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Experiences with sodium fluoroacetate (1080) poisoning, and the use of acetamide as an antidote

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Objective: Sodium fluoroacetate is a rodenticide that is illegal in China but sporadic cases of poisoning still occur. The features of sodium fluoroacetate poisoning reported include nausea, vomiting, abdominal pain, sweating, agitation, confusion, supraventricular or ventricular arrhythmias, QTc prolongation, hypotension, acute renal failure, hypocalcemia, hypokalemia, seizures, prolonged coma. A systematic review by Proudfoot in 2006 on sodium fluoroacetate poisoning stated that although several possible antidotes have been investigated, they are of unproven value in humans. The use of acetamide in treating patients with sodium fluoroacetate poisoning showed promising results in Chinese literature. We hereby report a series of sodium fluoroacetate poisoning cases and share our experience on the use of acetamide as an antidote in treating these patients.

Methods: In 2008- 2017, 5 confirmed cases of sodium fluoroacetate poisoning were recorded by Hong Kong Poison Information Centre.

Results: Hypocalcemia was found in 4/5 cases. Drowsiness, seizures and QTc prolongation (>498 milliseconds) were also noted in 3/5 cases of severe poisoning. Agitation, confusion, vomiting, impaired renal function and elevated creatine kinase were found in 2/5 cases. EEG was performed in 2/5 cases, one was normal and the other showed persistent and generalized delta wave in background with loss of reactivity to external stimuli suggestive of encephalopathy with severe cerebral dysfunction. Acetamide is centrally stored and readily mobilized to all hospitals within one day. Intramuscular acetamide was given in 3/5 cases with severe poisoning uneventfully. Serial MRI brain were performed in 2 cases and both showed reversible T2- hyperintense signals in white matter including the corpus callosum. Two cases with prolonged impaired conscious level (2 days and 1 week) regained consciousness shortly (within 1 day) after the use of acetamide. One case suffered from residual ataxia and cerebellar syndrome for 6 months afterwards.

Conclusion: Sodium fluoroacetate poisoning resulted in rhabdomyolysis, central neurological and cardiovascular manifestations. Hypocalcemia was associated with severe clinical outcomes. Serial MRI brain scans showed reversible T2-hyperintense signals in white matter in two severely poisoned cases. Acetamide appeared to be effective in the reversal of severe neurological symptoms. Residual cerebellar syndrome that improved over 6 months was observed in one case.