

Neem oil poisoning: Case report of an adult with toxic encephalopathy

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Abstract

Neem oil has widespread use in Indian subcontinent due to its many bioactive properties. Azadirachtin, an active ingredient, is implicated in causing the effects seen in neem oil poisoning. Neem oil poisoning is rare in adults. This report highlights the toxicity associated with neem oil poisoning in an elderly male. The patient presented with vomiting, seizures, metabolic acidosis, and toxic encephalopathy. The patient recovered completely with symptomatic treatment.

Keywords: Neem oil, poisoning, toxic encephalopathy

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Introduction

Neem oil is a vegetable oil obtained from the seed kernels of Neem tree (*Azadirachta indica*), an evergreen of the tropics and sub-tropics. It is deep yellow in color and has garlic-like odor. It contains active ingredients like azadirachtin, nimbin, picrin, and sialin. Azadirachtin, a complex tetranortriterpenoid, is implicated in causing the effects seen in neem oil poisoning.^[1] In children, neem oil is reported to cause toxic encephalopathy and Reye's-like syndrome.^[2] We report a case of neem oil poisoning in a 73-year-old male who presented with vomiting, seizures, metabolic acidosis, and toxic encephalopathy.

Case Report

A 73-year-old man with the medical history of diabetes mellitus type 2 and psoriasis presented to the emergency department with complaints of vomiting and drowsiness, 1 hour after accidental ingestion of 20 ml of Neem oil. The patient's current medication included human Mixtard insulin (30/70), 16 U before breakfast and 10 U before dinner.

At presentation, the patient was drowsy with Glasgow coma scale of 13/15. His pulse rate was 110/min, respiratory rate 28/min, oxygen saturation of 95% at room air, and blood pressure 150/90. Physical examination was unremarkable. Systemic examination showed: bilateral equal and reacting pupils with no meningeal signs and no focal neurological deficits. Respiratory, Cardiovascular and abdominal examination were unremarkable.

In the emergency department, the patient developed generalized convulsions with loss of consciousness. He was intubated and managed initially with intravenous lorazepam. In the intensive care unit, he was managed with insulin and symptomatic treatment.

Investigations at presentation showed: Hemoglobin 13.3 gm/dl, leukocyte count 16800/ μ l, red blood cell count 4.41 million/cmm, platelet count 375000/ μ l, blood sugar 298 mg/dl, serum osmolality 277 mosmol/kg, and normal serum lactate level. An arterial blood gas analysis showed metabolic acidosis: pH 7.34, pO₂ 81, pCO₂ 24, HCO₃ 16, and pO₂/FiO₂ 385. He had normal urine analysis, serum electrolytes, liver, renal, and thyroid function. MRI scans of brain showed chronic ischemic changes due to small vessel disease. ECG and radiograph of the chest were normal.

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Investigations repeated on day 2, 3, and on discharge showed: Normal electrolytes, arterial blood gas, liver, and renal function. During the course of illness, he remained drowsy for 4 days and recovered without any complications. He was discharged after 1 week.

Discussion

Neem oil, as a traditional medical remedy, is used as anti-bacterial, anti-fungal, insect repellent, and treatment of skin diseases. Traditional routes of administration of Neem extracts included oral, vaginal, and topical use. Neem oil comprises mainly triglycerides, steroids (campesterol, beta-sitosterol, stigmasterol) and many triterpenoids, of which azadirachtin is the most well-known and studied. The azadirachtin content of neem oil varies from 300 ppm to over 2500 ppm, depending on the extraction technology and quality of the neem seeds crushed.

In children, there are several case reports of Neem oil poisoning causing vomiting, hepatic toxicity, metabolic acidosis, and encephalopathy.^[2-4] Lai *et al.*, reported 22 cases of neem oil poisoning in infants, who were given single doses of Neem oil (few drops to 5 ml), presented with features of toxic encephalopathy, metabolic acidosis, and hepatic toxicity. The infants recovered completely with supportive treatment.^[3] Sundaravalli *et al.*, in a case series of 12 children with neem oil poisoning, who were given single dose of Neem oil (25-60 ml), reported fatality in 10 cases with features of toxic encephalopathy and metabolic acidosis.^[4] Sinnaih *et al.*, reported Reyes-like syndrome in fatal cases of Neem oil poisoning in a case series of 13 children.^[2]

In adults, there are few case reports of Neem poisoning. Iyyaduria *et al.*, reported a case of a 35-year-old female with suicidal poisoning, who presented after ingestion of 250 ml of Neem pesticide with encephalopathy and metabolic acidosis with no evidence of hepatic and renal complications. She recovered completely with supportive management.^[1] Bhaskar *et al.*, reported a 35-year-old female with Neem oil poisoning who presented with bilateral visual loss. Cranial MR imaging, showed symmetrical altered signal intensity bilaterally in the putamen region with extension to the posterior limb of the internal capsule. Laboratory findings were within normal limits, and she recovered completely with supportive management.^[5]

There is no specific antidote available, and gastric lavage is not recommended for Neem oil poisoning. The

management is primarily symptomatic.^[6] Our report highlights toxicity related to neem oil poisoning in an elderly male patient presenting with vomiting, seizures, metabolic acidosis, and encephalopathy.

He had no laboratory evidence of hepatic and renal complications. His symptoms resolved in 4 days with symptomatic treatment, and he was discharged after 1 week.

Azadirachtin ($C_{35}H_{44}O_{16}$) manifests its toxicity possibly by interfering with mitochondrial bioenergetics, resulting in inhibition of the generation of the electrochemical proton gradient (primary form of energy generated in mitochondria). Acute poisoning with inhibitors of electron transporting complexes causes symptoms such as muscle weakness, easy fatigability, hypotension, headache, facial flushing, nausea, confusion, and aggravation of latent myocardial angina. The inability to utilize oxygen is manifested as a cytotoxic hypoxia wherein the chemicals cause a metabolic acidosis and hyperpnea, despite normal pO₂. However, inhibitors of the supply of reducing substrates for the respiratory chain cause a similar metabolic syndrome that is difficult to distinguish from inhibitors of the electron transport chain.^[7]

Conclusion

This case highlights the toxicity associated with Neem oil poisoning in an adult. Treatment is primarily symptomatic and recovery is usually complete.

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