CORRESPONDENCE

Intravenous lipid-emulsion therapy in a patient with cardiac arrest after overdose of diphenhydramine

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Dear Editor,

Diphenhydramine overdose is characterized mostly by sedative and anticholinergic effects. Cardiotoxicity and circulatory collapse after massive ingestion of diphenhydramine have rarely been reported.1 Positive effects of intravenous lipid-emulsion (ILE) therapy for diphenhydramine overdose were reported in an animal study,2 although its role in humans remains uncertain.

A 33-year-old healthy male weighing 70 kg attempted suicide by witnessed ingestion of 4900 mg of diphenhydramine. He was found unconscious, with urination and generalized tonic-clonic seizure 30 minutes post-ingestion. The vital signs at the scene were blood pressure of 111/67 mmHg and a heart rate of 151 bpm. He arrived at the emergency department 48 minutes after ingestion, with cardiac arrest and a rhythm of asystole. Sodium bicarbonate of 128 mEq bolus and 1 mg of epinephrine every 3 minutes were given intravenously while cardiac compressions were continued. The patient regained a pulse 13 minutes later, with a blood pressure of 75/28 mmHg and a heart rate of 51 bpm.

A 12-lead electrocardiogram (ECG) revealed a sinus rhythm with marked intraventricular conduction delay (QRS interval: 224 ms). An echocardiogram showed global hypokinesia of the heart. Gastric lavage, administration of activated charcoal, and whole-bowel irrigation were performed. The patient was still hypotensive with persistent intraventricular conduction delay on the ECG monitor, despite treatment with sodium bicarbonate and inotropic agents (Figure 1). We prescribed a bolus of 110 mL of 20% ILE, followed by continuous infusion at a rate of 10 mL/min for 1 hour. Approximately 20 minutes after the bolus, the QRS width narrowed to 86 ms. His blood pressure improved within 1 hour, while inotropic agents were tapering off. Unfortunately, the patient was deeply comatose and ultimately died from hypoxic-ischemic encephalopathy. A study revealed that coma and seizures were significantly more frequent in patients taking >1.5 g as compared with those ingesting 1.0 g to 1.5 g.3 We believe that the dose-dependent toxicity and the cardiac arrest were the two major factors resulting in the patient’s death.

Tachycardia is common in diphenhydramine overdose, because of its anticholinergic effects. Bradycardia

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occurring in combination with a wide QRS is a sign of severe poisoning, indicating that the sodium-channel blockage is so profound that tachycardia does not occur. Our patient experienced resolution of wide QRS complexes, bradycardia, and hypotension within 1 hour after administration of ILE. The subsequent sinus tachycardia might represent the anticholinergic effect of diphenhydramine, while the effect of sodium channel blockade was suppressed by the ILE. Therefore, the positive effects of ILE cannot be overlooked in severe diphenhydramine poisoning, despite the absence of human studies.

In conclusion, we reported a novel application of ILE therapy in a patient with severe diphenhydramine poisoning who had a poor response to sodium bicarbonate. The administration of ILE resulted in apparent improvement in hypotension and QRS width. Its use may have a profound impact on hemodynamics if started earlier in life-saving circumstances.

References