Spinal myoclonus a rare complication following administration of spinal anaeasthesia – a case report

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Abstract

Spinal myoclonus is a rare complication following spinal anesthesia. Transient myoclonic jerks are referred as spinal myoclonus and it is diagnosed by exclusion. This case report describes a successful management of spinal myoclonus after spinal anesthesia.

A 48 year old, woman who had undergone incision and drainage under spinal anesthesia, developed truncal myoclonus 45 minutes following induction of anesthesia. It was effectively alleviated through administration of IV midazolam.

INTRODUCTION

Spinal segmental myoclonus is characteristic by involuntary, semi-rhythmic contractions of skeletal muscle groups innervated by a limited spinal cord region. It is a rare complication following spinal cord stimulation due to drugs administered via intrathecal or epidural routes.

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CASE REPORT

Our patient is a 48 year old, female, patient with background history of bronchial asthma and type II diabetes mellitus which is well controlled by metformin and gliclazide. There was no history of any neurological disorders. She was scheduled to undergo an incision and drainage of a peri-anal abscess under spinal anesthesia. She had undergone an open appendicectomy under general anesthesia. A lower segment caesarean section and an incisional hernia repair was done under spinal anesthesia, which had been uneventful. Her cardiovascular, respiratory and neurological examination were normal.

Pre operative investigations are shown in Table 1.

Table 1: Pre-operative investigation

Haemoglobin	11.0 g/dl
White blood cell count	11.37 103/µL
Platelet count	181 103/µL
Na+	138 mmol/1
K+	3.7 mmol/1
Blood urea	29mg/dl
S.creatinine	0.59 mg/dl
CRP	5.1 mg/l
ECG	Normal

Spinal anesthesia was administered with 0.5% hyperbaric bupivacaine 1.5ml without encountering any difficulties. T12 sensory level was achieved. Procedure was performed in the lithotomy position and lasted for 45 minutes. Intra-operative period was uneventful with normal parameters.

In the post-anesthesia care unit, 60 minutes following administration of spinal anesthesia she developed myoclonic jerks which was initially visible over the abdomen, sparing facial, upper and lower extremities. Patient remained conscious and rational. She was hemodynamically stable with BP between 110/60mmHg to 120/75mmHg and her SPO2 was 100%. There were four such episodes, each lasting for 30-40 seconds with 5-10mins intervals between each episode. Sensory level was at T11. Following 4th episode of myoclonic jerks, IV midazolam 3mg was administered and the jerks subsided instantaneously. Metabolic derangements such as hyper/hypoglycemia, hypomagnesemia, hypocalcaemia and hyponatremia were excluded as a cause. Her random blood sugar was 92mg/dl and arterial blood gas analysis showed Mg^{+2}-0.89mmol/L, Ca^{+2}-2.4mmol/L and Na^{+2}-137mmol/L. Local anesthetic toxicity was unlikely as low dose of bupivacaine has been administrated. There were no evidence to suspect neurological disorders, infections, autonomic diseases, paraneoplastic disorders, renal and liver failure during history, examination and investigations. Furthermore, the acute onset of symptoms and full resolution made above etiologies unlikely. Spinal myoclonic jerks due to spinal anesthesia was considered as the likely cause.

Patient was admitted to ICU for further observation. She had a complete recovery without residual neurological deficits. Her

post-operative investigations including serum calcium-2.4 mmol/L, magnesium 0.89mmol/L, sodium 137 mmol/L and potassium 3.8 mmol/L, AST- 30units/L ALT- 37units/L and Scr-0.66mg/dl were within normal range. Since she had no further episodes an electromyograph to exclude neuromuscular abnormalities was not performed. She was discharged from hospital of postop day 2.

DISCUSSION

Spinal myoclonus is a reaction to stimulus on a specific area of the spinal cord and they are focal, involuntary muscle contractions in repetitive, rhythmic or semi-rhythmic manner. It is characterized by myoclonic movements in muscles that originate from several segments of the spinal cord.

Causes of spinal myoclonus are degenerative disorders, systemic metabolic disorders, focal brain lesions, CNS infections, spinal cord injury and drugs. The pathophysiology remains speculative. But there is evidence that various possible mechanisms can be involved such as loss of inhibitory function of local dorsal horn interneurons, abnormal hyperactivity of local anterior horn neurons, aberrant local axons re-excitations and loss of inhibition from supra segmental descending pathways. There are main two types of spinal myoclonus, spinal segmental myoclonus and propriospinal myoclonus.[1]

Pathophysiology of spinal segmental myoclonus is due to abnormal hyperactivity of anterior horn interneurons and dysfunction of inhibitory interneurons leads to spinal segmental myoclonus but exact mechanism is not clear[2]. It resolves with complete dissipation of spinal anesthesia and there were several case reports that indicating spinal myoclonus settled following IV midazolam.

Propriospinal myoclonus may originate from propriospinal pathways which classically distinguished by their relative slow conduction. Axial musculature (head, neck and truncal muscles) is purely or predominately involved in propriospinal myoclonus.[3.] It can last from days to months. Benzodiazepines, Sodium valproate, carbamazepine and levetiracetam can be used to treat propriospinal myoclonus.[4]

Spinal myoclonus following spinal anesthesia is extremely a rare complication and there are only few cases reported in the medical literature.

Risk factors identified for spinal myoclonus are history of spinal cord pathology (such as compression, sepsis, trauma, degeneration, vasculopathy or neoplasm), epilepsy, toxicity and drug interactions. High doses of spinal opioids combined with spinal cord or nerve degeneration and intravertebral disease associated with increased risk of myoclonus. More over our patient received a very small dose of bupivacaine only. Local anesthetic neurotoxicity may cause spinal myoclonus. However, bupivacaine was the local anesthetic agent administered and it has a good safety record especially at low doses. Spinal myoclonus may result from local neuronal irritation or injury caused by spinal needle [9,10]. Procedural neurologic injury may precipitate spinal myoclonus by causing abnormal impulse transmission or aberrant

nerve root with consequent disturbance of spinal inhibitory neurons and hyperexcitability of anterior horn cells. There was no obvious neurologic trauma during the regional procedure as it was uneventful and patient was comfortable throughout the procedure. Vitamin deficiency may predispose to neuropathy and myoclonus [7,8]. But neither of cases have reported that. Chronic use of certain medications such as chronic diuretic therapy which precipitate electrolytes disturbance can cause neurologic dysfunction, but our patient was not on any long-term diuretic therapy. The acute onset of her symptoms and full resolution made it unlikely that her myoclonus was caused by autoimmune, paraneoplastic or central nervous system pathologies.

According to the existing evidence in literature some patients with type II diabetes mellitus developed spinal myoclonus but there are few patients who had under gone subarachnoid block without any previous co-morbidities experienced spinal myoclonus. Other factors we considered were age, gender, duration of surgery, level of anesthetic block and intraoperative hemodynamic stability. But we could not identify any evidences for above factors in the literature.

In our case report patient has undergone two previous uncomplicated spinal anesthetic blocks. In previous cases reports, some of the patients who had spinal myoclonus following spinal anesthesia had previous history of spinal anesthesia [2,5,6]. So it is difficult to predict the risk of spinal myoclonus according to the patient's previous anesthetic history.

Above two mentioned types of spinal myoclonus are the types which identified during the literature review. There were only few case reports related to segmental spinal myoclonus. The first case of myoclonus after spinal anesthesia was published by Fox et al in 1979 who used tetracaine[6]. Alfa and Bamgbade have documented a case of spinal myoclonus where a patient exhibited involuntary spastic movements in both lower limbs approximately 180 minutes following spinal anesthesia. It was effectively subsided with IV midazolam. This therapeutic approach further validated from case of S K Singhal et al and W. Sieffien et al. These cases provided evidences for using IV midazolam effectively as a treatment for spinal myoclonus following spinal anesthesia. Benzodiazepines act by facilitating the binding of inhibitory neurotransmitter GABA at various GABA receptors throughout the CNS. But exact mechanism of action in spinal myoclonus is not clear.

CONCLUSION

Even though it is rare, anesthetist should be aware of spinal myoclonus as a possible complication following neuraxial block. Other motor disorders, seizure disorders and local anesthetic toxicity should be ruled out in order to confirm the diagnosis and decide on appropriate management. After ruling out all possible causes, the diagnosis of spinal myoclonus following spinal anesthesia is

made. It is a diagnosis made by exclusion. Spinal myoclonus after spinal anesthesia can occur unexpectedly in patients without underlying diseases. Its occurrence is difficult to predict from past regional anesthetic history. Therefore, it is important to remember that spinal myoclonus is a potential complication following spinal anesthesia. Benzodiazepines considers as main stay of treatment for spinal myoclonus following spinal anesthesia. Midazolam was used to treat our patient as it is readily available in our setting.

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