

Case Report

Mania following organophosphate poisoning

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ABSTRACT

Organophosphate poisoning is the most common poisoning in developing countries. Although the acute muscarinic and nicotinic side-effects of organophosphate poisoning are well known and easily recognized, but neuropsychiatric changes are rarely reported. We are reporting a case of a 33-year-old female who developed manic episode following acute organophosphate poisoning.

Key words: Mania, organophosphate, poisoning

Introduction

Organophosphate (OP) poisoning is the most common poisoning in developing countries with half of the admissions to the emergency with poisoning being due to these compounds. In recent years, various investigations have demonstrated that OP exposure causes neurobehavioral changes after both acute and chronic exposure. The central nervous system effects of OP exposure have received less attention in the medical literature than peripheral effects. Tabershaw and Cooper^[1] noted that certain patients displayed vague mental changes such as irritability, memory disturbances and dream abnormalities for several months after their apparent recovery from OP poisoning. Gershon and Shaw^[2] described the development of schizophrenic and depressive symptoms after exposure to OP insecticides. Neurological manifestations like choreoathetosis, opisthotonos, torticollis, facial grimacing, extrapyramidal symptoms, and typical parkinsonism following OP exposure have been reported.^[3] Other behavioral disturbances such as learning deficits, increased anxiety, diminished activity and impulsivity have also been reported.^[4] No case of manic episode after acute intoxication by OP is till now reported. We are reporting a

case of 33-year-old female who developed manic episode following acute OP poisoning.

Case Report

Mrs. A., 33-year-old Hindu married female presented to Psychiatry outpatient department (OPD) with complaints of over talkativeness, big talks, irritability, over familiarity, decreased sleep and increased physical activity for last 7 days. On evaluation there was history of accidental ingestion of OP insecticide chlorpyrifos 5 days before the onset of psychiatric symptoms. Within 4 hours of ingestion of the insecticide, symptoms of acute OP poisoning developed and the patient was admitted to the hospital suffering from severe abdominal cramps, hyper salivation and excessive sweating. She was treated with atropine and recovered rapidly from the acute muscarinic and nicotinic symptoms. Day after her discharge from the hospital she developed the psychiatric symptoms. There was no history of any substance use. There was no contributory family history. Premorbidly she was fairly well adjusted. Her physical examination was within normal limits. Serum cholinesterase levels were low (650 U/L; normal: 3500-8500 U/L). Hematological and biochemical indices were normal as was computed tomography of the brain and EEG. She was treated with tablet olanzapine 10 mg per day and her symptoms improved significantly within next 10 days.

Discussion

The mechanism of neurobehavioral symptoms following OP exposure is yet unclear. It may be that in OP exposure

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the excess acetylcholine merely upsets the balance of transmitter systems active in cortical functions or alternatively leads to presynaptic inhibition of acetylcholine release via a negative feedback loop. Another mechanism that may be postulated to explain these symptoms is dopamine receptor hypersensitivity as seen after neuroleptic administration.^[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. Schizophrenic psychosis or depressive illness may occur after obvious acute OP intoxication or may follow chronic subliminal exposure. Recent studies^[7] have shown that acute OP intoxication may cause neurobehavioral deficits by producing inflammatory response. These neurobehavioral deficits may be due to direct neurotoxicity of proinflammatory cytokines (e.g. TNF α , IL-1 β , 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 evidences also suggest that the cholinesterase-based mechanism of OP toxicity cannot alone account for the neuropsychiatric symptoms. OP interactions with proteins involved in fundamental neuronal processes such as axonal transport, neurotrophin support, and mitochondrial function leading to these neuropsychiatric symptoms.^[8]

Although the acute muscarinic and nicotinic side-effects of OP poisoning are well known and easily recognized, but neuropsychiatric changes are rarely reported. This has important clinical implication for any community such as in India, where agriculture forms a large part of the economy and hence insecticide use is very common.

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