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01. Intermediate Syndrome' in Organophosphate Poisoning: Early Neuromuscular Predictors of Respiratory Failure in the Emergency Department

Ria Singh Rawat[1], Josh Reji Joseph[2], Ummey Arshiya[2], Srijamya[2]

[1] BGS Global Institute of Medical Sciences, India
[2] Tbilisi State Medical University: Tbilisi, Tbilisi, GE

Background: Intermediate Syndrome (IMS), caused by Acute Organophosphate Poisoning is characterized by muscular paralysis due to acetylcholinesterase (AChE) inhibition at the neuromuscular junction which typically emerges 24–96 hours post-consumption, following the initial cholinergic crisis. Key predictors of IMS include initial poison dosage, low acetylcholinesterase (AChE) levels, and specific electrophysiological abnormalities. Management in the Emergency Department (ED) necessitates early recognition, standardised antidote therapy (atropine and oximes), and prompt respiratory support if required.
Objective: To evaluate the predictive value of early neuromuscular and biochemical markers in identifying patients at risk for developing IMS and subsequently respiratory failure following acute organophosphate intoxication on arrival in the Emergency Department. **Methodology:** A comprehensive analysis of studies showing the various biochemical markers such as plasma glucose and serum cholinesterase and electrophysiology as predictors of IMS in ED were extracted and analysed.
Results: Synthesis of systematic review data found that RBC Acetylcholinesterase Activity <20% normal and elevated serum Creatine Kinase (CK) values were the most reliable biochemical indicators of developing IMS in the ED. While useful for identifying organophosphate (OP) exposures, several studies have demonstrated no significant correlations ($p = .061$ to $p = .147$) between serum Cholinesterase (S-ChE) and development of IMS. Additionally, electrophysiological studies employing single-fiber electromyography (SFEMG) jitter > 33.4 microsecond during the first 24 hours had a sensitivity rate of 86% for predicting respiratory failure. No statistical significance was demonstrated regarding admission serum cholinesterase ($p = 0.119$), complete blood cell count, plasma glucose values, or use of these markers in determining either time of onset of IMS or risk of death secondary to OPS. **Conclusion**: Therefore, in order to quickly identify patients who develop IMS in the ED, it is critical to utilize RBC Acetylcholinesterase Activity and serum creatinine kinase as biomarkers rather than less-sensitive markers such as serum cholinesterase and plasma glucose. Combining these biochemical markers with rapid electrophysiological testing (particularly SFEMG jitter) can provide a sensitive window for early detection of potential neuromuscular weakness leading to respiratory failure requiring mechanical ventilation. Utilization of evidence-based predictors will allow for better triaging of patients, earlier initiation of respiratory interventions and targeted treatments such as atropine to potentially decrease the substantial risks of mortality associated with acute organophosphate poisoning.

ISSN 2076-6327

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