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A Case Report On Cerbera Odollam Poisoning

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Abstract

Cerbera Odollam, also sometimes called Cerbera manghas/ suicidal tree/Pong- pong is a tree belonging to the poisonous Apocynaceae family. Cerbera odollam produces its poisonous results through a cardiac glycoside,"cerberin".The scientific manifestations encompass nausea, vomiting, thrombocytopenia, hyperkalemia and ECG abnormalities. Management of patients with C odollam is comparable to it in patients with digitalis glycoside poisoning. Here we report a case of an accidental ingestion of Cerbera odollam and was successfully treated at our hospital. Physicians ought to remember the clinical presentation, diagnosis and treatment related to C. Odollam poisoning and be prepared to save the patient's lives.

Keywords

Cerbera odollam, cerberin, poisoning, cardiac glycosides, ECG changes

Introduction

CERBERA ODOLLAM belongs to the poisonous Apocynaceae family which grows abundantly in streams, ponds,coastal areas and mangrove forests in southern India, Madagascar and Southeast Asia [1,2] . It is a small tree which bears fruits resembling unripe mangoes (Fig.1).Ingestion of the CERBERA ODOLLAM kernel is responsible for about 50% of plant poisoning cases and 10% of total poisoning cases in Kerala, India. Although it is largely found in these places, cases have also occurred outside the region, because the seeds are available on the internet at a modest cost. The primary ingredient responsible for toxicity is cerberin which is similar to digoxin [3] .The clinical manifestations of odollam

poisoning are vomiting, dizziness, abdominal pain, loose stools, palpitation, chest pain, drowsiness and abnormalities in pulse such as bradycardia, irregular pulse, hypotension, ECG abnormalities, and hyperkalemia.

Fig 1: Cerbera odollam

Case Report

Herein we report a case of a 24 year old boy with a 1 day record of chronic vomiting and dryness of mouth after accidental ingestion of odollam fruit. Initially he was taken to a Local Hospital, where conservative control alongside gastric lavage became done. His ECG indicates 2:1 AV Block-Wenckebach Phenomenon (Fig.2.1) and Inj. Atropine was administered. Then he was stated at our medical institution for in addition control. He had no records of fever, dysuria, belly pain, palpitations, cough, breathlessness, headache, seizures, LOC. On arrival, his blood pressure was 110/70mmHg, pulse rate was sixty five beats according to minute, breathing rate was sixteen breaths according to minute, body temperature at ninety eight ° F, and oxygen level of ninety two % on room air. On physical examination, he was alert and oriented to person, place and time with a Glasgow Coma Scale E4M5V6.Labs carried out and indicates He had a potassium 4.two mEq/L, phosphorus 2.00mg/dL, creatinine 1.04mg/dL,Sodium 135mEq/L, calcium nine.07mg/dL, Neutrophils elevated (80%), total count 8500/µL, magnesium two.27 mg/dL, first of all platelet remember became 1.81lakh/µL followed one lakh/µL on discharge.His ABG confirmed respiratory alkalosis with pH value of 7.532 and pCO2 value of 28.three mmHg. A temporary pacemaker was inserted and was admitted within the ICU then ECG was taken(Fig. 2.2).Consultants advocated to give Inj. Emeset 4mg IV and Inj. Tramadol 50 mg IV as stat medications, and to start Inj. Augmentin 1.2g BD IV with Inj. Pantoprazole forty mg OD IV. Digiband was recommended only if circumstance worsens. After three days, he became shifted to the medical ward, ECG was repeated and proven normal sinus rhythm(coronary heart rate:seventy three bpm), and Ist diploma AV block (Fig 2.3). After he became hemodynamically stable, a temporary pacemaker was removed and got discharged at his request.

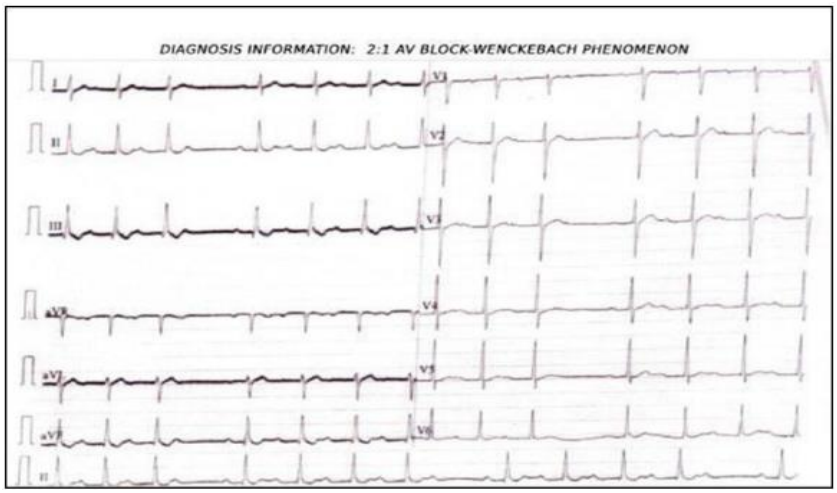

Fig 2.1: Sinus Rhythm, 2:1 AV Block-Wenckebach phenomenon, Low T wave

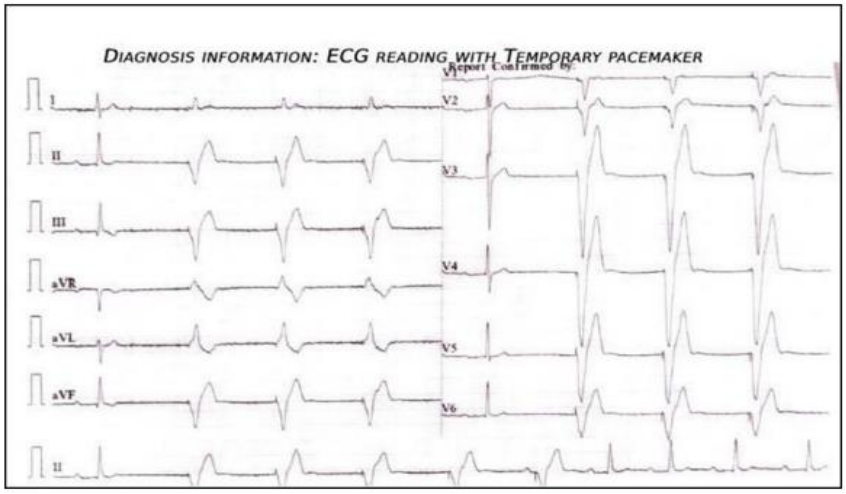

Fig 2.2: ECG readings with Temporary pacemaker, which shows Sinus Arrhythmia

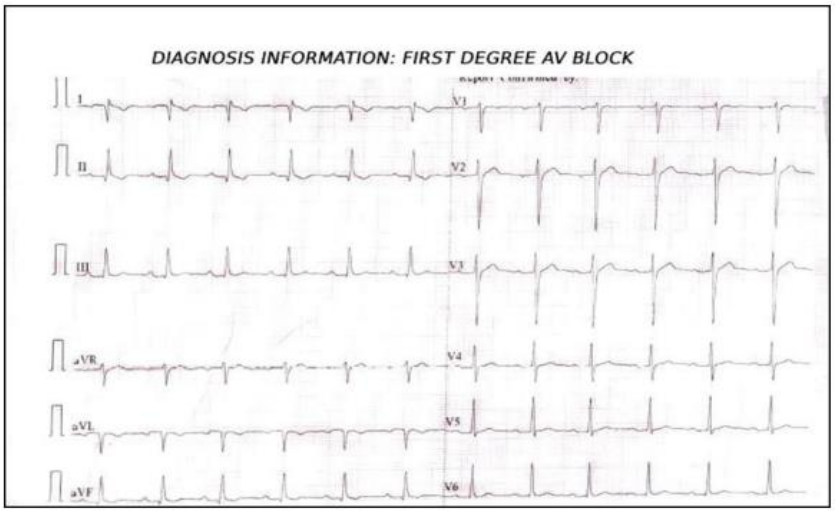

Fig 2.3: First degree AV block

Discussion

Cerbera Odollam with a far like unripe mangoes grows nicely in South India, particularly in Kerala.The poisonous part is the kernel of the fruit. The seeds incorporate Cerberin–cardiotoxins which is a colourless crystallisable glucoside. It's easy availability and excessive lethality makes it a not unusualplace suicidal agent, even in sufferers who take it unexpectedly with none suicidal intention [1] . In 2016,a single clinical centre of Kerala, India on my own stated 102 instances of odollam poisoning [3] . 10 years of examine from 1989 to 1999 indicates 537 stated deaths of odollam poisoning [6] . The stated signs of odollam toxicity consist of headache, muscle weakness, dizziness, altered intellectual status, abdominal pain, nausea & vomiting, chest pain, and palpitations, Bradycardia, hyperkalemia, and thrombocytopenia [3] .Our patient presented with new signs of dry mouth and his blood work showed decreased phosphorus and potassium levels. The ECG indicates a 2nd degree heart block that is just like the Electrocardiogram abnormalities of previous studies [1,3-4] .The mechanism of action of cerberin poisoning is based on the binding and reversible inhibition of the sodium adenosine exchanger triphosphatase in cardiac cells.This results in extracellular accumulation of potassium and intracellular accumulation of sodium.The increase in sodium disrupts the sodium-calcium exchanger which results in an increase in intracellular calcium, thus lengthening the cardiac action potential, which results in a drop in coronary heart rate. In turn, increased cytoplasmic calcium facilitates calcium uptake to the sarcoplasmic reticulum (SR) and allows greater release of calcium from the SR after stimulation, resulting in increased contraction of the heart. The building up of extracellular potassium is accountable for hyperkalemia inflicting weakness in the muscles and cause cardiac arrhythmias [1,2] .The only method for detecting odollam is by thin-layer chromatography [1,4,5] .However, here we agree with the patient's story of accidental consumption of "pong pong" seeds and hospital course, ECG changes, are sufficient to make a diagnosis of C. odollam poisoning. Some research indicates that a minimum lethal dose for humans is 1/2 of an odollam nucleus, while others indicate that a full nucleus is required for full lethal effect [3,7] . Our patient with a body mass index of 23.5 kg/m2 claim to have consumed 1/3 of the seeds and survive. Renymol B et al reported that patients were initially managed with stomach wash and activated charcoal which is parallel to our case.Supportive treatment of bradycardia and timely use of pacemaker has brought down the mortality. Digoxin specific antibody has been suggested as treatment only if the situation worsens but we do not experience it [1,3] .Most of cases reported were suicidal attemptes indicating severe poisoning but ours is an accidental intake of odollum with a minimal amount [1-6] .

Conclusion

C.odollum poisoning is a common suicidal method because of its wide availability. The intensity of poisoning is proportional to the seeds consumed.Early diagnosis and treatment prevents patients from Lethal effects.Due to the high risk of mortality, close cardiac monitoring is essential. Cases of odollam poisoning may be undiagnosed or underreported due to its difficulty in detecting blood glycoside level.Our case helps to implement awareness and treat physicians, chemists, coroners, and forensic toxicologists in light of the increasing incidence of attempted cerberin poisoning.

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