

Unrestrained Automaticity- A Peculiar Case of “Thevetia Peruviana (Yellow Oleander)” Poisoning

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Abstract:

Yellow Oleander/Kanaru (*Thevetia peruviana*) is a tropical poisonous plant with multitude of toxic effects, out of which cardiac toxicity is very challenging to manage. 23 year old boy was transferred to our unit for further management of symptomatic bradycardia following ingestion of four Kanaru seeds. Soon after admission his bradycardia worsens and advanced heart blocks develop. While planning on a temporary pacemaker (TPM) he swings towards tachyarrhythmias. Despite standard treatment according to available anecdotal evidence and multiple cardioversion attempts he progress in to a ventricular tachycardic storm and succumb at ICU while being intubated. Cardiac toxicity due to Kanaru (Yellow Oleander) can be both tachy and Brady arrhythmias. This means the available tool box for cardiologist is significantly limited. High dependency unit observation, TPM facilities and specific antidote (Digibind) is of paramount importance in reducing mortality.

Key words: kaneru; yellow oleander; plant poisoning in sri lanka; digitalis toxicity

Introduction

Yellow Oleander is shrub in the Dogbane family “Apocynaceae”. Poisoning due to deliberate self-harm (DSH) results in significant mortality in South Asia. In Sri Lanka most of the cases are reported in north central, eastern and northern districts (1). It was observed in threatening proportion around 2005-2010 where there was a wave of DSH attempts. Thanks to proactive preventive work of health officials and local administration which even included a significant reduction in number of plants the patient numbers has plummeted down during last few years.

The Plant contain highly toxic cardiac glycosides including thevetin A, B and neriifolin (2) which works directly on sodium/potassium ion channel (NaK) in sarcolemma. Ingestion causes a variety of cardiac, neurological and gastrointestinal effects (3). Ingestion of seeds causes predominantly cardiovascular effects such as bradycardia, varying degrees of heart blocks, atrial or ventricular ectopic and ventricular tachyarrhythmias (4). It is peculiar in that toxins can cause both Brady/tachy arrhythmias. Hyperkalaemia is a life-threatening sequel which is an indication for treatment with digoxin immune fab [Digibind]. Unfortunately this specific antidote is currently out of stock in Sri Lanka.

Case Report:

A 23-year-old was transferred from a regional hospital, 6 hours after ingestion of four kernels of Kanaru along with jaggery. They have already performed one cycle of activated charcoal. The indication for transfer was for observation of sinus bradycardia (45/min) with stable vitals. On admission to us patient was complaining of nausea and abdominal cramps occasionally. GCS was 15/15, non-agitated and no seizures were documented. Blood pressure was 124/88mmHg.

After admission to coronary care unit (CCU) in about two three hours he developed advanced AV (atrioventricular node) blocks including second degree and intermittent complete heart block. Potassium turned out to be 3.0mmol/l. We started him on another MDAC charcoal regime and slow Hartmann’s infusion. Initially two atropine boluses were given but only resulting in transient improvement of heart rate. Rate was around 35bpm with blood pressure dropping to 90/50mmHg.

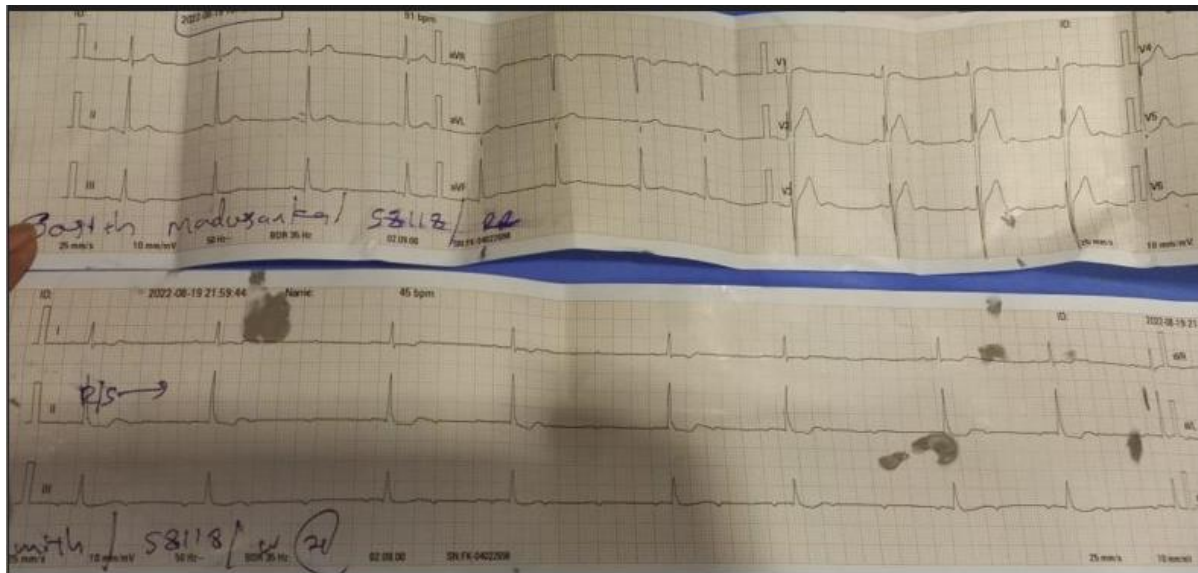


Figure 1: High degree AV block

While he was taken to cath lab for temporary pacing he developed fast atrial fibrillation (AF) with V rates over 130/min. As tachy-brady was alternating we implanted a TPM and kept it at a backup of 50/min. With the TPM, haemodynamics improved.

The boy next developed two GTC seizure attacks which were managed successfully with IV diazepam. Now he was persistently in tachy zone and reverberated between fast AF and bidirectional VT.

Most of the time when VT came in, we had to use multiple shocks in larger joule amount due to relative refractoriness. We loaded him with intravenous Lidocaine as it is preferred over Amiodarone in digitalis toxicity. By this time, he has become a bit delirious so to settle sympathetic drive we electively intubated. Despite our best efforts he went into refractory VT storm and shock state. Finally, the rhythm was a non-shockable PEA and ALS CPR was in vain at the end of 30 minutes.

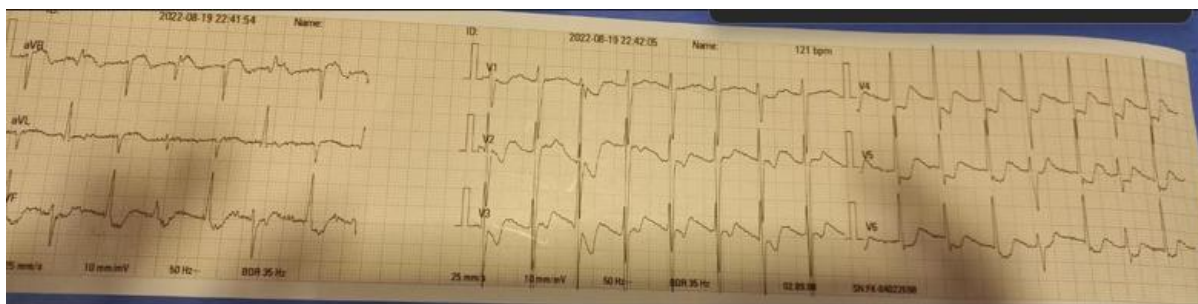


Figure 2- Fast Atrial fibrillation with digoxin effect

Discussion:

Cardiac effects of Kaneru poisoning could be very tricky to manage. Treatment involves a fine dynamic balance of brady-tachy treatment and high dependency/ICU care. Clinicians need to “think on their feet” on most occasions.

Due to long half-life and slow absorption of digitalis toxins, multiple dose activated charcoal (MDAC) has a very important place in initial management (5). Gastric lavage is recommended in the initial four hours of ingestion but most cases of DSH present later, attenuating its potential as a treatment modality. But activated charcoal can be even used up to 48 hours with proven benefits. It is the only treatment which reduces absolute amount of toxin ingested [5, 6]

Initial 72 hours is the critical period for cardiac complications. It is recommended that after ingestion of significant number of seeds (usually >3) patient's should be kept on ECG monitoring. There is a wide spectrum of presentations and patient can deteriorate in minutes to hours.

Due to this necessity most patients with sinus bradycardia following ingestion are transferred to tertiary centres with temporary pacemaker

capacity. It is rather advisable because of abrupt nature of complications and long transfer delays in aforesaid areas with high prevalence.

With regards to bradycardia management some authors recommend using atropine boluses (4) as when needed stating that it has a very short half-life. There is a general consensus that intravenous isoprenaline and chronotropic agents are detrimental because they trigger refractory ventricular tachycardia.

In our practice we observe these patients in high dependency setup and if there is persistent high degree AV blocks or decompensating sinus bradycardia we always insert a temporary pacemaker and program it to a low base rate, to enhance “on demand” function. TPM can be removed within 3 days in majority of cases.

When patient enters tachy zone the management is always challenging. If there is alternating tachy-brady as in our patient TPM is always helpful. Otherwise the VT/VF is usually resistant to cardio version. One can use intravenous Lidocaine as first line (1) and amiodarone is better avoided. Sympathetic block, paralysis with ventilation and IV Esmolol are all secondary options without proven benefits in larger trials.

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