Kerosene Poisoning - Varied Systemic Manifestations


Abstract

We report here unusual clinical manifestations in a case of kerosene poisoning. The patient presented with encephalopathy and in the course of stay in the hospital developed renal tubular acidosis, delayed first-degree burns and myocarditis. With supportive therapy the patient recovered completely and was discharged without any sequelae.

INTRODUCTION

Kerosene is a refined oil belonging to the hydrocarbon group of compounds, obtained from crude petroleum. Poisoning is usually due to inhalation of fumes or ingestion of small amounts accidentally or with suicidal intent. The usually encountered manifestations are due to the local caustic effects of kerosene. We report here a case of kerosene poisoning presenting with multiple systemic manifestations.

CASE REPORT

A 22 years female presented to us in the emergency ward with alleged history of consumption of a half bottle of kerosene followed by forceful administration of salt water by relatives to induce vomiting. On examination, she was unconscious, responding only to deep painful stimuli. She was normotensive (BP of 116/76) and had tachycardia (pulse rate 120/min) and tachypnoea (RR 36/min). There was no evidence of any fasciculations. Pupils were bilaterally equal and reacting to light. She had crackles in the left infraclavicular area. Blood gas analysis on day 1 revealed an oxygen saturation of 74% suggesting hypoxia. and her renal function tests, liver function tests and hemogram were normal. Her X-ray chest showed left upper zone pneumonitis. ECG was suggestive of sinus tachycardia.

In view of her pinpoint pupils and hypoxia, she was treated with pralidoxime and atropine in appropriate dosages, keeping in mind that organophosphorus compound contamination with kerosene, which is well known.4 The patient also received oxygen by mask. Invasive ventilation was not required and a few hours after admission, the patient regained consciousness. On day 2, an arterial blood gas analysis revealed metabolic acidosis with a normal anionic gap. Urine pH was 5.4. Serum electrolytes revealed a newly developed hypokalemia (K+ = 2.9 meq/l). This suggested a type II renal tubular acidosis.

On day 3, we noticed superficial excoriations with hyper-pigmentation over her buttocks and thighs, which were diagnosed to be first degree burns. In spite of omission of atropine, the patient was found to have persistent sinus tachycardia with a pulse rate >180/min. A 2D echo was done which showed global hypokinesia suggestive of myocarditis with a left ventricular ejection fraction of 45% and with minimal pericardial effusion likely to be of toxic etiology.

In due course of time, with antibiotics, daily silver sulfadiazine dressings and other supportive measures, the patient recovered completely. A repeat 2-D echocardiogram, three weeks later, revealed normalization of ventricular function. Repeated serial blood gas analysis over a period of time showed correction of acidosis and hypokalemia.

DISCUSSION

Kerosene poisoning is a commonly encountered emergency in general medical practice. The lethal dose of kerosene for a 70kg adult is 100 ml.

It causes a variety of systemic manifestations by a number of mechanisms. These can be enumerated as follows:

1. Hydrocarbons are lipophilic in nature, hence they dissolve the lipids in the stratum corneum making the skin more vulnerable to drying, thereby causing skin lesions varying from bullae, blisters maculopapular rash and first degree burns. As in our patient, these manifestations may not be evident on admission and have to be looked for, on routine examination.

2. Neural tissue, which is rich in myelin, a lipid component is acted upon by kerosene causing central nervous system depression and ventilatory drive suppression.

3. The myocardium is sensitized to endogenous catecholamines producing dysrhythmias. Myocardial
function may also be depressed resulting in a poor left ventricular ejection fraction.

(4) Hydrocarbons are excreted by the lungs. They initially replace the alveolar gas and may cause severe hypoxia resulting in cyanosis. They are also easily aspirated and as they spread to the lower levels of the respiratory tree, bronchospasam and chemical pneumonitis may develop.

(5) Renal damage may result in type 2 renal tubular acidosis, as was seen in our patient.

(6) Autonomic dysfunction like hypotension, excessive sweating may also occur.

(7) Chronic exposure to kerosene is known to cause polyneuritis, liver damage, bone marrow depression, acne and skin eruptions.

We report this case to bring to light, the unusual manifestations of a common clinical problem. This 22 years female with ingestion of kerosene, developed an encephalopathy, delayed first-degree burns, myocarditis and type 2 renal tubular acidosis during the course of her stay in the hospital. We would like to suggest scrupulous observation and intensive investigation in any patient of kerosene ingestion to reduce the morbidity and potential mortality due to these rare manifestations.

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REFERENCES

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