CASE REPORT
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ORGANOPHOSPHORUS POISONING PRESENTING WITH EXTRAPYRAMIDAL INTERMEDIATE SYNDROME

Internal Medicine

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ABSTRACT

Organophosphorus (OP) pesticide poisoning which is common in rural India, usually presents with acute cholinergic crisis, which is due to increased acetylcholine because of the inhibition of the enzyme acetyl-cholinesterase. Central Nervous System (CNS) manifestations are rare, and even rarer is the presentation with extrapyramidal symptoms. The authors report a case of organophosphorus poisoning presenting with torticollis 4 days after organophosphorus exposure. The patient had inhalational exposure to organophosphorus compound while spraying in the fields and developed intermediate syndrome 4 days later. But rather than having respiratory depression, the patient developed torticollis. The patient was successfully managed with Inj Pralidoxime and supportive care. We wish to bring awareness in the medical profession and the general public regarding this rare but life threatening presentation of organophosphorus poisoning.

KEYWORDS

Organophosphorous, poisoning, extrapyramidal, torticollis

INTRODUCTION

Organophosphate (OP) poisoning is a significant cause of morbidity and mortality in developing countries including India.[1] In recent years, various investigations have demonstrated that OP exposure causes neurobehavioral changes after both acute and chronic exposure. The most commonly encountered toxic effects in humans are peripheral (muscarinic and nicotinic effects). However, the central nervous system's effects of OP exposure have received less attention in the medical literature than peripheral effects. Any neurological complication in the form of the intermediate syndrome is rare and its presentation with extrapyramidal symptoms is even rarer. Neurological manifestations like choreoathetosis, opisthotonos, facial grimacing, extrapyramidal symptoms, and typical parkinsonism following OP exposure have been reported.[2] The delay in onset of these signs, following poisoning, varied from 4 to 40 days, and they disappeared spontaneously in about 1 to 4 weeks in those who survived.[3]

Case Report

A 20-year-old male farmer, who presented with torticollis was admitted to the hospital. He was exposed to OP compound (monocrotophos 36%) 4 days ago. Within 4 hours of inhalational exposure to the insecticide, while spraying over the field, the patient started experiencing mild abdominal cramps and had increased salivation, which resolved spontaneously in 4-5 hours and for which he did not seek any medical attention. 4 days later he started having abnormal neck muscle contraction and tilting of the head(torticollis), for which he presented to our hospital.

Figure 1: Presentation with torticollis

There was no history of fever, seizures, head trauma, drug intake, delayed developmental milestones or any neurocognitive decline. There was no history of substance use. On examination, he had increased tone in his neck muscles. Cranial nerves were normal. Tone, power and reflexes were normal in both upper limbs and both lower limbs. Babinski was negative. The rest of the examination was unremarkable. Sr Cholineesterase was reduced [1350 IU/L (Ref range: 4620-11500 IU/L)]. Other haematological and biochemical indices as well as computed tomography of the brain were normal. The patient was treated with Inj. Pralidoxime and Inj. Atropine SOS. The patient's symptoms improved after about 6 hours. Torticollis resolved. At follow-up after 1 month, no symptoms had recurred.

DISCUSSION

Our patient with organophosphorus poisoning initially had mild symptoms of increased cholinergic drive, but later on, he developed features of extrapyramidal involvement i.e. torticollis. It was on detailed enquiry, that a history of exposure to organophosphorus pesticide was elicited, and confirmed biochemically. He was diagnosed as having intermediate syndrome with extrapyramidal manifestations and managed accordingly. A similar case reported by Sumantra Sarkar et al[11] developed tremors, and intermittent jerky movements, in all four limbs with the extension of the neck after 4 days of OP compound consumption. The patient responded well to supportive management and all signs and symptoms had resolved on 3 month follow-up. These 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.[4,5] In the corpus striatum, a balance exists between dopamine as the inhibitory and acetylcholine as the excitatory system. The dopaminergic system inhibits the cholinergic neurons and vice versa[6]. Excessive acetylcholine, as can occur in OP exposure, suppresses dopaminergic activity, and hypersensitivity of postsynaptic dopaminergic neurons may result. Recent studies[7] have shown that acute OP intoxication may cause neurobehavioral deficits by producing an inflammatory response. These neurobehavioral deficits may be due to the direct neurotoxicity of proinflammatory cytokines (e.g. TNFa, IL-1b, and interleukin-6) or via interactions between these proinflammatory cytokines and excitotoxic glutamatergic pathways or due to excessive activation of NMDA receptors leading to neurodegeneration. Recent evidence also suggests that the cholinesterase-based mechanism of OP toxicity cannot alone account for the neurological symptoms. OP interactions with proteins involved in fundamental neuronal processes such as axonal transport, neurotrophin support, and mitochondrial function lead to these neurological symptoms.[8] Increased susceptibility of the basal ganglia

nuclei to the toxic products, in the absence of efficient detoxification pathways, may also be responsible.[9] Atropine or pralidoxime are ineffective in treating this condition.[10] Although abnormal signal intensities in the region of basal ganglia have been demonstrated in brain MRIs for some patients, most cases are reported to have normal neuroimaging.[3] The NCCT brain was normal in our patient.

CONCLUSION

As it is a very common poisoning in rural areas, identification of the syndrome and timely referral for appropriate supportive care can be life saving. Also, we suggest suspecting OP poisoning in cases presenting with the extra-pyramidal syndrome. Also, physicians should watch out for extra-pyramidal syndrome in cases of OP poisoning, to initiate early treatment.

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