Administration of succinylcholine for electroconvulsive therapy after organophosphate poisoning: A case study

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A 53-year-old man was admitted to the hospital psychiatric unit for evaluation and treatment following a recent suicide attempt, which involved ingestion of an unknown amount of Dursban (DowElanco, Indianapolis, Ind) and a self-inflicted knife wound to the abdomen. Dursban is a commercially prepared organophosphate insecticide in which the active ingredient is chlorpyrifos in a petroleum distillate solvent. The patient received 7 electroconvulsive therapy treatments during a 2-week hospital stay. The anesthetic regimen included methohexital for induction and succinylcholine for neuromuscular relaxation. Cholinesterase levels were low on admission at 5,780 IU (reference range, 11,000-15,000), yet succinylcholine was used successfully at low doses.

Key words: Electroconvulsive therapy, neuromuscular blockade, organophosphate poisoning.

Introduction
Organophosphate poisoning is relatively common in the United States. It is found in a variety of settings, including suicide attempts.1 The cholinergic crisis that ensues depends on the severity of the organophosphate poisoning. Anesthesia providers may be confronted with anesthetizing patients after a substantial exposure to organophosphates. Decisions about the use of neuromuscular relaxants routinely used in anesthesiology need careful consideration. We report a case involving a 53-year-old man who suffered acute organophosphate poisoning in a suicide attempt. After the acute phase, he required anesthesia for electroconvulsive therapy (ECT). Low doses of succinylcholine were used for muscle relaxation rather than nondepolarizing neuromuscular blocking agents.

Case summary
A 53-year-old man was admitted to the hospital psychiatric unit for evaluation and treatment after a recent suicide attempt, which involved ingestion of an unknown amount of Dursban (DowElanco, Indianapolis, Ind) and a self-inflicted knife wound to the abdomen. Dursban is a commercially prepared organophosphate insecticide in which the active ingredient is chlorpyrifos [o,o-diethyl o-(3,5,6-trichloro-2-pyridinyl) phosphorothioate] in a petroleum distillate solvent. He was admitted by transfer from an acute care hospital in which he had completed a 15-day hospitalization. During that hospitalization, he had suffered an initial cholinergic crisis followed by development of the intermediate syndrome. The patient had a history of emotional problems and previous suicide attempts. At the time of his present suicide attempt he was medicated with trihexyphenidyl hydrochloride, haloperidol, and lithium.

During the 14 days, the patient received 7
ECT treatments. For the first treatment, anesthesia was induced with 150 mg of methohexital followed by 40 mg of succinylcholine for paralysis. The patient then was intubated, as prolonged apnea was anticipated. Following the treatment, 3.5 mg of midazolam was administered. Anesthesia was maintained with incremental doses of methohexital while the patient breathed nitrous oxide in oxygen. One hour later, the patient was breathing spontaneously and demonstrated no decrease in muscle contraction in response to a single twitch stimulus with the peripheral nerve stimulator. He was allowed to awaken and then demonstrated that he could sustain a head lift. The patient was suctioned, extubated, and taken to the postanesthesia recovery unit where he had an uneventful recovery.

For the second treatment, anesthesia was induced with 170 mg of methohexital followed by 20 mg of succinylcholine for paralysis. Following induction, a laryngeal mask airway was placed for airway management. After the treatment, 2 mg of midazolam was administered. Anesthesia again was maintained with incremental doses of methohexital while the patient breathed nitrous oxide in oxygen. In 30 minutes, the patient was breathing spontaneously and demonstrated no decrease in muscle contraction in response to a single twitch stimulus with the peripheral nerve stimulator. The methohexital and nitrous oxide were discontinued, and the patient was allowed to awaken breathing 100% oxygen. The patient was then suctioned, extubated, and transferred to the postanesthesia recovery unit where he again had an uneventful recovery.

The anesthetics for ECT treatments 3 through 6 included 170 mg of methohexital for induction followed by 15 mg of succinylcholine for paralysis. Following induction, a laryngeal mask airway was placed or bag valve mask ventilation was used. This difference was related to anesthesia provider preference. After the ECT treatment, 1 to 2.5 mg of midazolam was administered, and the patient breathed nitrous oxide in oxygen. In 15 minutes, the patient was breathing spontaneously and demonstrated no decrease in muscle contraction in response to a single twitch stimulus with the peripheral nerve stimulator. The nitrous oxide was discontinued, and the patient was allowed to awaken breathing 100% oxygen.

Following removal of the laryngeal mask airway, when used, the patient was taken to the postanesthesia recovery unit and had an uneventful recovery. For each ECT treatment, muscle relaxation was satisfactory as evidenced by minimal muscle contraction with seizures. The patient experienced no complications related to inadequate neuromuscular relaxation.

Discussion

Organophosphate compounds are used predominantly as pesticides and chemical warfare agents. Various esterases are inhibited by organophosphates including pseudocholinesterase (plasma cholinesterase) 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 this accumulation and generally are classified as muscarinic, nicotinic, and central.

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

The possible sequelae of organophosphate poisoning include the well-known cholinergic crisis, an intermediate syndrome, and a delayed neuropathy2,3 (Table). Within minutes to hours of exposure, a cholinergic crisis occurs. Muscarinic and central nervous system symptoms should be treated with atropine and diazepam. Within 24 to 48 hours, an 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 the 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 has been suggested that either a delay in administering pralidoxime chloride or inadequate treatment may contribute to the development of this syndrome.3,5

Treatment of intermediate syndrome is symptomatic and often requires mechanical ventilation.6 Recovery occurs in a predictable pattern. Cranial nerve palsies resolve first, followed by improvement in respiratory function and renewed strength in proximal limb muscles, and, finally, neck flexion is strengthened. The time period for recovery, however, is variable, ranging from approximately 1 to 2 weeks.5 Late effects of organophosphate poisoning may occur within 1 to 3 weeks of the exposure. Organophosphate-induced delayed neuropathy (OPIDN) is characterized by polymyopathy affecting primarily the lower limbs. Some patients may exhibit spasticity and ataxia. In contrast with intermediate syndrome the respiratory muscles are spared in OPIDN.7 The development of OPIDN is unrelated to the anticholinesterase effects of organophosphates.
Many theories have been proposed to explain the development of OPIDN. The one most widely held is that it occurs as a result of inactivation of neurotoxic esterase or neuropathy target esterase.\textsuperscript{2,7} The concentration of organophosphate required for acetylcholinesterase inhibition is relatively low compared with the concentration required to inhibit neurotoxic esterase. Most compounds with neuropathic potential are no longer used, particularly in the Western market in which almost all organophosphates are incapable of causing OPIDN.\textsuperscript{2} The organophosphate tri-O-cresyl phosphate has been responsible for thousands of cases of OPIDN. Tri-O-cresyl phosphate was used as an adulterant in a prohibition era alcoholic beverage known as “Ginger Jake” and also in cooking oil. Partial or incomplete recovery usually occurs within approximately 1 year leaving only mild neuropathic damage.\textsuperscript{6}

Neuromuscular relaxants are used universally in the anesthetic regimen for ECT to prevent the trauma associated with violent muscular contraction. Two types of neuromuscular relaxants are available: depolarizing and nondepolarizing.
Succinylcholine is the only depolarizing neuromuscular relaxant in use in the United States; there are several nondepolarizing neuromuscular relaxants. Succinylcholine mimics the action of acetylcholine, leading to depolarization of the end plate. Succinylcholine is not metabolized as quickly as acetylcholine, so depolarization persists and neuromuscular transmission is blocked. Normally, succinylcholine is metabolized rapidly by pseudocholinesterase leading to a short duration of action of approximately 2 to 5 minutes. The depolarizing block attained with succinylcholine can be prolonged for a variety of reasons, including certain disease states, low plasma cholinesterase levels, drug-induced cholinesterase inhibition, and atypical pseudocholinesterase.

Nondepolarizing neuromuscular relaxants produce a competitive block. They combine with the nicotinic cholinergic receptor and competitively block the action of acetylcholine. Nondepolarizing neuromuscular block can be reversed by the administration of an anticholinesterase agent.

Plasma and erythrocyte acetylcholinesterase levels are the most widely used diagnostic tests in organophosphate poisoning. On admission, the patient’s erythrocyte cholinesterase level was 5,780 IU (reference range, 11,000-15,000 IU). There was concern among the anesthesia staff about the low cholinesterase level. The anesthesia providers involved in the case discussed the best overall anesthetic approach. Emphasis was placed on the safest and most efficient means to obtain adequate muscle relaxation of the required duration for ECT. The choices considered were as follows:

1. Delay ECT until cholinesterase level returns to normal.
2. Anesthetize the patient with methohexital for induction followed by rocuronium for neuromuscular relaxation.
3. Anesthetize patient with methohexital for induction followed by succinylcholine for neuromuscular relaxation.

Electroconvulsive therapy is indicated primarily for depressed patients who have not responded to antidepressants, yet it is the appropriate initial treatment for patients who are suicidal. Two weeks had passed since the patient’s suicide attempt, and there was no improvement in his cholinesterase level. There also was no assurance of a timetable for recovery of his cholinesterase level. The patient was still at high risk for suicide, and, therefore, delaying treatment was considered unsafe and not a reasonable option.

Administering rocuronium for neuromuscular relaxation following induction with methohexital also was considered. The duration of action of rocuronium depends on the dose administered. The dose considered was 0.45 mg/kg, which has a clinical duration of action of approximately 20 minutes, indicating that the block could be antagonized with a reversal (anticholinesterase) agent in approximately 20 minutes. The concern with this option was the need to reverse the block with an anticholinesterase agent. Administering an anticholinesterase agent could precipitate another cholinergic crisis.

The final choice considered was administration of small doses of succinylcholine for muscle relaxation following induction with methohexital. Succinylcholine is metabolized by pseudocholinesterase, which is inhibited by organophosphates. It was known that the patient could not metabolize succinylcholine in a normal fashion, and, hence, the duration of action would be prolonged. After discussion among the anesthesia providers and a review of the pharmacologic literature, the decision was made to attempt administering small doses of succinylcholine. It was thought that small doses of succinylcholine might effect a block and yet not be excessively prolonged. Whereas the time to full recovery following administration of rocuronium would be longer than 20 to 30 minutes if the block were not reversed. Administration of an anticholinesterase agent was considered unsafe.

Summary

We observed that succinylcholine can be used safely to achieve a neuromuscular block of short duration after organophosphate poisoning provided very small doses are administered. In this particular case, one fifth of the normal dose was used to achieve a neuromuscular block of short duration with adequate muscle relaxation. For cases such as ECT, in which it may be desirable to achieve a block of short duration without incurring potential risks associated with reversing nondepolarizing neuromuscular relaxants after organophosphate poisoning, succinylcholine may be an appropriate choice.

REFERENCES


**SUGGESTED READING**


**AUTHORS**

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