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Case Report

Lercanidipine Intoxication in A 16-Year-Old Adolescent

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ABSTRACT

Lercanidipine is a 1,4-dihydropyridine calcium channel blocker. For severe toxicity, treatment modalities are based on a restricted range of evidence and clinical experience. This case report describes our clinical experience regarding the treatment of Lercanidipine overdose in a 16-year-old girl. To the best of our knowledge, this is the first reported pediatric case overdose of lercanidipine.

Keywords: Lercanidipine, intoxication, child, treatment

INTRODUCTION

Calcium channel blockers (CCBs) are medications that inhibit the L-type calcium channels involved in myocardial and vascular smooth muscle contractility.[1] They are divided into two groups, dihydropyridine, and non-dihydropyridine, depending on their physiological effects.

Lercanidipine is a 1,4-dihydropyridine CCB.[2] Its selectivity for vascular smooth muscle is more significant than for cardiac smooth muscle.[2] It does not induce sympathetic activation or reflex tachycardia at therapeutic doses and exhibits no negative inotropic effects.[3] However, overdose leads to arterial vasodilation and reflex tachycardia. Additionally, peripheral selectivity is eliminated at severe toxic doses and can affect the myocardium, resulting in arrhythmias, bradycardia, and negative inotropy.[1,4] The time required for an overdose of Lercanidipine to achieve peak effects to wear off is more significant than that for other CCBs.[2]

Data concerning severe toxicity treatment are primarily derived from case series and animal studies. Treatment is therefore based on a restricted range of evidence and clinical experience.

This case report is presented to describe our clinical experience regarding treating Lercanidipine overdose in a 16-year-old girl. To the best of our knowledge, this is the first pediatric case report of an overdose involving the dihydropyridine CCB Lercanidipine.

CASE REPORT

A 16-year-old girl was brought to the pediatric emergency department due to vertigo approximately 2 h after ingestion of the suicide of 30-Lercadip 10 mg tablets used by her mother. At presentation, her Glasgow coma scale was 15, respiratory rate 16/min, heart rate 136/min, blood pressure 80/44 mmHg, capillary filling time <3 s, and SpO_2 100%.

Initial blood gas, fingertip glucose, and laboratory parameters were regular. Sinus tachycardia was present at the electrocardiogram (ECG). Her body weight was 50 kg (-1.35 SDS).

Activated charcoal (1 g/kg) was administered a nasogastric tube two hours after ingestion.

Because of the presence of hypotension and tachycardia at presentation, 0.9% sodium chloride was administered twice at 20 mL/kg. Despite fluid administration for 15 min, her blood pressure remained low (60/30 mmHg), and 10 mL 10% calcium gluconate was administered by intravenous (IV) infusion over 15 min. However, the hypotension persisted, and norepinephrine

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was issued at a starting dose of 0.1 mcg/kg/min and titrated to 0.3 mcg/kg/min. The patient was transferred to the pediatric intensive care unit (PICU).

Following central venous catheter placement in the PICU, insulin therapy at a dose of 0.5 IU/kg/h (hyperinsulinemic euglycemia), glucose infusion (100 mL/h of 10% dextrose), and potassium for preventing hypokalemia were initiated. One hour after hyperinsulinemic-euglycemic therapy (HIET), low ionized calcium levels were reported, and calcium gluconate infusion was started at 0.5 mL/kg/h and raised to 0,7 mL/kg/h. Epinephrine infusion at 0.1 mcg/kg/min was added to the treatment due to the persistence of hypotension. The vasoactive inotrope score was 28.[5] Serial EKGs were taken with serum glucose, calcium, and potassium measurements once every 2 h to prevent clinically significant hypoglycemia, hypercalcemia, and hypokalemia. These serial ECGs were normal apart from sinus tachycardia. No cardiac conduction disorders were observed. The calcium gluconate infusion was stopped on the 4th h due to the development of hypercalcemia. Adrenaline was administered for 20 h, and noradrenaline for 22 h. HIET was maintained for a further 7 h after discontinuation of inotrope therapy and was then tapered down and stopped. Treatments administered during hospitalization plotted with hemodynamic parameters are shown in Figure 1.

The patient was transferred to the pediatric ward 36 h after the presentation. She was finally discharged three days after admission following a psychiatric evaluation.

Informed consent was obtained from the parents of the patient.

DISCUSSION

CCB-related intoxication is associated with significant morbidity and mortality. Therefore, prompt treatment, plays a vital role in patient outcomes. The characteristic features of such intoxication include hypotension, bradycardia, and cardiogenic shock.

The management of CCB overdoses may involve administrating IV fluids, activated charcoal, IV calcium and/or or IV glucagon, IV high-dose insulin, IV lipid emulsion therapy, and IV vasopressor support. However, no set therapeutic algorithm exists for.[6]

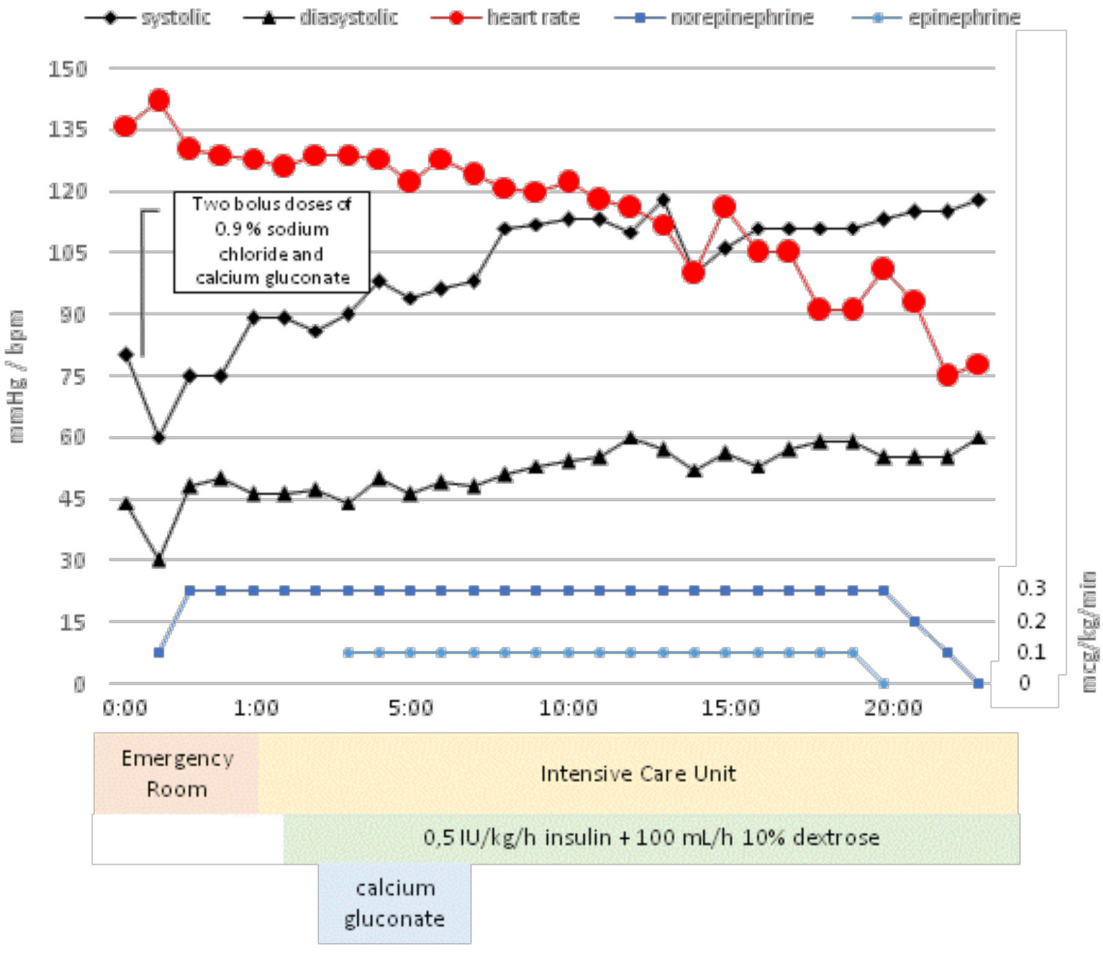

Figure 1. Treatments administered during hospitalization plotted with hemodynamic parameters

In this case, various treatments were required to establish tension stability. However, none of the therapeutic options appear superior to the others. The treatments applied are therefore discussed on the basis of the existing literature.

Activated charcoal[7] was administered since presentation occurred within the first 2 h after drug ingestion. IV fluids represent the initial treatment of hypotension. Our patient received a crystalloid fluid bolus, as recommended, for initial treatment. Since the crystalloid was ineffective, calcium gluconate was initiated to overcome the cardiovascular effects. However, hypotension persisted after calcium administration. Although there have been reports of hemodynamics improving the following glucagon administration[8], this was not applied in this case because glucagon's usefulness in overdose CCBs is doubtful and bradycardia was not observed.

Because of its powerful vasoconstrictive (alpha-adrenergic) effect, norepinephrine was started as the initial vasopressor. Infusion started at 0.1 mcg/kg/min but was titrated to 0.3 mcg/kg/min within 10 min to achieve a mean arterial pressure of 65 mmHg. High-dose insulin therapy was initiated due to the persistence of hypotension (mean arterial pressure 65 mmHg). This therapy exhibits positive inotropic effects in patients with CCB toxicity. Although the mechanism of HIET is not fully understood, its effectiveness and safety have been described in animal models, and case reports.[9-12] HIET was first described in humans in 1999 and improves circulatory shock in four instances of CCB overdose patients when applied as a combination of calcium, glucagon, and epinephrine therapy.[13] Another case study reported rapid hemodynamic stabilization with HIET.[14]

IV calcium gluconate infusion was initiated because of a low ionized calcium value and was maintained until clinically significant treatment-related hypercalcemia was observed. Adrenaline at 0.1 mcg/kg/min was created after 30 min for circulation support.

The options of methylene blue,[15,16] lipid emulsion,[17] and continuous venovenous hemodiafiltration and concomitant charcoal hemoperfusion[18] are available for treating hypotension secondary to CCB overdose refractory to multiple vasopressors and HIETs.

However, these were not employed in this case since the patient tension gradually increased, and her hemodynamic status was stable.

CONCLUSION

This is the first pediatric case of hypotension and reflex tachycardia developing following Lercanidipine overdose and recovery after a series of recommended early treatments.

Although Lercanidipine is safe at therapeutic doses, overdose can result in differing and even fatal effects. We, therefore, believe that early administration will be helpful in the knowledge of the drug's effect mechanism and all the therapeutic options.

Ethics

Informed Consent: Informed consent was obtained from the parents of the patient.

Peer-reviewed: Externally peer-reviewed.

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