such) to account for post-operative acute myelopathy. In addition, it is important to recognise the signs of myelopathy rather than attributing the findings to delayed wearing-off of the analgesic effect. We report the

unusual case of a young adult who developed spinal cord infarction in the setting of surgical evacuation of intra-abdominal (liver) haematoma and thoracic epidural analgesia.

Case Report

A 28-year-old woman, admitted to a hospital in Oman, tested positive for hepatitis C viral (genotype 4) infection by polymerase chain reaction assay. She had no vascular risk factors such as hypertension, diabetes mellitus, atrial fibrillation, and hyperlipidemia. To establish the extent of liver pathology and to plan further management she underwent an elective liver biopsy in another institution. Two days after the procedure, she developed right upper abdominal pain and vomiting. The haemoglobin (Hb) dropped from

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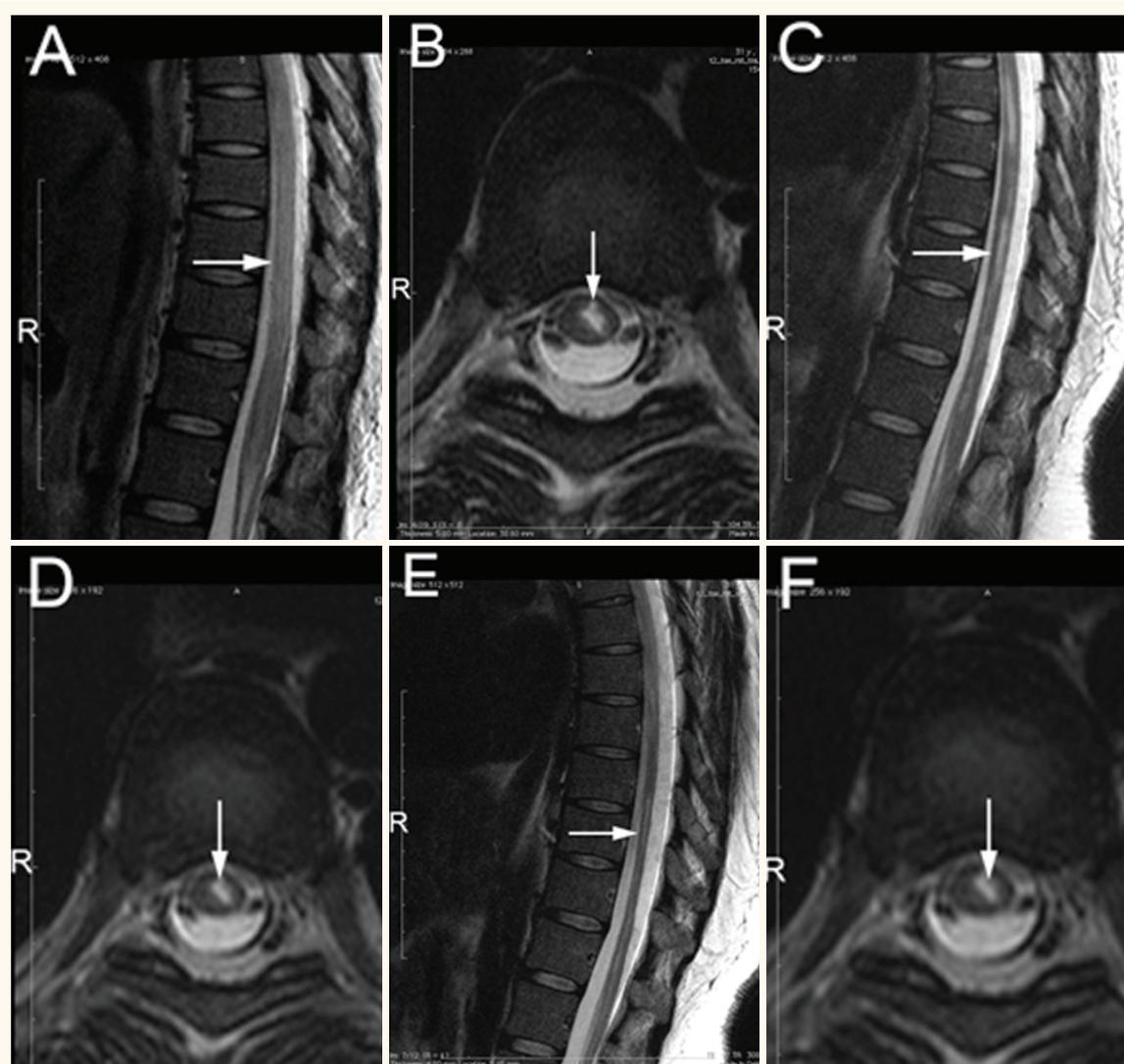


Figure 1a: Sagittal T2 weighted magnetic resonance imaging (initial scan) demonstrating diffuse bright signal (horizontal arrow) in the distal part of spinal cord. The corresponding transverse extent of the lesion with involvement of central gray on the left side (vertical arrow) is illustrated in **1b**: Follow-up scans at 2 months - **1c**: (sagittal image) and **1d**: (corresponding transverse image) and at 3 years- **1e**: (sagittal image) and **1f**: (corresponding transverse image) demonstrate progressive atrophy of the cord with myelomalacia (arrows) in the affected segments.

12 to 10 gm/l. An abdominal sonogram showed a hepatic subcapsular haematoma and she received 3 units of packed red cell transfusion. Despite the blood transfusion, the Hb remained low. Persistent intra-abdominal bleeding was suspected and an urgent abdominal computed tomography (CT) scan confirmed extensive haemorrhage in the peritoneal cavity and the pelvis. The following day (third day after the biopsy), she was transferred to our institution for further management including emergency laparotomy.

At pre-operative assessment in our centre, the blood pressure was 130/82 mmHg, and the heart rate ranged from 130 to 140 beats per minute. Hb remained stable at 10 gm/l and the coagulation

profile was within normal limits. The neurological examination was unremarkable. Intra-operatively she remained haemodynamically stable and there was no episode of hypotension. To arrest the continual oozing of blood from the liver surface, surgical packing of the liver was performed. At the end of the procedure, an epidural catheter was inserted at the thoracic (T)11-12 intervertebral space as per the standard technique of postoperative analgesia. A bolus of 10ml of 0.125% bupivacaine along with fentanyl 20 mcg was injected initially, followed by an infusion at 6ml/hr. She was extubated successfully in the intensive care unit an hour later and the rate of infusion was increased to 10ml/hr as she continued to experience pain.

She did not demonstrate hypotension during the procedure. Eight hours after the initiation of epidural analgesia, she complained of an inability to move her lower limbs and the epidural infusion was withheld. Neurologically, she demonstrated flaccid paraplegia (power of 0/5 in legs), sensory level at T8 dermatome (below which the primary modalities of sensation were lost) and areflexia in the lower limbs along with extensor plantar response and bowel and bladder disturbances. Emergent magnetic resonance imaging (MRI) of the spine revealed diffuse hyperintensity of the distal spinal cord, but sparing the terminal end and the conus [Figure 1a]. On the transverse T2 weighted image, it was observed that the signal abnormality corresponded to the central grey matter on the left side, while sparing the peripheral white matter [Figure 1b]. The ischaemic nature of the lesion was confirmed on diffusion weighted MR images. There was no evidence of epidural mass/compression, fluid collection, bone changes or acute traumatic injury. Serial clinical examination demonstrated persistent weakness and sensory signs indicating that the sensory changes were not due to delayed wearing-off of the analgesic effect.

A lumbar tap showed clear cerebrospinal fluid (CSF). CSF findings included glucose of 5.3 mmol/l (serum glucose 6 mmol/l), protein of 0.42 g/l, IgG 0.07 g/l and cell count of 28 cell/mm³ with 80% polymorphic lymphocytes. Auto-antibody screening for collagen vascular disease was negative. In view of recent recurrent surgical procedures, a course of intravenous immunoglobulin was preferred over high dose methylprednisolone to avoid the emergence of intra-abdominal sepsis, bowel perforation and in view of seropositivity for hepatitis C virus. She was re-explored surgically after 48 hrs and the liver packs were removed. The thrombophilia work-up (results obtained at a later date) was normal except for mild protein S deficiency. Haematological consultation was sought and it was felt that protein S deficiency was an incidental finding and not a causative factor for the cord infarction.

A follow-up MRI study performed two months later showed interval atrophy of the spinal cord as compared to the previous study with myelomalacia of the involved left central column [Figures 1c and 1d]. At a clinical follow-up a few months later, she had significant spastic paraparesis with ankle

clonus and a sensory level at T6-7. The patient was placed on regular physiotherapy. A subsequent follow-up MRI performed three years later showed progressive marked atrophy with persistent long segment T2 hyperintense lesions consistent with myelomalacia [Figure 1e]. These changes are also illustrated on the transverse T2 weighted images in Figure 1f.

Discussion

In this communication, we report the case of a young woman who developed extensive spinal cord infarction following surgical evacuation and packing for liver haematoma and epidural analgesia. It is to be noted that she did not have the conventional vascular risk factors for stroke. The extensive ischaemic myelopathy was unlikely to be due to the epidural analgesia *per se* and probably, the extensive intraabdominal haemorrhage prior to our hospital admission having set the stage for the ischemic injury. The tight packing of the liver (performed to arrest continual oozing of blood from the liver surface), may have compromised cord perfusion. Although venous stasis subsequent to the tight liver packing might also have contributed to congestive myelopathy, imaging studies did not demonstrate significant cord swelling, haemorrhagic lesions, or congestive changes in the intra-abdominal organs.

Regarding the vascular supply, the spinal cord is perfused by 3 main arteries that course vertically over its surface. The anterior two-thirds of the cord is supplied by a single anterior spinal artery and the posterior one-third by two anastomosing posterior spinal arteries. The anterior spinal artery is formed by the union of a branch from each vertebral artery. Along its course, the anterior spinal artery receives input from 6–9 unpaired radicular arteries in variable locations. These arteries are compromised in aortic lesions, but our patient did not demonstrate any aortic lesion. The major radiculo-medullary artery, the *arteria radicularis magna* (artery of Adamkiewicz) supplies the lower thoracic cord and lumbar enlargement; this site is particularly vulnerable to hypoperfusion and is referred to as the water shed area of the cord.²⁻⁴ In a multi-detector row CT angiogram study, acquisition of longitudinal imaging volume from the dome of liver at the level of eighth thoracic vertebra to the tip of the liver at the level of third lumbar vertebra

covered the location of artery of Adamkiewicz.⁵ With regards to the laterality of this radiculo-medullary artery, the artery arises from the lower intercostal and upper lumbar arteries on the left side in about two-thirds of cases and from the right side in one-third of cases.⁵ It is, therefore, conceivable that the unique vascular supply of the spinal cord and the anatomical proximity of vessels, such as the artery of Adamkiewicz, to abdominal organs like the liver render the cord particularly vulnerable to ischemia and infarction in the setting of complicated liver/abdominal surgery. Most spinal cord infarcts are located in the central territory of the anterior spinal artery with predominant involvement of the central grey matter and the adjacent central white matter, though some may have a more prominent involvement of the anterior grey matter.⁶ This central grey involvement was also observed more prominently on the left side in our patient. Occlusion of the *arteria radicularis magna* due to the pressure effect of tight liver packing and intra-abdominal haemorrhage was the most likely vascular mechanism based on the extent of anatomical lesion on the sagittal imaging [Figures 1a, 1c and 1e]. The sequential changes with progressive atrophy of the cord and myelomalacia [Figure 1c to 1f] confirmed the initial diagnosis of spinal cord infarction. Other focal non-mitotic intramedullary lesions of the cord, e.g. transverse myelitis, were unlikely to be limited to the central cord, and usually progressive improvement in the imaging appearances and neurological status of the patient might be expected in those conditions. Another technique that may be used for ascertaining the ischemic nature of the lesion is diffusion-weighted MRI of the spinal cord. As in the brain, acute spinal cord infarction demonstrates restricted diffusion.⁷ In keeping with this finding, severe diffusion restriction was also observed in the acute stage of cord infarction in our patient. In the late stage, the myelomalacic segment showed increased diffusivity.

Spinal cord ischemia following epidural anaesthesia is rare and usually a direct causal relationship is unlikely.⁸⁻¹⁴ Possible mechanisms of epidural analgesia-related myelopathy include cord compression due to epidural haematoma, cord trauma, injury to arteries supplying the cord, chemical contamination and iatrogenic meningitis.⁸⁻¹¹ Our patient did not have any evidence of compressive myelopathy, nor did

her CSF findings support chemical or iatrogenic meningitis.⁸⁻¹¹ Moreover, our patient did not have risk factors for premature atherosclerosis, in which setting a hyperlordotic or laterally flexed position may be a more common cause of spinal cord injury.^{12,13} Continuous infusion of local anaesthetic with sympathomimetics has been implicated in vasoconstriction and subsequent cord ischemia.^{14,15} This was less likely in our case as epinephrine was not used. Hypotension has been cited as a major contributor in the majority of reports of paraplegia complicating epidural anesthesia;¹⁵⁻¹⁹ however, the patient did not demonstrate systemic hypotension during the procedure. The presence of vasculitis and atherosclerotic and thrombotic risk factors render the cord more susceptible to a hypoperfusion insult.²⁰ Our patient did not have any clinical or serological evidence of vasculitis. Moreover, she was relatively healthy, apart from the hepatitis C infection prior to the liver biopsy. On many occasions, the association between epidural anaesthesia and serious neurological sequelae is only a temporal coincidence and might be inappropriately suspected as causative factor.²¹⁻²³ However, it is to be noted that in the post-operative setting the epidural analgesia may obscure the neurological assessment, especially the sensory examination.

Conclusion

In summary, we have described a young patient who developed ischemic myelopathy following surgical evacuation and packing of liver haematoma and postoperative epidural analgesia. We believe that tight packing of liver injury, apart from intra-abdominal haemorrhage might have compromised the blood supply and contributed to the ischemic injury of the cord. Our report underscores the importance of considering various possible mechanisms underlying spinal cord infarction, especially surgical factors, rather than attributing the signs of myelopathy to the delayed wearing-off of post-operative epidural analgesia.

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