Rare disease

Remote spinal epidural haematoma after spinal anaesthesia presenting with a 'spinal lucid interval'

Venkatesh S Madhugiri, Manish Singh, Gopalakrishnan M Sasidharan, V R Roopesh Kumar

Department of Neurosurgery, Jawaharlal Institute of Postgraduate Medical Education and Research (JIPMER), Pondicherry, India

Correspondence to Dr Venkatesh S Madhugiri, venkateshmadhugiri@gmail.com

Summary

An obstetric patient who had no significant risk factors developed a spinal epidural haematoma remote from the site of needle puncture (for administration of spinal anaesthesia). The clinical deficits were manifest after recovery from the motor blockade had started a phenomenon that we have termed as a 'spinal lucid interval'. The patient developed flaccid paraplegia with a sharp sensory level and urinary retention. The patient underwent emergency laminectomy and evacuation of the haematoma. She gradually recovered near normal power and was ambulant independently and had normal sphincter function at follow-up.

BACKGROUND

The occurrence of a spinal epidural haematoma (SEDH) following neuraxial blockade (NAB) is a devastating complication. SEDHs are described as most commonly occurring in the setting of epidural anaesthesia (EA)/analgesia, especially if an epidural catheter is inserted. SEDH may occur at the highest level that the catheter was inserted to, even after catheter withdrawal. SEDH can also occur at the level where a needle is inserted for administering spinal anaesthesia (SA). However, there are no documented reports of an SEDH occurring remote from the level of puncture for the administration of SA. Even more unusual is the delay in development of clinical deficits. This is a rare event and the possible causes are discussed and the relevant literature reviewed.

CASE PRESENTATION

A 23-year-old lady (G1, height 157 cm, weight 63 kg) with no comorbid illnesses and an uneventful antenatal period (ASA grade 1P) delivered a live male child by elective caesarean section at term under SA at another hospital. The indication for surgery was cephalopelvic disproportion. SA was administered with the patient in the left lateral position, in the L3–4 interspace with a 25G spinal needle. Only 2.5 ml of 0.5% isobaric bupivacaine was injected; no difficulty was documented in accessing the subarachnoid space. Surgery was uneventful and there was no documented episode of hypotension or hypertension during surgery. The patient began moving her lower limbs and regained a power of Medical Research Council grade 3/5 by about 10 h after surgery. The next morning (16 h after the procedure) the patient complained of paraesthesia over both lower limbs. She then developed weakness of both lower limbs that progressed to paraplegia over 4 h. She also developed urinary retention and had to be catheterised. On examination, she had flaccid paraplegia with areflexia in both lower limbs. She had graded sensory loss to all modalities below D7, with no sacral sparing.

INVESTIGATIONS

An urgent MRI of the dorsolumbar spine revealed a dorsally located SEDH extending from D6 to D8, (figure 1A) causing significant spinal cord compression (figure 1B,C). Thin T2 axial sections at the level of the lesion did not reveal any abnormal flow voids and therefore, spinal angiogram was not performed. Haemogram revealed haemoglobin of 8 gm% and platelet count of 220 000/mm³. Peripheral smear was normal. Bleeding time, clotting time and prothrombin time international normalised ratio were all normal. There was no history of antiplatelet, anticoagulant or oral contraceptive intake. There was no history of menorrhagia or evidence of other bleeding manifestations.

TREATMENT

She underwent emergency D6–D8 laminectomy through a dorsal midline approach under general anaesthesia. After

Figure 1 (A) T1-weighted sagittal image showing the dorsally placed hyperintense haematoma at D7–8. (B) Preoperative T2 and (C) preoperative T1 axial images showing the haematoma compressing the spinal cord. (D) Postoperative T2-weighted sagittal image showing evidence of laminectomy and complete evacuation of the haematoma.

laminectomy, a large blood clot was seen over the dura and was evacuated. Neither abnormal vessels nor any lesion was seen at surgery. The clot was subjected to histopathological examination. Microscopy revealed an organised haematoma with no lesional tissue.

OUTCOME AND FOLLOW-UP

The patient's neurological recovery was gradual but near complete. Seven months after surgery, she is ambulant independently with power grade 4+/5 in the lower limbs. She has regained control of bladder function. Mild hypoesthesia below S2 (in the perianal area) persists. MRI at follow-up showed complete evacuation of the haematoma and relief of spinal cord compression. No lesion or abnormal vessels were seen (figure 1D).

DISCUSSION

The exact incidence of SEDH after NAB is difficult to estimate because of its rarity. It is estimated that after EA, the incidence of SEDH is 1 in 150 000.^{1 2} A German study found an incidence of 1:6628 in *non-obstetric patients* who underwent procedures under EA.³ However, most of these patients had been on either antiplatelet therapy or on anticoagulation. The incidence of SEDH after SA is reported to be about 1:220 000.²

The epidural space contains loose fatty tissue and the epidural venous plexus, which is a network of large, thin-walled veins and therefore, an SEDH is more likely to be venous in origin unlike a cranial epidural haematoma.⁴ Tewari and Pandey⁵ have suggested that the rupture of the valveless veins in the internal vertebral plexus, even by the slightest change of posture during sleep, turning or coughing or due to Valsalva's manoeuvre, can cause epidural bleeding. Certain risk factors have been identified for the development of SEDH after NAB.⁶ These include advanced age, female gender, bony spinal pathology (that may necessitate multiple attempts at puncture) and coagulopathy. Pregnancy, arterial hypertension and vasculitis, especially systemic lupus erythematosus can lead to spontaneous SEDH.^{3 7} The use of thrombolytic agents in the perioperative period seems to increase the risk.⁸ The presence of occult arteriovenous malformations is another possible cause of SEDH after neuraxial blocks. Another important cause is the presence of undiagnosed diseases that predispose to bleeding. These may include leukaemia or lymphoma, both of which can produce tumour-like masses in the epidural space.⁹

There are two unusual features about this patient—the location of the haematoma and the clinical presentation. This patient was a pregnant woman. Besides these, she had none of the other risk factors that have been previously identified.³ She had not received antiplatelet therapy or anticoagulation. The spinal tap was described as uneventful and non-traumatic. The preoperative high-resolution MRI with thin T2-weighted images at the level of the lesion as ruled out tumours or vascular malformations as possible causes. Given the emergent nature of the situation and the need for immediate surgical intervention, a formal spinal angiogram was not performed.

There are reports of SEDH in patients with normal coagulation profiles. One of these reports postulates that the use of a double orifice spinal needle could have been responsible.¹⁰ However, a single lumen needle was employed in this case. Another patient with normal coagulation profile is

reported to have developed an L1–4 haematoma after SA administered at L3–4 level with a 27G needle.¹¹ Both these patients developed SEDH *adjacent to the level* at which lumbar puncture was performed. Spontaneous SEDH have been reported in pregnant women. However, in this instance, the SEDH did not occur during pregnancy but occurred in relation to the caesarean section and manifested several hours after the surgery. Labour (which could lead to an increase in intra-abdominal pressure which could then theoretically be transmitted to the vertebral venous plexus) did not occur in this patient since the caesarean section was elective. Thus, almost certainly the aetiology was administration of SA.

It is possible that occult injury to the epidural veins can occur during the administration of SA. However, in all these cases, the haematoma would be adjacent to the site of needle puncture. An extensive search of literature failed to reveal any documented case of an SEDH that occurred remote from the site of needle puncture. In this patient, the SEDH was seven levels removed from the L3–4 interspace where SA was administered.

The mechanism of such a remote SEDH is a matter of conjecture. It is possible that during pregnancy, the pressure of the gravid uterus on the inferior vena cava (IVC) causes a state of chronic engorgement of the epidural venous plexus. The lateral decubitus position employed for administration of SA could cause stretch of these engorged veins. This could cause a remote bleed in the thoracic area. Sudden decompression of the IVC after delivery of the fetus during caesarean section could lead to a sudden decompression of the epidural venous plexus; this could cause stretching and tearing of these veins, leading to an SEDH anywhere along the spine.

This patient had flaccid rather than spastic paraplegia (despite the D6–8 level) due to the acute spinal compression leading to a 'spinal shock' like state. Flaccid paraplegia could potentially be interpreted as a delay from recovering from motor blockade. Most patients may not recover from the motor block at all if an SEDH has developed. This patient manifested a very unusual phenomenon—that of a 'spinal lucid interval'. A lucid interval is characteristic of cranial epidural haematomas and is the period of normal consciousness after the patient recovers from the impact concussion.¹² This represents the time taken for the cranial epidural haematoma to grow to a size adequate to cause increased intracranial pressure and altered sensorium.¹³ This patient had grade 3/5 power before she developed sensory symptoms and subsequently paraplegia. It is postulated that this 'spinal lucid interval' or 'delayed neurological deficit' represents the period where the SEDH reached the critical volume to cause the devastating paralysis. This is a phenomenon that is absolutely unique and has not been reported hitherto.

The patient made a good recovery since surgery was performed within 48 h of the onset of paraesthesia. The preoperative status and timing of surgery affect patient outcome in SEDH.¹⁴ In a large series of SEDH, 83% of patients who were Frankel Grade D before surgery recovered completely compared with 25% of Frankel Grade A patients. The rapidity of surgical intervention also correlated with outcome. Patients taken to surgery within 12 h had better neurological outcomes than patients with identical preoperative Frankel grades whose surgery was

delayed beyond 12 h.¹⁴ Some authors have reported a possible role for conservative management of patients with a spontaneous SEDH, if they show spontaneous improvement within a few hours.¹⁵ However, in those patients with a definite aetiology (anticoagulant induced, post-SA, etc) if any neurological symptoms or deficits exist at the time of evaluation, urgent surgery is indicated.

Learning points

- ▶ Any patient who fails to recover from a neuraxial block within the expected time frame or develops fresh neurological symptoms should be investigated with a whole spine MRI.
- ▶ If a spinal haematoma is detected, urgent operative management is mandatory, and may lead to good outcomes.
- ▶ These haematomas can occur in the absence of risk factors.
- ▶ They may occur remote from the site of catheter or needle insertion and may present with deficits after motor recovery begins.

Competing interests None.

Patient consent Obtained.

REFERENCES

1. **Horlocker TT.** Low molecular weight heparin and neuroaxial anaesthesia. *Thrombosis Res* 2001;**101**:141–54.
2. **Tryba M.** Epidural regional anaesthesia and low molecular heparin: pro. *Anesthesiol Intensivmed Notfallmed Schmerzther* 1993;**3**:179–81. (in German)
3. **Volk T,** Wolf A, Van Aken H, *et al.* Incidence of spinal haematoma after epidural puncture: analysis from the German network for safety in regional anaesthesia. *Eur J Anaesthesiol* 2012;**29**:170–6.
4. **Groen RJ,** Ponssen H. The spontaneous spinal epidural haematoma. A study of the etiology. *J Neurol Sci* 1990;**98**:121–38.
5. **Tewari MK,** Pandey AK. Spontaneous spinal extradural haematoma. *Neurol India* 1999;**47**:159.
6. **Horlocker TT,** Wedel DJ. Anticoagulation and neuraxial block: historical perspective, anesthetic implications and risk management. *Reg Anesth Pain Med* 1998;**23**:129–34.
7. **Masski G,** Housni B, Ibahiouin K, *et al.* Spontaneous cervical epidural haematoma during pregnancy. *Int J Obstet Anesth* 2004;**13**:103–6.
8. **Ozgocmen S,** Yoldas T, Kocakoc E, *et al.* Spinal epidural haematoma associated with streptokinase treatment for myocardial infarction. *Spinal cord* 2004;**42**:374–7.
9. **Gottschalk A,** Bischoff P, Lamszus K, *et al.* Epidural haematoma after spinal anaesthesia in a patient with undiagnosed epidural lymphoma. *Anesth Analg* 2004;**98**:1181–3.
10. **Evron S,** Gurstieva V, Ezri T, *et al.* Transient neurological symptoms after isobaric subarachnoid anaesthesia with 2% lidocaine: the impact of needle type. *Anesth Analg* 2007;**105**:1494–9.
11. **Hasuwa K,** Nakahashi K, Kuzumoto N, *et al.* Spinal epidural haematoma after spinal anaesthesia using a 27-gauge spinal needle in a patient with normal coagulation profile. *Masui* 2009;**58**:456–9. (in Japanese)
12. **Rivas JJ,** Lobato RD, Sarabia R, *et al.* Extradural haematoma: analysis of factors influencing the courses of 161 patients. *Neurosurgery* 1988;**23**:44–51.
13. **Servadei F.** Prognostic factors in severely head injured adult patients with epidural haematomas. *Acta Neurochir (Wien)* 1997;**139**:273–8.
14. **Lawton MT,** Porter RV, Heiserman JE, *et al.* Surgical management of spinal epidural haematoma: relationship between surgical timing and neurological outcome. *J Neurosurg* 1995;**83**:1–7.
15. **Duffil J,** Sparrow OC, Millar J, *et al.* Can spontaneous spinal epidural haematoma be managed safely without operation? A report of four cases. *J Neurol Neurosurg Psych* 2000;**69**:816–19.

Copyright 2012 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Please cite this article as follows (you will need to access the article online to obtain the date of publication).

Madhugiri VS, Singh M, Sasidharan GM, Roopesh Kumar VR. Remote spinal epidural haematoma after spinal anaesthesia presenting with a 'spinal lucid interval'. *BMJ Case Reports* 2012;10.1136/bcr-2012-007258, Published XXX

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow