Rapidly fatal poisoning with an insecticide containing rotenone

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Introduction

Rotenone is a botanical pesticide derived from extracts of Derris roots, which is traditionally used as piscicide, but also as an industrial insecticide for home gardens. Its mechanism of action is potent inhibition of mitochondrial respiratory chain by uncoupling oxidative phosphorylation by blocking electron transport at complex I. Although its classification as mild to moderately toxic to humans (estimated oral lethal dose 300-500 mg/kg), there is a striking variety of acute toxicity of rotenone depending on the formulation (solvents). Human fatalities with rotenone-containing insecticides have been rarely reported in the literature (1,2), and a rapid deterioration within a few hours of the ingestion has been described previously in one case (1).

Case report

A 49-year-old Tamil man with a history of asthma, ingested 250 mL of an insecticide containing 1.24% of rotenone (3.125 g, 52.1-62.5 mg/kg) in a suicide attempt at home. The product was not labeled as toxic.

One hour later he vomited repeatedly and emergency services were alerted. He was found unconscious with irregular respiration and was intubated. On arrival at the emergency department, he was comatose (GCS 3) with fixed and dilated pupils, and absent corneal reflexes. Physical examination revealed hemodynamic instability with hypotension (55/30 mmHg) and bradycardia (52 bpm). Significant laboratory findings were lactic acidosis (pH 6.97, lactate 17 mmol/L) and hypokalemia (2 mmol/L). Cranial-CT showed early cerebral edema.

A single dose of activated charcoal was given. Intravenous hydration, ephedrine, repeated boli of dobutamine, and a perfusor with 90 µg/h norepinephrine stabilized blood pressure temporarily. Atropine had a minimal effect on heart rate (58 bpm). Intravenous lipid emulsion was considered with 90 mmol/L of norepinephrine and hypokalemia (2 mmol/L). Cranial-CT showed early cerebral edema.

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Quantitative analysis of serum performed by high resolution/accurate mass-mass spectrometry (GC-MS) and liquid chromatography–mass spectrometry (LC-MS/MS).

Discussion

In our case the rapidly fatal outcome is striking. A similar course has been described in the literature where a 3.5-years-old girl deteriorated rapidly and died 6 h after ingestion of rotenone (1). In contrast, a second case report with fatal outcome describes a 47-years-old woman who died only 3 days after ingestion (2). In all three fatal cases symptoms were similar with vomiting, coma, hemodynamic instability, and respiratory distress. Serum or blood concentrations in the three cases show great variability (Tab 2). It is difficult to compare them, because information on sampling time is lacking and the levels were measured in serum as well as in blood.

Conclusion

The clinical course was characterized by early severe symptoms and a rapidly fatal evolution, compatible with inhibition of mitochondrial energy supply. Although rotenone is classified as mild to moderately toxic, physicians must be aware that suicidal ingestion of emulsified concentrates may be rapidly fatal.