CLINICAL OUTCOME OF SEVERE AMLODIPINE INTOXICATION

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Objective: To characterize severe amlodipine poisoning in Ramathibodi Poison Center.

Methods: We retrospectively reviewed cases during 2010-2015. Demographic data, clinical effects, and treatment were analyzed. Patients who took co-ingestants with recognized cardiovascular effects were excluded. Severe amlodipine poisoning was defined as a patient presented with protracted hypotension and did not respond to fluid resuscitation alone.

Results: A total of 100 cases of calcium channel blocker (CCBs) were analyzed. There were 84% involved with the dihydropyridines, particularly amlodipine (69%). Severe CCBs intoxication were accounted for 14%, of which 12 cases were poisoned by amlodipine. After excluding 3 cases, 9 patients were analyzed. Five cases were single amlodipine exposure and the other 4 took co-ingestants.Median age was 48 years old and 55.6% of all were male.Mean time to hospital was 9.8 hours. At presentation, all patients developed hypotension with good consciousness. Some clinical data were unavailable, resulting in some variables less than the total number. Three out of 9 cases presentedwith bradycardia and all had heart rate less than 100beats/minute. Acute kidney injury (AKI) initially occurred in 6/6 cases with mean serum creatinine of 3.34 mg/dL. Other clinical features were found including metabolic acidosis (5/7 cases) and hyperglycemia (1/7 cases). Besides supportive treatment, intravenous calcium and vasopressors at very high doses were needed in all cases. Only one patient was improved after receiving 30 mL of calcium gluconate. For the patients who did not respond to the treatment, additional therapies were performed. The therapy included hyperinsulinemia-euglycemia therapy (HIE) in 5 cases, intravenous fat emulsion (IFE) in 2 cases, and glucagon administration in 2 cases. There were 4 cases who received either 'HIE and IFE', or 'HIE and glucagon'. Adverse events included allergic reactions from glucagon, hypoglycemia and hypokalemia from HIE, and skin necrosis from the extravasation of calcium. Mortality rate in this severe group was 55.6% (5/9 cases). One death was from hyperkalemia which developed after stopping HIE.One case was dead from hospital acquired pneumonia. Median hospital stays for surviving cases was 7 days (4-14 days), whereas median time to death was 2.5 days (1- 3 days).

Conclusions: Amlodipine caused high mortality in severe poisoning. Most patients initially presented with good mental status even in hypotensive state. The early detection of AKI, metabolic acidosis or hyperglycemia may reflect more severity. Supportive treatments alone may not be sufficient in severe conditions. Therefore, multi-faceted treatment must be performed promptly.