

Posterior Reversible Encephalopathy Syndrome in a patient of organophosphate poisoning

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Abstract

A 32-year-old male presented with a history of consuming some organophosphorous compound with suicidal intention. He was treated with atropine, pralidoxime, ventilator support. During stay patient had persistent irritability, tachycardia and hypertension despite sedation and labetalol infusion. He developed headache, visual blurring hemiparesis and focal seizures. Magnetic resonance imaging of the brain revealed multifocal hyperintensities mainly in subcortical areas of parietal and occipital regions in T2-weighted images, with increased values of Apparent Diffusion Coefficient, suggesting posterior reversible encephalopathy syndrome (PRES). The possibilities of PRES caused by organophosphorous poisoning either due to hypertension caused by autonomic deregulation or direct neurological toxicity has been discussed.

Keywords: Organophosphorous poisoning, poisoning, posterior reversible encephalopathy syndrome

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Introduction

Organophosphorous poisoning (OP) are known to have many neurological manifestations such as seizure and coma.^[1] We present a case of posterior reversible encephalopathy syndrome (PRES) caused by OP.

Case Report

Case

A 32-year-old male, with no other medical problems previously, presented with excessive salivation, sweating and breathlessness. Further history revealed that he had consumed OP with suicidal intention, though the precise name of the poison was not known. On examination, patient's breath had a smell of an OP compound. He was in a coma with Glasgow coma scale was E1 M4 V3, with bilateral pin point pupils. Examination revealed a heart rate of 80 beats/min, blood pressure of 140/90 mmHg, had had excessive secretions and crepitations in

the chest. Gastric lavage was performed with activated charcoal. Patient was started on intravenous Atropine bolus, followed by an infusion. Intravenous Pralidoxime 1g was given 6th h for 3 days. Patient was intubated and ventilated. Laboratory investigation revealed raised plasma cholinesterase levels, confirming the diagnosis of OP. During his stay in intensive care unit, patient had tachycardia with heart rate varying between 110 and 140/min and high blood pressure varying between 140/90 and 200/110 mmHg, presumably due to sympathetic over-stimulation caused by the OP. The patient was restless and irritable requiring sedation and analgesia with midazolam and fentanyl infusion. From the day 4 of poisoning, patient was complaining of headache, blurring of vision and weakness on the right side of the body. Pupils were equal bilaterally, miotic, sluggishly reacting to the light. On examination, motor power was 3/5 right limbs and 5/5 left limbs. His secretions had reduced and hence atropine was weaned off on the 6th day. Despite sedation with midazolam and propofol, the heart rate and blood pressure remained high. An infusion of labetalol was started for control of heart rate and blood pressure. Tracheostomy was carried out on 8th day and ventilator support was continued. On 9th day, patient developed focal seizures in the

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right upper limb and face. Intravenous Phenytoin was started. Focal seizures persisted next day; hence, tablet levetiracetam 500mg was started [Table 1].

Magnetic resonance imaging (MRI) of the brain performed on 10th day revealed multifocal hyperintensities mainly in subcortical areas of parietal and occipital areas in T2-weighted images [Figure 1]. Mild sulcal effacement was seen. To rule out ischemic infarct, apparent diffusion coefficient was calculated, which also showed increase values indicating PRES [Figure 2].

Oral metoprolol was started along with labetalol infusion for better control of blood pressure. Oral lorazepam 1 mg was started for control of agitation. Patient showed improvement in vision and mental state from the 12th day. Patient's blood pressure normalized from the 16th day; hence, metoprolol and labetalol were tapered off. He was weaned from ventilator, shifted to the ward and tracheostomy was decannulated over the next few days. Patient gradually recovered motor power over next 5 days and was discharged from hospital without any neurological deficits. Repeat MRI on 28th day

revealed complete resolution of radiological features of PRES.

Discussion

OP are known to cause three well-defined neurological syndromes.^[1] (a) Initial cholinergic crisis during, which mental obtundation, cognitive impairment, convulsions or coma may be present. (b) The intermediate syndrome, in which proximal muscle weakness, cranial nerve palsies and respiratory muscle weakness are seen. (c) Delayed organophosphate induced polyneuropathy that usually presents as calf and distal muscle weakness, foot drop or claw hand. Unilateral proximal and distal muscle weakness and focal seizures presenting about a week after poisoning are not seen in delayed neurotoxicity of OP. Development of latter symptoms along with a history of persistent high blood pressure raised the suspicion of intracranial hemorrhage. MRI imaging revealed multifocal hyperintensities mainly in the subcortical areas of parietal and occipital regions in T2-weighted images. Anything that is T2-hyperintense (e.g., cerebral edema as well as ischemia) can falsely elevate signals on diffusion

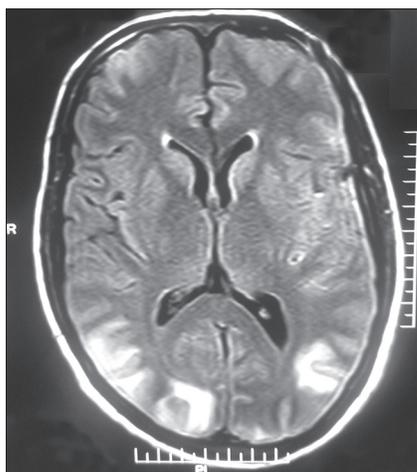


Figure 1: Multifocal hyper intensities mainly in subcortical areas of parietal, occipital and temporal cortex in T2-weighted images, with increased values of apparent diffusion coefficient

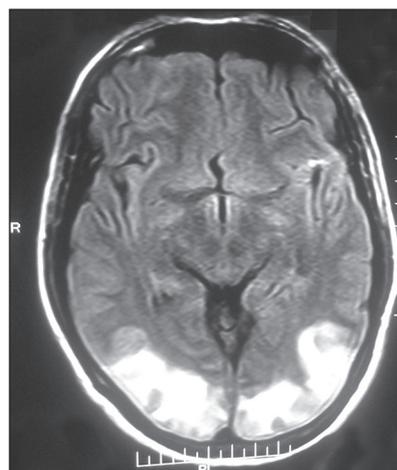


Figure 2: T2-hyperintense, elevated signal on diffusion weighted imaging ("T2-shine through")

Table 1: Course during of the patient Intensive care unit stay

	D1	D4-8	D9	D12-D16
Symptoms	Excessive salivation, sweating and breathlessness	headache, blurring of vision and weakness on the right side of body	Drowsy, focal seizure in the right upper limb and face	Improved alertness
Signs	HR: 80/min BP: 140/90 mm Hg	HR: 110-140/min BP: 140/90-200/110 mm Hg	HR: 100/min BP: 140/90 mm Hg	HR: 90/min BP: 120/80 mm Hg
Drugs	Atropine Pralidoxime	Midazolam Fentanyl, Propofol sedation Labetalol.	Phenytoin Levetiracetam lorazepam Metoprolol	Phenytoin Levetiracetam lorazepam Continued
Intervention	Ventilation	Stopped: Atropine Tracheostomy	Labetalol infusion MRI brain CSF analysis	Metoprolol labetalol stopped Stop ventilation Tracheostomy decannulated

MRI: Magnetic resonance imaging; CSF: Cerebrospinal fluid

weighted imaging (“T2-shinethrough”). To eliminate the effect of T2-signals was calculated that also showed increase values confirming the diagnosis of PRES. PRES is a clinical-neuroradiological entity involving mainly the posterior circulation of the central nervous system and appearing as vasogenic edema on MRI.^[2] The exact pathogenesis of vasogenic edema in PRES remains unclear; possibly it is due to the failure of autoregulation and endothelial damage.^[2] Hypertension may lead to disruption of the blood brain barrier and cytotoxic drugs like cyclosporine known to cause direct damage to the endothelium may lead to PRES.^[3] Hypertensive encephalopathy is the most common cause of PRES. We could not get any report of PRES caused by OP. Some of the organophosphates, especially those crossing the blood brain barrier are known to cause direct neurotoxic effects and convulsions.^[4] The exact organophosphorus compound consumed by our patient was not known. Hence, its direct neurotoxic effect can not be ascertained.

Though most of the organophosphates are more likely to cause bradycardia and hypotension, in a few patients tachycardia and hypertension may occur due to their nicotinic action.^[5] Our patient had tachycardia and hypertension from the time of presentation, which continued despite stopping atropine infusion and adequate sedation. Hence, autonomic dysregulation leading to persistent hypertension as well as direct

neurotoxic effects by OP, together might have contributed to the development of PRES.

Therefore, we conclude that in patients of organophosphate compound poisoning who develop hypertension, the possibility of PRES should be entertained. In patients with toxic encephalopathy MRI scan with Diffusion Weighted Imaging and apparent diffusion coefficient calculation should be carried out to differentiate vasogenic edema from PRES.

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