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Case Report Open Access

Acute Imidacloprid Poisoning Caused Prolong Depression of Butyrylcholinesterase

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Abstract

Background: Imidacloprid is currently one of the most widely used insecticides in the world. To the best of our knowledge, there is no study showing that imidacloprid decreases the butyrylcholinesterase activity in humans. However, here we present a case of suicide attempt by imidacloprid with prolong depression of butyrylcholinesterase activity.

Case Report: This is an observational case report of a patient attempted suicide by unknown pesticides. Pralidoxime and atropine were administrated under the impression of organophosphate poisoning based on the depression of butyrylcholinesterase activity. However, due to the patient's atypical presentation for organophosphate poisoning, pralidoxime was stopped and patient's condition was getting stable under supportive treatment. Patient's urine sample revealed the presence of imidacloprid but not other pesticides. Serial false depressions of butyrylcholinesterase activity were also detected in our patient.

Keywords: Imidacloprid; Poisoning; Butyrylcholinesterase; Organophosphates; Pesticides

Introduction

Imidacloprid is currently one of the most widely used insecticides in the world. It's used to control of sucking insects, soil insects, and some species of biting insects by binding to postsynaptic nicotinic acetylcholine receptors (nAChRs) in insect central nervous system [1]. Imidacloprid is highly selective for nAChRs in insects compared with mammals [2] and thus is less harmful to human [3,4]. At the presenting time, some articles stated that imidacloprid did not decrease the butyrylcholinesterase activity in humans [5-9]. However, here we present a case with prolong depression of butyrylcholinesterase activity due to suicide attempt with imidacloprid.

Case report

An 87-year-old male with prostate cancer drank 4 cups (approximately 400 cc) of unknown insecticide due to depressive mood 5 hours before transferring to our emergency department (ED). He was first sent to a local hospital and gastric lavage with active charcoal was performed before referral. On arrival, he was fully conscious and complained of dry mouth. He denied fever, dyspnea, chest pain and abdominal pain. His vital signs were as follows: body temperature 36.8°C, pulse rate 96/min, respiratory rate 21/min, and blood pressure 150/94 mmHg. On physical examination, mild miosis, bilateral crackle breathing sounds without wheezing, and normoactive bowel sounds were found. Organophosphate intoxication was suspected due to a low plasma butyrylcholinesterase activity (5.83 U/mL, reference range 7-13 U/mL) at ED and 1 gm pralidoxime (PAM) as the loading dose with 500 mg/hr as the maintenance dose were administrated intravenously. Atropine (1 mg) was also given intravenously. After admission to ICU, his conscious level was clear and bilateral lung field crackles remained. He was intubated and further doses of PAM were given. Unfortunately, his conscious level deteriorated (Glasgow coma scale E2VeM4) after intubation and profound weakness of four limbs was noted. Although the butyrylcholinesterase activity was 6.43 U/mL on the second day after his admission, there was no sign of organophosphate poisoning such as salivation, lacrimation or diarrhea. PAM was discontinued on day 2 not only because of his progressively limbs weakness which was suspected to be due to the effect of PAM but also lacking of signs of organophosphate intoxication. He was extubated on day 3 with full recovery in muscle power and conscious level and was transferred to an ordinary ward on day 4 to treat his aspiration pneumonia. He was discharged on day 15 and had been followed up for 2 months without any sequel.

The butyrylcholinesterase activities during hospital admission were as follows: 5.69 U/mL (day 3), 4.87 U/mL (day 7), 5.07 U/mL (day 9), and 4.76 U/mL (day 13). His urine sample was sent to Taiwan agricultural chemical and toxic substances research institute to confirm the offending agents. Imidacloprid (4 ppm) was detected by Liquid chromatography-tandem mass spectrometry but no other insecticides were detected.

Discussion

Data of human exposure to imidacloprid is limited, mild clinical effects such as nausea and vomiting, dizziness, mydriasis and abdominal pain were reported but severe symptoms, signs and sequelae such as tachycardia, hypotension,coma, respiratory failure, seizure, and even death can occur, too [10-14]. On the contrary to organophosphates poisoning, there is no laboratory test such as

butyrylcholinesterase activity to be used as the diagnostic tool for imidacloprid poisoning. Since some symptoms and signs of imidacloprid poisoning and organophosphates poisoning are similar, misdiagnosis may happen as we reported here. Mohamed et al reported a similar case treated with PAM that had acute respiratory failure but normal butyrylcholinesterase level [4]. Several reports stated that imidacloprid did not decrease the butyrylcholinesterase activity in humans [5-9].

To the best of our knowledge, we present the first observation that imidacloprid may cause prolong depression of butyrylcholinesterase activity in human poisoning.

The depression of butyrylcholinesterase activity is usually a diagnostic rule of acute organophosphate poisoning. Several possible causes may explain the depression of butyrylcholinesterase activity in this patient. The first one is laboratory error. However, the prolong depression of butyrylcholinesterase activity of our patient eliminates this possibility. A long-term organophosphate user may have chronic depression of butyrylcholinesterase activity. However, our patient already retired from his job and had no history of organophosphates pesticides exposure. Second, since butyrylcholinesterase is a liver enzyme, any conditions that deteriorate liver function such as liver cirrhosis, malnutrition may have lower butyrylcholinesterase activity. However, our patient has no history of liver disease. Therefore, the high imidacloprid dose our patient took may be the reason that caused the inhibition of human butyrylcholinesterase activity. However, further investigation is needed. The possibility of idiosyncratic reaction of this depression of butyrylcholinesterase activity in our patient cannot be ruled out.

"Why an emergency physician should be aware of this"

Depression of butyrylcholinesterase activity usually leads to the diagnosis of organophosphate poisoning and pralidoxime would be used to treat such kinds of patients. In our case and as Mohamed et al described in their report [4], pralidoxime weakened patient's muscle power and thus led to acute respiratory failure. We stopped PAM on day 2 and patient had rapid clinical improvement in the next day. Lacking of signs of organophosphate intoxication made us to stop PAM infusion. To treat patients according to their toxidrome is mandatory in poisoning patients. To have proper treatment modalities in pesticides poisoning patients, an emergency physician should be aware of the possibility that imidacloprid may decrease butyrylcholinesterase activity in human poisoning.

Conclusion

Careful interpretation of decreased butyrylcholinesterase activity is crucial in treating imidacloprid-poisoned patients. Although the majority of imidacloprid poisoning patients did not have decreased butyrylcholinesterase activity, the false depression of butyrylcholinesterase activity in our patient is an exception. We should treat pesticide poisoning patients not just rely on butyrylcholinesterase activity but on patients presentation as toxidromes.

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