Commentary

In resource-poor settings and in well-developed countries the organophosphate (OP) poisoning is associated with high morbidity and mortality rates.[1] Acute OP poisoning can manifest three different phases of toxic effects, namely, acute cholinergic crisis, intermediate syndrome (IMS), and delayed polyneuropathy.[2] The IMS is a delayed onset of muscular weakness and paralysis that occurs 1-4 days after the resolution of acute cholinergic syndrome in acute OP poisoning.[3] It was first reported by Wadia *et al*. in 1974[4] as "type II paralysis after organophosphate poisoning". In 1987, Senanayake and Karalliede termed this pattern of weakness as "intermediate syndrome" due to the fact that it arises between the period of early cholinergic syndrome and the late onset peripheral neuropathy.[3] The IMS occurs in 20-68% of patients following oral exposure to OP pesticides. Various factor accounts for this difference, including the nature of OP compound, severity of poisoning and inadequate Oxime therapy, etc.[5] Certain OP like parathion, methylparathion, malathion, and fenthion are commonly associated with this condition.[1] Some clinicians suggest that IMS may result from inadequate oxime therapy (subdosage, shorter duration of therapy, and modality of administration). There are other proposed mechanisms of IMS, which include different susceptibility of various cholinergic receptors, muscle necrosis, downregulation or desensitization of postsynaptic acetylcholine receptors, failure of postsynaptic acetylcholine release, and oxidative stress-related myopathy.[2,4,6] Clinically, IMS is characterized by weakness in the ocular, neck, bulbar, proximal limb respiratory muscles and motor cranial nerves with occasional dystonic posturing, and was attributed to muscle fiber necrosis following acute cholinergic crisis. Extra pyramidal symptoms (EPS) such as tremor, rigidity, or dystonia as a part of IMS are very rare.[7] Although this syndrome has been described for decades, due to sometimes diverse clinical picture, it often remains undiagnosed, at least until the occurrence of significant respiratory weakness. The controversy exists regarding not only the question of whether IMS is a clearly defined entity, but also its exact etiology, risk factors, diagnostic parameters and required therapy.[6] The authors report a case of a 12-year-old adolescent girl with OP poisoning who[8] was successfully treated with gastric lavage, atropine, antibiotics and intravenous fluid, in the acute phase of poisoning but 4 days later suddenly developed clinical features of IMS that include loss of speech, tremor and intermittent jerky movements, in all four limbs with extension of neck, followed by coma and referral to intensive care anticipating respiratory failure. Although the patient regained full alertness during the next 10 days, the extra-pyramidal symptoms such as tremor and intermittent jerky movements persisted and the patient developed rigidity, mask like face, and intermittent hypertonia with slurring and a monotonous speech. At follow up after 3 months, all her signs and symptoms had completely reversed. Continuous treatment with atropine and other supportive measures with close monitoring did not prevent the development of EPS, most likely due to lack of oxime therapy and because of chemical structure and lipophilicity of ingested fenthion. The IMS with extra-pyramidal symptoms in an adolescent following organophosphorous poisoning presented in this study emphasizes the need for careful monitoring and sufficient treatment of those patients with atropine and oximes. Mortality from respiratory paralysis can be prevented by early recognition of the syndrome and prompt ventilatory support.

Mostsfa Amr

Department of Psychiatry, College of Medicine, Mansoura University, Egypt

Address for correspondence:
Dr. Mostsfa Amr,
Department of Psychiatry, College of Medicine,
Mansoura University, Egypt.
E-mail: mostafapsy@yahoo.com

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