

Monoplegia Following Obstetric Epidural Anaesthesia

C C Loo,*^{MBBS, FRCA}, K F Cheong,**^{MBBS, M Med (Anaes)}

Abstract

We describe a case of transitory monoplegia following obstetric epidural anaesthesia which we believe to be either due to direct trauma to the nerve roots or radicular artery spasm resulting in spinal cord ischaemia both secondary to the epidural catheter. An obstetric epidural was performed on a 34-year-old lady with no significant past medical history. Transient radicular pain down her left leg was experienced during the insertion of the catheter. She had unilateral block on her left side which could not be rectified despite injecting local anaesthetics in the right lateral position, resiting the epidural catheter, and administering a total dose of 6ml of 1.5% plain lignocaine and 19 ml of 0.25% plain bupivacaine. There was no hypotension, and no vasoconstrictor was used epidurally. She still had left-sided numbness and weakness 9 hours after removal of the catheter, but without urinary or bowel dysfunction. A magnetic resonance imaging scan excluded any space occupying lesion. She was managed conservatively and had complete recovery 35 hours after the last dose of local anaesthetics.

Ann Acad Med Singapore 1997; 26:232-4

Key words: Catheter, Complications, Labour, Paresis

Introduction

Neurological complications following epidural anaesthesia are relatively rare, but nonetheless well recognised potential complications. Holdcroft et al¹ reported an incidence of 1 in 13 007 obstetric epidural anaesthesia, while Crawford² reported an incidence of 1 in 2400. Dawkins³ reported an incidence of 0.1% transitory paresis and 0.02% permanent sequelae in 32 718 cases of epidural anaesthesia for various procedures. We describe a case of transitory monoplegia following obstetric epidural anaesthesia which we believe to be either due to direct trauma to the nerve roots or ischaemia of the spinal cord both secondary to the epidural catheter.

Case Report

A 34-year-old gravida 2 para 1 parturient at 39 weeks of gestation was admitted in labour. She had an uneventful antenatal history. There was no history of bleeding diathesis, backache or sciatica and was not on any anticoagulants or antiplatelet drugs. The patient consented to epidural anaesthesia for pain relief during labour. The patient was preloaded with one litre of Hartmann. The epidural anaesthesia was performed with the patient placed in a sitting position. Her back was cleaned with povidone iodine 1 %, methylated spirit

70%, and a sterile gauze was used to remove the anti-septic before injection. The epidural set (Braun: 18G Tuohy needle, Perican®; catheter and syringe for test of loss of resistance, Perifix®; injection filter, Sterifix®), needles and plastic syringes used were all disposables. The epidural space was identified between the 3rd and 4th lumbar intervertebral space at first attempt using a loss of resistance to air technique (2 ml of air was injected) and midline approach. Upon passage of the epidural catheter in a cephalad direction, she felt transient sharp radiating pain down her left leg. The pain was not present however, during the insertion of the Tuohy needle, injection of the test dose of 3 ml 1.5% plain lignocaine (Astra) or during the bolus dose of 8 ml 0.25% plain bupivacaine (Marcain™ 0.5%, Astra, diluted with normal saline 0.9%), 5 minutes later. A 4-cm length of the catheter was left in the epidural space. She did not experience any more pain once the epidural catheter was in position. An epidural infusion of 0.125% plain bupivacaine and 0.0001% fentanyl was commenced at 6 ml/hr. No vasoconstrictor was used epidurally. Her blood pressure and heart rate was monitored via a non-invasive blood pressure monitor at 3-minute intervals. After 30 minutes, the sensory block was noted to be unilateral, confined to the left side of the body extending

* Registrar

Department of Anaesthesia
Kandang Kerbau Hospital

** Senior Registrar

Department of Anaesthesia
National University Hospital

Address for Reprints: Dr Loo Chee Chuen, Department of Anaesthesia, Kandang Kerbau Hospital, 1 Hampshire Road, Singapore 219428.

from the T10 dermatome downwards. The lower limit of the block was not tested. There was a concomitant motor blockade of the left leg with power at the hip and knee being grade 2/5 (movement which is possible with gravity eliminated, Medical Research Council). There was no hypotension during this period. The patient was turned to the right lateral position with the unblocked segments dependent and a further 5 ml of 0.25% plain bupivacaine was given. This however, did not produce any sensory blockade of the right side of the body. One hour after insertion of the epidural catheter, in view of the persistent pain of contraction, it was decided that resiting the epidural catheter might alleviate the situation. This was performed again in a sitting position, at an interspace one level higher than the previous attempt. The epidural space was also easily identified at first attempt and the bevel of the Tuohy needle was directed 45° towards the right side of the body and in a cephalad direction before the epidural catheter was threaded through the needle. However, similar transient radiating pain down the left leg was again experienced by the patient which lasted only during the insertion of the catheter. A 4-cm length of the catheter was left in the epidural space. After an uneventful test dose of 3 ml 1.5% plain lignocaine and another bolus of 6 ml 0.25% plain bupivacaine 5 minutes later, it was noted that the sensory and motor blockade was still limited to the left side of the body. We did not however, assess the height of the block after the first top-up or after resiting the epidural catheter. After discussion with the patient, it was decided that intramuscular pethidine and entonox should be provided for the rest of the labour and the epidural catheter removed, 2 hours after the insertion of the second epidural catheter. Her systolic blood pressure was never below 100 mmHg throughout the duration of the labour. She delivered a healthy male infant some 4 hours thereafter. There was still significant numbness and weakness of her left leg about 11 hours since the last dose of local anaesthetics and a neurologist was consulted. He noted a sensory loss (pain and touch) of the left leg extending from the second lumbar to the second sacral dermatome and diminished muscle power of grade 2/5 of the hip and knee movements with hyporeflexic knee and ankle reflexes. There was no ankle clonus and the plantar reflex was normal. No testing of temperature or vibration senses was done. Anal tone was normal. There were no urinary or bowel dysfunction. Backache was not present. An immediate magnetic resonance imaging (MRI) scan to exclude an epidural haematoma was negative. In view of this, expectant management was carried out. The residual weakness and numbness gradually improved and she had full recovery 35 hours after the last dose of local anaesthetics, and was eventually discharged on the third postnatal day. A follow-up visit at 6 weeks postnatal did not show any residual neurological deficits.

Discussion

We believe that the cause of the unilateral neurological deficit was due to the epidural catheter as it corresponded to the paraesthesia radiating down her left leg experienced twice during the insertion of the catheter. The mechanism may be either due to direct trauma to the nerve roots or radicular artery vasospasm resulting in spinal cord ischaemia secondary to the catheter.

The spinal cord and nerve roots can be injured by the Tuohy needle and epidural catheter. The Tuohy needle may penetrate the ligamentum flavum too laterally, and the subsequent attempts to pass a catheter may injure the spinal roots and cause immediate complain of a unilateral paraesthesia. Cases of nerve trauma due to epidural catheter have been reported.⁴ Resiting of the epidural is therefore advisable. However, transient radicular pain or paraesthesia down the leg during epidural catheter insertion is not uncommon, and the opinion of the anaesthetists in our department is that they would leave the catheter in situ if the pain or paraesthesia resolve immediately after insertion. All agreed that they will remove the catheter if there is persistent pain or paraesthesia after the insertion.

The spinal cord receives its blood supply from 2 posterior and 1 anterior spinal artery and reinforced by radicular arteries arising from the aorta. Henson and Parsons⁵ pointed out that the old idea of the spinal cord being nourished by a long anterior spinal artery running the entire length of the cord and being reinforced by frequent radicular arteries whose sacrifice is unimportant, owing to a supposed rich anastomotic supply, is false. The sacrifice of a single radicular artery may lead to ischaemia as there are many watershed areas between the areas supply by all the arteries. Richardson and Bedder⁶ reported a case of transient anterior spinal cord lesion secondary to the radicular artery vasospasm due to irritation by the epidural catheter. Their patient also had unilateral limb numbness and weakness without bladder and bowel involvement. Computerised tomographic scan revealed the tip of the epidural catheter to be lying in a left-sided intervertebral foramen where the radicular vessels enter and leave the spinal canal, ipsilateral to the neurological deficit. Their patient recovered 12 hours after removal of the catheter. The catheter in our patient was removed prior to the MRI scan but it is highly likely that the catheter tip could also be in a left-sided intervertebral foramen causing vasospasm of the radicular artery. The other causes of ischaemia were unlikely as there was no hypotension and no vasoconstrictor was used.

Haematoma, abscess, incidental disc prolapse may lead to radicular pain, paraplegia and urinary and bowel involvement. Haematoma is more likely if the patient is receiving anticoagulants or has a bleeding diathesis.⁷ Our patient was not on any anticoagulant or antiplatelet

drugs and a space occupying lesion was excluded by the MRI scan.

Neurotoxic reaction may be due to the local anaesthetic per se, preservatives in the preparation or chemical contaminants resulting in arachnoiditis or cauda equina syndrome. Modern local anaesthetics like bupivacaine and lignocaine have not been shown to be neurotoxic in clinical doses. Accidental dural puncture or subdural injection causing adhesive arachnoiditis or cauda equina syndrome were also unlikely as there were no hypotension and "high" block. The absence of urinary and bowel involvement and the complete recovery after 36 hours were also not typical of adhesive arachnoiditis or cauda equina syndrome. Preservatives in multiple dose vials of local anaesthetic solution like methyl and propyl-paraben are suspected to be neurotoxic and are no longer recommended in caudal or epidural anaesthesia.⁸ Sodium bisulphite, a frequently used antioxidant in local anaesthetics containing adrenaline, was found to be neurotoxic in concentration 2 to 4 times higher than clinical doses.⁹ The lignocaine and bupivacaine used in our patient did not contain any preservatives nor antioxidants. Chemical contaminants like detergents (tribasic sodium phosphate) used for washing needles and syringes were found to be neurotoxic. Disposable epidural set, needles and plastic syringes were used in our patient and residual antiseptic on the skin was removed with a sterile gauze before injection.

Neurological deficits may also occur in normal vaginal or instrumental delivery in the absence of an epidural.¹⁰ The foetal head may compress the lumbosacral trunk or lumbar plexus resulting in postpartum foot drop, femoral and/or obturator neuropathy. The foetal head may also compress the blood vessels that supply the conus medullaris and cauda equina. There is a dual blood supply to the terminal part of the spinal cord. The major supply descends from the artery of Adamkiewicz and a secondary source ascends from the internal iliac arteries via the iliolumbar and lateral sacral arteries. The two sources meet in a small circulus that surrounds the tip of the conus medullaris. In the majority (85%) of the population, the major supply is from the artery of Adamkiewicz and the supply from the internal iliac arteries is expendable. In the remaining 15% of the population, the ascending iliac contribution assumes a

greater role in maintaining the vitality of the tip of the conus medullaris and the cauda equina. These ascending arteries lie close to the lumbosacral trunk. Therefore, the foetal head may cause obstruction to arterial flow, or obstetric instruments may cause arterial injury. The presentation is usually paraplegia. Our patient's neurological deficit extends from the second lumbar to the second sacral dermatome of her left lower limb and is not typical of peripheral nerve injury or paraplegia secondary to the compression of the lumbosacral trunk or lumbar plexus or blood supply to the conus medullaris.

In conclusion, we believe this case of acute transitory monoplegia with no urinary and bowel dysfunction, following obstetric epidural anaesthesia, was either due to direct trauma to the nerve roots or radicular artery vasospasm resulting in ischaemia of the spinal cord secondary to the epidural catheter. A catheter that causes persistent paraesthesia should certainly be removed before irreversible damage is done but management of transient paraesthesia experienced only during insertion of the catheter is less certain.

REFERENCES

1. Holdcroft A, Gibberd F B, Hargrove R L, Hawkins D F, Dellaportas C I. Neurological complications associated with pregnancy. *Br J Anaesth* 1995; 75:522-6.
2. Crawford J S. Some maternal complications of epidural analgesia for labour. *Anaesthesia* 1985; 40:1219-25.
3. Dawkins C J M. An analysis of the complications of extradural and caudal block. *Anaesthesia* 1969; 24:554-63.
4. Birkhahn H J, Heifetz M. A complication following epidural anaesthesia. *Anesth Analg* 1961; 40:650-2.
5. Henson R A, Parsons M. Ischaemic lesions of the spinal cord: an illustrated review. *Q J Med* 1967; 36:205-22.
6. Richardson J, Bedder M. Transient anterior spinal cord syndrome with continuous postoperative epidural analgesia. *Anesthesiology* 1990; 72:764-6.
7. Spurny O M, Rubin S, Wolf J W, Wu W Q. Spinal epidural hematoma during anticoagulant therapy. Report of two cases. *Arch Intern Med* 1964; 114:103-7.
8. Barnhart E R. Physicians' Desk Reference. 42nd ed. Oradell, New York: Medical Economics Company Inc, 1988:2233-9.
9. Ready B, Plummer M H, Haschke R R, Austin E, Sumi M. Neurotoxicity of intrathecal local anesthetics in rabbits. *Anesthesiology* 1985; 63:364-70.
10. Bademosi O, Osuntokun B O, Van de Werd H J, Bademosi A K, Ojo O A. Obstetric neuropraxia in the Nigerian African. *Int J Gynaecol Obstet* 1980; 17:611-4.