Note of the editors: This is a BOWMAN-generated English translation of the officially indexed Spanish-language article, which should be cited as Emergencias 2026;38:318-320. In this translated version, the editors have supervised the process; however, it cannot be ruled out that some errors resulting from the artificial intelligence translation process may have gone unnoticed.

LETTERS TO THE EDITOR

Euglycemic hyperinsulinemic therapy in shock caused by beta-blockers and calcium channel blockers: Is less more

Terapia hiperinsulinémica euglucémica en el shock *por betabloqueantes y calcioantagonistas: ¿menos es más?*

To the Editor,

Acute poisoning with beta-blockers and calcium channel blockers is uncommon but severe and potentially fatal, accounting for up to 65% of deaths due to cardiovascular drugs.[1-3] Therapeutic options include glucagon, vasoactive drugs, and high-dose insulin euglycemic therapy (HIET), which has been proposed in recent years and consists of administering high doses of insulin, with an initial bolus of 1 unit/kg (maximum, 10 units) followed by an infusion of 0.5-1 units/kg/h, which can be increased up to 10 units/kg/h.[3]

We present the case of a 60-year-old man transferred to the emergency department with suspected intentional drug overdose. His medical history included hypertension, dyslipidemia, obesity, chronic ischemic heart disease treated with revascularization and preserved left ventricular ejection fraction, peripheral vascular disease, and recurrent major depressive disorder with a previous suicide attempt. His usual medication included carvedilol and nitrendipine.

On arrival, he was obtunded, with poor peripheral perfusion, blood pressure of 47/36 mmHg, and a heart rate of 40 bpm, with a junctional rhythm on electrocardiography. Glucagon 3 mg and atropine 1 mg were administered without a significant clinical or hemodynamic response. Because of hemodynamic instability and impaired consciousness, orotracheal intubation was performed and a norepinephrine infusion was initiated. Laboratory testing showed acute renal failure (estimated glomerular filtration rate, 30 mL/min/1.73 m^2), potassium 5.42 mmol/L, and hyperglycemia of 223 mg/dL. Cranial computed tomography showed no acute findings, whereas chest computed tomography suggested pulmonary consolidation, probably secondary to aspiration. Bedside echocardiography showed preserved ventricular function without valvular disease. The patient recovered sinus rhythm at 50 bpm, with no subsequent arrhythmias. Magnesium (12.2 mEq as a bolus) and calcium gluconate (2 boluses of 4.6 mEq) were administered, together with restrictive fluid resuscitation (500 mL of balanced crystalloid followed by 1 L of normal saline every 24 hours) and empirical intravenous amoxicillin/clavulanate (2000/125 mg every 8 hours).

Because of persistent refractory shock, norepinephrine was continued at doses of up to 1 µg/kg/min, an additional 5 mg of glucagon was administered, dopamine was added, and HIET was initiated with an intravenous bolus of 0.5 units/kg followed by an infusion of 0.1 units/kg/h, both below the recommended doses. A minimum dose of argipressin was also added. To maintain blood glucose within the normal range, 30% glucose solution was required at rates of up to 63 mL/h, with close potassium monitoring. Intravenous replacement with 40 mEq of potassium chloride was required, administered at a maximum rate of 10 mEq/h. After initiation of HIET, vasopressors could be progressively withdrawn. The insulin infusion was discontinued after 24 hours. A transient hemodynamic deterioration required reintroduction of low-dose norepinephrine and dopamine. Thereafter, the clinical course was favorable, with extubation at 48 hours and complete withdrawal of vasopressors at 60 hours.

This case shows that HIET, even at doses lower than those recommended, may be effective in refractory shock secondary to beta-blocker or calcium channel blocker poisoning. The high glucose requirements observed underscore the need for close metabolic monitoring. To our knowledge, this is the first case reported in Spain documenting its efficacy. This case raises the question of whether doses as high as those currently proposed for HIET are necessary to achieve clinical benefit, while supporting its early and protocolized consideration in initial treatment algorithms.

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