

Spinal myoclonus following spinal anesthesia.

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Citation

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Abstract

Spinal myoclonus is a rare form of non-generalized movement disorder. Myoclonus has been defined as sudden, brief, lightning-like jerks, twitching or spasm of a muscle or a group of muscles and can be generated by any area in the central nervous system. Drugs administered through intrathecal and epidural routes can occasionally cause myoclonus. The differential diagnosis of myoclonus is extensive. Local anaesthetic neurotoxicity may be responsible for acute myoclonus in our patient.

IMPLICATIONS STATEMENT

Spinal myoclonus is a rare form of non-generalized movement disorder. Spinal myoclonus following spinal or epidural anaesthesia is extremely rare. Local anaesthetic neurotoxicity may be responsible for acute myoclonus in our patient.

INTRODUCTION

Myoclonus has been defined as involuntary, repetitive, instantaneous, irregular contractions of a group of muscles or occasionally of a single muscle. Spinal myoclonus is a rare syndrome that has been reported in association with several types of spinal cord pathology. We report a case of spinal myoclonus possibly induced by intrathecal local anaesthetics.

CASE REPORT

A 65 yr-old female was admitted for the removal of a Backer's cyst in the right knee. Previous surgeries included an operation of a cholesteatoma in the left ear eleven years ago under general anaesthesia. She had no other significant medical history, any allergies and she had no previous neurological disease. The patient wasn't on any medication and there wasn't any positive neurological family history for myoclonus. Preoperative parameters were within the normal limits (ECG, blood tests and thorax radiology).

Midazolam 2 mg was given IV before the initiation of spinal anaesthesia as well as 500 ml of lactated Ringer's solution. The usual monitoring was used (ECG, non invasive blood pressure and pulsioximetry). Spinal anaesthesia with a 25-gauge-pencil point Whitacre needle (B.D.®) was performed at the L3-L4 interspace with the patient in the seated position.

Ten mg of hyperbaric bupivacaine 0.5% was injected through the needle after freely flowing cerebrospinal fluid was obtained. No opioids were added. The spinal procedure was performed without incidences. When adequate sensory block was achieved (T-10), the pneumatic ischemia tourniquet was inflated 300 mm Hg at the level of tight and then the patient was moved to the prone position. Oxygen was administered through a Venturi mask (nominal inspired oxygen (O₂) concentrations of 35%). The surgery proceeded uneventful, as well as the intraoperative course. The pneumatic tourniquet was inflated for 50 minutes. When deflated, the patient had an episode of hypotension which reversed with ephedrine (10 mg) and fast infusion of crystalloids. When arriving Post Anaesthetic Care Unit (PACU), the patient developed involuntary jerky movements of both lower limbs. These movements were shock-like, sudden, short burst of muscles contractions with lifting of the legs. They were clinical diagnosed of spinal myoclonus. The patient was alert and conscious and there were no other neurological manifestations. Ten minutes after arriving PACU our patient felt nausea and ondansetron 4 mg was given IV. The motor block resulted in a Bromage score of 1. The sensory block, confirmed by pin-prick and cold sensation, was up to L3 dermatoma. The frequency (every minute) and severity of the myoclonus movements increased gradually. Diazepam 8 mg was administered IV but myoclonus did not subside. Sodium valproate 15mg/kg was given IV and myoclonus disappeared after 15 minutes. The patient was discharged from PACU 90 minutes after her arrival and returned asymptomatic to the Intensive Care Unit. Two more episodes of myoclonus movements occurred during the following five hours, responsive to sodium

valproate.

A magnetic resonance imaging (MRI) of the lower thoracic and lumbar spine was performed the following day, as well as a head MRI, but no abnormalities were reported. She commenced on carbamazepine orally to prevent the reappearance of the jerky movements. A delayed EEG (forty eight hours post-crisis) was performed for differential diagnosis of subcortical focal abnormalities, anoxia and metabolic encephalopathy. An electromyography performed 48 hours after the episode, ruled out fibrillation potentials and showed normality in F wave latency. The patient was followed-up for three days and then discharged with no recurrence of myoclonus.

DISCUSSION

Myoclonic disorders are a type of hyperkinetic movement disorders different of tremor, chorea, dystonias and tics. Myoclonus has been defined as sudden, brief, lightning-like jerks, twitching or spasm of a muscle or a group of muscles and can be generated by any area in the central nervous system [1].

A diagnostic approach to myoclonus can be organized following the next classification (table 1) [2,34].

Spinal myoclonus is a rare form of non-generalized movement disorder. It is defined as a repetitive, often rhythmical, myoclonic jerking restricted to a limb, or even to a few muscles of an arm or leg. Spinal myoclonus may develop in response to infection, spinal cord compression, tumours, demyelinating diseases, trauma to spinal cord and paraneoplastic syndromes. Drugs administered through intrathecal and epidural routes can occasionally cause myoclonus [5]. It is not affected by peripheral stimuli and often persists in sleep. The spinal generator of propriospinal myoclonus is usually at the thoracic level and recruits axial muscles via slowly conducting polysynaptic propriospinal pathways both rostrally and caudally from the generator [6].

The differential diagnosis of myoclonus is extensive. Spinal myoclonus following spinal or epidural anaesthesia is extremely rare and there are only a few cases in the medical literature [5,7,8,9,10,11]. There was no obvious trauma during the spinal block procedure, which was uneventful. The patient was reinterviewed about previous history of disorders movements, with negative results. Myoclonus has been reported when using high dose spinal or epidural opioids, but in our patient no opioids were administered through intrathecal route. The patient did not have any electrolyte

disorder which may predispose to neurological dysfunction. Metabolic causes such as liver failure, renal failure and hypoglycaemia were excluded. Viral myelitis or bacterial infections were unlikely as the patient had a white cell count within the normal limits and was afebrile. Acute dystonic reaction after administration of dopamine blocking antiemetic group of drugs is well recognized. Dystonia is a neurological movement disorder characterized by involuntary muscle contractions, which force certain parts of the body into abnormal, sometime painful, movements or postures. Dystonia may appear in all muscle groups but it is observed mainly in the head and the neck area. The brief, shock-like movements of myoclonus should be distinguished from dystonia. Metoclopramide may cause extrapyramidal side effects but we use ondansetron instead of metoclopramide to avoid nausea in order not to create confusion in the diagnosis [12].

In most types of myoclonus, the pathophysiology is unclear, although abnormalities or deficiencies in neurotransmitter receptors, including serotonin, GABA, opiate, glycine, and dopamine, seem to be likely [13,14].

A variety of mechanisms have been proposed for spinal myoclonus, including dysfunction of segmental spinal-cord circuitry, deficient inhibitory glycinergic transmission in the spinal cord and subsequent release of synchronous motor neuron oscillations within segments of the cord and vitamin B12 deficiency causing vacuolar swelling of the myelin layers in the midthoracic cord, resulting in degeneration of the descending pyramidal and ascending posterior column tracts [15].

Local anaesthetic neurotoxicity may be responsible for acute myoclonus in our patient, even considering that bupivacaine has a good clinical record, especially at low doses (< 15 mg). Local anaesthetic agents administered through intrathecal route may penetrate into the spinal cord, more so in the posterolateral cord than in the anterior parts of the cord. The effect of the local anaesthetic on inhibitory neurons could have led to loss of inhibitory function in the spinal cord [8].

CONCLUSION

Spinal myoclonus is a rare complication which can be induced by drugs administered in the subarachnoid space, such as local anaesthetics. Spinal myoclonus is remarkably responsive to medical therapy with benzodiazepines, sodium valproate and carbamazepine.

Table 1: Myoclonus classification

1. clinical distribution:
 - a. Generalized myoclonus.
 - b. Multifocal myoclonus.
 - c. Segmental myoclonus.
 - d. Focal myoclonus.
 3. clinical presentation:
 - a. Spontaneous.
 - b. Action.
 - c. Reflex.
 5. neurophysiological origin
 - a. Cortical myoclonus
 - b. Brainstem myoclonus
 - a. Hyperekplexia
 - b. Brainstem reticular myoclonus
 - c. Palatal myoclonus
 - d. Spinal myoclonus
 - e. Propiospinal myoclonus
 7. aetiology
 - a. Physiological myoclonus.
 - b. Essential myoclonus. The cause may be:
 - a. unexplained: "Idiopathic essential myoclonus"
 - b. hereditary: "Hereditary essential myoclonus"
 - d. Epileptic myoclonus.
 - e. Symptomatic (secondary) myoclonus). It occurs as a result of an underlying medical problem:
 - g. Diseases of the nervous system
 - i. Creutzfeldt-Jakob disease
 - j. Alzheimer's, dementia with Lewy bodies
 - k. Parkinson's disease
 - l. Huntington's disease
 - m. Wilson's disease
 - n. Multiple sclerosis.
9. Infections
 11. HIV
 12. Malaria
 13. Siphilis
 14. Bacterial infections.
1. Metabolic disorders
 3. Hyperthyroidism
 4. liver failure
 5. Renal failure
 6. Hypoglycemia
 7. Vitamin E deficiency.
1. Chemical or drug reaction
 2. Damage to the nervous system
 3. Disorders that affect the ability to digest nutrients (malabsorption disorders)

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