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Case Report

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CERBERA ODOLLAM POISONING: A CASE REPORT

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ABSTRACT

Cerbera odollam also sometimes called Cerbera manghas, belongs to the notoriously poisonous Apocynaceae family. C. odollam produces clinical manifestations by its active principle, a cardiac glycoside "cerberin". The clinical manifestations of C. odollam include nausea, vomiting, hyperkalemia, thrombocytopenia, ECG abnormalities, syncope, chest pain and palpitations. Management of patient with cerebra odollum is similar to that in patients with digoxin poisoning and includes gastric decontamination, supportive therapy such as administration of atropine followed by temporary pacemaker insertion and antiarrythmic agents. Administration of digoxin immune Fab may be considered in severe cases. Life threatening hyperkalemia is treated by intravenous glucose, insulin, calcium gluconate, sodium bicarbonate, levosalbutamol and oral potassium binding resins.

KEYWORDS: Cerbera odollam, cerberin.

INTRODUCTION

Cerbera odollam also sometimes called Cerbera manghas L, belongs to the notoriously poisonous Apocynaceae family. The vernacular names of the odollam tree are: othalanga maram in the Malayalam language used in Kerala; kattu arali in the adjacent state of Tamil Nadu; famentana, kisopo, samanta or tangena in Madagascar; pong-pong, buta-buta or nyan in Southeast Asia.[1]

Human poisoning with the cardio toxic plant Cerbera odollam [Figure 1] is common in the muddy river deltas of Southeast Asia and tropical Pacific Islands as far east as French Polynesia. In the South Indian state of Kerala, it accounts for half of the state's plant poisoning deaths and 1 in 10 of all fatal poisonings. More than 500 cases of fatal Cerbera poisoning between the decade 1989 and 1999 have been documented in the state, but the exact fatality may be much higher, given the failure of detection of the toxin readily by clinical or chemical methods.[1]

Figure 1

The rate of poisoning appears to be increasing. It is largely unknown to the western world, given the geographic distribution of this plant. *C. odollam* produces clinical manifestations by its active principle, a cardiac glycoside "cerberin" binding and inhibiting Na+, K+ ATPase in cardiac myocytes. The resultant increased intracellular Na+ and Ca2+ levels leading on three effects: (1) Increased automaticity, (2) increased vagal tone, and (3) hyperkalemia. Increased automaticity leads on to cardiac arrhythmias. Increased vagal tone produces heart blocks and sinus pauses. Hyperkalemia causes muscle weakness or paralysis and cardiac arrhythmias.[2]

Exposure to high doses of Cerbera odollam carries the highest risk of mortality. Management of patient with cerebra odollum is similar to that in patients with digoxin poisoning and includes gastric decontamination, supportive therapy such as administration of atropine followed by temporary pacemaker insertion and antiarrythmic agents. Administration of digoxin immune Fab may be considered in severe cases, although efficacy is variable and data limited to isolated case reports. Life threatening hyperkalemia is treated by intravenous glucose, insulin, calcium gluconate, sodium bicarbonate, levosalbutamol and oral potassium binding resins.[5]

CASE REPORT

A 17 yr old female patient came with complaints of multiple episodes of vomiting following alleged consumption of cerbera odollum on day before arrival. She had no history of loss of consciousness, bleeding or breathing difficulty. On arrival, her blood pressure was 110/70mmHg, PR was 112/minute and RR was

16/minute. Her initial ABG shows respiratory acidosis and ECG shows severe bradycardia, third-degree atrioventricular block (Figure 2) and was admitted in ICU for observation. Other physical examination and routine laboratory data were unremarkable except serum potassium of 7.4mmol/L.

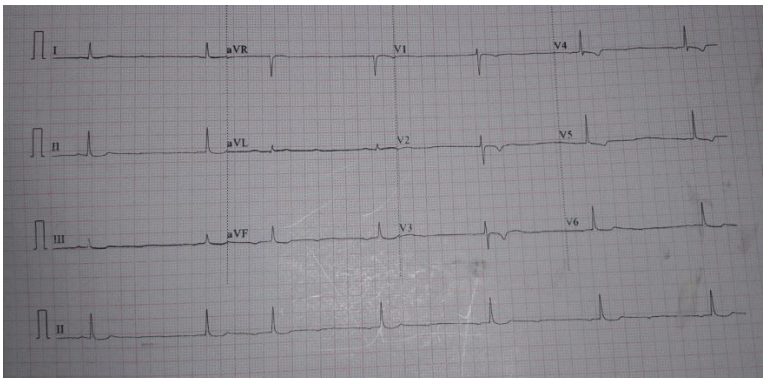

Figure 2

The patient was placed on oxygen, intravenous fluids and other supportive measures. Atropine was given because of persistent bradycardia (HR-40/minute) and patient was shifted to CCU as she is at the risk of developing complete heart block and temporary pacemaker insertion was done. Because of increased serum potassium level, treatment with glucose, insulin, sodium bicarbonate, calcium gluconate and levosalbutamol was commenced and planned for haemodialysis, but on repeating potassium level after twenty four hours post ingestion, her potassium level decreased to 4.2mmol/L and haemodialysis was withheld.

She was shifted to medical ward after 3 days as she become haemodynamically stable, hence temporary pacemaker removed after monitoring vitals while keeping temporary pacemaker switched off for a period of time. Psychiatry consultation was done and patient was diagnosed as emotionally unstable personality disorder and advised psychotherapy .Repeat electrocardiogram shows normal sinus rhythm and patient was discharged.

DISCUSSION

Cerberaodollam is a poisonous plant present in the southern coastal parts of Kerala. The kernel of the fruit contains cardiac toxins like Cerberin, Cerberoside; intestinal irritants like Odollin, Odollotoxin and cytotoxic molecules. It is a common suicidal agent in Kerala with 25% mortality rate. Its incidence is next to the insecticidal poisoning. It comprises 50% of the plant poisoning cases. The clinical profile of odollam poisoning varies according to the number of the kernels of plant ingested. Number of kernels of the plant ingested is a useful measure of dosage as it has associations with vomiting, need for cardiac pacing and death.

Cerbera odollam toxicity manifests similar to acute digoxin poisoning, commonly reported manifestations include nausea, vomiting, hyperkalemia, thrombocytopenia and ECG abnormalities. Syncope, chest pain and palpitations are rarely reported. With the advancements in treatment modalities and the emergence of cardiac pacing, cases presenting even with late manifestations are potentially salvageable.

Odollam has been previously evaluated for its anti‑cancer properties, found that the leaf extracts of C.odollam has cytotoxic activity against two kinds of breast cancer cell lines which are T47D and MCF7 with two ovarian cancer cells and a normal usual cell line.[6]

Initial management of cerbera odollum poisoning includes supportive therapy and administration of atropine followed by temporary pacemaker insertion and treating hyperkalemia with intravenous glucose, insulin, calcium gluconate, sodium bicarbonate, levosalbutamol and oral potassium binding resins.

Calcium salts are generally avoided because it is widely believed that calcium administration may enhance myocardial calcium load and ultimately lead to arrhythmia or cardiac arrest. Hyperkalemia in our patient was initially treated with calcium gluconate and the patient did not develop any arrhythmia. Although we agree that calcium salts should better be avoided in digoxin or similar poisonings, the dangers of calcium therapy might have been overstated.[7]

Digoxin-specific Fab fragments are generally considered the first-line therapy for patients with severe digoxin toxicities. For plant cardiac glycoside or toad venom poisonings, the antidote has also been shown to be effective in treating severe cardiac dysrhythmias and hyperkalemia.[7] Although our patient did not receive antidotal therapy, she may have had a smoother hospital course had she received the antidote.

CONCLUSION

Deliberate self-harm with C. odollam is major clinical problem in the developing world; however, it is relatively unknown to the western physicians. Ease of availability makes C.odollam kernels, the cause of deaths

in more than half of Kerala's ingested plant poisonings. The predictors of mortality include vomiting, bradycardia, hypotension, hyperkalemia, and the presence of severe ECG changes. Such poisoning cases need monitoring and treatment in ICU with facilities for ECG monitoring and temporary pacemaker insertion. Formulation and implementation of management protocols for C.odollam poisoning is the need of the hour.

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