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Case Report

A Rare Case of Transient Foot Drop Post Spinal Anesthesia

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ABSTRACT

The preferred anesthetic technique for cesarean section is spinal anesthesia. Neurological complications following spinal anesthesia are very rare and often transient. Here we present the case of 23 yr old female who underwent emergency LSCS under spinal anesthesia and developed foot drop 48 hours post operatively. Patient was thoroughly evaluated and started on rehabilitation and followed up regularly. Early clinical assessment and diagnostic interventions is of prime importance to establish the etiology and to start appropriate management.

Keywords: Spinal anesthesia; Neurological complications; Rehabilitation

INTRODUCTION

The most common technique of anesthesia for caesarean section is subarachnoid block also known as spinal anesthesia. Neurological complications following regional anesthesia are rare. The prevalence of these complications following epidural anesthesia and spinal anesthesia is about 0–36 per 10,000 and 35 per 10,000 cases respectively [1,2]. In a study conducted by Scott and Tunstall, the neurological deficits were reported in 8 out of 14,856 deliveries with subarachnoid block (0.054%), all of which were transient [3]. Foot drop is caused by injury to lumbosacral trunk and damage to common peroneal nerve. The symptoms of this neurological disorder include unilateral movement disorders of the ankles with sensory impairment or paresthesia. However, this type of complication is rare after caesarean section [4].

CASE REPORT

A 23 yr old primigravida at 40 weeks of GA with 150 cm height and 57 kg weight was posted for emergency caesarean section due to grade 3 meconium stained liquor. Thorough pre-anesthetic check-up was performed and routine investigations performed according to institutional protocol were checked. Patient was categorized into ASA physical status 2. After taking written informed consent, patient was shifted to operation room, standard ASA monitors were attached and IV fluid started via 18G cannula in left upper limb. Patient was positioned in left lateral decubitus position for spinal anesthesia. Under aseptic precautions spinal anesthesia (2 ml of 0.5% hyperbaric bupivacaine) was performed at L3-L4 intervertebral space using a 25G Quinke needle after confirming clear and free flow of CSF. Level of blockade was confirmed with sensory level attained till T4 and motor modified Bromage score 3, after which surgery was started. Duration of surgery was 45 minutes with blood loss of 700 mL and 1500 mL of Ringer Lactate was given i.v. Intra operative period was uneventful and patient was shifted to post anesthesia care unit with stable hemodynamics. After 90 minutes sensory level was T10 and motor blockade returned to modified Bromage score of 0 in 180 mins. Patient was monitored in PACU for a period of 24 hours before shifting towards.

On post op day 2 patients complained of weakness and par aesthesia in the left lower limb. On examination patient had grade 2/5 power of left ankle dorsiflexion and sensory deficit along L4, L5 and S1 dermatomes. Right lower limb was normal on examination. Lumbar MRI was done which was inconclusive. Motor Nerve conduction study of the left lower limb was done (Table 1). Left peroneal nerve F-waves were absent (Table 2). Patient was started on steroids (intravenous methylprednisolone 500 mg daily for 3 days and gradually reduced over the next days), anti-inflammatory drugs, electrical stimulation and physiotherapy. Complete recovery of sensory deficit was achieved by post-operative day 5. Patient was followed up every 7 days for a period of 3 months. Two months post -operatively, patient recovered to power 4/5 and was able to walk without support. Written informed consent was taken from patient for publication of her case details as per journal guidelines.

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Nerve	Site	Latency 1 (ms)	Duration (ms)	Amplitude (mV)	Nerve conduction velocity ($m \cdot s^{-1}$)
Left peroneal nerve	Ankle	3.5	10	6.9	52.8
	Knee	8.8	10.4	5.6	57.1

Table 1: Motor nerve conduction study.

Nerve	Fmin latency (ms)	Fmax latency (ms)	Fmean latency (ms)
Right Peroneal	41	3	38
Left Tibial	43.5	3.4	40.1
Right Tibial	43.4	3.2	40.2

Table 2: F- wave.

DISCUSSION

Spinal anesthesia is frequently employed technique for caesarean delivery. The most encountered adverse effects of this technique include hemodynamic changes, back pain, nausea, vomiting, and Post Dural Puncture Headache (PDPH). Neurological complications due to spinal anesthesia are uncommon and short-lived. The prevalence is about 3.5% [1]. The causes for neurological adverse effects are direct needle trauma, local anesthetic-induced neurotoxicity and hematoma.

In a study, 24 of the 71,053 patients with neuraxial blocks had neurological deficits [5]. Repeated attempts, needle placement or injection contributed to lumbosacral nerve injury leading to paraesthesia [6,7]. A similar study showed that two-thirds of patients who had neurological adverse effects complained of pain during needle placement or local anesthetic injections.

Foot drop usually occurs as a result of injury to common peroneal nerve, sciatic nerve injury radiculopathy, lumbosacral lesions, or cauda equina syndrome [8]. Epidural hematoma, epidural abscess, meningitis, and anterior spinal artery syndrome are some more causes of foot drop. A retrospective study showed that 17 out of more than 10,000 patients with subarachnoid blockade had permanent nerve deficits for up to 1 year [9]. Nerve paralysis following labor is three to four times more common after regional anesthesia. The common cause for postpartum nerve injury includes pressure between the fetal head and sacral trunk, which occurs as a result of an inappropriate lithotomy position.

The early diagnosis of postoperative foot drop can be made in susceptible patients by thorough follow up and clinical examination. Magnetic Resonance Imaging (MRI) has to be done to confirm spinal injury. The Nerve conduction study and electromyography is advised to know its neurological or muscular origin respectively. Timely start of anti-inflammatory agents and physiotherapy form an integral part of treatment of these complications.

The onset, recovery time rate, and extent of neurological symptoms vary among patients. In a study by Auroy et al. on 1,03,000 patients with subarachnoid blockade, the neurological deficits began to appear within 48 hours, and their recovery took from 2 days to 3 months [5]. In our case, the complication appeared 48 hours after recovery from spinal anesthesia and the patient regained 75% of ankle function 2 months post operation.

CONCLUSION

We advise a thorough neurological examination of the lower limbs before performing spinal anesthesia. It is important for immediate diagnosis of acutely developed foot drop following spinal anesthesia as early initiation of treatment is essential for improved long term outcomes in such patients.

REFERENCES

1. Brooks H, May A. Neurological complications following regional anaesthesia in obstetrics. BJA CEPD Rev. 2003;3(4): 111-114.
2. Moen V, Irestedt L. Neurological complications following central neuraxial blockades in obstetrics. Curr Opin Anaesthesiol. 2008;21(3): 275-280.
3. Scott DB, Tunstall ME. Serious complications associated with epidural/ spinal blockade in obstetrics: A two-year prospective study. Int J Obstet Anesth. 1995;4(3): 133-139.
4. O'Neal MA, Chang LY, Salajegheh MK. Postpartum spinal cord, root, plexus and peripheral nerve injuries involving the lower extremities: A practical approach. Anesth Analg. 2015;120(1): 141-148.
5. Auroy Y, Narchi P, Messiah A, Litt L, Rouvier B, Samii K. Serious complications related to regional anesthesia: Results of a prospective survey in France. Anesthesiology. 1997;87(3): 479-486.
6. Faccenda KA, Finucane BT. Complications of regional anaesthesia incidence and prevention. Drug Saf. 2001;24(6): 413-442.
7. Horlocker TT. Complications of spinal and epidural anesthesia. Anesthesiol Clin North America. 2000;18(2): 461-485.
8. Reynolds F. Damage to the conus medullaris following spinal anaesthesia. Anaesthesia. 2001;56(3): 238-247.
9. Dripps RD, Vandam LD. Long-term follow-up of patients who received 10,098 spinal anesthetics: Failure to discover major neurological sequelae. J Am Med Assoc. 1954;156(16): 1486-1491.