

Intrathecal Dexmedetomidine for Anaesthetic Management of a Patient with Chronic Inflammatory Demyelinating Polyneuropathy

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ABSTRACT

Chronic demyelinating disorders have multifactorial origin but common important physiologic and anaesthetic considerations. Choice of anaesthesia technique and the drugs used, understanding the pros and cons of using central neuraxial blocks will help in successful management of such patients. We describe the anaesthetic management of a 34-year-old male with chronic inflammatory demyelinating polyneuropathy posted for cystolithotripsy.

CASE REPORT

A 34-year-old male was admitted for cystolithotripsy. He was a diagnosed case of Chronic Inflammatory Demyelinating Polyneuropathy (CIDP) and at admission, gave history of 'cold attacks' over knee joints, numbness over right lower limb and intermittent sweating all over the body. History revealed that patient had difficulty in urination followed by retention, over one year ago and was managed conservatively with bladder catheterisation and medications. He had developed tingling over the sole of both feet and later over the legs which extended towards gluteal region as well. He had difficulty in walking and developed giddiness and blurring of vision. There was blunting of sensation in legs with a patchy distribution. There were no cranial nerve palsies, disturbed deep tendon reflexes or ophthalmic demyelinating signs.

Visual evoked potential study was insignificant. Dermatologist opinion was sought and it was in favour of the diagnosis of demyelinating polyneuropathy. Magnetic Resonance Imaging (MRI) plain and with contrast had revealed acute demyelination of lumbar spinal cord and cerebral cortex. Patient was also found to have hypovitaminosis D3. Diabetes mellitus, trauma and viral infections were ruled out. Serum electrolytes, autoimmune antibody levels and tumour necrosis factor levels were normal. Patient was managed with rest, steroids, antibiotics, multivitamins (including vitamin D) and lorazepam. Follow-up MRI brain and spine 3 months later showed complete resolution of previous brain lesions but new scattered bifrontal subcortical white matter lesions, with L4-L5 and L5-S1 mid posterior disc bulge. He was subsequently put on oral gabapentin, pregabalin, baclofen, acetaminophen. At discharge, patient had persistent, minimal paraesthesia over right lower leg and on lower back and was off catheter.

At the time of present admission, the investigation reports were normal and patient had been only on oral gabapentin and vitamin D supplements. Patient was on urinary catheterisation for two days and preoperative antibiotics. Subarachnoid block was planned and informed written consent was obtained. Routine nil per oral precautions were taken and patient was preloaded with Ringer lactate (500 ml) IV before shifting to operation theatre. Routine monitors were connected. Basal blood pressure (BP) was 130/90 mm Hg and pulse rate (PR) 66 per min. Patient was administered subarachnoid block (SAB) with bupivacaine 0.5% heavy, 7.5 mg (1.5 cc) with 10 µg of dexmedetomidine (0.5 cc volume) (Dextomid®, Neon Laboratories, India, 50 µg / 0.5 cc ampoule, diluted to 2.5 cc

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with normal saline)(total 2 ml volume)in supine left lateral position at L4-L5 interspace.

The sensory block at 5th min reached T12 dermatomal level and at 10th min, to T8 level. Motor blockade reached Bromage scale grade IV by 5th min. Patient developed 4 episodes of hypotension (fall of systolic BP>25% from basal) in first 30 min and was managed with mephentermine (45 mg in total) and increased rate of infusion of Ringer lactate (RL). Pulse rate varied from 62 to 81/min. Overall, patient received 550ml of RL till end of procedure, lasting 50 minutes. When patient was positioned supine from lithotomy, BP fell to 92/40 mmHg and one more dose of mephentermine was administered. Tendency for hypotension was otherwise observed till next one hour, as monitored in postoperative period. Motor recovery reached Bromage grade III, 3 hours after SAB and total recovery (grade 0) was reached in 4 hour 30 minutes. Sensory block receded to T10 at 90 minutes and L1 by 2 hours, which persisted till 4 hours and there was total recovery by 5 hours 30 minutes. Patient continued to have numbness below thigh on right side till discharge on 3rd day and he was advised to continue neurologist consultation. Patient was also advised to report for any precipitation of sensory deficit after discharge and during the visit after one month, patient had persistence of previous deficits only.

DISCUSSION

Demyelination is the loss of myelin with relative preservation of axons [1]. Confirmation of diagnosis of demyelination is by histopathology but in majority of cases clinical diagnosis is the norm. Chronic demyelination can be associated with inflammation, viral diseases, metabolic derangements, hypoxia-ischemia and focal compression [1]. Cardiorespiratory complications are of major concern and anaesthetic management is influenced by both the disease and interactions of drugs. Hence, thorough assessment and preparation are needed before patients are subjected to anaesthesia [2]. Choice of anaesthesia and management of patients with demyelinating diseases has to take into consideration the neurological status and associated pathology. Use of muscle relaxants carries risk of altered and prolonged response [3] and possible medico-legal implications. Hyperkalaemia may be precipitated with succinylcholine [4] but strict titration and use of neuromuscular monitor can result in successful outcomes [5].

Cases have been managed without the use of muscle relaxants [6,7]. Successful management of a 43-year-old with CIDP

undergoing thoracoscopic mediastinal lymph node biopsy under General Anaesthesia (GA) with propofol, remifentanyl and high-dose sevoflurane is described [7]. Supraglottic Airway Devices (SADs) permit use of GA without the need for relaxants; indeed the case discussed here did not need muscle relaxation. Classic Laryngeal Mask Airway (cLMA™) has limitation that it cannot protect against aspiration of regurgitated gastric contents but devices with gastric drain tubes such as Proseal™ LMA would be better choices. We had only cLMA™ and not other SADs with gastric drain option at the time of management of the patient.

Precipitation of neurological deficits after Central Neuraxial Block (CNB) is a risk [8], with medicolegal implications but successful management of patients under CNBs have been reported [9]. Hence, pros and cons of CNB have to be discussed with the patient before surgery, based on evidences and clinical situation. A 29-year-old pregnant lady in remission from multiple sclerosis was managed by subarchnoid block with hyperbaric bupivacaine (10 mg) for Caesarean Section (CS) and she was symptom free for next 19 months [9]. Prolonged recovery after spinal anaesthesia with hyperbaric bupivacaine (0.5%) for CS in a 19-year-old has been described where partial motor block persisted for a day. Patient was not able to dorsi ex both feet for 15 hours, which became normal over next few hours [10]. A retrospective cohort study however, found that CNBs can be used successfully for labour analgesia and CS in patients with multiple sclerosis [11]. We could attain sensory block till T8 by 10th min with bupivacaine heavy 0.5% (7.5 mg) along with dexmedetomidine (10µg). Cystolithotripsy procedure requires block upto T10. Postoperative pain is not a major concern, this being an endoscopic procedure. Hence, a dose of 7.5mg of bupivacaine with dexmedetomidine (10 µg), which is known to have a dose dependent duration of sensory block, was chosen [12,13]. Dexmedetomidine (highly selective α_2 agonist), a useful analgesic, sedative and anxiolytic, also provides haemodynamic stability. It is approved for use as a sedative in intensive care units by FDA but the off-label use has become widespread in anaesthesia practice with multiple research publications in last few years, including by intrathecal route [14].

We found that both sensory and motor recovery was moderately prolonged with the doses of drugs used. Total motor recovery (Bromage grade 0) was reached in 270 minutes (4 h 30 min) and sensory recovery, at 330 minutes (5h 30 min). His preoperative sensory deficits persisted at discharge. Presence of autonomic neuropathy can contribute to hypotension (due to inadequate compensatory responses) or hypertension (excessive stimulation of cardiovascular system). Bhirud et al., reported a 47-year-old patient with post infective demyelinating disease with persistent neurological features and difficult airway was operated for dynamic hip screw repair under epidural anaesthesia using bupivacaine and top ups with lignocaine [15]. The authors reported significant fall in blood pressure needing ephedrine treatment, unaccompanied by tachycardia.

Dexmedetomidine use, however, may not always be safe; severe hypertension and bradycardia have been described in an 18-year-old male with acute transverse myelitis, attributed to the initial stimulating effect of dexmedetomidine [16]. Dexmedetomidine has been reported to be used beneficially in a 10-month-old infant with familial dysautonomia under general anaesthesia with propofol, rocuronium and air-oxygen for insertion of a gastrostomy tube via laparoscopy [7].

In the present case, we were careful not to administer larger volumes of IV fluids because of risk of autonomic lability; hypotension needing treatment occurred frequently within the first 30 minutes. It is possibly related to the pre-existing autonomic neuropathy in the patient, not to intrathecal dexmedetomidine. Addition of dexmedetomidine allowed use of lower dose of bupivacaine; otherwise, use of routine doses (10-15 mg) would have possibly resulted in greater falls in blood pressure. Repositioning in supine position at the end of procedure (50 min) also resulted in significant fall and was treated with additional dose of vasopressor.

We also wanted to follow-up and rule out possible precipitation of neurological deficit, even though patient had no fresh complaints at discharge at 3 days after the procedure, including for medicolegal purposes. Patient was asked to report any such events and at follow-up at one month to us, he mentioned no worsening of deficits or new onset deficits. The patient was also referred back to the neurologist for further consultation.

CONCLUSION

In a patient with CIDP, thorough evaluation with recording of the pre-existing neurological deficits is important for medicolegal reasons. Regional anaesthesia has been used successfully as per many published reports. Subarachnoid block with low dose bupivacaine and low dose dexmedetomidine was performed in the present case. Dexmedetomidine may contribute to effectiveness of a reduced dose of intrathecal local anaesthetic with effective analgesia, sedation and attenuation of cardiovascular effects of demyelination.

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