Transverse myelitis following spinal anesthesia

Sanjeev Jha, Rajesh Kumar

Department of Neurology, Sanjay Gandhi PGIMS, Lucknow, UP, India

Spinal anesthesia is widely used during surgical procedures. It is generally safe and the frequency of severe, permanent neurological complications associated with it has been reported to be extremely low. We report a patient, who developed paraplegia following spinal anesthesia. A 29-year-old male was referred with acute, flaccid, sensory motor paraplegia, with bladder and bowel involvement. He developed this immediately after an operation for inquinal hernia under spinal anesthesia. Spinal magnetic resonance imaging revealed hemorrhagic myelitis in the conus at D₁₂. He was referred after he did not respond to intravenous methylprednisolone for 10 days. This case brings up the difficulty encountered in determination of the interspace used for spinal anesthesia and the potential for traumatic injury to the spinal cord. It also demonstrates the tragic outcome after a clinician violates some important, standard and established guidelines.

Key words: Hemorrhagic myelitis, spinal anesthesia, spinal cord injury, transverse myelitis

Introduction

Spinal and epidural injections for anesthesia and analgesia are widely used during surgical procedures. They are generally regarded as safe and the frequency of severe, permanent neurological complications related to them, based on prospective and retrospective studies, seems to be extremely low (roughly less than 0.01%). A wide array of temporary and permanent neurological complications has been reported with spinal anesthesia which includes cauda-equina syndrome, sciatic nerve palsy, transient paraplegia, quadriplegia, brain damage and even death.^[1,2] This injury can result from direct trauma by needle, toxicity of anesthetic agent, epidural hematoma and ischemia from arterial injury or severe hypotension.^[2,3]

We report a patient who developed transverse myelitis following spinal cord injury, secondary to spinal anesthesia. It is important for anesthesiologist, surgeon and neurologist to be aware of transverse myelitis as a complication of spinal anesthesia. This case also brings up the difficulty encountered in determination of the interspace used for spinal anesthesia and the potential for traumatic injury to the spinal cord.

Case Report

A 29-year-male was operated for left side inguinal hernia, under spinal anesthesia in the night around 22.00 hrs. As soon as the needle was inserted, the patient felt a severe, painful, electric shocklike sensation in both lower limbs, which was bilaterally symmetrical, lasting for one to two minutes. The operation continued and the hernia was repaired. The next morning, approx 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 so that he was unable to move them at all. As there was no response with high-dose (1 g daily for 10 days) of methylprednisolone (MPS), he was referred after two weeks of operation to our tertiary care center.

Examination revealed stable vitals. He had paraplegia with power Grade 0/5, hypotonia Grade 1 and areflexia in both lower limbs. Planters were nonelicitible. There was complete loss of all modalities of sensation below T_{11} cord level. Upper limbs were normal.

A well-trained and experienced anesthesiologist had performed the procedure. When contacted he informed that he had used appropriate, external landmarks (iliac crest) for lumbar puncture at L_3 - L_4 interspace and undiluted, 5% lignocaine was administered for spinal anesthesia using a 25-gauge needle for lumbar puncture which was attained in a single pass using a midline trajectory. The cerebrospinal fluid (CSF) was clear, flowing freely and no aspiration had been done. Patient had paresthesias after insertion of needle, but neither he nor any member of surgical team could decipher whether it was before or after the injection of lignocaine. The patient perhaps did not realize the seriousness, hence did not report to the surgeon at that time, hence the surgery proceeded. According to the anesthesiologist, possibly the patient had some recovery from spinal anesthesia prior to development of the profound motor and sensory deficit.

Sanjeev Jha Department of Neurology, Sanjay Gandhi PGIMS, Lucknow, India. E-mail: sjha@sopgi.ac.in

Routine investigations like ECG, hematology (including coagulation profile) and biochemistry were normal. The spinal MRI revealed cord edema from lower end of D₁₁ vertebral level extending up to the upper end of L, vertebral level, predominantly in the central region with mild contrast enhancement [Figure 1]. There were heterogeneous signal changes in T₂-weighted images, suggestive of lesion in conus at D_{12} . There was also evidence of hemorrhage which was suggestive of hemorrhagic myelitis subsequent to spinal cord injury [Figure 2]. Associated tethered cord was ruled out using standard radiological indices.

Discussion

Lack of information regarding temporal sequence of needle trauma, injection of lignocaine and paresthesias limits the ability to ascertain the etiology of such profound neurological deficit. The severe pain and the electric shock-like sensation passing symmetrically through both lower limbs immediately after insertion of needle, during the procedure were highly suggestive of direct



Figure 1: MRI of the spinal cord demonstrating the hemorrhagic myelitis subsequent to spinal anesthesia



Figure 2: Transverse section of the spinal cord demonstrating the linear injury to the spinal cord

cord injury by the needle. Paresthesias associated with spinal cord injury can occur at the time of needle placement but has also been reported at the time of injection or secondary to irritation, edema or hematoma.^[4,5]

The assessment of the lumbar intervertebral space by palpation of anatomical landmarks for lumbar puncture can be grossly wrong, which explains the rather rostral lesion in our patient. Surveys have shown that the correct position was often not clearly identified and errors can range from one to several spaces above the presumed level of puncture site.^[6] Similar discordance has been observed between actual level of puncture site and those recorded in operative notes.^[6,7] Some such accidents occur when this is tried under sedation and the patient is unable to react to the initial pain on touching the nerve root of the spinal cord but cases have also been reported where paraplegia occurred during injection in an awake patient as evident in our case.^[7,8] Pain is more common and severe in extra-axial lesions affecting the nerve roots or blood vessels that are innervated by sensory neurons mediating pain. Surprisingly, some patients do not experience any pain during the puncture of spinal cord and pass unnoticed even if the procedure is done under fluoroscopy.^[6,7]

In addition to direct needle trauma, neurotoxicity of the anesthetic agent or arterial occlusion or hypoperfusion could have contributed to paraplegia in this patient, since cytotoxicity of local and regional anesthesia is well established.^[9] This patient had paresthesias immediately after needle had traumatized the spinal cord and the injection of anesthetic agent most likely worsened the damage resulting in such a dense paraplegia.

North March The possible value of high-dose steroid treatment in this setting is unknown but given the poor prognosis, treatment by MPS should be considered. If used within eight hours, it has been shown to improve the outcome in other forms of injury.^[5]

This case demonstrates the tragic outcome when important, established, standard guidelines (nonaspiration of CSF, undiluted CSF, decision to proceed in spite of paresthesias and probably wrong site of injection in view of rostral location of lesion in MRI) are violated by clinicians. We conclude that while the frequency of severe, permanent neurological complications associated with spinal anesthesia has been reported to be extremely low, such complications can occur. Any patient in whom there is a suspicion of direct trauma to the spinal cord during attempted spinal anesthesia should immediately undergo detailed neurological assessment.^[7,8] If neurological deficit is present, a MRI should be done which may demonstrate the evidence of cord swelling and extent of injury.^[6,7]

References

- Tripathi M, Nath SS, Gupta RK. Paraplegia after intracord injection during 1. attempted epidural steroid injection in an awake patient. Anesth Analg 2005.101.1209-11
- 2. Houten JK, Errico TJ. Paraplegia after lumbosacral nerve root block: Report of three cases. Spine J 2002;2:70-5.
- 3. Tsui BC, Armstrong K. Can direct spinal cord injury occur without paresthesia? A report of delayed spinal cord injury after epidural placement in an awake patient.

Jha S, et al.: Transverse myelitis

Anesth Analg 2005;101:1212-4.

- Kao MC, Tsai SK, Tsou MY, Lee HK, Guo WY, Hu JS. Paraplegia after delayed detection of inadvertent spinal cord injury during thoracic epidural eatheterization in an anesthetized elderly patient. Anesth Analg 2004;99:580-3.
- Furman MB, Giovanniello MT, O'Brien EM. Incidence of intravascular penetration in transforaminal cervical epidural steroid injections. Spine 2003;28:21-5.
- Reynolds F. Damage to the conus medullaris following spinal anaesthesia. Anaesthesia 2001;56:238-47.
- Horlocker TT, Abel MD, Messick JM Jr, Schroeder DR. Small risk of serious neurologic complications related to lumbar epidural catheter placement in anesthetized patients. Anesth Analg 2003;96:1547-52.
- Bulow PM, Biering-Sorensen F. Paraplegia, a severe complication to epidural analgesia. Acta Anaesthesiol Scand 1999;43:233-5.
- Johnson ME. Neurotoxicity of lidocaine: implications for spinal anesthesia and neuroprotection. J Neurosurg Anesthesiol 2004;16:80-3.

Accepted on 25-05-2006

Source of Support: Nil, Conflict of Interest: None declared.

this asie not www.neeknow.com