CLINICAL, ELECTROPHYSIOLOGICAL AND BIOCHEMICAL STUDIES OF INTERMEDIATE SYNDROME IN ACUTE ORGANOPHOSPHOROUS POISONING

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Objectives: (1) To examine if Type I paralysis and intermediate syndrome (IMS) represent distinct forms of paralysis or a clinical continuum. (2) To examine the roles of neuromuscular block and myopathy in development of IMS. ***Methods:*** Forty patients with acute OP poisoning underwent focused neurological examination for muscle weakness and cholinergic crisis. Patients were grouped into 4 categories: Category 1 − No muscle weakness, Category 2 − Type I neuroparalysis resolving with no IMS, Category 3 − Type II neuroparalysis evolving into IMS, Category 4 − No Type I weakness, develops IMS. Electrophysiological studies including serial Repetitive Nerve stimulation (RNS) and single Compound muscle action potential (CMAP) and Direct Muscle stimulation (DMS) tests were done on 10 out of 40 patients. Blood acetyl cholinesterases (AChE) and muscle iso enzymes (LDH and CPK) were studied on alternate days and their temporal profiles analysed according to the severity of poisoning and the development of IMS. OP compound levels were measured on alternate days.

Results: 15 out of 40 patients (37.5%) developed IMS (Cat 3) and 7 out of 40 patients developed only Type I neuroparalysis (Cat 2). Distribution of weakness was similar in both the groups (Cat 2 & Cat 3) but was more severe (p=0.006 shoulder flexion) and prolonged (p=0.00) in category 3 patients. The most commonly consumed insecticide was Monocrotophos- 17 cases (51.5%). Higher mean levels of Monocrotophos levels at admission were observed in patients who developed IMS (Cat 3) − 23.33 microgm/ml (p=0.20) . AChE levels were uniformly suppressed in all patients. 46.6% of the IMS (Cat 3) patients had undetectable AChE (<5 mU/umol Hb) on 3rd day. While there was elevation in muscle iso enzymes seen in all patients, there was no statistical correlation between elevation of muscle enzymes and the occurrence of IMS. Electrophysiological findings on RNS showed serial changes corresponding to the severity of weakness. Patients who developed IMS had severe and progressive decrement response and Decrement Increment (DI) pattern seen at lower frequencies in their RNS studies in comparison to the DI pattern seen only at higher and intermediate frequencies in patients who didn't develop IMS. Normal muscle excitability ruled out primary myopathy.

Conclusions: The clinical and electrophysiological results support a Type I -Type II continuum of neuromuscular weakness in acute OP poisoning and the concept of clinical spectrum disorder. The primary pathophysiology of IMS is a neuromuscular block due to inhibition of AChE and not a myopathy.