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Neurocognitive dysfunction in patients with poisoning: Is it toxin specific?

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Neurocognitive dysfunction in patients with critical illness is very well documented. We have studied neurocognitive dysfunction at our centre in envenomation, organophosphate poisoning and Aluminium phosphide and have encountered interesting results.

Three different studies were carried out in the patients admitted to the medical emergency with organophosphate poisoning, aluminum phosphide poisoning or neurotoxic snake envenomation. Details of the history, clinical evaluation including detailed neurologic examination, hematological and biochemical parameters were recorded. All the patients received treatment as per the protocols in the high dependency unit attached to our emergency department. They were subjected to a detailed neurocognitive testing before discharge from the hospital. These tests were chosen to assess memory, attention, concentration, executive functions and perceptual motor skills. Tests were repeated at follow-up up to three months. The longer follow up was intended but too many patients were lost to follow-up.

In the first study, 28 patients of acute organophosphate poisoning were included. The study revealed significant deficits on neurocognitive testing at baseline. The performance on neurocognitive tests improved at 3 months follow up. The improvement was not statistically significant. Significant deficits persisted but they did not correlate with any particular area in brain.

In the second cohort, 23 cases of acute aluminium phosphide poisoning were included. Cognitive dysfunction involving the domains of attention, executive function, memory and concentration was observed in survivors at base line. Verbal new learning, language/ speech and visuospatial functions were impaired in the acute phase. All the parameters showed a trend towards improvement, yet significant residual defects persisted even at 3 months.

In the most recent snake bite cohort, 48 patients were included. At Baseline, all the tests were abnormal as compared to the healthy controls. At 3 months follow up there was improvement in all score but statistically significant improvement in PGI Memory scale ($p<0.001$). Significant deficits continued to persist in relation to the frontal and temporal lobe functioning when compared to healthy controls.

Discussion: Both in organophosphate poisoning and neurotoxic snake envenomation, acetylcholine or its receptor dysfunction could have resulted in dysfunction. In neuroparalytic snake envenomation, hypoxia may have contributed as well. In aluminium phosphide poisoning, although acetylcholine or any other neurotransmitter is not directly affected but hypo tension may have resulted in altered blood flow to the brain resulting in reversible neurocognitive dysfunction. Since all the patients had reversible cognitive decline, there may be a unifying factor like ICU stay or hypoxia/hypotension which could have been responsible. The fact that at 3 months none had near normal cognition, may point to some toxin mediated damage but it is difficult to establish.

Conclusion: Reversible neurocognitive dysfunction seems to be a norm in different poisoning. Although dysfunction in cognition is partly reversible, yet return to normality is not seen at 3 months interval.