CASE REPORT

Dichlorvos Poisoning: A Mystery Case of Distributive Shock Unraveling with Atropine

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Abstract

Hypotension can be explained by the cardiotoxic effects of an organophosphate poison, but a distributive shock is a rare event. This is a case report of a young north Indian man who presented to the emergency room in a comatose state and distributive shock. He was initially managed with intravenous crystalloids but required inotropic therapy to maintain the desired mean arterial pressure and organ perfusion and also required mechanical ventilation. He improved during the hospital stay only after 4 days when cocktail treatment of atropine was started considering the possibility of organophosphorus toxin exposure and had tapered off the inotropes and mechanical ventilation. Dichlorvos ingestion was confirmed later on after recovery from the coma. At 4-week follow-up, he developed delayed neuropathy. This case is a torchlight toward organophosphorus poisoning presenting as a distributive shock. Atropine may be used as a cocktail treatment in distributive shock where the diagnosis is uncertain.

Keywords: Autonomic dysfunction, Coma, Poisoning, Shock, Survival.

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Introduction

Organophosphorus (OP) compounds are commonly used as pesticides in agriculture, and India being an agricultural-developing country, its population is more prone to poisoning with the same. According to the National Poison Information Centre India, suicidal poisoning with household agents, such as OPs, pyrethroids and carbamates, being cheap, highly toxic, capable of being taken along with food or drink, and easy availability, is the most common modality of poisoning. The data from the National Crime Bureau shows that the ingestion of poisonous substances contributed to 26.7 and 25.8% of all the suicides in the year 2018 and 2019, respectively.[1] Since the under-reporting of poisoning cases is another major issue at present, these numbers are going to be still on the higher side. In a previous study that happened in Haldwani (Uttarakhand), pesticide ingestion was the most common method of poisoning (58.5%).[2]

Irreversible binding of OP to acetylcholinesterase in the cholinergic synapses in the central nervous system (CNS) and peripheral nervous system (PNS) leads to high concentrations of acetylcholine in the synaptic clefts that cause initial excessive stimulation and later blockade of synaptic transmission.[3] The acute manifestations of OP poisoning are mainly neurological due to the cholinergic crisis that occurs in the CNS and PNS. Cardiovascular manifestations associated with the poisoning are not so uncommon, which includes sinus bradycardia or tachycardia, hypotension or hypertension, supraventricular and ventricular arrhythmias, ventricular premature complexes, and electrocardiogram (ECG) finding of QTc prolongation, ST–T-segment changes, and T-wave abnormalities, of which that leads to death are mostly arrhythmias or severe and refractory hypotension. Hypotension can be explained by the cardiotoxic effects of the poison; the distributive shock is also a manifestation.[4] Hypotension is seen with dimethoate (one OP compound) poisoning as commonly as in 40% of the deaths whereas it is 5% in chlorpyrifos poisoning and not common in other OP compounds.[5]

We report the instance where acute dichlorvos poisoning presents as a distributive shock.

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Case Presentation

A 21-year-old man from Uttar Pradesh, India was referred to our emergency room in coma (Glasgow coma scale [GCS] of E1VTM2). His history was incomplete as he was well before and was found in an unconscious state with frothing from his mouth by his peers 3 hours before the presentation. His birth, developmental, and history were unremarkable. He had no addictions or psychiatric diseases. He had low blood pressure (70/40 mm Hg) with a normal heart rate but with warm extremities. He had no evidence of bruises or fresh external injuries or bite marks. His pupils were mid-dilated and were reacting to light. He had no smell of poisonous substances. His chest was clear on auscultation. He was immediately given fluid resuscitation with crystalloids. The central line was secured in the right internal jugular vein and the central venous pressure (CVP) was measured to be 3 cm blood column. Fluid therapy was continued according to CVP charting.

His initial evaluation revealed normal blood glucose levels with a normal liver function test. The kidney function test was slightly deranged with an estimated glomerular filtration rate of 54 mL/minute (creatinine clearance) with normal serum

electrolytes, thyroid functions, and fasting cortisol. Arterial blood gas analysis revealed mild normal anion-gap metabolic acidosis. His initial counts were elevated without neutrophilic predominance. His ECG showed normal cardiac activity including a normal 2D-echocardiogram and cardiac markers. He had a normal chest radiograph and ultrasonography of abdomen. His urine toxicology screen was negative for amphetamines, barbiturates, benzodiazepines, cocaine, opiates, and marijuana.

With a lack of any clue to etiological findings, a distributive shock following possible toxin intake was kept in the first place because of acute-onset unconsciousness and suspicious presentation to the emergency.

His blood pressure did not improve with fluid therapy and he was started on noradrenaline infusion and the dose was gradually increased. Foley's catheterization and Ryle's tube drainage were done. He was given mechanical ventilation because of altered sensorium and shock. His CVP remained very low despite liberal fluid therapy and inotropic support (noradrenaline 50 µg/minute). Atropine was given on trial as initial bolus doses followed by continuous infusion of 1 mg/hour; due to the possibility of vasodilator shock and unknown poisoning (OP?), pralidoxime was not administered as the type of toxin ingested was not clear. He had no bradycardia, diarrhea, chest secretions, pinpoint pupils, or fever spikes during the hospital stay. On the 4th day of admission, his blood pressure improved and inotropes were tapered off so did his sensorium, after which he was liberated from mechanical ventilation. On reviewing the history from the patient, he confessed suicidal ingestion of 30 mL of dichlorvos 4 hours before presenting to the hospital.

His renal function improved later on and he was shifted to the general ward. Autonomic function tests were performed to know the pathophysiology of the effect of poisoning that showed a normal sympathovagal response, decreased parasympathetic tone, and decreased sympathetic reactivity. The nerve conduction study showed normal sensory and motor nerve velocity and latency.

On follow-up, 4-weeks after the ingestion of dichlorvos he developed symmetric lower limb followed by upper limb sensorimotor neuropathy (delayed neuropathy). However, autonomic function tests had improved to normal.

Discussion

Though cardiovascular complications due to the above-mentioned common killer in Indian population are not uncommon, in this case we have come across a patient whose only presenting symptom was a distributive shock after dichlorvos ingestion. He was treated with liberal intravenous crystalloids and required inotropes and ventilatory support for 4-days. In dichlorvos poisoning, hypotension can be explained by cardiotoxicity or arrhythmias. However, cardiac investigations did not show any abnormality. CVP monitoring showed consistently low values despite fluid therapy after a negative history of any fluid loss. Also, we failed to elicit other common signs and symptoms of OP poisoning.

A well-known toxidrome in OP poisoning consists of salivation, lacrimation, urination, defecation, gastric cramps, and emesis symptoms. The manifestations are based on the type of receptor that is being dramatically stimulated by acetylcholine. The nicotinic receptors of neuronal subtype Nn or N2 on activation lead to tachycardia and hypertension whereas the receptor subtype Nm or N1 leads to weakness, fasciculations, and cramps. The muscarinic receptor subtype M1–M5 stimulation leads to anxiety, restlessness, ataxia, convulsions, insomnia, dysarthria, tremors, coma, respiratory depression, and circulatory collapse. Also, the M2 receptor activation leads to bradycardia and hypotension.[4] Animal study for the vascular effects of OP compounds noted peripheral vasodilatation due to the effect of acetylcholine on muscarinic receptors on the vascular endothelium and these compounds may also block nicotinic transmission at the sympathetic and parasympathetic ganglia further leading to inhibition of the baroreceptor reflexes. Chronic OP exposure may cause oxidative damage to the vascular endothelium as noticed on the histopathological examination in the animal models.[6] The similar effects in acute OP exposure are questionable. However, in our present case, we see it.

Organophosphate-induced delayed polyneuropathy (OPIDN) appears to follow the phosphorylation and subsequent aging of an enzyme in axons called neuropathy target esterase.[7] OPIDN usually sets in after 7–21 days of exposure. The spectrum of symptoms is paresthesias and calf pain. The weakness of subacute onset and slow progression over 2 weeks involving the distal leg muscles causing foot drop is the initial presentation followed by small muscles of the hands which later involve even the truncal muscles while the cranial nerves and the autonomic nervous system are not involved. Deep tendon jerks are absent. Clinical involvement of the corticospinal tracts and the dorsal columns becomes apparent when the peripheral neuropathy improves showing brisk ankle and knee jerks. The prognosis of patients with mild neuropathy is good though those with severe neuropathy are usually left with persistent deficits like claw hand, foot drop, persistent atrophy, spasticity, and ataxia. Delayed OP neurotoxicity, especially with triorthocresyl phosphate, has been reported following the outbreaks in Mumbai where 58 patients were affected. After that, four outbreaks have been reported from Bengal And in 2015–2016, eight cases were reported from Western India; however, the substances of OP were not characterized.[8]

Based on the experience of managing two patients with atropine in OP-associated vasodilatory shock, Buckley et al. summarize atropine therapy is preferable as compared to inotropic therapy as OP compounds have variable responses on the blood vessels.[9] It causes vasodilation as well as vasoconstriction of a few organs. Administering inotropic agents causes more harm by deranging the organ functions. This undesired effect is not expected with atropine administration, even in larger doses. The same thing happened in our case where the requirement of inotropes decreased and stopped before the atropine.

Another case report of three patients who had distributive shock following dimethoate ingestion, all of them presented with cholinergic features that responded to atropine administration but continued to have a shock that required high-dose inotropic support. All three expired unfortunately but a special note was made of the first two patients who had peripheral vasodilatation as noticed by the distended veins in their extremities. The second patient's blood pressure responded to the addition of dobutamine to a dopamine infusion, but the initial presentation of the patient was poor and he developed cardiac arrest during intubation.[10]

The clinical course of patients in terms of the duration of intensive care unit stay, mechanical ventilation, and inotropic support is not predictable. Various factors including pseudocholinesterase (PChE), GCS score, acute physiology and chronic health evaluation II score, and creatine phosphokinase are used by various clinicians to assess the severity of OP poisoning. A study by Pradeep Kumar et al. showed

PChE levels <1000 IU/L were associated with a more morbid course in acute OP poisoning with a need for prolonged mechanical ventilation and vasopressor support as well as a longer hospital stay.[11]

In a cohort study by Jayasinghe et al. to see autonomic function anomalies in acute OP poisoning, a statistically significant autonomic dysfunction was seen in the first assessment of patients compared to the controls in the change of diastolic blood pressure three minutes after standing, heart rate on deep breathing (HR-DB), sympathetic skin response–amplitude, latency, postvoid urine volume, and size of the pupils.[12] At 6 weeks, recovery of autonomic dysfunction was observed except in HR-DB similar to our case. However, sensory–motor neuropathy appeared as a delayed phenomenon. The reason for the same is unclear, maybe due to the initial presentation of a distributive shock.

This syndromic presentation of dichlorvos poisoning is dramatic, can mimic many other medical conditions, and will be a challenge for physicians from a diagnostic point of view as well as in the management. A lot more evidence needs to be sought for in the management of this type of syndromic case.

Conclusion

- Dichlorvos poisoning can present with distributive shock without any classical features of OP poisoning.
- Atropine trial in distributive shock in unknown diagnosis rewards sometimes.
- Distributive shock in OP poisoning patients may suggest the future possibility of delayed neuropathy.

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