



Paraparesis Following Spinal Anaesthesia in a Patient with an Undiagnosed Spinal Tumour

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Abstract: Although extremely rare, paraparesis can be a complication following spinal anaesthesia, in a patient with unrecognized spinal tumour. We describe a 45-year old female patient who underwent arthroscopy under spinal anaesthesia and developed paraparesis in the postoperative period. Magnetic resonance imaging (MRI) revealed intradural extramedullary tumour at C7-D1. The aim of this case report is to highlight the importance of neurological examination of patients undergoing neuraxial block both in the pre-anaesthetic check-up and postoperative examination in the recovery period. Careful observation of the postoperative course is essential to exclude any possible neurological complications. If motor and sensory functions do not satisfactorily recover, an MRI examination should be undertaken without delay to determine whether the underlying pathology is treatable or reversible.

Keywords: Anaesthesia, spinal; paraparesis; postoperative complications; spinal neoplasms.

Neurological complications following spinal anaesthesia, although uncommon, always imply significant underlying morbidity. In this report, we present a patient who was free of any neurological signs and symptoms relating to spinal cord disorder or root compression, developed paraparesis following spinal anaesthesia, possibly as a consequence of spinal cord compression by a previously undiagnosed spinal tumour.

Case Report: A 45 year old female with diagnosis of osteoarthritis right knee joint was posted for arthroscopy, under spinal anaesthesia. There had been no prior signs and symptoms of neurological deficits, such as low back pain, sciatica or lower-extremity numbness. She had not taken any anticoagulants in the previous 3 months. Physical examination and routine investigations revealed no abnormality. Premedication included tab. alprazolam 0.25 mg, tab. ranitidine 150 mg and tab.

metoclopramide 10 mg on the night before and 2 hrs prior to surgery with a sip of water. On arrival in the operating room, intravenous line was secured with 18 G cannula and monitoring with EKG, NIBP and SpO₂ was initiated. Her HR was 90 beats/min and BP was 124/70 mmHg. Following infusion of 500 ml normal saline, she was placed in the right lateral decubitus position and spinal anaesthesia was given in the L3-4 interspace, via median approach using a 25-gauge spinal needle (BD spinal needle, Quincke type, Becton Dickinson S.A., S Agustin del Guadalix Madrid Spain). The spinal puncture was accomplished smoothly at the first attempt and no paraesthesia was elicited. Clear and free flow of cerebrospinal fluid was obtained and 10 mg of hyperbaric bupivacaine (AstraZeneca Pharma India Limited) was injected intrathecally. After the spinal anaesthesia, patient was turned supine and the upper level of sensory blockade was T10 by pinprick test. After preliminary preparation, the arthroscopic examination started and osteophytes were removed from knee joint uneventfully. The total operative time was 60 minutes. Vital signs were stable throughout the surgery. At the end of surgery she was transferred to the post-anaesthesia care unit for further observation. One hour later, when the sensory block faded by two segments downward to the T12 level, she was shifted to the ward.

Postoperatively 4 hrs after the surgery, the patient complained of motor weakness and numbness of the lower extremities. Muscle power was 2/5 in bilateral lower limbs. There was loss of sensory response to pain and thermal stimuli bilaterally up to nipples. Bowel function was intact, but urinary retention was noted so the urinary bladder was catheterized. Considering that the effect of spinal block had not regressed totally, aggressive management was not initiated at this time. However, her condition did not improve even after 24 hours of surgery. A magnetic resonance imaging (MRI) scan at the advice of the neurosurgeon was carried out which revealed intradural, extramedullary tumour at C7-D1 level. (Fig. 1) Emergency laminectomy with excision of the tumour was done. Histopathological examination of tumour revealed meningioma. Postoperatively, motor and sensory functions gradually improved with almost complete recovery at 1 month follow-up.

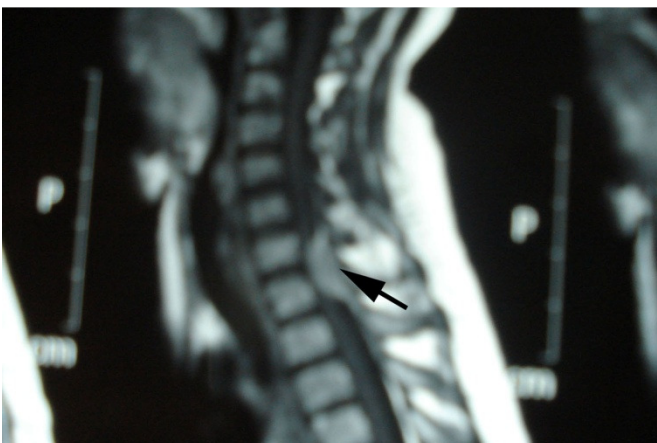


Fig. 1: Sagittal T2-weighted magnetic resonance image showing intradural, extramedullary tumor at C7-D1 level (arrow)

Discussion: Neuraxial blockade, either by spinal or epidural anaesthesia, may be associated with a variety of complications, among which neurological sequel, including paraesthesia, cauda equina



syndrome and paraplegia are the most serious. They are possibly caused either by direct needle injury, toxicity of local anaesthetics, spinal cord ischemia or a space-occupying lesion e.g. haematoma, abscess or tumour.^{1,2} Large studies are required to determine the incidence of severe neurological complications. Auroy et al prospectively evaluated a regional multicenter anaesthesia series in France and found 34 neurological complications (radiculopathy, cauda equina syndrome, paraplegia) out of 103,730 cases (3.3 per 10,000 cases). Spinal anaesthesia had a higher rate of neurological injury (6 ± 1 per 10,000 cases) than other types of neuraxial block.³ In another series, Dahlgren and Tornebrandt retrospectively studied 17 cases of neurological injuries (13 permanent, 4 transient) out of 17,733 cases with central blockade, three of whom were paraplegic resulting from hematoma.⁴ In contrast to Auroy et al, they had almost a three-fold higher rate of neurological complications (9.6 per 10,000 cases), and the epidural group had a higher incidence than the spinal group (0.1% vs 0.03%). Cherng et al in 2008 also reported a case of paraplegia 24 hrs following spinal anaesthesia in a patient with an undiagnosed metastatic spinal tumor.⁵ Paraparesis and paraplegia due to intradural extramedullary tumour compression which is incidentally found is extremely rare, and only sporadic cases have been presented. The patient presented here did not have a history of neurological complaints before surgery, and no neurological signs were found on routine physical examination. So, preoperative recognition of the potential risks was difficult, and this resulted in a postoperative delay in making the exact diagnosis. The actual cause of the neurological damage after lumbar puncture in patients who have a spinal tumour remains unknown. Some postulations have been made in the literature to explain the possible mechanism. Nicholson and Eversole considered that the high concentration of the injected local anaesthetic is responsible for causing neurotoxicity.¹ The injected material cannot be properly diluted if the CSF flow is obstructed by a space-occupying lesion at a higher level of the spinal cord. Another theory of neurological deterioration is coined "spinal coning", which means the influence of a spinal cord tumour after leakage of CSF.^{6,7} This type of tumour may lead to complete or nearly complete spinal subarachnoid blockade, which creates a pressure gradient between the two compartments above and below the blockage. There is a relatively isolated high-pressure compartment below the block. When a lumbar puncture is performed in the lower area, the CSF continues to leak out through the puncture hole. Thus, the downward spinal coning is exacerbated, causing serious complications. The incidence of spinal coning is between 14% and 26%. Another possible mechanism is the compression effect of the engorged epidural veins. When CSF is released from a site below the tumour, the reduction in intrathecal pressure causes further engorgement of the epidural veins, thus enhancing their compressive effect on the spinal cord.⁷ In consideration of the sensitivity to identify a spinal tumour or hematoma, MRI is more favourable than computed tomography (CT). MRI can provide immediate and accurate visualization of the underlying neurological pathology with good resolution.⁸ In the event of neurological deterioration, no time should be wasted on less sensitive diagnostic tools, such as CT scans or myelography. They may fail to demonstrate the developing neurological lesion, such as a growing hematoma or abscess. Despite the low incidence of neurological complications, this case and the relevant literature review demonstrate that serious morbidity is associated with mortality. Central neuraxial block should be avoided in patients with neurological deficits, such as low back pain, sciatica or lower-extremity numbness. We suggest that in addition to the preoperative history review, a neurological examination is essential, especially in patients with signs



or symptoms of root or cord compression. This report also serves as a reminder of the importance of close observation of the postoperative course following spinal block. If motor and sensory functions do not satisfactorily recover, sequential neurological assessment and an MRI examination should be done immediately to determine whether the underlying pathology is treatable or reversible.

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