

Case Report

Spinal epidural hematoma associated with bolus injection during patient-controlled epidural analgesia

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- Patient-controlled epidural analgesia
- Bolus injection; Recovery

Abstract

Although spinal epidural hematoma is rare, it can have critical consequences. We experienced a case of spinal epidural hematoma after bolus injection of local anesthetics during patient-controlled epidural analgesia (PCEA). A female patient was presented for management of herpes zoster related pain. An epidural catheter was placed at the thoracic level for PCEA. On day 6 after epidural catheterization, after bolus injection, she complained of bilateral lower extremity motor deficits, and vesicorectal disturbance. A spinal MRI demonstrated spinal epidural hematoma at the T6 level. The hematoma spontaneously disappeared without invasive treatment and the patient recovered completely without any neurological sequelae.

ABBREVIATIONS

PCEA: Patient-controlled Epidural Analgesia

INTRODUCTION

Spinal epidural hematoma is a rare but devastating complication of neuraxial blockade. In general, the risk of spinal epidural hematoma is thought to be greatest at the time of insertion a block needle and catheterization or at the time of removal an epidural catheter. To our knowledge, there is no report of spinal epidural hematoma caused by bolus injection of local anesthetics during patient-controlled-epidural-analgesia (PCEA). We have a case of spinal epidural hematoma associated with bolus injection during PCEA for pain management. We obtained the patient's consent of this case-report.

CASE PRESENTATION

A 27-yr-old female patient [weight 56 kg, height 164 cm], with no significant past history or prescription therapy, presented for management of herpes zoster related pain in the right thoracic 6-7 regions 1 month after its onset. The pain scores of Visual Analogue Scale (VAS, range: 0-100 mm) were from 69 mm to 90 mm. Blood

tests showed a platelet count of $232 \times 10^3 / \mu\text{L}$, hemoglobin of 14.2 g/dL. Blood coagulation test of prothrombin time (international normalized ratio) was 0.98; activated partial thromboplastin time was 31.5 second. There were no abnormalities of liver and renal function in blood chemical values. We planned to reduce her pain using combined treatment of epidural blockade and medications under admission. An 18-G epidural catheter (Portex® Epidural Catheter, 3 Lateral Eyes, Smiths Medical Inc., Keene, NH, USA) was placed at T 7/8 on the first attempt using a "loss of resistance" technique with a 18-G Tuohy needle inserted by a left paramedian approach without traumatic needle and catheter insertion (the length of 10 cm into epidural space). The program of PCEA with 0.3% levo-bupivacaine was set at a continuous injection speed of 2 ml/hr, with 3 ml patient controlled bolus injections (bolus injection speed of 175 ml/hr) and a 60-min lockout interval. We used PCEA devices of CADD-Legacy® PCA Pump (Smiths Medical Inc.). Ten days after epidural catheterization, the catheter spontaneously dislodged. As the patient still complained of severe pain, we re-inserted an epidural catheter the length of 9.5 cm into epidural space at the T 7/8 level by median approach without any trouble on the same day. The same devices and program of PCEA were used in following pain

management. Local anesthetic was not changed in concentration and volume. Thereafter, she required 5-8 boluses of the patient-controlled analgesic for pain control over a 24-hour period. Oral medications were as follows: pregabalin 250 mg/day, celecoxib 200 mg/day, amitriptyline 10 mg/day, codeine phosphate 40 mg (potion, 1-2 times a day), diazepam 4 mg/day, paroxetine 10 mg/day, metoclopramide 15 mg/day, fexofenadine 120 mg/day, magnesium oxide 1800 mg/day. On day 6 after epidural re-catheterization, immediately after bolus administration of local anesthetics during PCEA, she abruptly complained of warmth and discomfort in the upper abdominal region, bilateral lower extremity motor deficits, numbness on bilateral lower extremity and vesicorectal disturbance. Disturbances in cold sensation were confirmed between levels T6 and T10. Motor powers of the lower limbs were 3/5 in all muscle groups. There was no complaint of back pain, nuchal pain and headache. The body temperature was 36.3 degree Celsius. We confirmed the absence of backflow of cerebrospinal fluid and blood from the catheter. We considered the cause of this symptom due to subdural blockade and removed the epidural catheter an hour after the event, at which time she complained of difficulty of urination. The catheter was positioned 5 cm in the epidural space at the time of its removal. As the bilateral lower extremity motor disturbances gradually improved, we had been keeping her under neurological surveillance. Difficulty of urination and constipation persisted to the next day. A spinal MRI was performed 19 hours after the onset of neurological symptoms that demonstrated a spinal epidural hematoma at the T6 level (Figure 1a, 2a). We immediately consulted with orthopedists about the indication of surgical decompression. She complained dysuria but bowel movement recovered at consult to orthopedists. Motor functions of lower limbs were recovered and she came to be able to walk at their inspection. They examined her and assessed that she

was not needed to operate immediately because the symptom was in recovery period. MRI performed the next day showed a reduction in the size of the spinal epidural hematoma (Figure 1b, 2b. twenty hrs after the 1st MRI). Eight days after the appearance of the symptom, dysuria also improved and thus, her condition was conservatively followed without any invasive procedures. Twelve days later, we confirmed resolution of the spinal epidural hematoma on MRI (Figure 1c, 2c). There was no residual sensory, motor deficit and vesicorectal disturbance on the day. Thereafter, the herpes zoster pain improved with oral medication.

DISCUSSION

Spinal epidural hematomas are idiopathic in most cases (29.8%). Other frequent causes of spinal epidural hematomas are anti-coagulant therapy (17%), regional anesthesia (10.4%) and vascular malformation (9.1%) [1]. The frequency of spinal epidural hematoma due to epidural blockade is affected by patient background and the use of peri-operative anticoagulants, with a reported incidence of 1 in 3,600-200,000 cases [2]. Spinal epidural hematomas secondary to epidural or spinal blockade can cause sensory and motor deficits and vesicorectal dysfunction, in which symptoms appear and be disappeared within 24 hrs in most cases [1]. Although the early symptoms of spinal epidural hematoma are back pain, there are a few reports of cases with no back pain [3]. Other report presents cases with symptoms of motor deficit rather than back pain [4].

The spinal epidural hematoma may have occurred at the following times:

1. At the time of epidural catheterization.
2. At the time of epidural catheter withdrawal.
3. At the time of bolus injection of the local anesthetic.

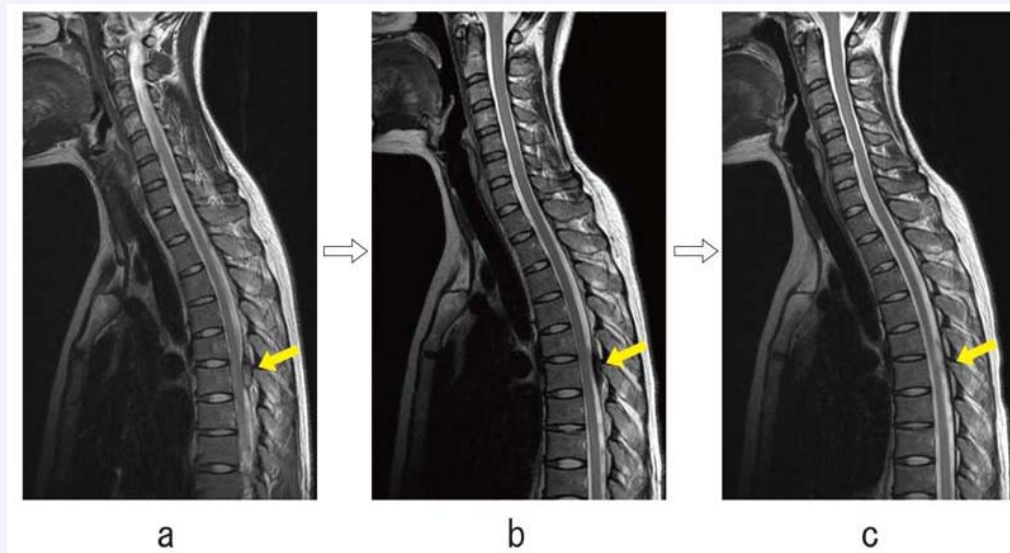


Figure 1

(a) Sagittal MRI (T2-weighted imaging, T2WI) was carried out 19 hrs after the event, showed a hyperintense hematoma in the posterior epidural space at T6 spinal level (large arrow). It compressed spinal cord, but abnormal intensity imaging was not seen in the spinal cord.
(b) Second MRI was carried out 20 hrs after 1st MRI. The hematoma revealed hypointense imaging on T2WI and was a slight decrease in the size.
(c) Twelve days after its identification, the hematoma had disappeared.

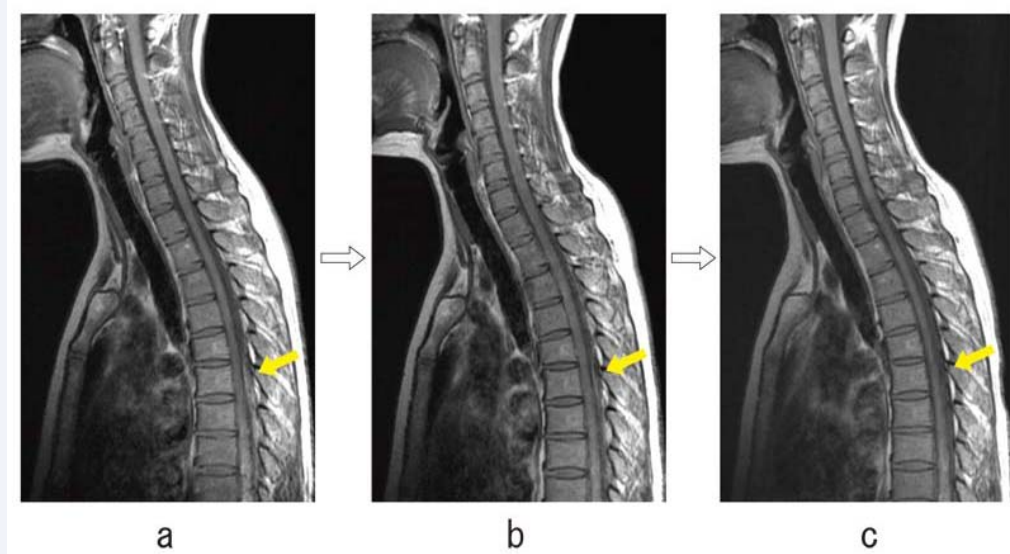


Figure 2

MRI (T1-weighted imaging, T1WI) was performed at the same time of Figure 1.

(a) Sagittal MRI (T1WI) was carried out 19 hrs after the event, showed anisointense hematoma in the posterior epidural space at T6 spinal level (large arrow). It compressed spinal cord, but abnormal intensity imaging was not seen in the spinal cord. The size of the hematoma measured on T1WI was 65mm (length), 13mm (breadth) and 5mm (thickness).

(b) Second MRI was carried out 20 hrs after 1st MRI. The hematoma revealed isointense imaging on T1WI and was a slight decrease in the size.

(c) Twelve days after its identification, the hematoma had disappeared.

Spinal epidural hematoma occurs in many cases within 24 hrs of epidural catheterization. In our case, symptoms were confirmed 6 days after catheterization. Furthermore, subacute spinal epidural hematomas (3-7 days after onset of hematomas) appear on MRI as high intensity areas with T1-weighted imaging (T1WI) and low intensity areas with T2-weighted imaging (T2WI) [5]. In our case, MRI carried out 19 hrs after the event presented isointense shadows on T1WI and high intensity shadows on T2WI, suggesting the hyperacute stage hematoma, it means the onset of the hematoma within the previous 24 hrs [6]. The second MRI taken 39 hrs after the event showed isointense on T1WI and low intensity on T2WI, suggesting was not in conflict with the images on acute stage hematoma. The acute stage hematoma (1-3 days after onset) mainly consists of deoxyhemoglobin is slightly hypo-/isointense on T1WI and hypointense on T2WI [5-6]. Since neurological symptoms appeared before removal of the epidural catheter, it is highly unlikely that its removal caused the spinal epidural hematoma. Rather, both lower extremity motor and vesicorectal dysfunction improved after epidural catheter removal. We need to provide differentially diagnosing local anesthetics or abscess, which may cause abnormal findings in epidural space on MRI. Local anesthetics appear on MRI as high intensity with T2WI same as the intensity of water. Twenty hrs after the first MRI, the second MRI revealed low intensity area on T2WI. It did not indicate water intensity of imaging. Epidural abscess has low or intermediate signal intensity on T1WI and high signal intensity on T2WI [7]. The signal intensity of epidural abscess would not change without antibiotics within only 20 hrs. She had no infectious symptom such as fever and meningeal irritation sign on the day. Spinal epidural hematoma at thoracic level may involve mechanical spinal compression and spinal circulatory disorder. Signs of spinal compression appear,

presenting as progressive paraplegia and loss of sensory function and cauda-equina syndrome [8]. Sensory disturbance on thoracic nerve level, muscle weakness on lower limbs and vesicorectal dysfunction occurred in this case. These disturbances happened to the patient abruptly after bolus injection during PCEA. Therefore, we concluded that the spinal epidural hematoma occurred following bolus injection of the local anesthetics. But in this case, there is a possibility that the hematoma was formed probably small immediately after bolus injection and that the local anesthetics accumulated on the epidural space. It is possible that the relatively prompt recovery of the symptoms could have been associated with the absorption of the local anesthetics. As the other cause of spinal epidural hematoma, patients with arteriovenous malformations in spinal epidural region are at risk of hematoma. Since our patient refused further examination, arteriovenous malformation remains a possible cause of the spinal epidural hematoma after bolus epidural anesthetic drug administration. A point to be aware of this case, we consider if we took MRI immediately after the first neurological symptom at bolus injection, the diagnosis of spinal epidural hematoma has been got earlier.

According to a literature on the operative management of spinal epidural hematomas, neurological prognosis is comparatively good when surgery is performed within 6 hrs of symptom onset [1]. Complete recovery is found in more than 50% of cases if surgery is performed within 12 hrs of symptom onset.¹ Furthermore, full recovery is not expected in cases where surgical intervention is performed more than 12 hrs after symptom onset.⁹ However, there is a report of spontaneous recovery from a thoracic spinal epidural hematoma accompanied with epidural anesthesia.¹⁰ In this report, motor power of the

lower extremities was 2/5 in almost muscle groups and no signal abnormalities of the cord itself were seen on MRI. In our case, when the spinal epidural hematoma was confirmed on MRI, the remaining neurological symptom were dysuria and constipation, the improvement in her condition of motor and sensory disturbances leading to our decision to treat the patient conservatively. There would be a possibility to get recovery from neurological disturbance caused by spinal epidural hematoma without invasive treatment, if the neurological functions would be in recovery phase.

In conclusion, we experienced a patient who developed a spinal epidural hematoma accompanied by neurological symptoms immediately after bolus injection during PCEA. The spinal epidural hematoma spontaneously disappeared without invasive treatment and the patient recovered completely without any neurological sequelae.

REFERENCES

1. Kreppel D, Antoniadis G, Seeling W(2003) Spinal hematoma: a literature survey with meta-analysis of 613 patients. *Neurosurg Rev*26:1-49.
2. Moen V, Dahlgren N, Irestedt L(2004) Severe neurological complications after central neuraxial blockades in Sweden 1990-1999. *Anesthesiology*101:950-959.
3. Li SL, Wang DX, Ma D(2010) Epidural hematoma after neuraxial blockade: A retrospective report from China. *AnesthAnal*111:1322-1324.
4. Vandermeulen EP, Van Aken H, Vermeylen J (1994) Anticoagulants and spinal-epidural anesthesia. *AnesthAnal*79:1165-1177.
5. Braun P, Kazmi K, Nogués-Meléndez P, Mas-Estellés F, Aparici-Robles F (2007)MRI findings in spinal subdural and epidural hematomas. *Eur J Radiol*64:119-125.
6. Bradley WG Jr (1993) MR appearance of hemorrhage in the brain. *Radiology*189:15-26.
7. Kricun R, Shoemaker EI, Chovanes GI, Stephenes HW (1992) Epidural abscess of the cervical spine: MR findings in five cases. *Am J Roentgenol*158:1145-1149.
8. Alexiadou-Rudolf C, Ernestus RI, Nanassis K, Lanfermann H, Klug N(1998) Acute non-traumatic spinal epidural hematomas. An important differential diagnosis in spinal emergencies. *Spine*23:1810-1813.
9. Meikle J, Bird S, Nightingale JJ, White N(2008) Detection and management of epidural haematomas related to anaesthesia in the UK: a national survey of current practice. *Br J Anaesth*101:400-404.
10. Schwarz SK, Wong CL, McDonald WN(2004) Spontaneous recovery from a spinal epidural hematoma with atypical presentation in a nonagenarian. *Can J Anaesth*51:557-561.

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