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LETTER TO THE EDITOR

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To the Editor:

The use of intravenous fat emulsion (IFE) has been well described in a systematic review of human and animal studies. Cases of successful early use of lipid emulsion for cardiotoxic effects induced by local anesthetics or other lipophilic drugs, such as calcium-channel antagonists or beta-blockers, have recently been published.^{2,3} We report the first case of IFE therapy for the treatment of a voluntary flecainide poisoning with refractory shock.

A 72-year-old French female patient, whose main medical history included a rhythmic heart disease treated with flecainide and a depressive syndrome with multiple suicide attempts, was rescued at home by a first-aid team after voluntarily ingesting drugs. At the scene, an empty plate of 15 tablets of flecainide (100 mg), a plate of 15 tablets of oxazepam (10 mg), and a plate of 30 tablets of levothyroxin (50 μg) were found; the time of ingestion was unknown. The patient was taken to the intensive care unit of the nearest hospital. Clinical examination revealed a drowsy patient, with an enolic breath and without coma. Respiratory rate was 30 breaths per minute, oxygen saturation was 88% without oxygen supply, blood pressure was 70/50 mmHg, and heart rate was 55 beats per minute. An electrocardiogram revealed a widening of QRS complexes longer than 0.2 sec with a prolongation of the QT interval. However, despite being treated with aggressive supportive care, including fluid resuscitation (750 mL of 4.2% sodium bicarbonate with hydroxyethyl starch 1500 mL), mechanical ventilation, and an increasing dose of vasopressors (up to 6 mg/h of epinephrine), it was decided to transfer her to the university reference hospital for the possible implementation of circulatory assistance.

After the failure of conventional pharmacological therapy and regarding the similarities of flecainide with molecules of the class of local anesthetics from the electrophysiological point of view,4 an infusion of Intralipid® 20% in the form of a bolus of 1.5 mL/kg associated with a continuous infusion of 0.25 mL/min was started. The patient was then transferred with a medical team (emergency physician, nurse, and ambulance driver) to the university hospital. During the following 30 min, hemodynamic stabilization allowed a dramatic decrease in the dose of epinephrine (1 mg/h during the transfer) without any further complication. On arrival at the reference hospital, the patient's blood pressure was 111/80 mmHg and her pulse rate was 75 beats per minute. An ECG showed a marked shortening of the QRS complexes. The evolution was a progressive hemodynamic improvement without implementation of circulatory assistance. After 20 days of hospitalization in ICU because of aspiration pneumonia, the patient was weaned from oxygen therapy without after effects. She was secondarily hospitalized in a psychiatric unit for the management of her major depressive disorder.

According with De Roock, 5 who reminds us that lipid emulsion therapy does not entirely fulfill the criteria for antidotal therapy, this case is a reminder that lipid emulsions should be considered as a second-line treatment for poisonings with cardiotropic drugs after conventional treatment and can even be used during medical transfer between two hospitals.

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