Anaesthetic management and implications of a case of chronic inflammatory demyelinating polyneuropathy

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ABSTRACT

A 60-year-old man with chronic inflammatory demyelinating polyneuropathy (CIDP) was posted for surgery of the neck femur fracture and was successfully managed. We discuss the anaesthetic considerations during regional and general anaesthesia of this patient with CIDP. A brief review of the available literature reveals no consensus on the choice of anaesthetic management.

Key words: Chronic inflammatory demyelinating polyneuropathy, myopathy, steroid, subarachnoid block

INTRODUCTION

Chronic inflammatory demyelinating polyneuropathy (CIDP) is a predominantly motor polyneuropathy of unknown aetiology. There is proximal muscle weakness with a relapsing—remitting course of disease. There is paucity of literature regarding the anaesthetic management in patients with CIDP. We discuss the anaesthetic management in a patient with CIDP.

CASE REPORT

A 60-year-male was posted for surgery of the neck femur fracture. His past medical history revealed CIDP 20 years previously which was manifested by weakness and paraesthesia of all four limbs (power 3/5) for the last 2 months. Electrophysiological studies (EPS) confirmed the diagnosis of polyneuropathy. Haematological investigations for secondary causes from systemic diseases such as human immune deficiency virus, cytomegalovirus, chronic active hepatitis, Lyme disease, multiple myeloma, thyroid disease, diabetes mellitus and neurosyphilis were done and were negative. He was treated with steroids and

had complete recovery after 3 weeks. He developed recurrence of symptoms 4 years later for which steroids were restarted which resulted in complete remission of disease. Thereafter, the patient resumed self-medication whenever the symptoms recurred. The patient developed adverse effects of steroids which manifested as Cushingoid facies, skin friability, poor wound healing, recurrent infection, back pain and increase in blood pressure. He developed proximal muscle weakness with paraesthesia in all limbs since last few months. Bone marrow densitometry was suggestive of osteoporosis. Hypertension was treated with tablet amlodepine 5 mg daily.

On the pre-anaesthetic check-up, the patient was conscious and oriented with Cushingoid facies; a pulse rate of 84 beats per min, regular; blood pressure (BP) 154/86 mmHg and a breath holding time (BHT) of 14 s. Neurological examination revealed bilateral symmetrical wasting with a normal tone, and with a power of 4/5 in all the limbs. Proximal muscle weakness was more than distal. Deep tendon reflexes (DTRs) were present whereas vibration and fine touch were reduced in all limbs in a graded manner. There

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were no cerebellar signs. The bladder and bowel were spared. The systemic examination and laboratory investigations were within normal limits. Steroid (prednisolone, 50 mg/day) was resumed after neurology consultation. Subarachnoid (SAB) was administered in lumbar space (L3-4) with a 25G spinal needle. Ten millilitres of cerebrospinal fluid (CSF) was collected for analysis and thereafter 7.5 mg bupivacaine (heavy with 8% dextrose) and 25 μ g fentanyl was administered intrathecally. Sensory blockade up to the T12 level achieved. The intra-operative course was uneventful. There was no change in the neurological status in the immediate post-operative period. CSF analysis revealed an increased protein level (61 mg/dl, normal = 15-45). Three days later, he was discharged from the hospital with steroids and anti-hypertensive treatment and advised to follow up.

DISCUSSION

CIDP is a predominantly motor polyneuropathy of unknown aetiology with symptomatology of at least 2 months. There tends to be a relapsing-remitting course, with benefits using immunotherapy and periodic intravenous immunoglobulin therapy. [2] The onset may occur at any age. The estimated prevalence of CIDP in populations from the UK, Australia, Italy, and Japan is 0.8-3.6 per 100,000^[3-5] The time course of weakness progression is one of the criteria that contrasts Guillain-Barre Syndrome (GBS) from CIDP. By definition, unlike CIDP, the nadir of GBS is reached within 4 weeks.^[6] CIDP may occur as a relapsing-remitting condition with episodes of symptoms alternating with periods of remission or it may occur as a monophasic/progressive disease. Nerve conduction abnormalities as seen in this patient must meet four electrophysiological criteria to provide a definitive diagnosis of CIDP: slowing of conduction velocity in distal nerve segments to at least 60%, prolongation of the F wave latency, prolonged distal motor latencies and conduction block in one or more nerves.^[7] The CSF protein level is usually increased. Depending on the severity of symptoms, treatment includes intravenous steroids, plasma exchange, and intravenous immunoglobulin. Many patients reach remission either spontaneously or with therapy.

It is important for the anaesthesiologist to know the current immunotherapeutic regimen and the baseline neurologic examination of the patient. There is paucity of the literature concerning anaesthetic considerations in a patient with CIDP. Joy E. Schabel used SAB for emergency caesarean section delivery in a patient of CIDP.[8] Central neuraxial blockade (CNB) in a patient with coexistent neurological dysfunction is not without concerns. Relapses have been reported in multiple sclerosis (MS) patients after spinal anesthesia after prolonged epidural analgesia or after exposure to a high dose of local anaesthetics. Another concern regarding regional anaesthesia administration to patient with a neurological dysfunction is respiratory function impairement if higher sensory levels paralyze intercostal muscles. [9] There might also be unpredictable block height in patients with neurological dysfunction. A Medline search revealed only one report of general anaesthetic management of a patient with CIDP. In 2000, Hara et al.[10] showed a prolonged effect of vecuronium in a man with CIDP undergoing partial gastrectomy.

The benefits of regional anaesthesia in our patient were considered to outweigh the theoretical risk of further neurological damage. SAB avoided some of the problems associated with obesity and hypertension including difficult airway, risk of airway manipulation and aspiration, rapid desaturation with apnoea and exaggerated hypertensive response to laryngoscopy and intubation. The possibility of the detrimental effects of depolarizing drugs and hypersensitivity to non-depolarizing drugs was also avoided. A SAB was instituted to obtain CSF for analysis and to achieve a sensory blockade up to the T12 level. Since the level of blockade required for surgery of the neck of the femur was below T12, the respiratory mechanics were unaltered and it proved beneficial to our case with possible coexisting steroid-induced myopathy, low functional oxygen reserve and reduced BHT. The CSEA technique is an alternative as it could have allowed us to titrate the anaesthetic block and postoperative analgesia in a more controlled manner.

CONCLUSION

The choice of anaesthesia for a patient with CIDP is difficult given the paucity of the available literature for either regional or general anaesthesia techniques. The anaesthesia technique needs to be individualized till we have strong recommendation in the medical literature for an optimal anaesthesia technique. This case report is just our experience with a patient of CIDP.

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