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A survival case of amlodipine poisoning

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ABSTRACT

Calcium channel blockers are commonly used in various cardiovascular diseases, but its poisoning may lead to fatal outcomes. We report a case of alleged poisoning of amlodipine in a 25-year-old female who consumed 20 tablets of 5 mg amlodipine. She presented with cardiogenic shock, hypoglycemia, hypokalemia, constipation, and central nervous system symptoms which were managed with intravenous crystalloids, calcium gluconate, 5% dextrose, dopamine, and noradrenaline infusion after which patient responded well and discharged.

Key words: *Amlodipine intoxication, Calcium channel blockers, Cardiogenic shock, Dihydropyridine*

Calcium channel blockers (CCB) are the leading cause of overdose death among all cardiovascular medicines [1]. Toxicity associated with CCBs may produce serious, life-threatening complications, including bradycardia, dysrhythmias hypotension, metabolic acidosis, shock, or cardiovascular collapse. Intoxication with CCB has primarily been described for drugs such as diltiazem, verapamil, and nifedipine; whereas, there is limited clinical experience with amlodipine intoxication [2]. There are very few case reports from India related to amlodipine poisoning. We report a survival case of amlodipine intoxication with no other concomitant substance ingestion who is managed conservatively without using hyperinsulinemia euglycemic therapy.

CASE REPORT

A 25-year-old female nurse without any past medical or psychiatric history had suicidal ingestion of 20 tablets of 5 mg amlodipine. She had received gastric lavage within 30 minutes at the hospital where she was working. Then, she was shifted to Intensive cardiac care unit of our Institute. She had headache, abdominal pain, vomiting, fatigue, restlessness and was confused. She also had severe constipation for 3 days which improved later on. On presentation, her pulse was feeble with the rate of 100/min, the intra-arterial line showed BP of 68/46 mmHg. She was afebrile with the respiratory rate of 18/min, and normal jugular venous pressure. On cardiac examination, heart sounds were normal without any murmurs. The lungs were clear to auscultation. All other system examinations were within normal limits.

On routine blood examination, her hemoglobin was 9.7 g/dl, white blood cell count 23000 cells/mm^3, neutrophils 88%, lymphocytes 7%, and erythrocyte sedimentation rate 55 mm/h. Random blood sugar was 66 mg/dl for which intravenous (IV) dextrose 5% was given. Serum Na^+ was 129 mEq/L, serum K^+ 2.2 mEq/L, serum Ca^{++} 6.9 mg/dL, and serum Mg^{++} 2.4 mg/dL but Renal and Liver function tests were within normal limits. ABG analysis showed metabolic acidosis with pH 7.25, PCO_2 31.3 mmHg, PO_2 121 mmHg, HCO_3^- 14 meq/L. IV potassium chloride, bicarbonate, and calcium gluconate were given to correct potassium, bicarbonate, and calcium levels, respectively. SPO_2 was 84% for which intermittent continuous positive pressure ventilation was given. Electrocardiogram (ECG) shows sinus tachycardia with heart rate of 104/min, PR interval 140 ms, QRS interval 102 ms, QTc-394 ms, rSr in V1, ST depressions present in II, III, aVF, V2-V6 leads and ST elevation in aVR which mimics with myocardial infarction (Fig. 1). However, her cardiac enzymes (TropT and CK MB) were normal. Echocardiography shows normal left ventricular function without any regional wall motion abnormality.

She was given a rapid infusion of 1 L of IV fluid followed by maintenance infusion, but mean arterial blood pressure remained 55 mmHg. In addition, 10 ml of 10% calcium gluconate was administered every 15-20 min with ECG and serum calcium monitoring, but there was no improvement in arterial blood pressure. Dopamine was uptitrated to 15 µg/kg/min and noradrenaline 15 µg/min to increase the patient's inotropy. 12 h later, the patient's blood pressure increased to 80/56 mmHg and after 48 h blood pressure reached to 100/60 mmHg. Then, tapering of inotropes started and stopped on the 3rd day and patient was discharged in stable condition on the 6th day when her all symptoms relieved, electrolytes and ECG (Fig. 2) became normal. An informed written consent was taken from the patient for the publication of this case report.

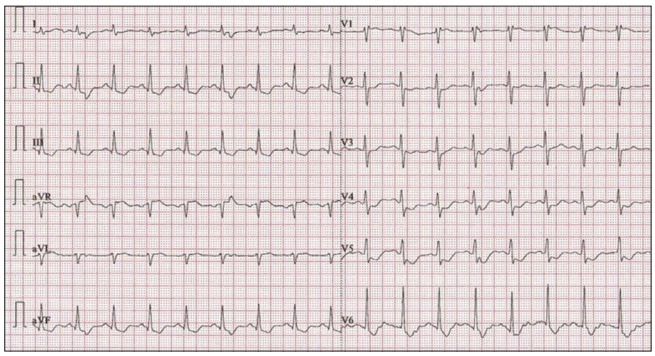

Figure 1: Electrocardiogram on presentation showing ST-T changes

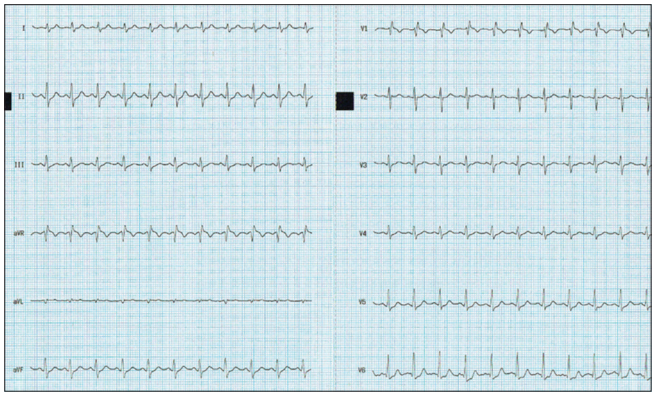

Figure 2: Electrocardiogram at the time of discharge with resolution of ST-T changes

DISCUSSION

CCBs can be divided into two major categories: Dihydropyridines and nondihydropyridines. Dihydropyridines (e.g. Amlodipine) act preferentially on vascular smooth muscles and leads to relaxation. These drugs have little myocardial depressant activity at therapeutic levels and in fact, may increase cardiac output due to the reflex tachycardia. As dihydropyridines are smooth muscle selective, not smooth muscle-specific, in toxic concentrations they may lead to myocardial depression and impaired cardiac conduction. In addition, at high doses, they can block sodium channels and can cause QRS prolongation, similar to tricyclic antidepressants [3]. Intoxication with amlodipine results in nonspecific symptoms such as headache, nausea, vomiting, constipation, abdominal pain, dizziness, confusion and edema, and other non-specific symptoms.

The management of CCB overdose includes gastrointestinal decontamination with activated charcoal and total gut lavage with polyethylene glycol along with IV fluids, vasopressors; glucagon and/or calcium infusions [4]. Specific antidote is calcium gluconate or chloride. Other measures which can be tried include hyperinsulinemia-euglycemia therapy [5]. Some authors reported the use of vasopressin analogues in cases of hypotension due to sustained released calcium antagonists. In fact, using Terlipressin, hemodynamic stabilization could be achieved and the patient survived [6]. Hyperinsulinemic euglycemia in CCBs overdose was beneficial in several case reports. This approach was usually started after calcium gluconate and vasoactive drugs failure. Insulin acts by increasing plasma levels of ionized calcium, improving the hyperglycemic acidotic state, myocardial utilization of carbohydrates, and exerting its own independent inotropic effect [7]. However, we have not used hyperinsulinemic euglycemia as our patient presented with hypoglycemia and severe hypokalemia, which could have been worsened and may have detrimental effects. Although in patients with shock refractory to these therapies, anecdotal therapies have been tried with some success. These include continuous venovenous hemodiafiltration without charcoal hemoperfusion [8], IV lipid emulsion [9], levosimendan [10], methylene blue [11], intra-aortic balloon pump [12], and extracorporeal life support [13,14].

CONCLUSION

Early recognition, decontamination, IV fluids, calcium infusion, electrolytes correction, supportive measures such as inotropes and ventilation can successfully treat amlodipine intoxication. While in case of refractory shock, other new therapies can be considered.

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