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Case Report

Spinal myoclonus following combined spinal and epidural anesthesia: An unusual complication

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Abstract

Introduction: Spinal myoclonus under regional anaesthesia necessitates awareness about its manifestation with a framework for immediate plan of action.

Case: A 52 year old male patient posted for open reduction and internal fixation with Interlocking nail for both bone leg fracture lower limbs. Combined spinal and epidural anesthesia (CSE) was administered. He developed abnormal movements in lower limb after 2hrs of subarachnoid block. These uncoordinated movements initially thought to be due to weaning of the effect of spinal anesthesia were later found to be spinal myoclonus which resolved over a period of three hours after giving GABA agonists.

Conclusion: Spinal myoclonus, a rare disorder requires high clinical suspicion of the entity for diagnosis and can be managed successfully with GABA agonist drugs.

Introduction

Spinal myoclonus is an uncommon disorder characterized by sudden onset myoclonic jerks involving a group of muscles supplied by a few contiguous segments of the spinal cord.[1] Spinal myoclonus may be due to trauma, tumor, infection, spinal cord compression, demyelinating diseases, vascular lesions. Drugs used for spinal anesthesia, subarachnoid catheter in-situ and contrast material may also cause such type of spinalclonus.[2,3] We present the case report of spinal myoclonus following Combined spinal and epidural anesthesia (CSE) which was managed successfully.

Case Report

A 52 year old male patient presented to PAC for open reduction internal fixation with Interlocking nail in a case of both bone leg fracture in left lower limb. Patient gave history of road traffic accident 10 days before. There was no history of loss of consciousness, ENT bleed, seizures, vomiting or head injury. Patient was a known case of hypertension since 3 years. He was on Tab. Amlodipine 5mg once a day, Tab. Ecosprin 75 mg once a day, Tab. Atorvastatin 20 mg once a day. Exercise tolerance was good with no history of chest pain, syncope, breathlessness, palpitation or seizure disorder. All the investigations were within normal limits. Patient was planned to be taken under combined Spinal & Epidural anaesthesia.

Patient was premedicated with Tab. Alprazolam 0.25 mg night before surgery and Tab. Amlodipine 5mg on the morning of surgery with sips of water. Patient was transferred to Operation Theater and standard monitors applied which showed Pulse- 80/min, B.P- 130/80 mmHg, room air saturation 99%. In sitting position, epidural catheter was placed aseptically in L2-L3 epidural space. Catheter fixed at 9cm. Subarachnoid block was given in L3-L4 spinal space with 25G Quincke Babcock spinal needle. 2.4 ml of 0.5% Bupivacaine (heavy) was given. Sensory level up to T8 was achieved. Surgery was started on left lower limb with tourniquet placed. Surgery was uneventful until 2hrs when patient developed involuntary abnormal movements of both the lower limbs every 5 to 10 minutes. Sensory and motor blockade appeared to be adequate with normal vital parameters. Patient was conscious, oriented but had no control on those involuntary movements. 5 ml of 0.5% bupivacaine was given through epidural catheter in view of suspected weaning of spinal block. However, Myoclonic movements persisted even after 15 minutes of epidural top-up. Inj. Midazolam 1 mg intravenously given which decreased the frequency of myoclonus. Patient remained hemodynamically stable. Blood sample of the patient was sent for serum electrolytes and blood sugar, which came out to be in normal limits. Post-surgery patient was shifted to ICU for monitoring. EEG and CT scan of brain done and were found to be normal. Neurology advice was sought. No definitive diagnosis could be established and thus these movements were clinically attributed to Spinal myoclonus. Inj. Midazolam 1 mg and Tab. Clonazepam o.5 mg given in ICU in consultation with neurologist. Finally, myoclonic movements stopped after 3 hours of its first onset. Patient was discharged one week after surgery.

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Discussion

Spinal myoclonus is an abnormal movement of a group of muscles due to a stimulus on a specific area of spinal cord. The patient remains alert and conscious while the contractions are repetitive, rhythmic and may be synchronous in several muscles. Spinal myoclonus are of two types, it can be segmental or propriospinal. In 1881, Fridreich suggested that Spinal myoclonus could be of spinal origin. In 1991, Lhermitte established spinal origin of myoclonus in a case of traumatic transection of spinal cord.[4] There are few case reports in literature mentioning myoclonus after spinal anesthesia.[5] Although rare, possibility of neurotoxicity due to drugs especially with bupivacaine cannot be overlooked.[6] The local anesthetic may induce spinal cord irritation, resulting in spontaneous and repetitive discharges of the anterior horn cell groups. This may lead to a loss of inhibitory function in the spinal cord and sudden irritability of the α-motor neuron, leading to myoclonus.[7,8] In their extensive study, Yashmita et al. compared the effects of intrathecal bupivacaine, tetracaine, lidocaine and ropivacainefor increased glutamate concentrations in micro-dialysate of the cerebrospinal fluid (CSF) to elucidate mechanisms of neurotoxicity due to local anaesthetics injected intrathecally and leading to possible myoclonus. They found the vacuolation of the dorsal funiculus and hence toxicity and side effects was in the order of lidocaine = tetracaine > bupivacaine > Ropivacaine.[9]

Differentiating from other forms of myoclonus, in spinal myoclonus the patient remains alert, conscious, without muscle weakness. Spinal myoclonus is stimulus sensitive, not affected by sleep, coma or anesthesia. In our patient abnormal movements were confined to lower limbs following combined spinal epidural, with well-maintained sensorium, leading to the possibility of spinal myoclonus.

Since our patient had no prior history of any specific disease and had unremarkable neurologic imaging and laboratory findings, the cause of spinal myoclonus was likely intathecal bupivacaine. Spinal myoclonus due to bupivacine toxicity is usually a self-limiting adverse event and usually resolves after the disappearance of the drug's effect as was observed in our case too.[8] In our patient we gave GABA agonist drugs like midazolam and clonazepam for symptomatic treatment to which patient responded well. A clinical suspicion of the entity and symptomatic treatment with GABA agonist drugs, baclofen or anticonvulsants remains the mainstay of treatment.[10]

To conclude, spinal myoclonus following combined spinal epidural is a rare complication with lesser-known pathophysiology, which can be treated symptomatically using GABA agonist drugs. Anesthesiologists should keep in mind about this unfamiliar situation. A careful history of similar episodes in the past and avoiding spinal anesthesia in such patients is the key for management.

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