Case Report

Intramuscular pyrethroid with organophosphorus (cypermethrine 3% + quinolphos 20%) mixed poisoning, its clinical presentation and management

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Abstract

Organophosphorus compounds are absorbed by any route including intramuscular injection. Pyrethroid compounds are less toxic for human but in combination with organophosphates, its toxic effect potentiates due to inhibition of its metabolism. In this case report, a 40-year-old female patient presented with acute onset of pain abdomen, recurrent vomiting, and excessive salivation followed by altered mental status, on evaluation her clinical findings were suggestive of organophosphorus poisoning. Her treatment was started with injection atropine, anti-emetics, and adequate parenteral rehydration. After regaining consciousness, the patient confessed that she had taken herself intramuscular injection of pesticide which contained 3% cypermethrine plus 20% quinolphos. It was further confirmed by a very low level of plasma cholinesterase level. Intramuscular injection acted as a depot that leads to prolong intoxication and in the due course of illness, she also developed delayed onset intermediate syndrome that was managed appropriately.

Keywords: Cypermethrine poisoning, intramuscular, organophosphate

Introduction

Organophosphorus compounds are absorbed by any route including transdermal, transconjunctival, inhalational, across gastrointestinal tract, genitourinary mucosa, and direct injection.[1] The majority of the patients have a good prognosis but those who develop organophosphorus induced delayed polyneuropathy will have lasting sequelae. Cypermethrin, a pyrethroid compound is widely used due to its high insecticidal potential and slow onset of resistance in pest. It is considered less toxic for humans, because of poor absorption, rapid metabolism, and less tissue accumulation.[2] Organophosphates can inhibit the detoxification of pyrethroid and increase the toxicity of combination.[3] There is no antidote for cypermethrin poisoning and treatment consists of preventing further exposure together with supportive and symptomatic measures.[4] Here, in the case of mixed intramuscular poisoning with notably intermediate syndrome is described in different course of time.

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Case Presentation

A 40-year-old female patient was admitted through the emergency department with acute onset of pain abdomen, recurrent vomiting, and excessive salivation followed by altered mental

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status. The physical examination revealed bilateral constricted pupil, muscle fasciculations all over the body with a pulse of 58 beats per minute (bpm), blood pressure 100/70 mm Hg, soft abdomen with no localize tenderness, and organomegaly. Her initial investigations with atrial blood gas analysis and serum electrolytes were within normal limits. Gastric lavage was done and the content did not have a typical smell of organophosphates. Her ultrasonography of the abdomen and computed tomography (CT) of the brain were within normal limits. Although her attendants repeatedly denied any history of intoxication at that time, considering strong suspicion of organophosphorus poisoning, emergency treatment with atropine 5 mg intravenous (iv) bolus followed by 1 mg hourly infusion along with parenteral hydration were started. Her report showed very low level of plasma cholinesterase (100 units/L; reference range 4260-9120 units/L). With this measure, patient regained consciousness over the next 12 hours and on interviewing, the patient confessed that she had taken pesticide not orally but by intramuscular injection of around 3 ml over her right hypochondrium. She did not tell any family member about this incident. On re-examination of the abdomen, there was prick mark over the right hypochondrium [Figure 1] with mild tenderness and there was no sign of inflammation or abscess formation. The bottle and a syringe [Figure 2] containing the pesticide was recovered from the residence of the patient by her attendant and it contained cypermethrine 3% and quinolphos 20%. Her treatment was continued with atropine 1 mg hourly infusion and pralidoxime 500 mg iv twice daily was added. A dose of the atropine was gradually tapered to 0.5 mg every 4 hour infusion in the next 6 days and the iv pralidoxime was stopped. Initially, the patient was doing well but on day 7 the condition of the patient deteriorated and she was complaining of breathlessness, weakness, and pain abdomen. She started having more pronounced fasciculations all over the body, weakness of all four limbs, and was unable to elevate her head from the bed. At this presentation, she developed delayed onset intermediate syndrome. The patient was shifted to the intensive care unit and her atropine dose was intensified from 0.5 mg every 4 hour to 1 mg per hour infusion and pralidoxime 500 mg iv twice daily was restarted. On day 9 morning, her symptoms improved gradually and she was continued with the same dose of atropine and pralidoxime. Despite 1 mg atropine infusion every hour, her pupils remained constricted and heart rate was 120 bpm for the next two days, although she did not have any tracheo-bronchial secretions. As there were no tracheo-bronchial secretions and gradual improvement in this patient, the dose of atropine was gradually tapered to 0.5 mg every hour infusion and pralidoxime 250 mg iv twice daily in the next two days. On day 11, her plasma cholinesterase level was remeasured. The level of plasma cholinesterase did not increase and remained 100 units/L as the previous value. The patient improved gradually over the next week with the return of power and sensation in the limbs. On day 18, she was able to hold her neck and sit up on the bed by her own, plasma cholinesterase level was remeasured and it raised to 545 units/L. Her pupillary size increased and become semi dilated. Her injection pralidoxime was stopped and the dose of atropine was further tapered off to 0.1 mg/hour infusion and gradually stopped on day 21 and plasma cholinesterase was increased to 1530 units/L. With this regimen followed by a complete psychiatric evaluation, the patient improved gradually. She was able to walk without support before discharge from the hospital on day 22. On 6-week follow-up, she was able to do her routine work.

Discussion

In our case, clinical signs and symptoms of systemic toxicity resulting from the intramuscular injection manifested for prolonged duration. This may have been due to the slow release of poison from the site of injection (acting as a depot) to the circulatory system and due to the combination of organophosphate with a pyrethroid, as organophosphate delays the metabolism of pyrethroids and potentiates its toxicity on the muscular and nervous system. These manifestations were recurring and the patient was kept on high dose atropine for three weeks. Similarly, Chia-Chang *et al.*[5] described in a case report of 28-year-old Taiwanese woman with prolonged cholinergic features for many days and needed atropine up to 80 mg in an hour with a total dose of 11,665 mg in 17 days who also took pralidoxime. This case differs from a previous case report[6] of intramuscular organophosphorus poisoning, in which there was a cicatrix formation at the site of injection. Immediate surgical debridement, incision, and drainage were carried out. In our case, intermediate syndrome developed on the 7th day after organophosphate poisoning. It seemed to be delayed more than

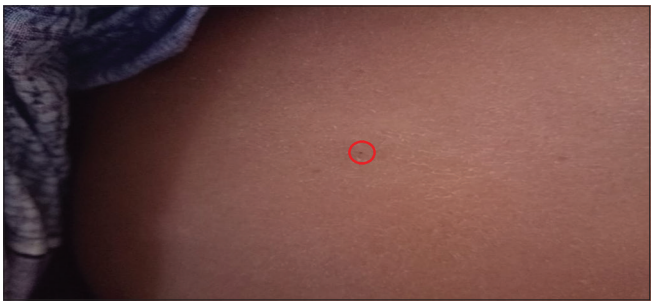

Figure 1: Injection mark over the right hypochondrium

Figure 2: The bottle and a syringe containing the pesticide

the usual manifestation. This delayed-onset intermediate syndrome might result from the intramuscular injection of organophosphate and the organophosphate being released for longer duration in the patient's body. The duration of atropine infusion has to be 'tailored' in individual cases to maintain a state of mild atropinisation.[7]

Conclusion

Clinical presentation and complications of parenteral organophosphate compound poisoning persist for a longer duration. So, the treating physician should be vigilant and appropriate treatment has to be administered.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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