Anaesthetic management of a woman who became paraplegic at 22 weeks’ gestation after a spontaneous spinal cord haemorrhage secondary to a presumed arteriovenous malformation

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Summary
A 19-yr-old woman developed a paraplegia with a T10 sensory level at 22 weeks’ gestation. The spinal injury was caused by spontaneous bleed of a presumed arteriovenous malformation in the spinal cord. She presented for Caesarean section at term because of the breech position of her fetus. The successful use of a combined spinal epidural–regional anaesthetic is described and the risks of general and regional anaesthesia are discussed. (Br. J. Anaesth. 1998; 81: 976–978).

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The anaesthetic management of pregnant paraplegic patients has been well documented. However, there is no previous report of the anaesthetic management of a woman requiring a Caesarean section who had become paraplegic during her pregnancy after a subarachnoid bleed in the spinal cord.

Historically, subarachnoid bleeds have been implicated in 5–12% of maternal deaths,1–3 although more recent estimates suggest a slightly lower incidence.4 5 However, this mortality is almost entirely caused by intracranial bleeds. Spontaneous subarachnoid haemorrhage can also occur in the spinal canal. While spinal canal bleeds are less likely to be fatal, they may cause devastating neurological sequelae. These bleeds are usually caused by arteriovenous malformations (AVM).

The incidence of spinal canal AVM is 3.4–11.5% of all spinal tumours.6 7 They are either epidural or intradural, with intradural lesions being on the surface or in the substance of the cord. The average age at presentation is 57 yr for epidural AVM and 37 yr for spinal cord AVM. AVM are nine times more common in males than females.8

AVM have the potential for spontaneous haemorrhage or to cause cord ischaemia. Characteristic symptoms at presentation are leg weakness, sensory deficit and a disturbance of micturition and defecation. Nerve root pain may also occur. When these symptoms are caused by cord ischaemia, onset is usually slowly progressive, sometimes taking more than 1 yr to develop. In contrast, haemorrhage presents with an acute onset of symptoms and is the cause of presentation in approximately 10% of dural AVM8 and up to 45% of spinal cord AVM.9

The presence of a known spinal AVM is generally considered a contraindication to neuroaxial anaesthesia, although Ong and colleagues reported the successful management of a woman with a large asymptomatic cervical AVM who had a Caesarean section performed under spinal anaesthesia.10

Case report
A previously healthy 19-yr-old woman in her first pregnancy presented at 22 weeks’ gestation with radicular pain in both legs and a urinary tract infection. She was treated with antibiotics and analgesics and discharged. She returned 7 days later with leg weakness and urinary retention. Over the following 12 h she developed a T10 sensory level and near complete flaccid paralysis of her legs. She was referred for a neurosurgical opinion and magnetic resonance imaging, which revealed a bleed in the spinal cord at T10 consistent with a small AVM (fig. 1). Oedema caused mild swelling of the cord from T9 to T12, but no surface abnormality was seen on the cord or in the epidural space.

Surgery was not indicated and the patient was transferred to the regional spinal rehabilitation unit. Over the following weeks she had some recovery of power in her legs and could stand with the aid of a frame. She was eventually discharged home at 32 weeks’ gestation.

Her pregnancy was further complicated by the breech position of her fetus and therefore an elective operative delivery was planned. She was seen in our anaesthetic assessment clinic where she expressed a strong preference to be awake for the delivery.

A literature review was undertaken, the MRI scan was discussed and opinions sought from neurologists, neurosurgeons and anaesthetists at two spinal injury centres who advised first that regional anaesthesia was not contraindicated and second that the patient had a small risk of developing autonomic hyperreflexia in the perioperative period.

Women with spinal injuries commonly go into premature labour. Depending on the neurological deficit, the patient may be unaware of labour starting. This patient presented because her urinary catheter fell out. Early labour was diagnosed and a semi-elective Caesarean section performed.

A careful assessment was made of her neurological
case.

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Caesarean section after spinal cord haemorrhage

Deficit before anaesthesia was started. Routine monitoring of ECG, arterial pressure and oxygen saturation was established. If general anaesthesia had been used, we would have inserted intra-arterial pressure monitoring but felt that non-invasive arterial pressure monitoring at 1-min intervals was sufficient for the anticipated slower changes in arterial pressure associated with regional anaesthesia.

We wished to extend the regional block gradually to maintain haemodynamic stability. Additionally, to minimize pressure changes in the epidural space, we wanted to avoid using large volumes of epidural local anaesthetic. We therefore elected to use a combined spinal–epidural technique with a slightly lower dose than usual of intrathecal hyperbaric bupivacaine. By elevating the upper thoracic spine we could protect against rapid spread of local anaesthetic.

The patient was positioned in the left lateral position with a shoulder wedge to raise the upper thoracic spine. A “through needle” technique was used. At the L3–4 interspace, 2 ml of 0.5% hyperbaric bupivacaine with fentanyl 12.5 µg was injected intrathecally using a 25-gauge pencil point spinal needle. The spinal needle was withdrawn and the epidural catheter was gently inserted. Blood could not be aspirated through the catheter. The patient was then turned to the full right lateral position, again with a shoulder wedge.

After 20 min the block had extended to T9. A test dose of 3 ml of 0.5% bupivacaine was given via the epidural catheter. Five minutes later the block was assessed again and further increments of epidural bupivacaine were administered over 10 min until a total of 8 ml of 0.5% bupivacaine had been given. Forty minutes after the spinal injection, the block had extended to T2 to cold, T3 to pinprick and T5 to light touch. No ephedrine was given and systolic arterial pressure was maintained at 110–140 mm Hg. The efficacy of the block below the level of the lesion was confirmed by loss of ankle clonus and muscle tone in the legs. Interestingly, the patient complained of some dysesthesia at T12 despite anaesthesia to cutaneous stimulation. Surgery was uneventful and a live female infant was delivered.

After operation the patient was observed closely. As autonomic hyperreflexia could occur in the post-operative period, arterial pressure was assessed regularly and the patient was instructed to report any severe headaches. The regional block receded over a “normal” time course and neurological assessment confirmed that no new deficit had developed. The epidural catheter was left in situ in the event that it was required to control a hyperreflexic episode, but was removed after 12 h.

The patient made an uncomplicated recovery from her surgery and was discharged home 5 days later.

Discussion

Neurological deficits in patients with previously unknown and asymptomatic AVM have been reported after epidural11–13 and spinal14 anaesthesia in pregnant13 and non-pregnant patients.11 12 14 These deficits can be devastating and permanent. However, in each of these reports, cord ischaemia was suspected to have caused the neurological deficit. In our patient, spinal cord haemorrhage had already occurred. Our principal concern was to prevent further neurological damage and we suspected that, although a critical change in spinal cord hemodynamics could result in cord ischaemia, the greater risk was a re-bleed from her spinal cord lesion.

Regional or general anaesthesia could have been used. Both techniques carried potential risks. Regional anaesthesia can be associated with hypotension, especially when the onset of block is rapid. Epidural pressure may also be increased by injection of fluid into the spinal canal. Huselmeyer and White found that injecting 10 ml of 2% lidocaine over 30 s generated a mean increase in epidural pressure of 28.1 cm H$_2$O and in one patient produced a peak elevation of 73 cm H$_2$O.15 The combination of hypotension and increased epidural pressure may cause a critical decrease in perfusion pressure of an already compromised segment of cord, and treating hypotension with vasoconstricting agents could worsen cord ischaemia.

However, with a combined spinal–epidural technique, large volumes of epidural local anaesthetic are not usually required, minimizing any change in epidural pressure. In addition, by using a low dose of intrathecal local anaesthetic and carefully positioning the patient, onset of block could be controlled, minimizing haemodynamic instability. Indeed, during surgery, no vasoactive agents were necessary.
Regional anaesthesia may also cause haemorrhage. As the site of the AVM was known, direct trauma to the lesion itself was unlikely, but trauma to an epidural vein was possible. Extensive AVM generate high flow rates and can enlarge epidural veins along the whole length of the spinal canal. Disruption of an “arterialized” vein could result in a significant epidural haematoma. However, in this instance, the lesion was small and therefore the epidural vessels were unlikely to be enlarged.

Haemorrhage has been reported after diagnostic lumbar puncture. Loss of CSF from the dural sac can change spinal cord anatomy and place AVM under tension. However, with a 25-guage pencil point needle, the incidence of CSF leak sufficient to cause a postdural puncture headache is approximately 1%, and therefore the risk of producing significant tension on a spinal lesion would probably be much lower than this.

General anaesthesia was the alternative technique. We considered that the risk of further haemorrhage was greater with general anaesthesia because of the potential for hypertension, particularly during induction and intubation. Suppression of the hypertensive response to intubation with high doses of opioids or short-acting antihypertensive agents could have been used, but these techniques might themselves cause problems. A high-dose opioid technique would have compromised the standard rapid sequence induction which would have been modified to avoid using succinylcholine (suxamethonium) because of potential hyperkalaemia. However, in this patient, who was scored as Mallampati grade 1 and was adequately starved, the increased risk of aspiration would have been small. Using antihypertensive agents might have caused a period of rebound hypotension with the associated risk of cord ischaemia, particularly as deep anaesthesia would have been required to suppress autonomic hyperreflexia.

Autonomic hyperreflexia is a condition characterized by a disordered autonomic response to a stimulus below the level of a spinal cord injury. The common features are episodes of severe hypertension with or without reflex bradycardia, headache and occasionally profuse sweating. The higher the level of the spinal injury, the greater the risk of autonomic hyperreflexia. In patients with a T7 lesion there is an 85% incidence of autonomic hyperreflexia. Episodes may start any time from 3 months to 12 yr after the injury has occurred. By the time of surgery, our patient had not had a hyperreflexic episode. However, the lesion had developed recently and surgery would be a potent stimulus. General anaesthesia does not reliably control autonomic hyperreflexia unless deep anaesthesia is used, while regional anaesthesia, particularly spinal anaesthesia, does.

Trying to quantify these relative risks is almost impossible with little literature published. It must be remembered that regional anaesthesia is being performed regularly in patients who have undiagnosed lesions without problems. In this instance we felt that because the AVM was small and had already bled, the risks from general anaesthesia were at least as great as those from regional anaesthesia and therefore after discussion with the patient, we opted for regional anaesthesia.

We remain cautious about using regional anaesthesia in the presence of an AVM, and would always advise careful assessment of the potential benefits. In this particular instance, we have reported the successful management of a primiparous breech delivery by Caesarean section under combined spinal– epidural anaesthesia of a patient who had become paraplegic at 22 weeks’ gestation secondary to haemorrhage in the spinal cord.

References