Blood
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Selected Abstracts

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Adsorption therapy in patient with calcium chanel blocker poisoning

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We present a 22-year-old patient on chronic hemodialysis, who was admitted to the emergency room 24 hours after his last HD treatment, with severe hypotension 60/40, sinus bradycardia, stupor, no response to vasopressors, and required mechanical ventilation. He had moderate tricuspid insufficiency history. For this reason, an echocardiogram was performed with LVEF 37%, moderate tricuspid insufficiency. Persistence with MAP 55mmHg despite norepinephrine and vasopressin at maximum doses. Admission laboratories were BUN 75 mg/dl, Creatinine 14 mg/dl, Hb 11.5 g/dl, Na 135 mEq/L, K 5.6 mEq/L, Cl 110 mEq/L, CRP<6 mg/L, procalcitonin <0.5 ng /mL.
Later, we found out that he voluntarily ingested 50 immediate-release 5 mg amlodipine pills, approximately 6 hours prior to admission. Calcium gluconate was initiated and insulin + glucose infusion for 8 hours without MAP improving. CRRT was started, as there was no MARS in our center, so we performed single pass albumin dialysis technique (SPAD) without any improvement, and the same dose of vasopressor was continued.
Approximately 14 hours after admission we get a Cytosorb filter and was started. After 2 hours of Cytosorb treatment the dose of amines was reduced to 50%, and at 12 hours later they were completely withdrawn and removed from mechanical ventilation.
Unfortunately, we didn't find a laboratory in Mexico to measure amlodipine levels. Currently patient is in good shape and attends his chronic hemodialysis therapy on his own.

Literature review: Amlodipine is a drug with high affinity for proteins and a large volume of distribution, therefore, its clearance from hemodialysis is negligible (<5mL/min) regardless of the dialyzer used (1).
First-line therapy of symptomatic calcium chanel blockers (CCB) poisoned patients, it is recommended the use of iv calcium, high-dose insulin therapy, vasopresors in the presence of shock, with preferential use of norepinephrine in the presence of vasodilatory shock. (2)
There are in vitro reports of the use of hemodiafiltration membranes and molecular adsorbent recirculating system (MARS) in CCB poisoned patients. (3)
Various indications have been described for the use of adsorption membranes, such as sepsis, cardiac surgery, heart transplantation and drug removal. (4) We only found evidence of a single case report with Cytosrob in CCB poisoning (5).

Conclusion: In the case we present supports this positive evidence with Cytosorb and adds information about the null response with in vivo techniques as SPAD and hemodiafiltration.

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