Case Report

Prolonged Apnea Following Modified Electroconvulsive Therapy with Suxamethonium

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ABSTRACT

A 36-year-old male from an urban middleclass family with strained relationship among family members was referred from a corporate hospital for further management of psychological problem. As he was attempting suicide repeatedly, Electroconvulsive Therapy (ECT) was planned. After preoperative assessment and preparation, modified ECT was done with thiopentone and 0.5 mg/kg of suxamethonium. Apnea following suxamethonium was prolonged for 2 hours. Subsequent enquiry revealed that patient was treated for organophosphate poisoning and was on ventilator support for 15 days. This was concealed by the relatives. On searching patient previous records, Butyrylcholinesterase levels were very low, i.e., 350 u/l (normal reference range is 5 500 – 12 500 u/l). Prolonged suxamethonium apnea should be anticipated in patients with recent history of organophosphate poisoning; it is advisable to estimate the levels of butyrylcholinesterase and avoid suxamethonium in patients with low enzyme levels.

Key words: *Direct electroconvulsive therapy, modified electroconvulsive therapy, neuromuscular blockade, organophosphate poisoning*

INTRODUCTION

Electroconvulsive therapy (ECT) is standard practice for psychiatric patients with suicidal tendencies. For modified ECT, suxamethonium is commonly used for muscle relaxation. Prolonged apnea following suxamethonium can occur in patients with qualitative and quantitative abnormalities of enzyme. Quantitative abnormalities are seen in patients with liver disease, anemia, patients treated with ecothiopate eye drops and antimetabolite drugs. Organophosphate poisoning is an important cause for prolonged apnea following suxamethonium. We report a case of prolonged apnea following organophosphorus poisoning, which was not revealed to treating doctors.

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CASE REPORT

A young adult patient aged 35 years who has been suffering with major depression with suicidal ideation was referred from a private hospital for psychiatric evaluation and treatment. He was evaluated and in view of suicidal tendencies, modified ETCs were planned by the psychiatrist. Medical evaluation was done by the internist and anesthesiologist. Patient had uneventful anesthetic procedures under general anesthesia in the past.

An informed written consent was obtained from the patient and the attender. After confirming fasting status, the patient was advised to void the bowels and bladder. The patient's vital data and laboratory investigations were within normal limits.

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Intravenous access was secured and general anesthesia was administered with intravenous thiopentone sodium (2.5% solution) titrating dose, along with 0.6 mg of atropine, was administered till the patient was induced. Succinylcholine 25 mg was administered as bolus dose for neuromuscular relaxation. After subsiding of fasciculation, a rubber mouth gag was inserted between the upper jaw and lower jaw, so as to prevent tongue bite and injury to the buccal mucosa. All the joints were restrained while applying the bi-temporal stimulus using brief pulse ECT machine. 120 mille coulombs of energy was delivered and patient had thrown a therapeutically effective seizure for 30 seconds, simulating the generalized tonic clonic seizures (GTCs), as observed in grand mal type of epilepsy. Other signs of a seizure were also noted like pupillary dilatation, congestion of conjunctiva, masseter muscle spasm, and goose skin appearance was observed.

The patient was in relaxation phase; presumably expecting him to come out of the relaxant effect, mask ventilation continued with 100% oxygen. After 15 minutes, as there was not even flicker of respiratory attempt, suxamethonium apnea was suspected. On repeated questioning, attenders revealed that 20 days back, patient consumed unknown quantity of organophosphate (chlorpyrifos 40%) and was on a ventilator for 15 days. There was similar attempt 1 year back and was treated in the same hospital with ventilator support for 1 month. The gap between the two suicidal attempts was 15 months. In view of the long-term ventilation, percutaneous tracheostomy was done and closed after recovery. The tracheostomy wound was masked by a scarf around the neck and being winter, a high neck vest was worn by him. On searching patient previous records, butyrylcholinesterase levels were very low, i.e., 350 u/l (normal reference range is 5 500 – 12 500 u/l).

Then endotracheal intubation was performed with oral cuffed endotracheal tube and cuff inflated and connected to Bains circuit and continued intermittent positive pressure ventilation with oxygen and 2 mg of midazolam intravenously.

Intravenous fluids were administered. Inj. Frusemide 60 mg was given to produce diuresis. Bladder was catheterized and connected to a urine bag and input, output was monitored.

Hydrocortisone 100 mg was given to prevent laryngeal edema. Apnea lasted for two hours. After adequate tidal volume and good muscle tone, and recovery of consciousness and reflexes endotracheal tube extubated, the vitals were stable and patient was shifted to recovery room. The psychiatrist advised IV neuracetam which would help him to decrease the memory impairment.

Patient was observed in the observation room with a multichannel monitor. After full recovery, patient was advised to have his breakfast and was sent to his room.

The next sessions of ECTs were planned for direct ECT avoiding succinylcholine; only thiopentone sodium and atropine were used.

Patient was discharged after 4 ECT sessions as he showed signs of improvement and advised not to go for modified ECTs in future. He was also advised to inform Anesthesiologist about prolongation of suxamethonium apnea for any future surgical procedures.

DISCUSSION

The prolonged paralysis after succinylcholine can result in two conditions. First, the patient may have a genetically determined plasma cholinesterase functional abnormality, or deficiency which is predominantly seen in Vysya community (baniya) in our region. But, it can be seen in many other communities. Second, there may be acquired decrease in the plasma cholinesterase activity. Acquired abnormalities can be seen with hepatic dysfunction, pregnancy, or medication with ecothiopate eye drops and some antimetabolite drugs. The plasma cholinesterase is mainly synthesized in the liver, circulates in the plasma, and it facilitates the metabolism of intermediate ester products formed during fatty acid metabolism. Organophosphate poisoning can produce irreversible inhibition of the activity of circulating plasma cholinesterase and result in prolonged respiratory paralysis.[1]

Anti cholinesterase agents may be used as therapeutic agents in certain disease states, such as ecothiopate in glaucoma, neostigmine in myasthenia gravis, and cyclophosphamide in cancer treatment.

Organophosphate compounds are used predominantly as pesticides and chemical war fare agents. Because of its easy availability, many use it as oral poison to commit suicide or to draw the attention of others. The cholinergic crisis that ensures depends on the severity of organophosphate poisoning. Decision about the use of neuromuscular relaxants needs careful consideration by anesthesiologist.

Various esters are inhibited by organophosphate poisoning including pseudocholinesterase and particularly acetylcholinesterase. The inhibition of acetylcholinesterase allows acetylcholine to accumulate at peripheral and central nervous cholinergic sites.

Initial signs and symptoms are, therefore, attributable to the accumulation and generally are classified as muscarinic, nicotinic, and central.[2]

The possible sequelae of organophosphate poisoning include the well-known cholinergic crisis, an intermediate syndrome, and a delayed neuropathy.[2,3]

- Within minutes to hours of exposure, cholinergic crisis occurs. Muscarinic and central nervous system symptoms should be treated with atropine and diazepam.
- Within 24 to 48 hours, and enzyme reactivator, such as pralidoxime (also called 2-PAM) chloride, should be administered.
- An intermediate syndrome may develop 1 to 4 days after exposure and initial cholinergic crisis. Intermediate syndrome is characterized by sudden weakness of the proximal limb muscles, neck flexors, and respiratory muscles and by cranial nerve palsies.[3] It may be attributed that a delay in administration of PAM or inadequate treatment may contribute to the development of the syndrome.[3,4]
- Treatment of intermediate syndrome is symptomatic and often requires mechanical ventilation. Recovery occurs in predictable manner; cranial nerve palsies is resolve first, followed by improvement in the respiratory function and renewed strength in proximal limb muscles, and finally neck flexion is strengthened. The time period for recovery is variable from 1 to 2 weeks.
- Late effects of organophosphate poisoning may occur within 1 to 3 weeks of exposure. Organophosphate-induced delayed neuropathy (OPIDN) is characterized by polyneuropathy affecting primarily the lower limbs. Some patients may exhibit spasticity and ataxia. In contrast with intermediate syndrome, the respiratory muscles are spared in OPIDN.[2]

Melanie Dillard and Jack Webb of Joplin, Missouri, have used low doses of succinylcholine for ECT even after succinylcholine apnea.[5] However, we have deferred the administration of modified ECT in the next sessions, as it again causes succinylcholine apnea. Direct ECT without muscle relaxant adequately restraining the joint movements facilitated well in subsequent procedures without any untoward incidents.

SUMMARY

A case of depression with suicidal tendencies was given modified ECT using low-dose suxamethonium. Apnea following suxamethonium was prolonged due to recent intake of organophosphate poisoning, which was concealed by attenders. It is imperative to take detailed history including drug history to avoid such recurrences. It is mandatory in all cases with recent history of organophosphate poisoning to estimate the plasma cholinesterase level prior to any anesthetic procedure.

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