Case Report

Cauda equina syndrome after spinal anaesthesia in a patient with asymptomatic tubercular arachnoiditis

Address for correspondence:
Dr. Divya Sethi,
A-2B/118-B,
Paschim Vihar,
New Delhi - 110 063, India.
E-mail: divyasth@gmail.com

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Divya Sethi, Madhu Gupta, Suvidha Sood

Department of Anaesthesia and Intensive Care, Employees' State Insurance Postgraduate Institute of Medical Sciences and Research (ESIC PGIMSR), Basaidarapur, New Delhi, India

ABSTRACT

A 14-year-old boy underwent emergency debridement surgery of right foot under spinal anaesthesia. Four hours after the surgery, the patient developed symptoms of cauda equina syndrome (CES). Postoperative magnetic resonance imaging of the patient's spine suggested underlying tubercular arachnoiditis. The boy was started on intravenous methylprednisolone and antitubercular therapy. He responded to the therapy and recovered completely in 2 weeks without any residual neurological deficits. We suggest that underlying pathological changes in the subarachnoid space due to tubercular arachnoiditis contributed to maldistribution of the local anaesthetic drug leading to CES.

Key words: Cauda equina syndrome, spinal anaesthesia, tubercular arachnoiditis

INTRODUCTION

We report a case of cauda equina syndrome (CES) after a spinal anaesthesia in a patient with underlying asymptomatic tubercular arachnoiditis. In this case, this disease pathology may have contributed to maldistribution of intrathecally administered local anaesthetic leading to CES.

CASE REPORT

A boy aged 14 years presented at our hospital with cellulitis of right foot. He had fever with swelling of the right ankle for the past 2 days. The boy had no previous history of any major illness or hospital admission. Preoperative laboratory blood tests yielded normal results. He was posted for emergency debridement of the right foot. After informed consent of the patient's parents, the resident anaesthesiologist elected to give him spinal anaesthesia for the surgery. With the routine monitors attached to the patient in sitting position, lumbar puncture was done in a single attempt with a 25G spinal needle in L3-4 interspace. There was no bloody tap and the cerebrospinal fluid (CSF) drawn was clear and colourless. Thereafter, 50 mg of 5% hyperbaric lignocaine was injected in subarachnoid space after barbotage (dilution to 2% concentration and a total volume of 2.5 ml). Paresthesias or pain at the time of lumbar puncture or injection of the drug was absent. The level of sensory block at the start of surgery was at T10. Intraoperatively, intravenous midazolam 1 mg was given for sedation. The surgery lasting for 45 minutes was completed uneventfully with all the haemodynamic parameters remaining stable. The patient was then shifted to recovery room.

After four hours in the recovery, the patient complained of inability to move his legs. He had also developed paresthesias in medial parts of both thighs and urinary retention. There had been no regression in his sensory block, which had remained at T10. Similarly there had been no recovery of the motor power in the lower limbs and deep tendon reflexes were absent. Fever, headache, vomiting or neck rigidity was absent. As patient's clinical features were suggestive of CES, he was started on intravenous methylprednisolone (500 mg/day) and shifted to intensive care unit (ICU) for further management. A neurologist's opinion was sought who confirmed the finding of CES and advised for an urgent magnetic resonance imaging (MRI) of the spinal cord.

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The MRI imaging of lumbosacral region of spinal cord revealed arachnoiditis with dural ectasia, myelitis with intradural granulomas, clumping of cauda equina nerve roots and fluid collection close to the right posterior paraspinal muscle [Figure 1]. As the MRI findings were indicative of tubercular arachnoiditis, antitubercular drugs (isoniazid, pyrizinamide, ethambutol, rifampicin and pyridoxine) were started while continuing intravenous methylprednisolone (500 mg/day). A serum sample for polymerase chain reaction (PCR) test for tuberculosis was sent which was later reported positive.

In the ICU, on postoperative day 2 the patient showed gradual recovery of sensations in lower limb. Return of motor power was seen on postoperative day 6 and recovery of bladder functions on day 8. A repeat MRI scan done during second week showed significant reduction in size and number of intradural granulomas and resolution of arachnoiditis with normal cauda equina nerve roots. There was mild reduction in the paraspinal collection but dural ectasia still persisted. The dosage of steroid was gradually tapered. On postoperative day 15, the patient was transferred to the ward with complete recovery of motor power of lower limbs and bladder functions, and with no residual sensory deficits. He was discharged from hospital a week later with advice to continue antitubercular treatment and to follow-up in neurology clinic.

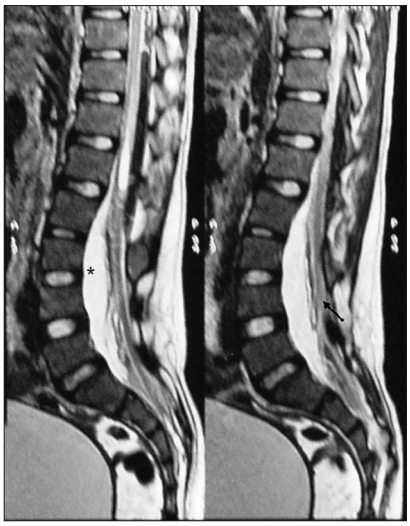

Figure 1: Sagittal T2-weighted MR image of dorsolumbar spine shows CSF loculation (asterix) and clumped cauda equine nerve roots (arrow)

DISCUSSION

We describe a case of CES that occurred when a patient with previously undiagnosed tubercular arachnoiditis underwent an uneventful surgery for debridement of right foot under spinal anaesthesia with hyperbaric lignocaine. With the postoperative MRI suggesting the diagnoses of tubercular arachnoiditis, later confirmed by a PCR test for tuberculosis, the patient was put on high-dose steroid therapy and antitubercular drugs, to which he responded very well, gaining complete neurological recovery in 2 weeks. In our review of the literature, we did not find any previous reports of diagnosed or undiagnosed tubercular arachnoiditis complicating spinal anaesthesia.

Although tubercular arachnoiditis is the most common type of infectious arachnoiditis in India, increasing incidence of the disease is being seen in the developed nations in conjunction with acquired immunodeficiency syndrome (AIDS).[1] Frequent involvement of nerve root and spinal cord in the subarachnoid space differentiates tubercular arachnoiditis from arachnoiditis due to other causes like intrathecal administration of contrast agents, antibiotics, local anaesthetics; traumatic punctures or 'bloody taps' occurring after a neuraxial block; trauma; surgery; disc disease; etc. Hence the disease process is sometimes termed 'tubercular radiculomyelitis'.

Tubercular arachnoiditis may occur as a primary lesion, or as secondary to tubercular meningitis or vertebral tuberculosis. Most patients present with backache (86%) and fever (67%), and smaller numbers with radicular pain, paraesthesias, subacute paraparesis, bladder and bowel disturbances, and vertebral kyphosis. In some cases, however, the disease may be entirely asymptomatic, as in our patient.[2,3] In early stages of this disease, inflammation leads to the clumping of cauda equine nerve roots and granulomas formation. The later stages are marked by irregular CSF loculations and blockage of CSF flow due to formation of arachnoid bands. Unchecked, the disease may progress to vascular involvement causing cord infarction and cavitation. MRI is the primary modality used for screening patients with suspected spinal tuberculosis.[4] Early diagnosis and institution of antitubercular therapy is important to ensure good recovery.

It has been seen that in patients receiving central neuraxial block, undetected underlying spinal pathologies such as spinal canal stenosis[5,6] spinal metastatic lesion[7,8] and subarachnoid cyst[9] can cause CES due to the local anaesthetic drug being maldistributed in the compromised subarachnoid

space. The maldistribution in turn causes high concentrations of the local anaesthetic drug in CSF leading to structural and functional changes in the neural tissue. The same mechanism is responsible for CES from use of spinal microcatheter[10] or with repeat spinal injections in the event of failed or patchy blocks.[11]

Although CES has been reported with several local anaesthetics, it is frequently associated with lignocaine. Yet lignocaine remains a useful drug for the procedure in short ambulatory surgery due to its fast onset, intense sensory and motor blocks, and rapid recovery,[12] with dosage below 60 mg considered safe for subarachnoid block.[13] Other identified causes of CES after spinal anaesthesia include direct needle trauma, spinal cord ischaemia, abscess and spinal haematomas. Cases of CES complicating spinal block have been typically seen in the immediate postoperative period, but these may occur later as well, as documented in two cases reports where CES due to subdural haematoma[14] and epidural haematoma[15] was seen 96 hours and 3 months respectively, after uneventful spinal anaesthesia.

In the present case, we presume that the underlying disease pathology of tubercular arachnoiditis may have caused neurotoxicity of lignocaine by restricting the spread of the drug in the subarachnoid space, eventually resulting in CES, even though a safe dosage (50 mg lignocaine) was used. Besides, the use of small bore spinal needle, the hyperbaric drug, or the sitting position could have been additional contributory factors in the CES. However, direct damage to the spinal cord by dural puncture did not seem a likely factor as there was no neurological pain or dysethesia at the time of needle insertion.

In conclusion, this case highlights that like any undetected pre-existing spinal pathology, asymptomatic tubercular arachnoiditis can contribute to CES even after carefully administered spinal anaesthesia. Therefore, after surgery under spinal anaesthesia, careful monitoring for recovery of sensory and motor functions is important for early detection of a neurological complication such as CES. In a case where CES is suspected (1) the patient should be investigated thoroughly and an MRI done to identify and suitably address any underlying contributory spinal pathology; and (2) appropriate treatment should be immediately instituted for the same.

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