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A CASE SERIES REVIEW ON CERBERA ODOLLAM POISONING

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ABSTRACT

Cerbera odollam, popularly known as the suicide tree, is well-known for its cardiotoxic properties. Three odollam poisoning cases were investigated. Cerberin's mode of action is similar to that of digoxin, hence Cerbera odollam intoxication looks like acute digoxin overdose. Its kernel causes nausea, vomiting, hyperkalemia, thrombocytopenia, and ECG irregularities when consumed. All were presented with similar complaints of vomiting and bradycardia. ECG demonstrated a second degree heart block in 2 cases and a third degree heart block in one patient. Among the 3 cases we discussed, two patients were given Digibind out of which one expired and the other survived. Remaining one patient was managed with a temporary pacemaker insertion. Atropine was given in view of persistent bradycardia and was treated with glucose, insulin, sodium bicarbonate, calcium gluconate and levosalbutamol for hyperkalemia.

KEYWORDS: Cerbera odollam, Mortality, poisoning, Temporary pacemaker.

HIGHLIGHTS

- Cerbera odollam poisoning is a common method of deliberate self-harm.
- Cases of C. odollam poisoning may be undiagnosed in areas of the world where this plant is not seen as the doctors may be unaware of the clinical features.
- Awareness regarding the odollam poison, its clinical and ECG manifestations and its treatment options at the right time can save the patients.

INTRODUCTION

Cerbera odollam is a member of the Apocynaceae family of plants. Because of its powerful cardiotoxic effects, it is commonly referred to as the "suicide tree." Pong-Pong and Othalanga

are common names for the plant, which grows in damp places in South India, Madagascar, and Southeast Asia. In the developing world, deliberate self-harm with this plant's fruit is a big clinical problem.[1] This plant's poisonous portion is the kernel, which is found at the centre of its fruit. Cerberin, neriifolin, and diacetyl tanghinin are the main cardiac glycosides found in C. odollam seeds. Cerberin's mode of action is similar to that of digoxin, hence cerbera odollam intoxication looks like acute digoxin overdose. Its kernel causes nausea, vomiting, hyperkalemia, thrombocytopenia, and ECG irregularities when consumed.

Management of patients with cerebra odollam poisoning is similar to that in patients with digoxin poisoning and the initial management includes supportive therapy and administration of atropine followed by temporary pacemaker insertion.[2] Administration of digoxin immune Fab may be considered in severe cases.[3] The total number of deaths due to odollam poisoning in the state of Kerala over a period of 11 years was 537.[2]

Case 1

A 32-year-old woman with a history of depression presented to the hospital with nausea and lethargy, as well as one incident of non-bloody emesis. The patient had taken one Cerbera odollam seed with an intention of ending her life. On the way to hospital, the patient experienced a junctional rhythm and hence administered a total of 10 vials of Digibind (digoxin immune fab). There was no history of headache, visual disturbances, chest pain, palpitations, shortness of breath, abdominal tenderness, diarrhea, or constipation. Her vital signs were noted and found to have a blood pressure of 105/74 mmHg, heart rate of 106 beats per minute, respiratory rate of 18 breaths per minute, body temperature at 98.6 degrees F, and oxygen level of 98% on room air. Urine toxicology showed positive results for cannabis. Other parameters were digoxin level of 0.2 ng/mL, white blood cell count 8,100/µL, hemoglobin 13.9 g/dL, hematocrit 40.8%, platelets 165/µL, sodium 138 mmol/L, potassium 4.3 mmol/L, chloride 103 mmol/L, bicarbonate 29 mmol/L, blood urea nitrogen 10 mg/dL, creatinine 0.84 mg/dL, glucose 107 mg/dL, total bilirubin 0.5 mg/dL, AST 37 U/L, ALT 61 U/L, and alkaline phosphatase 48 eU/L. ECG demonstrated a significant decrease in heart rate from a range of 106 bpm to 36 bpm and sinus bradycardia. 5 more vials of Digibind could be given in case of slowing of heart rate. Her probability of getting toxicity was low since her level of digoxin was 0.2 ng/mL and normalized potassium levels.

Case 2

A 17 yr old female patient presented with complaints of multiple episodes of vomiting

following consumption of cerbera odollam with suicidal intention. She was conscious with no history of bleeding or breathing difficulty. On arrival, her blood pressure was 110/70mmHg, PR was 112/minute and RR was 16/minute. Her initial ABG showed respiratory acidosis and ECG showed severe bradycardia, third-degree atrioventricular block and was shifted to ICU for further observation. On laboratory examination, her serum potassium was 7.4mmol/L and all other parameters were found to be normal. Initial management included oxygen supplement, intravenous fluids and other supportive measures. Atropine was given in view of persistent bradycardia (HR-40/minute). A temporary pacemaker insertion was done as she was at the risk of developing complete heart block. Considering her increased serum potassium level, she was treated with glucose, insulin, sodium bicarbonate, calcium gluconate and levosalbutamol. After twenty four hours of post ingestion, her serum potassium level was found to be 4.2mmol/L. After 3 days, she became hemodynamically stable hence the temporary Pacemaker was removed after monitoring her vitals.

Case 3

A 22-year old patient presented to hospital after a suicide attempt. The patient admitted that he had ingested “pong-pong” seed approximately seven hours prior to arrival which he had purchased online. He vomited two hours after seed ingestion and presented with chest pain and a weird feeling. He had a past history of depression, post-traumatic stress disorder, and one previous suicide attempt. His vital signs revealed a temperature of 36.2° Celsius, pulse rate of 53 beats per minute (bpm), respiratory rate of 16 breaths per minute, blood pressure 114/54 mmHg and Spo2 of 99% on room air. On physical examination, the patient was alert and conscious with a GCS of 15. His cardiovascular examination revealed bradycardia with a regular rhythm and no murmurs. Lungs were clear bilaterally. The patient’s initial electrocardiogram (ECG) demonstrated second-degree heart block with 2:1 atrioventricular (AV) conduction and ST-segment depression with biphasic T-waves. Five vials of digoxin immune fab were given intravenously and the patient’s heart rate improved to 90bpm. An improvement to a sinus rhythm with first-degree AV block was shown in the second ECG that was obtained after the first dose of digoxin immune fab. Laboratory parameters displayed a potassium level of 5.2 mEq/L and troponin I <0.05 ng/ml. The patient experienced a bradycardia again and the 3rd ECG demonstrated a high-degree heart block. Five more vials of digoxin immune fab were administered and thirty minutes later the patient became unresponsive and lost pulses. He was resuscitated and intubated. Ten additional vials of digoxin immune fab were given. Unfortunately, the patient expired.

DISCUSSION

C. Odollam is a tiny tree that grows abundantly around canals and backwaters of Kerala. It reaches a height of 5-10 metres and has whorled branchlets with lanceolate leaves at the ends. It features enormous, fragrant white flowers and drupe-like green fruits. The active principles, cerberin, cerebroside, and odollin, are glycosides found in the kernels. Cerberin is a monoacetyl nerifolin with strong cardiotoxic properties. The clinical findings emphasise the common occurrence of bradyarrhythmias like SA and AV blockages, as well as AV dissociations on occasion. Cerberin, a cardiac glycoside found in the seed, reversibly inhibits the sodium–potassium adenosine triphosphatase (Na–K– ATPase) exchanger in myocardial cells. This produces an increase in automaticity, increased vagal tone, and hyperkalemia in cardiac myocytes, resulting in elevated calcium levels.[4] All of these lead to cardiac arrhythmias and heart blockages, which eventually lead to death. Management of cerberin toxicity is similar to that of digoxin toxicity, consisting of supportive treatments for bradycardia and hyperkalemia as well as administration of digoxin immune fab. The reported presenting symptoms of odollam toxicity include headache, muscle weakness, dizziness, nausea, vomiting, abdominal pain, chest pain and palpitations. Bradycardia, hyperkalemia and thrombocytopenia are often present. ECG findings include sinus bradycardia, sinus pauses, wandering pacemaker, atrial fibrillation, ventricular tachycardia, digoxin like ST changes and first, second and third degree heart block.

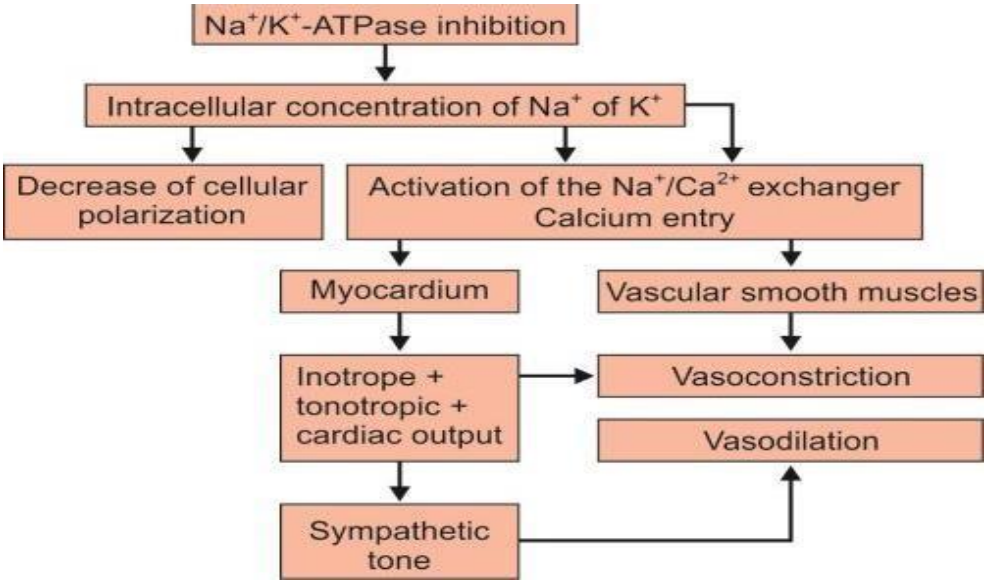

Figure 1: Mechanism of action of odollam.

In the Asia-Pacific region, cerberin toxicity is well-known. A single medical hospital in Kerala, India's southwest state, recorded 102 cases of odollam poisoning in 2016.[1] Another study

showed a total of 537 deaths during the period from 1989 to 1999 due to *odollam* poisoning.

In the 3 of the cases we discussed, the most affected age group was 20-25. But the poisoning can occur regardless of age and gender. The presentations of the patients to the hospital were almost similar. Vomiting and bradycardia were the common symptoms spotted in these 3 cases. Two out of three patient's ECG demonstrated a second degree heart block and the third one showed a third degree heart block. All the three patients had suicidal intention. None of the patients experienced thrombocytopenia, confusion, headache and vision changes. Hyperkalemia, one of the major symptoms of odollam poisoning, was seen only in one patient.

Generally odollam poisoning is managed with stomach wash and activated charcoal. Supportive treatments for bradycardia and hyperkalemia are atropine, temporary pacemaker therapy and potassium-lowering agents with insulin-dextrose infusions respectively as well as administration of digoxin immune fab. In a study conducted by B. Renymol *et.al.* patients were initially managed with stomach wash and activated charcoal.[1] Hyperkalemia was corrected. Atropine was given for bradycardia and heart blocks. Temporary cardiac pacing was done for patients with severe bradycardia not responding to atropine and higher degrees of heart block.

Among the 3 cases we discussed, two patients were given Digibind out of which one expired and the other survived. Remaining one patient was managed with a temporary pacemaker insertion. Atropine was given in view of persistent bradycardia and she was treated with glucose, insulin, sodium bicarbonate, calcium gluconate and levosalbutamol for hyperkalemia. Her temporary pacemaker was removed as she became hemodynamically stable.

CONCLUSION

Treatment approach to cerberin toxicity depends on many factors such as quantity of seeds consumed, BMI, severity of hyperkalemia, and ECG changes. Cases of C. odollam poisoning

may be undiagnosed in many areas of the world where this plant is not seen. This case series serves as an alerting tool among health care professionals regarding Cerbera odollam poisoning and its management. Creating awareness among the general public helps to identify the fruit and avoid accidental consumption of the same.

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