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Case Study

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OLEANDER POISONING: A CASE REPORT ON NON-FATAL OLEANDER POISONING

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ABSTRACT

Oleander poisoning is very rarely evaluated in clinical practice. The roots, leaves and fruits of the oleander plant contain cardiac glycosides. Oleanderin and neriine are the two powerful cardiac glycosides that are extracted from every part of the plant and are the harmful ingredients [1]. We report a case of oleander poisoning in an adult male presented with multiple episodes of vomiting, burning sensation over the throat, chest and stomach after the ingestion of oleander leaf and fruit. This is a non-fatal oleander poisoning case in which the patient recovered with conservative management. [2]

KEYWORDS: Oleandrin, cardiac glycosides, bradycardia, activated charcoal, orciprenaline.

INTRODUCTION

Nerium oleander, often known as oleander, is an evergreen shrub that grows across the Mediterranean region as well as subtropical and tropical areas. It is a member of the Apocynaceae family.[3,4] The leaves are leathery, very green and grouped in whorls of three. They can have either a linear or narrowly elliptic form. The fragrant blooms are funnel-shaped with five lobes and occur in clusters at the tips of branches. They range in size from 2.5 to 5 cm and come in a variety of colours, including pink, red, white, peach and yellow. Thevetia peruviana is its scientific name and its seeds contain nerifoline and the very poisonous cardiac glycosides thevetins A and B.[1]

Oleandrin is the most relevant toxin of the Oleander plant and it is the only one available as a pure standard. CGs are secondary compounds found in plants and amphibians, widely distributed in nature with a potential cardiovascular action. The most common CGs are digoxin, ouabain, oleandrin and bufalin. Due to their similar chemical structure, CGs share well-known similarities in the mechanism of action and their cardiovascular toxicity usually restricts their clinical use.[4] Ingestion of these plants results in a clinical presentation closely resembling digoxin poisoning. Symptoms primarily affect the heart and gastrointestinal system and include vomiting, diarrhea, dizziness, hypotension, hyperkalemia, sinus bradycardia, atrioventricular blocks (AV blocks) and other arrhythmias. The poisoning occurs due to the inhibition of the sodium-potassium pump in the heart and increased vagal tone, leading to both positive inotropic and chronotropic effect.[1,4,5]

CASE DESCRIPTION

A 43- year old male patient was admitted in the emergency department with nausea, multiple episodes of vomiting & burning sensation over the throat, chest and stomach after ingestion of oleander leaf and fruit after 6 hours.

The patient was under the influence of alcohol with no other noted comorbidities. During the initial examination, vitals were monitored: pulse rate was 84 beats per minute, blood pressure was 140/90 mmHg, respiratory rate was normal and oxygen saturation was 98% on room air. The patient was conscious with a Glasgow Coma Scale score of 15/15. Systemic examination revealed clear chest and lungs, with normal audible S1 and S2 heart sounds.

For further assessment and monitoring, the patient was transferred to the nephrology and toxicology ICU. An electrocardiogram revealed T wave inversion in leads V2-V4., The patient experienced a drop in heart rate to 56 beats per minute and blood pressure to 100/60 mmHg. However, routine examination showed no electrolyte imbalances. The serum potassium was 3.6 MEq/L (normal range: 3.5-5.5 MEq/L). Over the next hour, the patient became increasingly bradycardic with the heart rate falling to 42 beats per minute.

Management and outcome

After the initial assessment, the patient was immediately given a bowel wash with activated charcoal and an IV drip of DNS (1 pint over 1 hour). An injection of Emeset 4 mg was administered as a stat medication to stop vomiting. Upon cardiology consultation, the patient was prescribed Tab Orciprenaline 10 mg three times daily for asymptomatic sinus bradycardia, with instructions to administer an injection of atropine if the heart rate dropped below 35 beats per minute. Additionally, an injection of Deriphylline (1 amp IV) was administered every 8 hours.

After one day, the patient's heart rate responded, and the bradycardia resolved. The injection of Deriphylline was discontinued on the third day. By the third day, the patient's heart rate had improved to 62 beats per minute, and blood pressure normalized to 110/70 mmHg. Routine lab investigations were normal, so the patient was transferred from the ICU to the ward. The patient was continued on Tab Orciprenaline upon discharge. The patient was symptomatically better and discharged in stable condition.

DISCUSSION

Cardiac glycosides such as oleandrin, oleandroside, nerioside, digitoxigenin, thevetin and thevetoxin are found in most plants, including foxglove and oleander. Due to oleander's strong gastrointestinal tract irritation, the cardiac glycosides in it have a greater gastrointestinal impact than those in digoxin. Symptoms might include cramps, bloody diarrhoea, nausea and vomiting. Additionally, oleander irritates mucosal membranes, which results in increased salivation and burning around the mouth. Furthermore, it may result in neurological symptoms such as mydriasis, disorientation, tiredness, weakness, and vision abnormalities.

The most dangerous and sometimes fatal side effects of oleander overdose include cardiac irregularities, such as heart blockages, bradycardia and several types of ventricular dysrhythmias. T wave flatness or inversion, a reduced QT interval and an elevated PR interval are frequently seen in ECG readings. Direct cardiac glycoside poisoning and elevated vagal tone are the causes of these clinical symptoms. The empirical basis for treating oleander poisoning involves the management of symptoms associated with digitalis glycoside toxicity. Initially, gastric lavage with activated charcoal is effective for preventing absorption.

Madhurantakam SR et al, reported a case of oleander poisoning and management of acute cardiac toxicities of oleander leaf poisoning. The patient was immediately taken to hospital after 2 hours of ingestion. They managed initially with gastric lavage and atropine intravenously. The serum electrolye imbalances were corrected with injection potassium chloride and injection magnesium sulphate for management of hypokalemia. Fluid resuscitation was also done.

Although our patient was brought in 6 hours after ingesting the toxin. Activated charcoal 50gm every 6th hourly was used for our patient to remove any residual toxin from the stomach. The patient then followed by symptomatic management. Hypotension was corrected by adequate fluid resuscitation and electrolyte monitoring escpecially potassium and sodium was done. Cardiology opinion was taken for bradycardia. Inj. Atropine is the first line therapy for symptomatic bradycardia and its given if the heart rate falls below 35 beats per minute. Tab Orciprenaline 10mg thrice daily reported to be effective in such cases. Deriphylline can be beneficial for severe recurrent bradycardia. Placing a temporary pacemaker, direct current cardioversion also can be suggested in severe persistent cases of bradycardia and ventricular arrhythmias. Digoxin-specific antibodies (digoxin-specific Fab fragments) are the treatment of choice for severe bradyarrythmias with hypotension unresponsive to atropine and life threatening ventricular arrhythmias.

CONCLUSION

Poisoning due to oleander is rarely seen. Although most cases with oleander poisoning are fatal. This is a non-fatal oleander poisoning in which the patient recovered following conservative management only. All compounds of N. *oleander* are toxic. N. *oleander* produces typical clinical signs of cardiac glycoside poisoning and the most common and potentially fatal signs are cardiac. The patient approach to treatment depends on the patient's neurological, cardiac and circulatory stability. Most cases can be managed by multidose activated charcoal and supportive care with IV fluids and symptomatic treatment.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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