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Case Report

Neurological deficit following spinal anaesthesia for caesarean section: an anesthetist's nightmare

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ABSTRACT

Neurological deterioration after regional anaesthesia, though extremely rare has been reported worldwide. A number of mechanisms like intraspinal hematoma, meningitis, cauda equina syndrome, etc. have been attributed to this dreadful complication. We present a case of a 26 year old female with no past medical history who was posted for emergency caesarean section. After an uneventful surgery under a subarachnoid block, the patient was discharged on the third day. However, on tenth postoperative day, the patient presented in casualty with bilateral lower limb weakness and backache. What was initially thought of being a rare post-operative complication of spinal anaesthesia was found to be an incidental intradural lesion on subsequent evaluation. Even a thorough pre-anaesthetic checkup may not prevent the occurrence of such incidents postoperatively.

Keywords: Caesarean section, Neurological deficit, Spinal anaesthesia

INTRODUCTION

The choice of anaesthesia in caesarean sections for good intraoperative and post-operative pain relief is spinal neuraxial blockade.

The extension of the subarachnoid space into the intra-cranial cavity can cause mechanical changes, in the intra-cranial hemodynamics and precipitate presentation of a dormant intra-cranial space occupying lesion. However it is fortunate that this is an extremely rare complication in today's medical scenario.

Literature says there are few other causes for spinal anesthesia related neurological deficit other than direct needle injury to the spinal cord or nerves which includes spinal cord ischemia, neurotoxicity due to drugs administered, spinal hematoma formation and infection.[1] Here we report a case of sudden onset neurological deficit due to an intradural spinal cord lesion discovered incidentally post operatively.

CASE REPORT

A 26 year old female patient was posted for emergency caesarean section, the indication being previous caesarean section with failed induction. After a routine pre anaesthetic checkup and aspiration prophylaxis with injection metoclopramide 10 milligrams i.v., the patient was taken up for surgery.

With the patient in the left lateral position, spinal anesthesia was performed at the L3-L4 interspace with a 25G quincke's spinal needle using all aseptic precautions. Clear, free flowing cerebrospinal fluid was obtained in a single atraumatic attempt and 1.8 mL of 0.5% heavy bupivacaine was injected and surgery was concluded uneventfully with the T4 dermatome being the maximum sensory height.

There was no pain or paresthesia during the needle placement or drug injection. She was admitted in the Post-Anesthesia care Unit until the regression of the

motor block. Patient was discharged on third day following the surgery. However, on the tenth postoperative day, the patient presented in the emergency room with severe lower backache and bilateral lower limb weakness. On examination, motor power in bilateral lower limb was 1/5 and superficial and deep tendon reflexes were depressed.

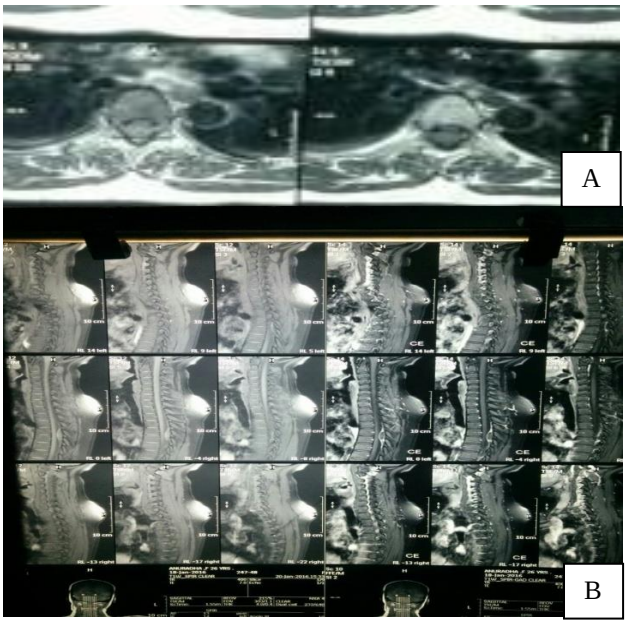

Figure 1: MRI shows a well-defined fusiform extramedullary intradural lesion (22x14x8 mm) at the level of D5-D6 vertebra along the posterior aspect of spinal cord appearing heterogeneously hyperintense on long TR/TE sequence and iso intense in T1W image with respect to spinal cord. Peripheral rim enhancement present with mild thickening of dura (Dural tail sign).

A referral was sent to the anaesthetist as well as physician on call. After consulting with neurologist, the patient was started on intravenous steroids. On the following day, MR imaging of the spine was done which revealed a well-defined, oval shaped extramedullary intradural mass lesion which was 22 ×14× 8 mm at the level of D5-D6 vertebrae along the inner aspect of spinal cord, with rim enhancement with significant secondary spinal canal narrowing at same level with mild compression and anterior displacement of spinal cord (Figure 1). The patient showed signs of improvement from the third day of receiving intravenous steroids. Patient was referred to higher centre for further management where the lesion was removed surgically.

DISCUSSION

Neurological complications of regional anaesthesia have been recognised since Bier reported the first spinal anesthetic over 100 year ago. Incidence of neurological central neuraxial blockade (CNB) complications is estimated to be between 1/1000 and 1/1,000,000. The incidence of complications was comparatively higher for spinal than epidural anesthesia.[2] Spinal anesthesia has been the anesthesia of first choice for caesarean sections. Though it has been considered a relatively safe form of anesthesia, it still can have complications like hypotension, post dural puncture headache, backache, total spinal block, cardiovascular collapse and various neurological complications. These neurological complications are fortunately rare amounting to 0.03%.[3]

In 1946, Nicholson et al mentioned the possible neurological complications of spinal anesthesia in the presence of previously undiagnosed spinal space occupying tumors. The authors considered the neurological complications mainly attributable to the occurrence of neurotoxic effects caused by the injected anesthetic agent which could not be diluted appropriately, due to impaired flow of cerebrospinal fluid caused by the lesion.[4] However, the precipitation of such symptoms occurs within a few hours of giving the intrathecal drug. In present patient, the occurrence of the neurological impairment after 10 days rules out the above mechanism.

Serious neurologic complications as anterior spinal artery syndrome, transverse myelitis, caudaequina syndrome with an incidence of one in 10,000 cases following either spinal or epidural anaesthesia. However these neurologic lesions can occur spontaneously in absence of anaesthesia or following general anaesthesia.[5] In year 2000 in a study Johnson ME stated that spinal lidocaine is also implicated in the syndrome of transient neurologic symptoms (previously referred to as transient radicular irritation), in which patient presented with pain or dysesthesia in the buttocks or legs after recovery from anesthesia. Although the pain typically resolves within 1 week without lasting sequelae, it can be severe in up to one third of patients with the syndrome.[6] Following many such reports, the use of hyberbaric lidocaine has been discontinued in spinal anesthesia.

Arachnoiditis, another rare complication of neuraxialanaesthesia may appear as transient nerve root irritation, caudaequina, and cones medullaris syndromes. Patients usually presents with pain in the lower back, dysesthesia and numbness not following the usual dermatome distribution on the contrary our patient presented with symmetrical weakness with dermatomal distribution.[2]

A case of patient who developed transverse myelitis following spinal cord injury, secondary to spinal anesthesia has been reported. In this case as soon as the needle was inserted, the patient felt a severe, painful, electric shock-like sensation in both lower limbs, which was bilaterally symmetrical, lasting for one to two minutes. Approximately six hours after the operation, the patient developed retention of urine and more than 1.2 liters of urine was evacuated when the indwelling catheter was passed. The patient observed his lower limbs had become absolutely numb and weak.[7]

CONCLUSION

Study concludes that while the frequency of severe, permanent neurological complications associated with spinal anesthesia has been reported to be extremely low, such complications can occur. The regression of sensory as well as motor block in all patients receiving regional anesthesia should be monitored in the post anesthesia care unit and the nursing staff should be well aware of the alarming signs to watch for. Any patient in whom there is neurological deficit is present post regional anesthesia, appropriate imaging studies such as an MRI should be done which may demonstrate the exact pathology.

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