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Case Report

Management of Acute Cardiotoxicity of Oleander Leaf Poisoning at Tertiary Care

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Abstract: Poisoning due to deliberate self-harm with the leaves or seeds of pink or yellow oleander results in significant morbidity and mortality. A wide variety of symptoms like nausea, vomiting, bradyarrhythmias and tachyarrhythmias occur following ingestion. Usually more cases will be seen in suburban parts of India and South Asia. We present an unusual case of poisoning by the ingestion of oleander leaves manifested with refractory bradycardia at tertiary care.

Keywords: Oleander poisoning, Nerium oleander, Thevetia peruviana, Kaner, Cardiotoxic poisoning.

INTRODUCTION

Nerium oleander is an ornamental evergreen shrub belonging to the family Apocynaceae, widespread in the Mediterranean area, but also in subtropical and tropical regions like India. Oleander contains, in each of its parts (seeds, roots, leaves, flowers, fruits, branches and stems). Cardiac glycosides (CGs), also defined as cardenolides [1].

The two types of oleander commonly encountered in India are pink oleander (Nerium oleander) and yellow oleander (Thevetia peruviana, T. neriifolia, Cerbera thevetia). Common names include soland, lorier bol, rosebay, and rose laurel and kaner [2]. Both are predominantly cardiotoxic. Most such cardiotoxic plants contain various cardiac glycosides which act in similar fashion. Some of these glycosides are useful in pharmacotherapeutics (e.g., digitalis).

All parts of the pink oleander are poisonous, especially the leaves, stem, seeds, and root. The following active toxins are present—oleandrin, neriin, folinerin, rosagenin, and digitoxigenin. These are cardiac glycosides and have digoxin-like effects, acting by inhibiting the sodium–potassium adenosine triphosphatase (Na^+-K^+ ATPase) pump [3]. All the parts of the yellow oleander, especially the seeds and root are poisonous. The active toxins include thevetin B, cerberin, nerifolin, thevetin A, ruvoside, and peruvoside.

The two potent cardiac glycosides, oleanderin and neriine, can be isolated from all parts of the plant. Both have positive inotropic, negative chronotropic and cross reactivity [4]. Toxicokinetics and toxic effects are similar in both types of oleander plants. The most commonly seen manifestations in poisoning include gastrointestinal (GI) distress, bradycardia with AV block, hypotension, lethargy, and dizziness [5]. We report a case of deliberate self-harm by ingestion of oleander leaves.

CASE REPORT

A 27 year old female, presented to the emergency room (ER) with history of consumption of seven oleander leaves, two hours ago. She had multiple episodes of vomiting with which she was brought to the ER of private care facility, where gastric lavage was done and injection(Inj.) atropine was given for bradycardia, following which she was shifted to our hospital for further care. In ER, her Glasgow coma scale (GCS) was 15/15, heart rate (HR)-51 beats per minute(bpm), blood pressure (BP)-90/60 mm of Hg. Patient received Inj. Atropine and 50 grams(g) of

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activated charcoal and was shifted to medical intensive care unit.

In the ICU, she was restless with HR-50 bpm, BP- 100/60 mm of Hg, respiratory rate (RR) - 24/min. 2D ECHO showed normal chamber dimensions with left ventricular ejection fraction of 60%. Adequate hydration and electrolyte monitoring especially sodium (Na^{+}), potassium (K^{+}), calcium (Ca^{2+}) and magnesium (Mg^{2+}) was done.

Meanwhile, cardiologist opinion was taken for refractory bradycardia after maximum dose of Inj. Atropine (Figure-1). Patient was intubated for restlessness and temporary cardiac pacemaker insertion (TPI) was done. First 24 hrs she required intermittent pacing, following which she regained her intrinsic rhythm (Figure-2). As patient was hemodynamically stable with normal intrinsic rhythm, pacemaker was removed and she was extubated after 72 hrs of intubation.

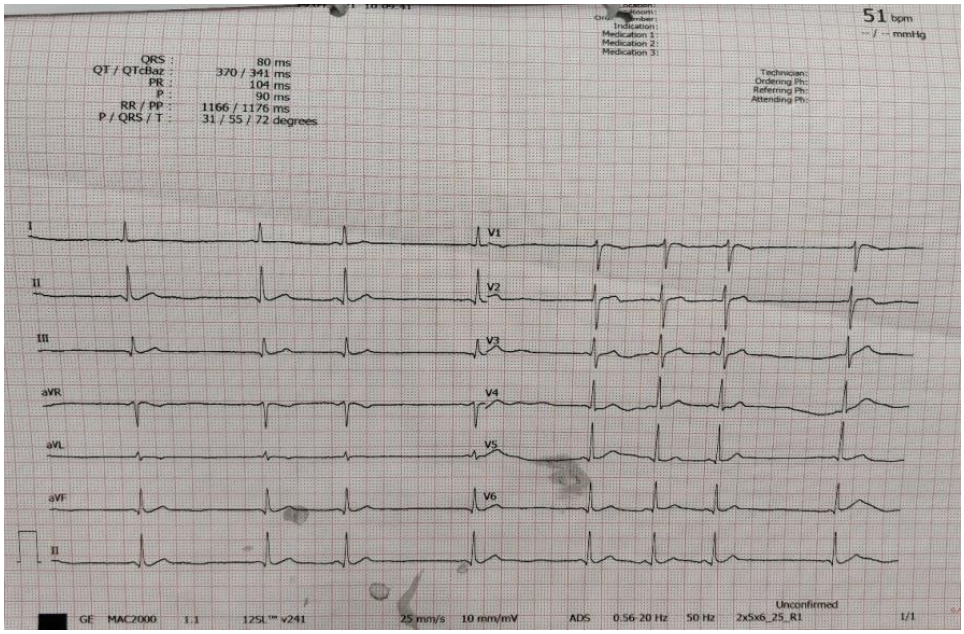

Figure-1: Electrocardiography (ECG) showing refractory sinus bradycardia with occasional junctional escape beats after medical management

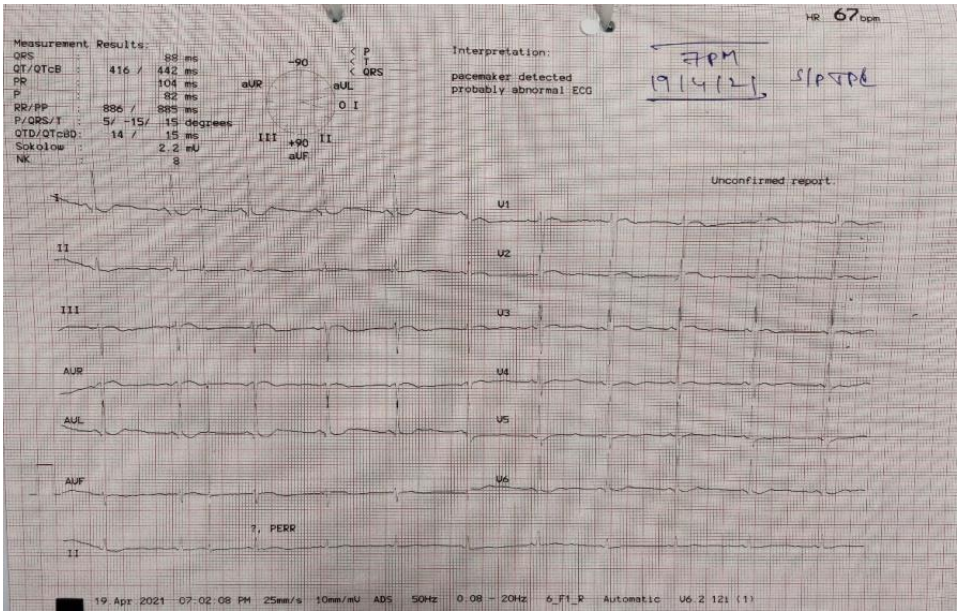

Figure-2: ECG showing intrinsic rhythm after 48 hrs

During her ICU stay, metabolic acidosis was corrected with bicarbonate supplementation. Hypokalemia was corrected with Inj. Potassium chloride (KCl) and Inj. Magnesium sulfate ($MgSO_4$).

DISCUSSION

Pink oleander belonging to the family Apocynaceae, with long, lanceolate, leathery leaves and clusters of white or pink flowers. The leaves produce a

clear, thick sap. In Indian traditional medicine, the roots and leaves are used to prepare decoctions that are said to be useful in the treatment of various skin conditions. The root is sometimes used as an abortifacient by rural people. Pink oleander is also a popular ornamental plant that is grown in gardens as well as on the dividers of national and state highways across India [6].

Yellow oleander is also an ornamental shrub (grows up to 30 feet in height) belonging to the family Apocynaceae, with yellowish funnel-shaped flowers and longish leaves yielding a milky sap [6]. As in the case of pink oleander, yellow oleander is also used in traditional Indian medicine. The bark contains cardenolides, which have been investigated as cytotoxic agents for the treatment of cancer. In the past, yellow oleander glycosides have been used in the treatment of heart failure and atrial fibrillation, but subsequently this was discontinued due to high toxicity. The seeds of this plant have been used in suicidal and homicidal cases [7].

The cardiac glycosides in oleander produce more gastrointestinal effects than those in digoxin, and the symptoms range from nausea and vomiting to cramping and bloody diarrhea. Also, it causes irritation to the mucosal membranes, resulting in burning around the mouth and increased salivation. Confusion, dizziness, drowsiness, weakness, visual disturbances, and mydriasis are central nervous system manifestations of toxicity [8].

Gastrointestinal (GI) manifestations are followed by cardiovascular features such as marked bradycardia with PR and QRS prolongation. Sinus arrest, varying degrees of atrioventricular (AV) block with dissociation, or escape rhythms may occur, including paroxysmal atrial tachycardia with AV block, junctional tachycardia, frequent ventricular ectopics, and bigeminy. Many people present with different cardiac arrhythmias.

In 1989, Driggers DA et al., reported a case of atrial fibrillation with nonspecific ST segment changes and intraventricular conduction delays were seen more than 12 hours after ingestion [9]. In 1998, Khasigian P et al., reported a case which involved inhalation of smoke from a burning oleander plant, a middle-aged patient suffered from sinus bradycardia (without AV block), severe dizziness, and vomiting [10]. In 1999, Bose T. K et al., did a study of 300 yellow oleander seed ingestions with suicidal intent and found 12% of the patients had palpitations, while 46% had some type of arrhythmia; sinus bradycardia was present in 49% of the patients, and ischemic ECG changes were noted in 39% of the patients [11].

In suspected cases of oleander poisoning, resuscitation should be initiated immediately, supporting the patient hemodynamically and continued for at least 1 hour as good outcomes have been reported on prolonged resuscitation. This must be followed by baseline ECG and 24-hour intensive care unit observation. In all patients, the levels of urea, electrolytes, magnesium, and creatinine must be estimated.

Gastric lavage is of limited benefit due to the large size of plant parts in relation to the lavage tube. In fact, the procedure may worsen bradycardia secondary to vagal stimulation. Whole gut lavage is said to be more beneficial. Administration of activated charcoal (50 g for adults; 1 g/kg for children) is recommended for patients presenting to the ER within 4 hours of ingestion.

Serum potassium concentrations must be checked, preferably every 6 hrs. Since hypokalemia can worsen digitalis toxicity and predispose to dangerous arrhythmias, hypokalaemia should be corrected with intravenous potassium [12]. Hyperkalemia is common as a result of blockade of the Na^+-K^+ ATPase pump and potassium shift from the intracellular to the extracellular space, which is a marker of toxicity and is associated with a worse outcome [13]. Hyperkalemia should be corrected.

Theoretically, since intracellular calcium concentrations are already high in the setting of digoxin toxicity, administration of calcium may worsen arrhythmias, and it is generally held that intravenous calcium administration should be avoided [14] or used with caution and it must be given by slow infusion [13].

The place of activated charcoal (AC), as a single dose (SDAC) and as multiple doses (MDAC), is a subject of considerable controversy. Activated charcoal reduce toxicity by preventing absorption of glycosides soon after ingestion and by interrupting their enterovascular and enterohepatic circulations thereby increasing elimination [15]. In our patient we used activated charcoal, 50g, every 6th hourly for 48 hours.

Hypotension is corrected by adequate fluid resuscitation with a crystalloid and brady- and tachyarrhythmias can be treated appropriately. Vasopressors or inotropes can be initiated in emergency if required. Metabolic acidosis should be treated by correction of hypoxia and adequate fluid resuscitation. In severe acidosis sodium bicarbonate can be used. In our patient, we used sodium bicarbonate to correct metabolic acidosis.

Bradyarrthymias are an important cause of death in yellow oleander poisoning [11]. They are commonly treated with atropine, isoprenaline, salbutamol, and temporary cardiac pacing [16]. In our case, as patient was not responding to repeated doses of atropine, we did temporary transvenous cardiac pacing.

Tachyarrhythmias are more dangerous and are more difficult to treat. Limited data on the use of antiarrhythmic drugs in yellow oleander poisoning, and evidence is from digitalis toxicity. Ventricular tachyarrhythmias are best treated with lidocaine. Amiodarone, quinidine, and calcium-channel blockers are contraindicated because they may increase digitalis concentrations, and b-blockers may worsen heart block[18]. Ventricular tachycardia is often resistant to electrical cardioversion. Electrical cardioversion can also result in ventricular fibrillation or asystole and is best avoided [17].

Intravenous magnesium may be considered in cardiac arrhythmias caused by digoxin poisoning where there is likely to be a delay in the availability of digoxin-specific antibody fragments even in the presence of elevated serum magnesium [18]. Magnesium is required for the proper functioning of the Na^+–K^+ pump and opposes the action of digoxin. Similarly, magnesium useful in oleander poisoning.

Hemodialysis or hemoperfusion is not likely to increase the elimination of cardiac glycosides as they have large volume of distribution in the body and are highly bound to protein. However, in the presence of renal failure, they may be beneficial for severe hyperkalemia/acidosis [6].

Digoxin-specific antibodies (digoxin-specific Fab fragments) are the treatment of choice for severe bradyarrhythmias with hypotension unresponsive to atropine and life-threatening ventricular arrhythmias [19].

CONCLUSION

Ingestion of oleander plant or it's parts can be accidental but also intentional. Practicing physicians and intensivists should understand the potential lethal properties of oleander, it's availability throughout the world and it's management.

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