CASE REPORT

Gadolinium enhancement of cauda equina after combined spinal–epidural anaesthesia

¹M TAKASU, MD, ²M OKITA, MD, ³T ARAKI, MD, ¹N TANITAME, MD, ¹A TAMURA, MD, ⁴N SUWAKI, MD and ⁵K ITO, MD, PhD

¹Department of Radiology, Hiroshima Red Cross Hospital, 1-9-6, Sendamachi, Nakaku, Hiroshimashi 730-8619, Japan,

²Department of Anesthesiology, Hiroshima Red Cross Hospital, 1-9-6, Sendamachi, Nakaku, Hiroshimashi 730-8619,

Japan, ³Department of Neurology, Hiroshima Red Cross Hospital, 1-9-6, Sendamachi, Nakaku, Hiroshimashi 730-8619,

Japan, ⁴Department of Obstetrics and Gynecology, Hiroshima Red Cross Hospital, 1-9-6, Sendamachi, Nakaku,

Hiroshimashi 730-8619, Japan and ⁵Department of Radiology, Division of Medical Intelligence and Informatics, Programs for Applied Biomedicine, Graduate School of Biomedical Science, Hiroshima University, Japan

ABSTRACT. The occurrence of neurological symptoms after spinal anaesthesia has been reported with several local anaesthetics including lidocaine, prilocaine, mepivacaine, tetracaine and bupivacaine. Although hyperbaric bupivacaine is known to induce neurological symptoms less frequently than lidocaine, a few cases of cauda equina syndrome (CES) following the intraspinal injection of bupivacaine have been reported in the English literature. We describe lumbar MRI findings for a 29-year-old woman presenting with CES after caesarean section.

Received 5 March 2009

Revised 5 July 2009

Accepted 1 August 2009

DOI: 10.1259/bjr/32314637

© 2010 The British Institute of Radiology

Cauda equina syndrome (CES), a rare complication of spinal or epidural anaesthesia, results from injury to sacral roots in the neural canal. Because the nerves in the cauda equina lack a protective sheath as they pass through the distal end of the dural sac, they are particularly prone to injury from high concentrations of local anaesthetics. Although laboratory findings have suggested that lidocaine, mepivacaine and ropivacaine may have greater potential for neurotoxicity than bupivacaine [1], several cases of CES have been reported in association with the intrathecal injection of bupivacaine [2]. To the best of our knowledge, no spinal MRI findings have been presented for nerve root injury caused by spinal anaesthesia.

Case report

A 29-year-old multiparous woman was admitted to the hospital in the 38th week of pregnancy for her second caesarean delivery. Her past medical history was unremarkable. On the second day of hospitalization, she went to the operating room. Combined spinal–epidural anaesthesia (CSEA) was performed in the left decubitus position with 2.2 ml 0.5% hyperbaric bupivacaine administered intrathecally at the L3–4 interspace, followed by placement of a multiorifice epidural catheter at the T8–9 interspace. An 18-gauge epidural catheter

was positioned 5 cm into the epidural space oriented in the cephalad direction to optimise cephalic spread of the anaesthetic. Surgery was allowed to proceed after sensory block from T5 to S5 was achieved. A total dose of 5.1 ml of 0.2% ropivacaine was administered using a patient-controlled epidural analgesia device over 75 min. Oxygen 3 l min⁻¹ was given through a face mask; 20 min elapsed between induction of anaesthesia and uterine incision. The surgical procedure was completed without incident and the estimated blood loss was 800 ml.

The patient did not fully recover from the motor and sensory blockade induced by CSEA, and at about 48 h post-partum was unable to walk because of lower extremity weakness. Neurological assessment confirmed sensory deficit to pinprick and vibration in the T3 to L5 distribution bilaterally with preservation of position sense. On the following day, she still exhibited hypaesthesia bilaterally in the T6 to L2 distribution with lower extremity weakness, which had shown minor improvement. Results of lumbosacral MRI without contrast medium were within normal limits, ruling out the possibility of spinal epidural haematoma or spinal infarction. Because the levels of sensory and motor dysfunction were nearly identical to the extent of the CSEA, a neurological complication related to lumbar epidural or subarachnoid infusion of anaesthetic was deemed the most likely cause of her symptoms; 7 days postoperatively, she continued to be unable to walk, void or defaecate spontaneously. A repeat MRI study with contrast medium was performed, which detected enhancement of the nerve roots from the T11–T12 to L3–L4 level (Figure 1). At the level of T12–L1 and L1–L2, the ventral nerve roots exhibited higher signal intensity than the dorsal nerve roots on post-contrast axial T₁ weighted images.

Address correspondence to: Miyuki Takasu, MD, Department of Radiology, Hiroshima Red Cross Hospital, 1-9-6, Sendamachi, Nakaku, Hiroshimashi 730-8619, Japan. E-mail: my-takasu@syd.odn.ne.jp

Figure 1. A 29-year-old woman with cauda equina syndrome after combined spinal–epidural anaesthesia. Contrast-enhanced sagittal (A) T_1 weighted magnetic resonance (MR) image reveals abnormal enhancement in the cauda equina. Contrast-enhanced axial T_1 weighted MR images show enhancement of the ventral nerve roots at the level of T12–L1 (B) and the level of L1–L2 (C).

Neither thickening of the nerve roots nor spinal cord abnormalities were demonstrated. Results of testing for autoantibodies such as antinuclear antibody, anti-ds-DNA antibodies, anti-Sjögren's syndrome A, anti-Sjögren's syndrome B and anti-neutrophil cytoplasmic antibodies were negative. An autoimmune process with nerve root involvement was therefore considered unlikely. Haematological examination revealed no abnormality suggestive of infection (white blood cell count, 6500 mm^{-3} ; C-reactive protein, 0.2 mg dl^{-1}). The patient was treated with high-dose corticosteroids, with gradual improvement of signs and symptoms. By the end of treatment, she was able to walk without assistance, but 6 months later she still had mild sensory disturbance of the lower extremities and pelvic area.

Discussion

Persistent neurological injury is a rare but significant complication of spinal anaesthesia. Four cases of CES after continuous spinal anaesthesia with 5% lidocaine or 1% tetracaine were reported in 1991 and generated concern regarding the neurotoxic potential of local anaesthetics administered intrathecally [3]. Although bupivacaine may be less neurotoxic than other local anaesthetics [1], 11 patients with CES attributable to bupivacaine were reported among the 1 710 000 cases of spinal and epidural blockade reviewed by Moen et al [4].

Several mechanisms have been proposed for neurological sequelae of spinal and epidural administration of local anaesthetics [1]. Sakura et al [5] intrathecally infused normal saline, 10% lidocaine or 2.13% bupivacaine in rats, and found that the specimens from rats given lidocaine or bupivacaine included axonal degeneration, disintegrated myelin lamellae and myelin ovoid. Yamashita et al [6] compared the effects of local anaesthetics on glutamate concentrations in cerebrospinal fluid microdialysate and neurological and histopathological outcomes in rabbits. They observed characteristic types of histopathological injury to the cauda equina and the spinal cord, with chromatolytic injury of motor neurons and vacuolation in the dorsal funiculus.

It is unclear why intense contrast enhancement was observed in the ventral nerve roots in our patient, despite the presence of dorsal root symptoms as well as motor disturbance. One possible explanation is unmyelinated fibres in the ventral nerve roots. Ko et al [7] reported that approximately 20% of human spinal ventral root fibres were unmyelinated. This suggests the possibility that the ventral nerve roots may be more severely affected than the dorsal roots by anaesthetics. Another possible explanation is that some agents injure the ventral nerve roots more strongly than the dorsal roots. Anderson et al [8] reported the case of a 3-year-old girl diagnosed with acute lymphoblastic leukaemia who developed subacute anterior lumbosacral polyradiculopathy following intrathecal methotrexate. Her spinal MRI revealed

lumbosacral ventral root enhancement, which the authors suggested could indicate selective ventral nerve root vulnerability to intrathecal chemotherapy. Their patient's electrodiagnostic studies demonstrated motor axonal loss, rather than demyelination. An effect of uneven dispersion of local anaesthetics in the subarachnoid space cannot be excluded as another possible cause, although the puncture level of spinal anaesthesia, which was more caudal than the levels of increased enhancements of the ventral roots, and the absence of the denticulate ligament below the level of T11, which could hamper homogeneous spread of anaesthetics, make this unlikely in our patient. The possibility that CSEA has an intrinsic potential to cause nerve root enhancement not correlated with our patient's clinical findings could not be completely excluded either.

The differential diagnosis of our patient's findings includes the pure motor and axonal variant of Guillain-Barré syndrome (acute motor axonal neuropathy (AMAN)). Byun et al [9] reported MRI findings for the spine in eight patients with Guillain-Barré syndrome and concluded that enhancement of the anterior spinal nerve roots alone is strongly suggestive of AMAN. However, the absence of an antecedent event, the short duration of time between the onset of symptoms and CSEA, the concordance in distribution of neurological disturbances with the extent of local anaesthetic delivery, the sphincteric involvement, and the prolonged neurological impairment did not support a diagnosis of AMAN in the present case.

Radwan et al [1] assessed the neurotoxicity of local anaesthetics including bupivacaine using dorsal root ganglia of chick embryos, and found that all local anaesthetics tested produced growth cone collapse and neurite degeneration. Although their findings cannot be directly extrapolated to *in vivo* conditions, the detrimental effects of local anaesthetics on growing or

regenerating neurons may have played a role in prolonging the clinical course in this case.

In conclusion, we have described a case of CES following the intrathecal injection of bupivacaine with contrast enhancement of nerve roots on spinal MRI. Although the reason for this finding is unclear, it may have involved ventral root vulnerability to bupivacaine.

References

1. Radwan IAM, Saito S, Goto F. The neurotoxicity of local anesthetics on growing neurons: a comparative study of lidocaine, bupivacaine, mepivacaine, and ropivacaine. *Anesth Analg* 2002;94:319–24.
2. Kubina P, Gupta A, Oscarsson A, Axelsson K, Bengtsson M. Two cases of cauda equina syndrome following spinal-epidural anesthesia. *Reg Anesth* 1997;22:447–50.
3. Rigler ML, Drasner K, Krejcie TC, Yelich SJ, Scholnick FT, DeFontes J, et al. Cauda equina syndrome after continuous spinal anesthesia. *Anesth Analg* 1991;72:275–81.
4. Moen V, Dahlgren N, Irestedt L. Severe neurological complications after central neuroaxial blockades in Sweden 1990–1999. *Anesthesiology* 2004;101:950–9.
5. Sakura S, Kirihara Y, Muguruma T, Kishimoto T, Saito T. The comparative neurotoxicity of intrathecal lidocaine and bupivacaine in rats. *Anesth Analg* 2005;101:541–7.
6. Yamashita A, Matsumoto M, Matsumoto S, Itoh M, Kawai K, Sakabe T. A comparison of the neurotoxic effects on the spinal cord of tetracaine, lidocaine, bupivacaine, and ropivacaine administered intrathecally in rabbits. *Anesth Analg* 2003;97:512–19.
7. Ko HY, Shin YB, Sohn HJ, Chang JH, Ahn YH, Ha YH. Unmyelinated fibers in human spinal ventral roots: C4 to S2. *Spinal Cord* 2009;47:286–9.
8. Anderson S, Baquis G, Jackson A, Monteleone P, Kirkwood JR. Ventral polyradiculopathy with pediatric acute lymphocytic leukemia. *Muscle Nerve* 2002;25:106–10.
9. Byun WM, Park WK, Park BH, Ahn SH, Hwang MS, Chang JC. Guillain-Barré syndrome: MR imaging findings of the spine in eight patients. *Radiology* 1998;208:137–41.