Case Report

Severe Hypothermia Causing Ventricular Arrhythmia in Organophosphorus Poisoning

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Abstract

Organophosphorus poisoning cases are routinely treated across all Intensive Care Units adjoining the rural areas where agriculture is the main source of income. We present a unique case of severe hypothermia seen in a case of organophosphorus poisoning, which led to electrocardiogram disturbances and life-threatening arrhythmias.

Keywords: Atropine, hypothermia, organophosphorus poisoning, Osborn waves, ventricular tachycardia

Introduction

Organophosphorus (OP) poisoning patients are seen commonly in rural parts of developing countries.[1] Its varied clinical manifestations are due to its action at the neuromuscular junction.[2] Though hyperthermia is a well-known entity among them, severe hypothermia causing ventricular arrhythmia is less known. OP poisoning leading to temperature dysregulation has been described in this case report.

Case Report

A 26-year-old male was admitted with endotracheal tube *in situ* after alleged consumption of OP poison 7 days ago. He was treated at a local hospital where he was ventilated in view of respiratory failure and altered sensorium. Pralidoxime and atropine daily were given along with antibiotics and other supportive care. On admission, the patient had tachycardia without any excessive secretion, henceforth decision to stop atropine was taken. On day 2 in Intensive Care Unit (ICU), the patient developed high-grade fever followed by sudden drop of body temperature. Temperature was monitored with nasopharyngeal probe which showed reading of 28°C. The patient received active as well as passive methods of warming with the help of warm saline and hot air blower. Electrocardiogram (ECG) showed the presence of Osborn waves [Figure 1] with second-degree heart block and prolonged QTc. Subsequently, the patient developed ventricular tachycardia [Figure 2] and was treated with amiodarone. Body temperature was increased gradually over the next 12 h [Figure 3]. The patient laboratory parameters showed decreased serum cholinesterase of 0.05 KU/L, hypokalemia (2.3 meq/l), and hypernatremia (154 meq/l). Other investigations such as arterial blood gas, liver function test, and complete blood picture were normal. Magnetic resonance imaging brain showed hyperintense foci in the right parietal lobe and bilateral inferior peduncles. He was tracheostomized on day 3 of ICU admission and ventilated for the next 4 days and weaned on day 5. After patient regained consciousness, neurological examination revealed bilateral weakness of lower limbs. Electroneuromyography (ENMG) showed motor neuropathy with peroneal nerve involvement. He was decannulated and discharged on day 12 of ICU stay.

Discussion

OP poisoning has been recognized as a significant cause of morbidity and mortality in the third world countries.[3] The cornerstone therapies are still atropine, acetyl cholinesterase reactivators, and supportive care. Apart from causing respiratory failure, arrhythmias, and cardiomyopathies, OP poisons also affect the body temperature regulatory

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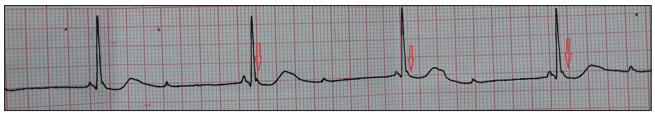

Figure 1: Electrocardiogram showing Osborn waves.

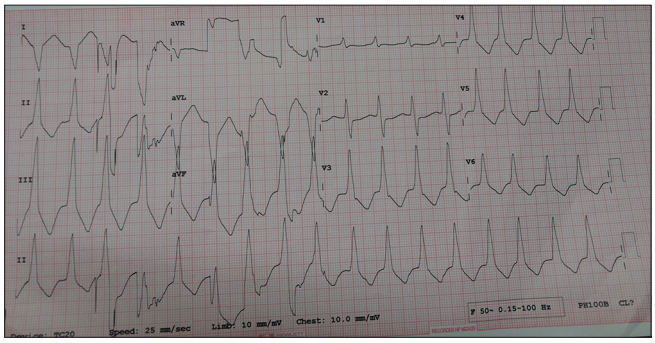

Figure 2: Ventricular tachycardia seen during hypothermia.

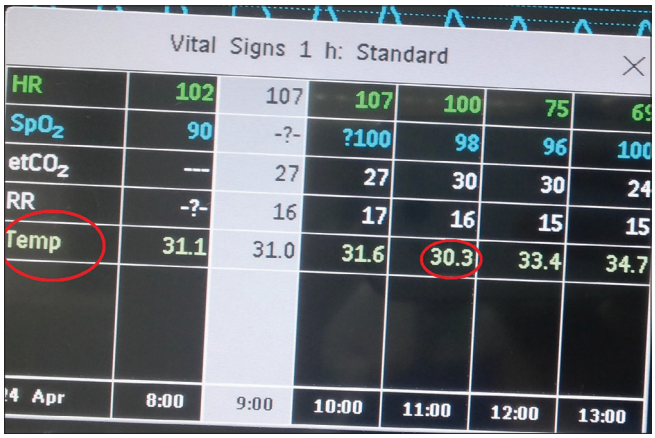

Figure 3: Trends of vital parameters on monitor showing hypothermia.

mechanism.[4] OP compounds act on hypothalamus, thereby affecting the homeostatic mechanism of temperature regulation.[5] It has been seen that the OP poisoning causes an initial fall in body temperature followed by normal-to-high body temperatures.[6] The rise in temperature has been attributed to the use of anticholinergics and concomitant ICU-acquired infections during the stay in ICU.[6] There are very few cases reported in literature regarding the occurrence of hypothermia of this magnitude with ECG changes. Muthu *et al*. reported similar case of hypothermia with cardiotoxicity in OP poisoning.[7] In this case, we had verified the side effects of all ongoing medications for causation of hypothermia before we could attribute it to OP compound. Catecholamine surge and various inflammatory mediators have been postulated to be the likely mechanisms for various cardiac arrhythmias.[8] The most common ECG abnormality seen in hypothermia is prolongation of QTc seen in 62% of individuals with ventricular tachycardia occurring in only 5.6%.[8] In this case, QTc was 695 ms and it led to ventricular tachycardia. It has been observed that ventricular tachycardia occurs in hypothermia due to triggered automaticity.[9] Sympathetic nerve activation by drugs, hypothermia, electrolyte abnormalities, and coronary ischemia may aggravate abnormalities in myocardial ion channel leading to triggered automaticity.[10] In our case, there was hypokalemia, organophosphorus compound, and hypothermia which could have been the triggering factors for arrhythmia.

Osborn waves were reported by Osborn in his hypothermia studies in dogs in 1953.[11] He attributed such occurrence to hypothermia-induced acidosis. Osborn waves are seen in 80% of patients with body temperature more than 35°C.[12] Brugada syndrome, cocaine use, acute ischemic events, and hypercalcemia also show the occurrence of Osborn waves.[13] The transmural voltage gradient created by increased epicardial flow of potassium when compared to endocardium during ventricular repolarization leads to occurrence of J waves or Osborn waves.[14] Osborn waves are seen in lead II, aVF, V5, and V6. Further involvement of anterior leads is seen when there is progressive hypothermia.[15]

Conclusion

The occurrence of hypothermia needs to be recognized as one of the complications of OP poisoning. It may not only occur during the early phase of treatment but also can occur at late stage as seen in the case report. The probable reasons could be the effect of the poison over the hypothalamus and tapering of atropine. Recognizing hypothermia by periodical monitoring of body temperature can prevent life-threatening arrhythmias.

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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Nil.

Conflicts of interest

There are no conflicts of interest.

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