Intravenous calcium therapy in calcium channel blocker poisoning – A double-edged sword

Dear Editor,

Co-ingestion of beta-blocker and calcium channel blockers (CCBs) as a case of poisoning poses a therapeutic challenge for the treating intensivist. Negative chronotropic effects, reduced cardiac output, and peripheral vasodilation may severely compromise the end organ perfusion in these patients. The initial modalities for management include decontamination and supportive treatment.[1] In this letter, we would like to stress the adverse effect of intravenous calcium therapy.

Our patient in late 20s was admitted with consumption of 28 tablets of atenolol 50 mg each (total 1400 mg) and 50 tablets of amlodipine 10 mg each (total 500 mg). Patient was initially managed with intravenous (IV) fluids, vasoactive agents, glucagon, high dose insulin euglycemic therapy, transvenous pacemaker, and intravenous calcium therapy. Initially, the patient was considered for Veno arterial extra corporeal membrane oxygenation (ECMO) on day 1 of illness. As atenolol is a dialyzable agent, hemodialysis was done as the initial strategy of management. Once the hemodialysis was done, it was seen that the contractility of heart had improved, which was poor in the beginning. Due to improvement in contractility after hemodialysis, the plan of ECMO was deferred. Intravenous calcium was given as an infusion to target higher ionized calcium levels (around 2 mmol/L), where the maximum calcium level recorded was 2.6 mmol/L. Continuous electrocardiogram (ECG) monitoring did not show any arrhythmias due to higher calcium levels, although 12-lead ECG monitoring showed a lower corrected QT interval.

Patient had persistent fever spikes and increase in total leucocyte count after initial improvement in hemodynamics. Cultures were sterile. Patient was evaluated for non-infectious aetiology of fever, and we noted that our patient had developed pancreatitis as evident by raise in biomarkers and imaging findings. Other aetiologies for pancreatitis were ruled out.

Hypercalcemia is a rare cause of pancreatitis. The role of calcium in the treatment of calcium channel blocker poisoning is controversial. Intravenous calcium acts by promoting calcium influx through unblocked L-type calcium channel and thereby improving the tone of vascular smooth muscles.[2] There are no definite randomized controlled trials (RCTs) indicating its importance, but there are many case reports indicating its role in CCB poisoning.[3,4] But these reports are often about non-dihydropyridines such as verapamil. The dose–response relationship is not linear, and human studies are not definitive. The definitive end point of calcium infusion is not clear. It could be titrated to clinical response in terms of heart rate and blood pressure or till the serum value is double that of normal.[5] There are few reports of intravenous calcium-induced calciphylaxis, pancreatitis, acute tubular necrosis of kidney, hepatic necrosis, and splenic infarcts.[2] In one such study of hypercalcemia causing pancreatitis, the maximum calcium level associated was found to be 11.0 ± 0.6 mg/dL. The serum ionized calcium level in our patient was 2.6 mmol/L. The pathogenesis of calcium-induced pancreatitis is poorly understood and is often attributed to acinar cell activation.

In conclusion, intravenous calcium is relatively safe with occasional complications as mentioned above, and therefore, it is to be used with caution. Serum ionized calcium level has to be monitored and has to be kept not more than twice the upper limit of normal level.

Author contribution

Rajathadri Hosur Ravikumar, Balaji Rajaram Shashikant Sharma, and Puneet Khanna were involved in the management of the case. Rajathadri Hosur Ravikumar and Balaji Rajaram prepared the manuscript. Puneet Khanna and Shashikant Sharma were involved in editing and proof reading of the manuscript. All the authors reviewed the manuscript.

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Conflicts of interest

There are no conflicts of interest.

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