

NEAR MISS AFTER SUBARACHNOID BLOCK - A MUST KNOW COMPLICATION

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Abstract

Myoclonus are defined as brief, involuntary, shock like movements. They can be of various types like cortical, subcortical and spinal. Spinal myoclonus can be caused by spinal cord trauma, syringomyelia, myelitis, vascular lesion, malignancy and drugs. Very rarely these myoclonic movements can occur after giving spinal anesthesia. We report a unique case of myoclonic movement of all limbs along with autonomic disturbances in a 62 year old male posted for cystoendoscopy after giving subarachnoid block.

Keywords: Bupivacaine, Intrathecal, Myoclonus.

Introduction

Myoclonus is defined as the sudden, brief, shock like movements. It can be classified in number of ways. Myoclonus are commonly classified as cortical, subcortical and spinal. Spinal myoclonus can be segmental and propriospinal. It can be caused by spinal cord trauma, syringomyelia, myelitis, vascular lesion, malignancy and drugs. They can persist during sleep, rest and may or may not be stimulus sensitive¹. Very rarely these myoclonic movements occur after giving spinal anaesthesia. We report a unique case of myoclonic movement of all limbs along with autonomic disturbances leading to near collapse of a 62 year old male posted for cystoendoscopy under subarachnoid block.

Case Report

A 62 year old male known case of carcinoma bladder with stricture urethra posted for cystoendoscopy under subarachnoid block (SAB). He gave history of diabetes mellitus since 6 years and was on tablet metformin 500 mg twice a day with tablet glimepiride 1mg once a day. His HBA_{1c} level was 6.0 %. He also had history of hypertension since 5 years and was on tablet telmesartan 40mg once a day. There was no history of seizures, myoclonus, weakness or wasting of lower limb muscles. All blood investigations were within normal limits. His ECG and CXR were normal. Protocol based anxiolytic and antacid were given one night before and on the day of the surgery. All ASA standard monitors were attached. His preoperative B.P was 130/90 mm Hg. An 18 G intravenous cannula was secured in left upper limb. Preloading was done with 500ml of ringer lactate solution. Spinal anesthesia was given with 26 G quinkes spinal needle in sitting position

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without any difficulty. After negative aspiration for blood and checking for free flow of cerebrospinal fluid, 2.5 ml of 0.5% hyperbaric bupivacaine was injected. Sensory level of T₁₀ with motor block of modified bromage grade 3 was achieved. After about 5 min he started complaining of severe itching over the abdomen. Following this he started having brief, involuntary jerky movements. Initially these movements involved both lower limbs followed by involvement of both upper limbs. Immediately 100% oxygen was given. Inj midazolam 2mg i.v was given thinking it as seizures. But movements did not stop and patient became agitated. His heart rate also increased to 160 beats per minute and B.P increased to 230/130 mm Hg. Inj esmolol 40 mg i.v was given. H.R came down to 100 bpm and B.P to 170/90 mm of Hg. In view of continuous involuntary movements and hemodynamic instability it was decided to secure the airway. Inj fentanyl 100µg i.v, propofol 150 mg i.v and inj atracurium 40 mg i.v was given. Patient was preoxygenated for 3 minute. Inj esmolol 40 mg i.v bolus was given 90 seconds before intubation. Airway was secured with 8.5 cuffed endotracheal tube (ETT). 20 G Intraarterial cannula was secured in left radial artery for beat to beat blood pressure monitoring. ABG sent to find out any electrolyte abnormality. Ultrasound guided 7 F triple lumen central line was put in right internal jugular vein. Infusion of NTG @ 1µg/kg/min was started and titrated according to B.P. Intermittent boluses of 15-20 mg of esmolol was given to control the heart rate. He also developed frank pulmonary edema with bilateral coarse crepts and frothy fluid in ETT. Inj lasix 60 mg i.v, inj morphine 6 mg i.v was given. He continued to had brief intermittent involuntary movements, so infusion of propofol was also started @ 30 ml/hr. After initial stabilization inside the operation theater, patient was shifted to post-operative ICU and was put on mechanical ventilation. Neurology consultation was obtained. EEG, MRI brain with spine and CSF examination showed no significant finding and clinical diagnosis of myoclonus was made. Inj valproate 500 mg twice a day and inj levetiracetam 500 mg twice a day was started. But patient continued to have myoclonic movement for next 2 days after which it got subsided. Cardiology consultation was also obtained. Echocardiography showed left ventricle dilatation with ejection fraction of 40% and global

hypokinesia. However ECG was normal without any ST segment changes. On third post-operative day patient was extubated uneventfully. There was no residual neurological deficit. He got shifted toward next day. Coronary angiography was done which showed no significant coronary artery lesion and got discharged from the hospital.

Discussion

There are very few case reports available describing the myoclonus after intrathecal drug administration. In most of the case reports wrong drug administration was the cause for myoclonus following intrathecal drug administration. As per our literature search, myoclonic movement of all four limbs with autonomic disturbances following intrathecal bupivacaine administration has not been reported till now.

In our case, 5 minutes after giving spinal anaesthesia patient had developed severe itching over the abdomen. This can be explained by the nerve root irritation caused by the expired or inadvertent drug administration. But in our patient both these factors were ruled out as the broken ampule was rechecked for its expiry and wrong drug administration. Moreover the hyperbaric bupivacaine ampule available in our institute differs greatly in color from all other ampules.

After about 10 minute of giving spinal anesthesia he developed brief shock like movements, initially of both lower limb followed by involvement of upper limb also. Intrathecal bupivacaine can induce spinal myoclonus by causing loss of inhibition of spinal interneurons leading to hyperexcitation of anterior horn cells and loss of inhibition of suprasegmental descending pathways². Segmental spinal myoclonus usually involves the limb muscles depending on the affected spinal segment which can explain the initial involvement of lower limb muscles followed by upper limb involvement. Its treatment includes clonazepam, carbamazepam, botulinum toxin, sodium valproate or levetiracetam. However they may or may not respond to the treatment and can subside spontaneously³. In most of the previous case reports these movements got subsided by second or third post-operative day. Diabetes can be associated with myoclonus but our

patient had myoclonic movements immediately after bupivacaine administration and there was no previous history of involuntary movements and wasting of lower limb muscles⁴. Therefore the possibility of diabetes induced myoclonus was ruled out. However the presence of long standing diabetes could have made our patient more prone for this incidence.

Nakamot et al reported myoclonic movements of upper limb only, after 40 min of giving spinal anaesthesia and these movements subsided after giving 2mg of i.v midazolam⁵. In contrast to this our patient had myoclonic movements of both upper and lower limbs which continued to occurred even after giving muscle relaxant. In another case report by Kusi et al patient had myoclonic movement of lower limb only followed by cardiac arrest in the ICU, after the accidental intrathecal tranexamic acid administration⁶. Abrao et al reported myoclonic movement of lower limb in ASA 1 patient while he was given lithotomy position following spinal anesthesia with bupivacaine. Along with myoclonic movements he also had increase of H.R to 120 bpm and B.P to 170/90 mm Hg. The movements subsided on third post-operative day⁷. Lee

JJ et al reported recurrence of spinal myoclonus after two episodes of spinal anesthesia at a 1 year interval in a 35 yr old female and the intensity of second myoclonus was more compared to the first episode⁸.

Our patient also had severe hypertension and tachycardia along with myoclonic movements. Following which he had developed left ventricular failure and pulmonary edema. Although the correct mechanism for this is difficult to explain but it can be due to the massive sympathetic discharge similar to that caused by intrathecal administration of drugs like tranexamic acid, potassium chloride and antibiotics⁹.

To conclude, this case report alarms the anesthetist about the possibility of having myoclonus with autonomic disturbances following intrathecal bupivacaine administration, for which he can be blamed, either for faulty technique or inadvertent drug administration. Although it's very difficult to predict which type of patients are more prone, as no risk factors have been described till now. But the sound knowledge and management of this complication can prevent grave prognosis.

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