Acute myoclonus following spinal anaesthesia

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EDITOR:

Spinal myoclonus is a rare form of non-generalized movement disorder that may be segmental or focal, affecting many groups of muscles, a muscle group or a single muscle [1,2]. It comprises sudden, shock-like, involuntary muscle contractions and is usually attributed to spinal cord pathology such as trauma, compression, infection, demyelination, tumours, vasculopathy and paraneoplastic syndromes [1,2]. Spinal myoclonus may also be induced by drugs administered through the intrathecal and epidural routes such as local anaesthetics, analgesics or contrast material [2–4].

A 53-yr-old Caucasian female presented for surgical repair of cystocoele and uterine prolapse. She had a medical history of hypertension, hyperlipidaemia, hiatus hernia, diverticular disease and rheumatoid arthritis. She denied any history of neurological disease. Regular long-term medications included oestradiol, bendrofluazide, atorvastatin and dexamethasone. She was allergic to erythromycin. Her surgical history included laparotomy and hysterectomy. She had uneventful spinal anaesthesia 2 yr previously. Previous general anaesthesia and morphine analgesia were uneventful. Serum vitamin B_{12} level was low at 191 ng L^{-1} (normal range 200–900 ng L^{-1}), but electrolytes, urea and other laboratory parameters were normal. Body mass index was 25, physical examination was normal and cardio-respiratory parameters were normal.

The patient made an informed choice of spinal anaesthesia. Perioperative monitoring included electrocardiography, non-invasive blood pressure and pulse oximetry. With the patient seated, a spinal block was performed aseptically at L3/4 spinal space with the aid of a 25-G pencil-point Whitacre needle and injection of 2.5 mL of heavy 0.5% bupivacaine plus 300 μg of diamorphine. The spinal-block procedure was uneventful and comfortable for the patient. Adequate sensory and motor block up to the T6 level was achieved after 5 min, and confirmed by loss of sensation to cold. The surgery and intraoperative course was uneventful and the patient was returned to the ward.

The anaesthesia team was subsequently called to the ward to review this patient about 3 h after spinal anaesthesia because she had developed involuntary jerky movements of both lower limbs. These myoclonic movements were sudden, shock-like, short bursts of muscle contractions with lifting of the legs. They initially occurred every 30–60 s, but subsequently increased in frequency and severity. There were no other neurologic manifestations. The patient was conscious, alert, could move her toes, felt light touch, but did not feel pin-prick or cold sensation up to L1 dermatome, indicating incomplete recovery from the spinal block. Post-anaesthesia myoclonus was unfamiliar to us, but based on our general knowledge of myoclonus therapy, we administered intravenous midazolam. The myoclonus subsided after titrating 4 mg of midazolam and disappeared completely after 30 min. The total duration of myoclonus was 2 h. Patient follow-up for 3 days before discharge and 10 days afterwards revealed no recurrence of myoclonus and no residual neuropathy.

Spinal myoclonus is very rare and involves musculature innervated by a few contiguous segments of the spinal cord [1,2]. Spinal myoclonus following spinal or epidural anaesthesia is extremely rare, although there are a few case reports [3–5]. The suggested pathophysiology of spinal myoclonus includes abnormal hyperactivity of local anterior horn neurons, aberrant local axon re-excitations, loss of inhibitory function of local dorsal horn interneurons and loss of inhibition from suprasegmental descending pathways [1,3]. Loss of inhibitory function in the spinal cord may account for the myoclonus in our case report, because this complication occurred when the spinal block was regressing.

The onset of myoclonus at 3 h following spinal anaesthesia in our case report is very quick and unusual. Previous case reports recorded much later onset times of 1–7 days [4,5]. There is no obvious explanation for the early onset of spinal myoclonus in our patient. There was no obvious neurologic trauma during the spinal-block procedure, which was uneventful and comfortable for the patient. Although acute spinal myoclonus may result from high-dose spinal or epidural opioids [3,4], our patient was administered a low dose of diamorphine. Local anaesthetic neurotoxicity may be responsible for acute myoclonus, but bupivacaine has a good clinical record, especially at low doses of ⩽15 mg such as administered to our patient. Pre-existing neuropathy may predispose to post-anaesthesia myoclonus, but our patient did not have any obvious pre-existing neuropathy. Despite being

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on chronic diuretic therapy, the patient did not have any electrolyte disorder, which may predispose to neurologic dysfunction.

The patient was on chronic dexamethasone and oestradiol therapy; and chronic steroid therapy may be associated with neuropathy [6]. Although difficult to prove, this may have been a contributory factor to the myoclonus in this case report. Another possible contributory factor to the myoclonus in this patient is the vitamin B_{12} deficiency that was reported. Vitamin B_{12} deficiency is associated with myelopathy and neuropathy [7]. However, the degree of deficiency in our patient was mild and the patient did not require vitamin B_{12} therapy for permanent resolution of myoclonus. Lower limb neuropathy after spinal anaesthesia has been reported in a patient with thiamine deficiency [8], but our patient had a normal thiamine level.

The treatment of spinal myoclonus includes detection of the aetiology, abolition or minimization of the aetiology, and symptomatic treatment with benzodiazepines, baclofen or anticonvulsants [3,4,5]. Benzodiazepines are effective and the mainstay of treatment. Diazepam and clonazepam have been reportedly used successfully [3,5]. Midazolam was used for treatment in our patient because it was readily available and in ready-to-use injectable form. It is very potent, of rapid-onset, painless on injection and relatively short acting. Thus, we believe that midazolam is the benzodiazepine of choice for treating perioperative spinal myoclonus. Intrathecal baclofen is effective therapy for spinal myoclonus [2,3], but was not attractive to our team and our patient. Anticonvulsants such as carbamazepine and sodium valproate are also effective [5].

In conclusion, spinal myoclonus following spinal anaesthesia is a rare complication with unclear pathophysiology that can be treated effectively with midazolam. Anaesthesiologists and the perioperative team should be aware of this distressing complication, especially in patients with vitamin deficiency or neurological dysfunction. Long-term patient follow-up is important to rule out latent or evolving neuropathy.

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A potentially fatal complication of postoperative vomiting: Boerhaave's syndrome

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EDITOR:

Herman Boerhaave, a Dutch physician, first described the spontaneous rupture of the oesophagus in 1724. He described a complete transmural laceration of the lower part of the oesophagus with the flow of gastric content into the mediastinum. Such patients may present following forceful protracted vomiting associated with sudden pain in the thorax and epigastrium, which may radiate to the neck or to the back together with progressive dyspnoea, tachypnoea, cyanosis and shock.

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