Case Report

Role of Magnetic Resonance Imaging in Diagnosing Neurological Complications in Intermediate Syndrome of Organophosphate Poisoning

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Abstract

Organophosphate poisoning (OP) is a very common mode of suicide in rural and urban areas due to the wide availability of pesticides. The identification of OP and timely referral for appropriate supportive care can be lifesaving. Injury to the central nervous system is a serious entity in acute OP. Application of modern imaging techniques like diffusion weighted imaging increases the diagnostic rate of brain injury in the early period and can provide evidence for medical treatment. We present the imaging features in the intermediate syndrome of OP.

Keywords: Diffusion-weighted imaging, infarcts, intermediate syndrome, magnetic resonance imaging, organophosphate, poisoning

Introduction

Organophosphate poisoning (OP) generally presents initially as acute cholinergic crisis which manifest as excessive salivation, lacrimation, sweating, vomiting, diarrhea, urination, pinpoint pupil, mental status changes and seizures. Treatment is targeted at clinical findings of cholinergic excess in OP.[1] In some patients, after acute cholinergic crisis is over an intermediate syndrome of muscle weakness develops leading to respiratory failure. Delayed complications such as organophosphate-induced delayed polyneuropathy (OPIDN) and chronic organophosphate-induced neuropsychiatric disorder (COPIND).[2] Well localizing magnetic resonance imaging (MRI) findings attributable to OP have not been previously reported. Here, we present the MRI findings of neurological complications in intermediate syndrome of OP.

Case Report

A 26-year-old man was brought to the Emergency Department by family 1 h after ingesting a bottle of an unknown insecticide. On arrival, he was having excessive secretion and multiple episodes of vomiting. He had two episodes of vomiting while in the emergency department. On examination, he was tachycardic and had pinpoint pupils. The patient was suspected to have OP based on clinical features. Nasogastric lavage was performed. He was also atropinized and started on pralidoxime and was subsequently shifted to Intensive Care Unit He initially presented with a cholinergic crisis, but later on, she developed features of extrapyramidal involvement such as temporary loss of speech, muscle tone abnormalities, tremor, and rigidity. He was diagnosed as having the intermediate syndrome with extrapyramidal manifestations. Routine laboratory tests were within normal limits, except for white blood cell count 18.9 × 109/L (reference range, 4.00–10.80 × 109/L). Blood gases showed acidosis (pH 7.212, pO_2 123 mmHg, saturatedO_2 97.2%, pCO_2 35.5 mmHg, actual base excess – 12.6 mmol/L, and lactate 1.1 mmol/L). However, serum transaminases, alkaline phosphatases, renal function tests, and serum electrolytes were in normal range. Furthermore, chest X-ray was normal and electrocardiogram showed prolonged QTc interval (49 ms). He developed respiratory distress with carbon dioxide retention. His pulse oximetry revealed hypoxia, and he was started on high flow oxygen through facemask. He then developed an episode of generalized tonic–clonic seizures during which he was

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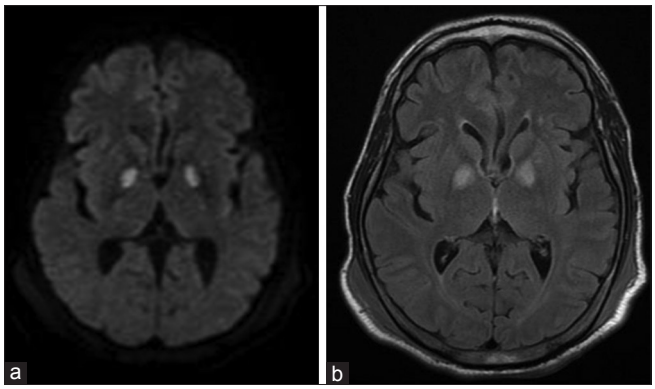

Figure 1: (a) Diffusion-weighted magnetic resonance image showing symmetrical restriction in bilateral globus pallidus. (b) T2 fluid-attenuated inversion recovery magnetic resonance image showing symmetrical hyperintensities involving bilateral globus pallidus with extension to posterior limb of bilateral internal capsules.

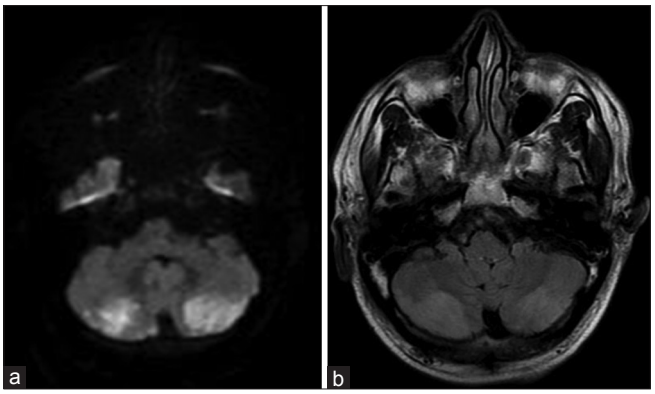

Figure 2: (a) Diffusion-weighted magnetic resonance image showing multiple foci of restriction in bilateral cerebellar hemispheres. (b) T2 fluid-attenuated inversion recovery magnetic resonance image showing multiple hyperintensities corresponding to areas of restricted diffusion in bilateral cerebellar hemispheres.

intubated. Glasgow coma score deteriorated, and patient became restless. The patient was sedated and MRI of the brain was requested. MRI revealed diffusion restriction with symmetrical T2/T2 fluid-attenuated inversion recovery hyperintensities involving bilateral globus pallidus with extension to posterior limb of bilateral internal capsules [Figure 1a and b] and bilateral cerebellar hemispheres [Figure 2a and b] suggesting acute infarcts.

Discussion

Four types of OP-induced neurological syndromes had been described. These are: (1) Cholinergic crisis/Type 1 syndrome; (2) intermediate syndrome/Type 2 syndrome; (3) OPIDN; and (4) COPIND.[3] Among these, cholinergic crisis is the most common following exposure to larger doses of OP compounds.[4] The extrapyramidal symptoms, which occur rarely as a part of the intermediate syndrome, are thought to be due to the inhibition of acetylcholinesterase in the human extrapyramidal areas.[5] Groups of nuclei in basal ganglia are more susceptible to toxic injuries in the absence of efficient detoxification pathway.[6]

Symmetrical basal ganglia involvement may occur due to metabolic disorders such as Wilson's disease, Hallervorden–Spatz, methylmalonic acidemia, hypoxic insults, emboli or toxins like methanol, cyanide, carbon monoxide, and toluene.[7] Basal ganglia infarcts are rare as an entity and even rare as an effect of OP poisoning.[8]

Intermediate syndrome develops 12–96 h after exposure and reflects a prolonged action of acetylcholine on the nicotinic receptors and is characterized by muscular weakness in the ocular, neck, bulbar, proximal limb, and respiratory muscles. Occasionally, dystonic posturing may be observed, and respiratory muscle weakness may be the first clue to the onset of this syndrome. The sensory functions characteristically remain normal and full recovery is evident in 4–18 days. Severe intoxication may cause emotional irritability, mental obtundation, cognitive impairment, coma, and convulsions because of central nervous system effects. In the cholinergic phase, paralysis usually passes off within 48–72 h but complete clinical recovery from all the effects may take up to a week after exposure to these compounds.[9] Phase I (acute cholinergic crisis) occurs secondary to continued depolarization at the neuromuscular junction. Phase II (intermediate syndrome) develops 24–96 h after Phase I resolution. It is characterized by weakness of respiratory muscles, proximal muscles, and cranial nerve palsies. Extrapyramidal symptoms are uncommon. Phase III (organophosphate-induced delayed polyneuropathy) presents 2–3 weeks after OP exposure.[10]

Conclusion

An intermediate syndrome that occurs between the early cholinergic crisis and late onset neuropathy is rare and often missed. Extrapyramidal symptoms as part of the intermediate syndrome are very rare and are due to the involvement of basal ganglia. Novel signal abnormalities on MRI help in timely recognition of neurological complications like basal ganglia infarcts in OP.

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Conflicts of interest

There are no conflicts of interest.

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