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CASE REPORT

Accidental fatal poisoning in a child due to ingestion of *Nerium oleander* leaf

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Abstract

Nerium oleander is an ornamental plant that belongs to the family Apocynaceae. It contains a cardiac glycoside named oleandrin, which is present in all parts of the oleander plant. Suicidal and medication-related deaths due to *Nerium oleander* poisoning are not uncommon. However, accidental deaths due to oleander leaf ingestion are most commonly encountered. We are reporting a case of an accidental ingestion of *Nerium oleander* leaf in a child by mistaking it for a guava leaf. The child presented to the casualty with vomiting, poor sensorium, hypotension, and shock. The child developed hyperkalemia, acute kidney injury, myocardial dysfunction, and bleeding manifestations. The urine output was decreased (< 0.5 ml/kg/h). Later, the child died after 36 h. On autopsy examination, periorbital puffiness and bluish discoloration of the nail beds were present. Petechial hemorrhages were present in the heart, kidney, and mesentery. The stomach mucosa was hemorrhagic. Histopathologically, the lung showed interstitial congestion, the liver showed centrilobular necrosis, and the kidney showed acute tubular necrosis. Toxicology analysis was positive for oleander poisoning. This case highlights the toxic nature of *Nerium oleander* ingestion and the importance of avoiding such plants around residential areas.

Keywords *Nerium oleander* leaves · Accidental · Autopsy · Poisoning · Hyperkalemia · Glycosides

Introduction

The oleander is an evergreen decorative plant belonging to the dogbane family, Apocynaceae. It is solely prevalent in the Mediterranean region and is grown all over the world, especially in mild temperate and subtropical climates [1]. There are commonly two types, *Nerium oleander* Linnaeus (pink oleander) and *Thevetia peruviana* Juss (yellow oleander), both having various cultivars and naturally occurring variants within each species [2]. All parts of the plant, such as leaves, flowers, fruits, branches, and the stem, contain more than 30 different cardiotoxic glycosides (digoxin, ouabain, oleandrin, bufalin, etc.) known as cardenolides [3]. Human exposure to oleander by suicide and intentional intake of natural preparations for medical purposes have both been commonly recorded in South Asian countries. Herbal extracts from its leaf are used to treat a wide range of conditions, including slimming, muscular enhancement, erectile dysfunction, malaria, epilepsy, psoriasis, herpes, dermatitis, thyroiditis, and cancer treatment [4]. Its abundance in the wild and around dwellings as an ornamental plant and its colorful flowers make it highly prone to accidental ingestion by young children (as they tend to play with the leaves and flowers of those plants) and animals. In India, pink oleander is widely grown in gardens, parking areas, and national and state highway road dividers [5]. In this case report, we discuss an accidental consumption of *Nerium oleander* leaf by a child mistaken for guava leaf. This also highlights the hazardous nature of such ornamental plants around the home.

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Case report

A 7-year-old female child and her 5-year-old male sibling were brought to a private hospital with an alleged history of accidental ingestion of *Nerium oleander* leaves by chewing, as both mistakenly consumed it for guava leaves, which is an ideal leaf for making a kind of edible preparation made by guava leaf put with tamarind and salt. Oleander poisoning was suspected before admission as the elder child informed the parents about the incident when they started developing abdominal pain. Both child presented to the casualty of a private hospital with multiple episodes of vomiting, abdominal distension, poor sensorium, and hypotension. Both were resuscitated with fluid boluses, followed by a stomach wash. The 5-year-old sibling died due to acute kidney injury and hepatic encephalopathy after 9 h of ingestion of oleander leaves despite resuscitation measures. Following this, the 7-year-old child was referred to our tertiary health care center for further management. On arrival at our emergency department, the child had poor sensorium and shock and was resuscitated with fluid and started on inotropes. The child developed myocardial dysfunction and acute kidney injury. On further evaluation, the child also had hyperkalemia, bleeding manifestations in the form of pulmonary hemorrhage, gastrointestinal bleeding, and haematuria with decreased urine output (< 0.5 ml/kg/h). Despite efforts, the child's health deteriorated, and she died 36 h after the ingestion.

The body was brought to the mortuary for postmortem examination. The deceased was moderately built, weighed 15 kg, and measured 110 cm in length. On external examination, there were no external injuries; bilateral periorbital puffiness and bluish discoloration of the fingernail beds and lips were present. On internal examination, the laryngeal, tracheal, and esophageal mucosa showed congestion with a purplish hue, and the bilateral pleural and peritoneal cavities contained around 750 ml and 1000 ml of straw-colored fluid, respectively. Petechial hemorrhages were present on all the surfaces of the heart, the anterior surface of the kidney, and the anterior and posterior surfaces of the mesentery, and the stomach mucosa was hemorrhagic (Fig. 1), and all other organs were congested. Histopathological examination of the lung showed interstitial congestion and peri-bronchial chronic inflammatory infiltrate, the liver showed features of centrilobular necrosis (Fig. 2), and the kidney showed acute tubular necrosis (Fig. 3). The toxicology screening was positive for oleander poisoning (Toxicology analysis report No. VPM/TOX.H/1986/2020). The cause of death was attributed to oleander intoxication and its complications.

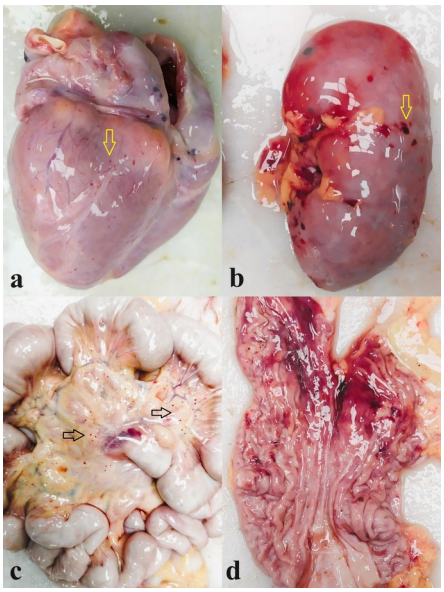

Fig. 1 **a** Petechial hemorrhages present over the anterior surface of the heart (yellow arrow), **b** kidney (yellow arrow), and **c** mesentery (black arrow), and **d** stomach shows hemorrhagic mucosa

Discussion

Nerium oleander plants usually produce white, pink, and red color flowers. Though all types are highly fatal when ingested, the plant that bears red color flowers is more poisonous as it contains a higher concentration of cardiac glycosides compared to the white flower-bearing oleander plant, particularly in the flowering stage [6]. The fatal amount of oleander leaf ingestion is around 5 to 15 leaves in adults, whereas ingestion of even a single oleander leaf can be fatal to children. However, the fatality due to *Nerium oleander* toxicity is connected not only to dosage but to various parameters, including the concentration of toxin in the swallowed part of the plant, the age of the person who consumed the plant, and the individual's health status [7].

The earliest symptoms after *Nerium oleander* ingestion are abdominal pain, nausea, vomiting, and diarrhea. This is followed by neurological symptoms such as headache, delirium, dizziness, lethargy, and mental confusion, as well as cardiac symptoms such as bradycardia with PR and QRS prolongation and the onset of atrioventricular (AV) block, which results in fibrillation, and apart from this, hyperkalemia is prevalent [8]. The cardiac glycosides in oleander plants work by the following mechanism: Inhibition of the Na-K ATPase enzyme leads to an increase in intracellular Na+ concentration and a decrease in K+ concentration. This affects the Na+/Ca++ exchange channels, resulting in an increase in intracellular Ca++ ions and an increase in concentration force (positive inotropic effect). However, inhibiting the Na/K+ ATPase pump also affects intracellular K+ transport, resulting in hyperkalemia. The degree of hyperkalemia corresponds with the severity of toxicity in acute myocardial glycoside poisoning [9].

Cardenolides and other cardiac glycosides may have direct toxic effects on the renal tubules through alteration

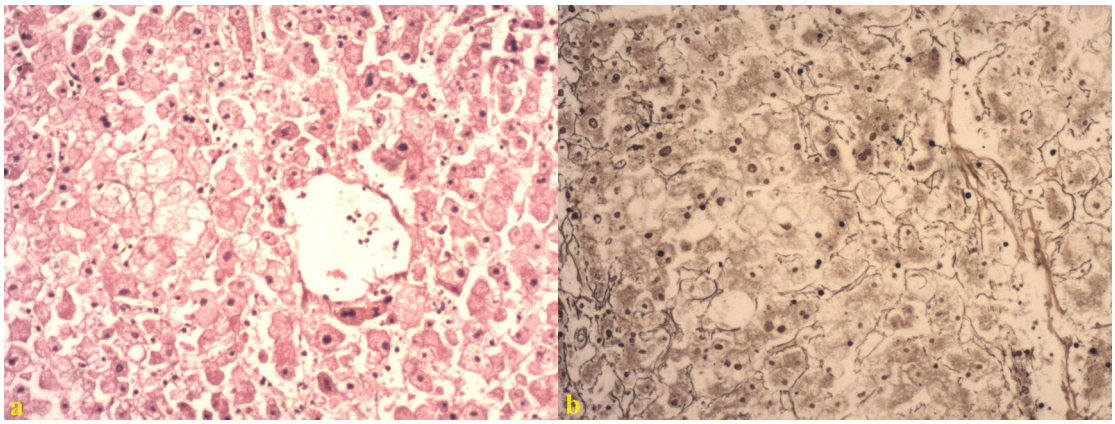

Fig. 2 **a** Low-power image of the liver showing centrilobular necrosis (H&E×10). **b** Low-power image of the liver showing loss of reticulin fibers suggesting centrilobular necrosis (reticulin stain×10)

of Na+/K+ ATPase transporters in the basal membrane of the thick ascending limb of the loop of Henle and the distal convoluted tubule. Alternatively, as the cortex is very susceptible to the effects of ischemia, acute tubular necrosis/injury may be attributable to renal hypoperfusion, as oleander intoxication causes cardiac failure with systemic circulatory impairment. Both mechanisms of renal impairment could occur simultaneously. The severity of renal lesions in the present case may be explained by the need to excrete an increased amount of oleandrin from the kidneys due to an increase in serum oleandrin concentration [10]. The biliary system is principally responsible for the elimination of oleandrin and its metabolites. A similar mechanism of cardiac dysfunction causes decreased blood supply to the liver and so the lack of adequate oxygen supply due to passive congestion, leading to centrilobular necrosis, which occurs predominantly in zone 3 of the hepatic lobule [7].

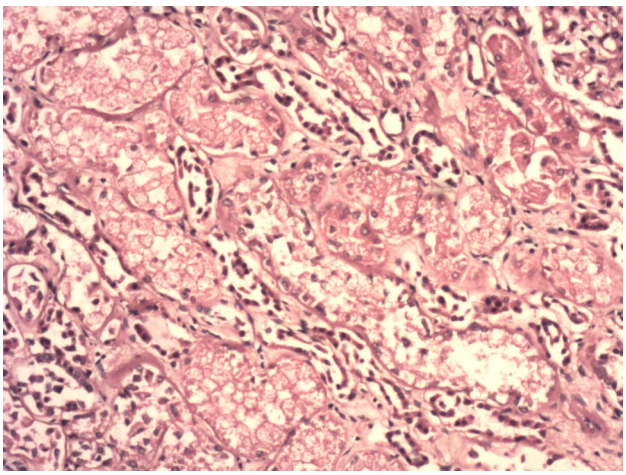

Fig. 3 Low-power image of the kidney showing acute tubular necrosis (H&E×10)

In a case reported by Wasfi et al. in 2008, a 49-year-old male drank an infusion made out of oleander leaves in an attempt to treat his diabetes mellitus, following which he developed heart block, cardiac arrest, ventricular fibrillation, and multiorgan failure and died. Even though the oleandrin extracted from the oleander plant has medicinal value when used as a cardiac tonic, diuretic, and in homeopathy, the chances of accidental deaths are higher [11]. Blum and Rieders reported the death of a young female who underwent weight gain treatment from a person practicing local Haitian medicine. As a treatment measure, she was given oleander leaf extract orally and rectally, following which she developed bloody diarrhea with generalized weakness and succumbed to death within hours [12].

Haynes et al. reported a case of a 30-year-old female who consumed herbal tea made from the extract of oleander leaves as she had mistaken it for eucalyptus leaves, following which she developed nausea, numbness in her tongue, and later ventricular fibrillation. The autopsy revealed pulmonary edema and congestion. The postmortem serum digoxin level was 6.4 ng/m [13]. Similarly, Papi et al. reported a case of two homeless young vegan people found dead in a malnourished state in a desolated forest. Further investigation found that they died due to accidental ingestion of oleander leaves mistaken to be another edible plant. On autopsy examination, both cases showed areas of hemorrhagic erosions in stomach mucosa and hemorrhagic pancreatitis. Histologic investigation showed degenerative changes of myocardial cells and necrosis with hemorrhagic infiltrate in the pancreas [14].

In a case reported by Tumram in 2014, a 65-year-old man suffering from hemiparesis for 6 months consumed a decoction made of oleander leaf extract as a part of herbal treatment. An autopsy examination revealed local subarachnoid hemorrhage in the brain, esophagus, and stomach mucosa, showing congestion with areas of hemorrhage.

Fig. 4 **a** *Nerium oleander* plant seen around the residential area and **b** in the dividers of highway roads

Histopathology showed pneumonitis features in the lungs, acute tubular necrosis in the kidneys, and periportal inflammatory infiltrate in the liver, and other organs were congested. The toxicology analysis was negative [15]. In 2000, two children adopted from an orphanage died from oleander poisoning after eating the leaves from a neighbor's plant in California. In this death, a question arose about how it is possible to eat a bitterly tasting oleander leaf to such a fatal amount. Finally, experts found that the children had acquired pica, a malnutrition-related illness that compels individuals to ingest otherwise inedible stuff [16].

In the present case, the child had consumed an oleander leaf by placing tamarind and salt in it, as she had mistaken it for a guava leaf. Thus, the child might not have felt the bitterness to such an extent. Eating guava leaves in combination with tamarind and salt is common in India. The possible reason for the children to consume oleander leaf mistakenly for guava leaf could be that children might not have enough knowledge about the edible leaf preparation and would have thought that it can be made with any plant leaf. The accessibility to the oleander plant is higher in India, particularly near home, as it is grown widely for decorative reasons, and its flowers are used for devotional purposes, making it very dangerous for accidental ingestion in children. Thus, awareness about the toxic nature of the oleander plant should be publicized to the people, and strict avoidance of the availability of this plant around residential areas and even in the dividers of highway roads (Fig. 4) should be ensured to prevent accidental ingestion not only in children but also in animals.

Conclusion

Though predominantly ingestion of oleander leaf is suicidal and medication-related, accidental is rare, mainly due to its bitter taste. However, its abundance is high near residential areas, making it dangerous for children. Thus, it is better to avoid such ornamental plants around the household.

Key points

1. Oleander is a cardiotoxic plant that contains oleandrin
2. Deaths due to *Nerium oleander* leaf poisoning are mostly accidental
3. Children are prone to accidental oleander poisoning because of its attractive flowers

Author contribution All authors contributed to the conception and design of the work, the interpretation of the findings, analysis, drafting of the work, and final review. All authors approved the final version of the manuscript for publication.

Declarations

Consent to participate Written consent is taken from the next of kin for publication without divulging the identity of the individual.

Conflict of interest The authors declare no competing interests.

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