CASE REPORT

Lower limb weakness and paresthesia after combined spinal epidural anesthesia for abdominal hysterectomy: a report of three cases

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ABSTRACT

Neurological deficits are the rare but unacceptable complications of neuraxial blockade. We report three cases of vaginal hysterectomy performed under combined spinal epidural anesthesia (CSE) using 3 ml of 0.5% hyperbaric bupivacaine (15 mg) in subarachnoid space followed by epidural analgesia top up after wearing off of spinal anesthesia. One patient complained of unilateral paresthesia and numbness on left thigh with no motor involvement in the evening postoperatively, two patients developed bilateral paresthesia and numbness over anterior thigh and knees and motor weakness in both lower limbs on next day morning. Epidural catheter was removed immediately and treated with oral tab prednisolone and tab methylcobalamin. All patients had complete recovery and were discharged after a week. Unrecognised mechanical irritation of the nerve roots by epidural catheter is thought to be the cause. We conclude that patients with epidural catheter should be monitored and on appearance of any neurological symptoms the catheter be removed to prevent permanent neurological sequelae.

Key words: Anesthesia, Epidural; Anesthesia, Spinal; complications; Paresthesia; Somatosensory Disorders; Sequelae


INTRODUCTION

In the recent years combined spinal epidural (CSE) anesthesia has become more popular as it provides anesthesia as quickly as single shot subarachnoid block, allows intraoperative epidural supplementation and prolonged postoperative analgesia.

Incidence of neurologic injury has been reported to be 0 to 0.16% of all spinal and epidural anesthesia; persistent paresthesia and limited motor weakness being the most common presentation of neurological sequelae.1 Higher incidence of paresthesia during CSE (0.9 – 11%) is a recognised factor.2 Neurological complications following neuraxial anesthesia can be caused by hematoma formation, infection, traumatic placement and drug toxicity. Other contributing factors are spinal cord ischemia due to the use of vasoconstrictors, prolonged hypotension and lithotomy position.1

The cases of sensory and motor impairment following epidural catheterisation have been reported. Some showed complete recovery after catheter removal;3 however, in few patients sensorimotor symptoms persisted.3,4

We report a case series of three female patients who underwent vaginal hysterectomy in lithotomy position under CSE anesthesia. They developed paresthesiae, numbness and motor weakness of lower limb within 24 hours postoperatively. Possible mechanisms, treatment and outcome are discussed.
neurological sequelae of CSE

CASE REPORT 1
A 62 year, 60 Kg female patient of ASA I, had uterovaginal prolapse with cystoele and was taken for vaginal hysterectomy with pelvic floor repair. Pre-anesthetic evaluation including history, physical examination and investigations were unremarkable. Patient was shifted to OR with 8 hour fasting status and preloaded with 500 ml Ringer lactate solution via 18G peripheral IV cannula. Standard ASA monitoring was done using noninvasive blood pressure, pulse oximetry and electrocardiography. After recording baseline vitals, patient was placed in right lateral position. Tuffier's line (line crossing anterior superior iliac spine) was identified and taken as a landmark to correspond to L4-5 interspace. Under aseptic precautions skin was infiltrated with 2% lidocaine (3 ml) using 5 ml disposable syringe with 24G needle in midline. Epidural space was identified with 18G Tuohy needle using loss of resistance to normal saline technique in a single attempt. Then a 27G Whitacre needle was passed via Tuohy needle to reach subarachnoid space. It took three attempts to locate subarachnoid space and get the free flow of CSF, then 3 ml of 0.5% hyperbaric bupivacaine (15 mg) was injected in subarachnoid space. Spinal needle was removed and 18G epidural catheter was inserted upto 5 cm into the epidural space. Sterile dressing was applied, catheter was fixed and the patient was turned supine. After 5 min, sensory level of T6 was achieved and she was placed in lithotomy position. Surgery was allowed to start after 10 min of spinal injection. Surgery lasted for 60 min and a total of 1500 ml Ringer’s Lactate solution was infused. As per institutional protocol of post-operative epidural analgesia, 50 µg fentanyl diluted in 5 ml was given through epidural catheter at the end of surgery, which was to be repeated 12 hourly till 48 hours. The patient recovered from spinal anesthesia completely in 3 hours.

When patient was attended for epidural drug administration in the evening, she complained of feeling of tingling and numbness in right anterior thigh and around the right knee. Motor examination was normal. Epidural catheter was removed and she was assured regarding reversible nature of the symptoms and treated with oral prednisolone 20 mg twice daily and methylcobalamin 1500 µg once daily for 5 days. She showed gradual recovery starting on next day of the treatment, recovered completely over 4 days and was discharged on 7th day.

CASE REPORT 2
A 50 years old 55 Kg patient underwent vaginal hysterectomy under CSE as described in case report 1. In this case all steps of CSE procedure were completed easily in single attempt and surgery lasted for 70 min. Sensory and motor recovery occurred completely in three and a half hours. She was pain and symptom free in the evening when she received booster dose of fentanyl through epidural catheter. On the next morning when she tried to stand up on the floor, she could not and fell down. On examination power of hip flexors was found grade 1 and that of knee extensors to be grade 2. There was tingling and numbness around both of her knees and anterior aspect of thighs. Epidural catheter was removed and treated with oral steroid and methylcobalamin for 5 days as in case 1. Signs of recovery started within 6 hours and she completely recovered over 5 days and was discharged after 7 days.

CASE REPORT 3
A 40 years old 50 Kg female patient underwent vaginal hysterectomy under CSE as described above. In this case also, all steps of CSE procedure were performed in single attempt and the surgery lasted for 75 min and she recovered completely from spinal anesthesia in 4 hours. She received two doses of 50 µg fentanyl diluted in 5 ml through epidural catheter on that day. On next day morning, she complained of bilateral numbness on thighs and knees. Neurological examination revealed hypoesthesia over anterior side of thighs bilaterally and around the knee joints. Bilateral knee jerks were absent. Ankle jerks were normal. Power of hip flexors and knee extensors was grade 3. Epidural catheter was removed and steroids and methylcobalamin were started. She recovered completely over 5 days and was discharged after 7 days.

In all the three cases CSE block was performed by II year resident anesthesiologist, who was well experienced with CSE technique, under direct supervision of consultant. None of the patients was on anticoagulation treatment and coagulation profiles were all normal. None of the patients complained of any paresthesia during epidural catheter placement or spinal needle placement. All patients received 50 µg fentanyl diluted in 5 ml normal saline, via epidural catheter at the end of surgery. Case 1 received only single dose, while case 2 and 3 also received the second doses in the evening. After removal of the epidural catheter in
all of the three patients, postoperative pain was managed with inj. diclofenac 75 mg IM and inj. tramadol 100 mg IV infusion was given as rescue analgesic as per institutional protocol of acute pain services.

DISCUSSION

Needle or indwelling catheter induced trauma and local anesthetic neurotoxicity have been described to be the etiology of most of the neurological complications following neuraxial block. Other significant causes described are infections, hematoma, transient neurological symptoms (TNS), lithotomy position, etc.¹

In our present case series most likely explanation of sensory and motor symptoms seems to be mechanical irritation of the nerve roots by epidural catheter, which remained unrecognised during placement because of prior spinal anesthesia in CSE. It has been reported that if paresthesia occurs during needle or catheter placement there is significantly increased risk of persistent paresthesia afterwards (P<0.001). In the single space, needle through needle technique of CSE, the insertion of epidural catheter after administration of spinal local anesthetics may prevent identification of paresthesia that may warn the anesthesiologist about catheter misplacement.²

We kept our first diagnosis as catheter impinging on nerve roots, which was removed immediately as neurological symptoms appeared. Steroids and methylcobalamin were administered for treatment as described by others.³ Corticosteroids have direct anti-inflammatory effects, reducing the tissue concentration of inflammatory mediators that activate nociceptors. They also reduce the aberrant firing that can originate from sites of nerve injury.

One important limitation of our case series was that MRI was not done to reach at definite diagnosis, as economic constraints remain a limiting factor to get MRI in every suspected case. We decided to proceed for MRI and neurologist opinion if the symptoms did not recover or got worst.

Hematoma formation is a rare (<1 in 150,000) complication of spinal or epidural anesthesia. Patients with hematoma generally do not recover from spinal anesthesia, persistent motor weakness is most common presentation and they need emergency surgical decompression. We excluded spinal/epidural hematoma in these cases without MRI because our cases showed complete recovery from spinal anesthesia in 3-4 hours, neurological symptoms were mainly sensory with mild motor weakness in two patients and all showed complete recovery after catheter removal.

Similar to our cases Zeidan et al⁴ described a case of transient postoperative neurological symptoms in a 6 year old male child who underwent radical nephrectomy. Under general anesthesia, epidural catheterisation was done for intra and postoperative analgesia using bupivacaine and morphine. Next morning on 1st postoperative day, he had severe pain in the right groin radiating to anterior surface of the thigh (L2 dermatome) with no motor block. The epidural catheter was removed and a complete neurological recovery was seen within 24 hrs. Mechanical irritation of the nerve root by the epidural catheter due to its longer length was described as the most likely explanation. The length of the epidural catheter introduced into the epidural space (5 cm) was too long for the child and the pain or paresthesia elicited during needle or catheter misplacement may have been hidden by general anesthesia. In our three cases epidural catheter was inserted for 5 cm in epidural space which is not too long for adults⁸.

As per institutional protocol Tuffier’s line was used as an anatomical landmark technique for identification of intervertebral space. There is evidence that using Tuffier’s line as a landmark may be misleading and unintentional cephalad needle insertion may occur damaging the cauda equina nerves or conus medullaris. Ultrasound guided space identification reduces chances of error. However we do not have ultrasound guidance as our hospital is in tribal area.

If the needle is off midline, nerve roots may be injured. Once mechanical damage has occurred, the spinal cord is more susceptible to further damage by agents that would under normal circumstances not be regarded as noxious, e.g. local anesthetics and epinephrine. However, in our case series symptoms of patients were not suggestive of cauda equina syndrome which include severe back pain, saddle anesthesia involving S3,4,5, bladder and bowel incontinence, sciatica type of pain including perineum, external genitalia, anus, lower limb paraplegia, absent ankle reflex, anal reflex and bulbocavernous reflex.⁹

Another differential diagnosis could be transient neurological symptoms (TNS) which is defined as pain and / or dysesthesia in the buttocks or legs following spinal anesthesia, with an incidence of 0 % to 37 %, with repeated high doses of lidocaine
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and tetracaine rather than bupivacaine. We used bupivacaine in spinal anesthesia which is less associated with TNS. Local anesthetics injected into epidural space may undergo transmeningeal transfer into the CSF mostly via the arachnoid villi in the dural cuff region. Repeated injections or continuous infusions may result in increased intrathecal concentration of the local anesthetic in the CSF causing this neurotoxic effect even after epidural analgesia. In our cases, we did not use local anesthetics in epidural analgesia excluding the drug toxicity as a cause of TNS. We used fentanyl for epidural analgesia. A variety of neurological symptoms after epidural and intrathecal fentanyl have been reported but they were dysphagia, perioral numbness, Horner’s syndrome, nystagmus, claw hand etc. These symptoms were attributed to cephalad spread of fentanyl exhibiting local anesthetic effect by reversible blockade of voltage gated sodium channels.

Another important differential diagnosis could be prolonged lithotomy position under regional anesthesia that had been blamed for femoral neuropathy and TNS. In this position there is flexion abduction and external rotation at hips, thus pressing the stretched inguinal ligament. Compression of its blood supply may lead to local ischemia of nerve trunk. Stretching of the lumbosacral nerves, render them vulnerable to the toxic potential of local anesthetics. Gupta et al described two cases of vaginal hysterectomy in epidural and spinal anesthesia lasting for 3 hours 30 min and 2 hours respectively, who developed bilateral numbness and weakness of lower limb on 2nd postoperative day. They were also treated successfully with methylcobalamin, amitryptiline and corticosteroids. They attributed neurological sequelae to prolonged lithotomy position causing femoral neuropathy. In our cases lithotomy position cannot be the sole etiological factor for the neurological symptoms as lithotomy position lasted only for 60 – 75 min.

Permanent neurological injuries have also been reported after spinal / epidural anesthesia. Wilkinson reported three cases who sustained intrinsic spinal cord lesions after attempted epidural catheterisation. In each case there was an early onset of motor and sensory impairment after the procedure and MRI demonstrated similar, extensive, paracentral, high signal intensity lesions within the cord on T2 weighted images. They proposed that the most likely cause of these lesions was direct trauma to the spinal cord during the procedure and subsequent injection of fluid into the spinal cord producing localised hydromyelia. The prognosis in each case was for a gradual recovery of motor function but spinthalamic sensory impairment and severe spontaneous pain over the affected area persisted.

In our three cases, as the neurological symptoms appeared, prompt removal of catheter and early treatment prevented the development of permanent neurological damage. Laboratory studies have demonstrated demyelination and inflammation adjacent to the catheter tract in both the spinal root and cord of rats following placement of indwelling subarachnoid catheters.

CONCLUSION

We conclude that postoperative epidural analgesia administered via single space needle through needle technique of CSE should be carefully monitored for development of sensory and motor symptoms in postoperative period because in these patients insertion of epidural catheter after administration of spinal local anesthetics may prevent identification of paresthesias that may warn about catheter misplacement. If neurological symptoms arise postoperatively, catheter should be removed immediately to prevent permanent neurological sequelae. MRI should be done to confirm diagnosis. Prior to epidural catheter placement, patients should be well informed regarding possible neurological complications and consent should be obtained to fulfil medicolegal requirements.
REFERENCES


