EXTENDED VERSION

(Supported by case reports from journal articles)

Common Poisonous Plants in Sri Lanka

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ATHTHNA (THORN APPLE) - අත්තන

Botanical Name: Datura stramonium




Most poisonous:

Seeds

Fatal dose: 50-75 seeds

Flowers

Stem

Fruit

Leaves

Roots

Toxin: Belladonna alkaloids

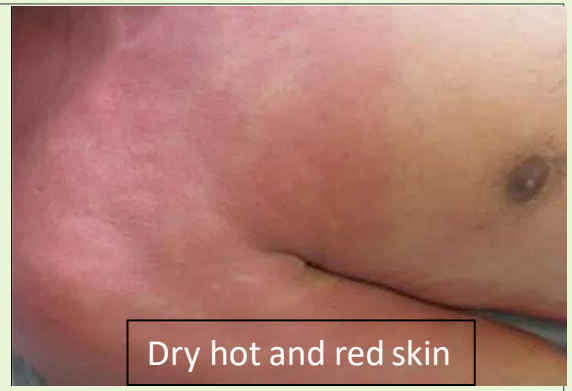
- Atropine
- Hyoscine
- Hyoscyamine
- Scopolamine

CIRCUMSTANCES OF DATURA POISONING

- Stupefying purpose
  - Mixed with cigarettes produce state of unconsciousness to facilitate robbery & rape
  - Mixed with sweets (gingerly) robbery & rape
- Accidental poisoning: Children
- Voluntary ingestion: Seeking its hallucinogenic and euphoric effects
- Suicidal
- Homicidal : Very rare

DATURA POISONING SIGNS & SYMPTOMS

- Dryness of mouth/mucosa
- Dysphagia
- Dysarthria
- Dry hot and red skin
- Ataxia
- Impaired short term memory
- Disorientation/ confusion/ delirium
- Hallucinations (visual and auditory)/ psychosis
- Agitation/ Seizures
- Drowsiness leading to coma
- Urinary retention
- Rarely: rhabdomyolysis, fulminant hepatitis
- Death : respiratory failure or cardiac arrythmias


Dry hot and red skin

Autopsy findings: Seeds in stomach & non-specific features

- Toxins act as competitive antagonists to peripheral and central muscarinic acetylcholine receptors.
- Produce characteristic manifestations of anticholinergic poisoning.

- Toxicity usually occurs within 60 min after ingestion.
- Clinical symptoms may persist up to 24-48 hours due to delayed gastric emptying.

Management:

- Mainly supportive
  - Gastric decontamination with activated charcoal.
  - Sedation to control agitation.
  - Hyperpyrexia control.

CASE 01– *Datura stramonium* poisoning

22 year old male was admitted to the Emergency Unit with history of deliberate ingestion of *Datura stramonium* 2 hours before the presentation.

At arrival, patient showed GCS of 7, mydriatic and areactive pupils, agitation, dry skin and mucosa, fever, tachycardia with right bundle branch block, and severe urinary retention in USS.

Rapid sequence induction and intubation was performed immediately.

After gastric lavage, which revealed Datura seeds, the patient received activated charcoal, in repeated doses. Toxicological screening was negative for other substances.

The blood tests revealed rhabdomyolysis and prevention of renal failure was initiated.

At the ICU, due to agitation, patient was sedated and kept on assisted-control mechanical ventilation. external and internal cooling was applied, and an infusion of neostigmine was started.

A proper neurological evaluation was done. After 36 hours of *Datura stramonium* ingestion, the patient was extubated without any signs of respiratory or circulatory impairment and transferred to the Psychiatric ward for further assessment and care.

Trancă SD, Szabo R, Cociş M. Acute poisoning due to ingestion of Datura stramonium - a case report. *Rom J Anaesth Intensive Care*. 2017;24(1):65-68. doi:10.21454/rjaic.7518.241.szb

DIVI KADURU (EVE'S APPLE) දිවි කදුරු

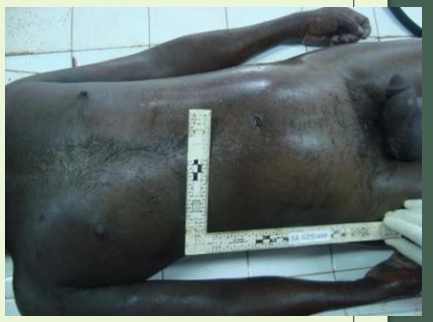
Botanical Name: *Pagiantha dichotoma*
Tabernaemontana dichotoma

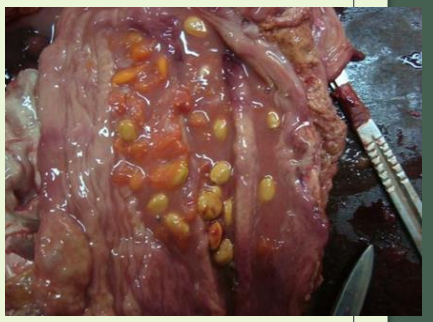
Most poisonous:
Latex
Seeds
Fruit

Toxin: Alkaloids
- Strychnine

DIVI KADURU POISONING SIGNS & SYMPTOMS

- White latex : inflammation of eye
- Ingestion :
  - Dryness of mucus membranes
  - Thirst
  - Dilatation of pupils
  - Rapid pulse
  - Psychomotor disturbances
  - Hallucinogenic effects





Autopsy findings: Seeds in stomach & non specific features

Circumstances of Divi Kaduru Poisoning

- Accidental poisoning: Children
- Suicidal poisoning

- Symptoms are similar to those caused by Daturu sp.

GODA KADURU (BITTER NUT) ගොඩ කදුරු

Botanical Name: Strychnos nux-vomica

Most poisonous:

Seeds

Bark

Leaves

(Although all parts are toxic)

Fatal dose: 1-2 seeds

Toxin: Alkaloids

- Strychnine
- Brucine
- Loganin

CIRCUMSTANCES OF GODA KADURU POISONING

- Strychnine injections are used to kill stray dogs/ seeds to kill animals, in pesticides/rodenticides (to kill rats)
- Suicidal
- Accidental : With consumption of herbal and native medicinal preparations
- Homicidal : very rare (mixed with alcohol)

STRYCHNINE POISONING SIGNS & SYMPTOMS

- Bitter taste
- Clonic (intermittent) & Tonic (sustained) seizers
- Affects flexor and extensor muscles simultaneously
- Facial Muscles: “risus sardonicus”, “trismus” (locked jaw)
- Opisthotornus
- Respiratory distress and death
- Cardiovascular manifestations: Tachycardia, hypertension, feeble pulse
- Agonizing death due to retained consciousness during spasms
- Severe poisoning: Lactic acidosis, hyperthermia, rhabdomyolysis

Autopsy findings: No macroscopic or microscopic features (non specific features),
seeds / powder in stomach, poison in blood & urine

- Strychnine primarily acts on the central nervous system as a competitive antagonist on the postsynaptic glycine receptors.
- The inhibitory effect of the spinal interneurons on the muscles is lost and this in turn leads to twitching, muscle spasms.

- Clinical features of toxicity may develop as early as
  - 5 minutes after inhalation,
  - 30 minutes after ingestion and
  - upto 24 hours after transdermal exposure.

Management:

- Mainly supportive
  - Initial stabilization with oxygen.
  - Avoidance of all extraneous stimuli, control of muscle spasm with benzodiazepines and barbiturates, and airway management including endotracheal intubation.
  - Intravenous fluids should be administered to maintain a brisk urine output.

CASE 01 - *Strychnos nux-vomica* poisoning

A 22-year-old male with no premorbidities, presented with severe myalgia and backache of acute onset. He also complained of one episode of backward arching of the entire body, consistent with opisthotonus.

On repeated questioning, he admitted to having consumed approximately 25 leaves of *Strychnos nuxvomica*) three hours prior to presentation with suicidal intent. Unconsumed leaves from the same plant were later produced for identification and confirmed to be those of the Strychnos nux-vomica plant.

General physical and systemic examinations were unremarkable except for severe thigh and calf muscle tenderness.

Routine laboratory parameters showed elevated serum creatine kinase levels (2 189 U/L), with normal renal function tests.

He was treated conservatively with intravenous diazepam and analgesics.

Except for one episode of transient opisthotonus immediately after presentation, he made a rapid recovery and was discharged.

Dasari S, Naha K. A rare case of strychnine poisoning by consumption of *Strychnos nux-vomica* leaves. *Asian Pac J Trop Biomed.* 2011;1:S303–4.

CASE 02- *Strychnos nux-vomica* poisoning

A 39-year-old man with no previous known comorbidities presented with acute onset myalgia and generalized body ache. He gave a history of consuming six leaves of the local "*etti*" plant after making it into a decoction with 100 ml of boiling water, with suicidal intent. Later, the plant was confirmed to be *Strychnine nux-vomica* by visualization

Following consumption, he had induced a few episodes of small volume nonbilous vomiting.

On presentation to the Emergency Department, he was apprehensive and restless with pulse rate of 60/min, respiratory rate of 16 and blood pressure of 100/80 mmHg. The rest of the systemic examination was within normal limits except for moderate muscle tenderness over the calf and thighs. Within hours of consumption, he began to develop twitching movements of the arm and forearm muscles that were more pronounced in his sleep. He had normal blood counts and metabolic panel.

While under observation, his heart rate dropped to 48 beats/min without any associated hypotension. Urgent electrocardiogram (ECG) showed PR interval prolongation (210 ms) with left anterior fascicular heart block. He was kept on maintenance intravenous fluids, and urine output was closely monitored. He was started on tablet lorazepam for the muscle twitches and injection atropine as and when required for bradycardia <50 beats/min.

The muscle twitches and hypervigilance reduced in severity and he was discharged on day 4.

Ponraj L, Mishra AK, Koshy M, Carey RAB. A rare case report of *Strychnos nux-vomica* poisoning with bradycardia. *J Family Med Prim Care*. 2017;6(3):663-665. doi:10.4103/2249-4863.222036

Diya Kaduru/Gon Kaduru/Wel Kaduru (Sea Mango)

Botanical Name: *Cerbera manghas*




Most poisonous:
Fruit Kernel

Fatal dose: 2 fruits

Toxin: Glycocides

- Cerberin
- Odollum
- Thevetin

- Early features:
  - Burning sensation and dryness of the mouth
  - Nausea, vomiting, diarrhea and abdominal pain
- Cardiac manifestations:
  - Bradycardia
  - Hypotension
  - Hyperkalaemia
  - 1st and 2nd degree heart block
  - Atrial and ventricular extrasystoles
  - Ventricular fibrillation
- Convulsions and coma

Autopsy findings: Non specific features, Kernal or the fruits in the stomach

Circumstances of Poisoning

- Suicidal : Common in Eastern province
- Accidental/Homicidal : Very rare

DIYA KADURU POISONING SIGNS & SYMPTOMS

- The seed's primary toxic ingredient is cerberin, which causes disruption of cardiac electrical activity and hyperkalemia by inhibiting the Na-K-ATPase exchanger in myocardial cells.

Management:

- Similar to that in patients with digoxin poisoning;
  - Gastric decontamination.
  - Supportive treatments such as atropine, cardiac pacing, and antiarrhythmic agents.
  - Life-threatening hyperkalemia is treated by intravenous glucose, insulin, and sodium bicarbonate and oral use of potassium-binding resins.

CASE 01 - *Cerbera manghas* poisoning

A 30 year old man presented to Batticaloa Teaching Hospital 4 hours after ingesting 6 natchchukkai seeds according to his family.

He had presented to a primary rural hospital 1.5 hrs after the ingestion where he had a pulse of 40 beats/ minute, a blood pressure of 100/60 mm Hg, and was noted to be drowsy. He was given a bolus dose and infusion of atropine (total 15 mg) and transferred to Batticaloa. On arrival his pulse was 74 beats/minute, BP 120/50 mm Hg, and had reduced consciousness, responding to pain (P on the AVPU scale).

A gastric lavage was done in case the seeds had not passed out of the stomach and atropine was administered. He rapidly became bradycardic and was transferred to intensive care where he was given isoprenaline. He thereafter developed 3rd degree AV block and ventricular fibrillation, before dying 80 minutes after admission. Blood biochemistry assays were not available out of hours and blood samples were therefore not sent before he died.

His family brought some natchchukkai fruit to the hospital. They were identified as *C. manghas* fruit from National Poisons Information Centre literature; this identification was subsequently supported by review of photographs by staff of the Royal Botanical Gardens, Kew.

Eddleston, Michael; Haggalla, Sapumal (2008). Fatal injury in Eastern Sri Lanka, with special reference to cardenolide self-poisoning with Cerbera manghas fruits. Clinical Toxicology, 46(8), 745–748. doi:10.1080/15563650701668617

CASE 02 - *Cerbera manghas* poisoning

A 50-year-old man experienced numbness of the face, throat, and upper extremities after ingesting three seeds of *Cerbera manghas* in a suicidal attempt. Nausea and vomiting ensued one hour later, followed by light-headedness, chest tightness, palpitation, and dyspnea. He presented to the emergency department two hours post-ingestion.

On arrival, his heart rate was 90 /minute and blood pressure was 142/72 mmHg. Examination ten minutes later revealed a slow, regular heart rate of 22 /minute, which was a complete atrioventricular block. Other physical examination and routine laboratory data were unremarkable except serum potassium of 5.4 mmol/L; serum digoxin concentration was 1.03 ng/ml. The patient was placed on oxygen, intravenous fluids, and other supportive measures.

Cont.

CASE 02 – *Cont.*

Post-ingestion 20 hours, his BP was 132/76 mmHg and a follow-up ECG revealed 1st degree AV block. Serum potassium level -5.9 mmol/L and serum digoxin concentration- 0.65 ng/ml. Hyperkalaemia was treated accordingly. Single-lead rhythm strip showed sinus bradycardia after the above treatment.

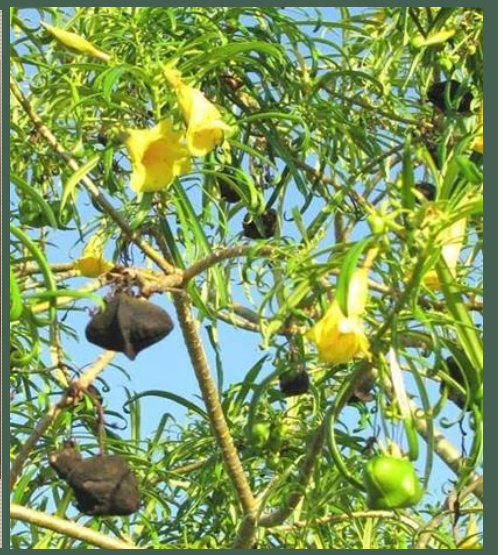
Post-ingestion 26 hours, his clinical condition remained stable, yet serum potassium level increased to 6.5 mmol/L. Glucose, insulin, sodium bicarbonate, and sodium polystyrene sulfonate were administered, which ameliorated both hyperkalemia and the clinical manifestations. Repeat ECG showed normal sinus rhythm at a rate of 60 beats/minute.

Serum digoxin concentration was undetectable on day four and the patient was discharged without sequellae.

Yi-Cheng Tsai, Chun-Yu Chen, Ning-I Yang & Chen-Chang Yang (2008) Cardiac glycoside poisoning following suicidal ingestion of Cerbera manghas, Clinical Toxicology, 46:4, 340-341, DOI: 10.1080/15563650701291766

KANERU (YELLOW OLEANDER) කනේරු

Botanical Name: Thevetia peruviana



Most poisonous:

Fruit

Seed

All parts of tree

Fatal dose: 2 seeds

Toxin: Cardiac Glycocides

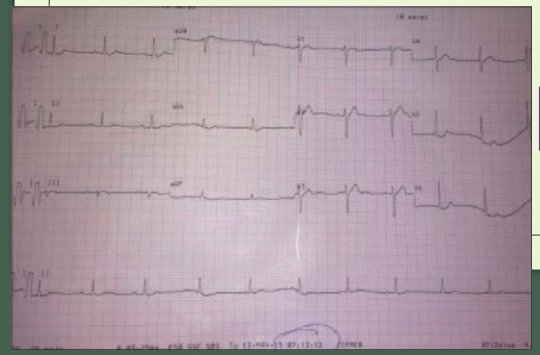
- Thevetin A
- Thevetin B

CIRCUMSTANCES OF KANERU POISONING

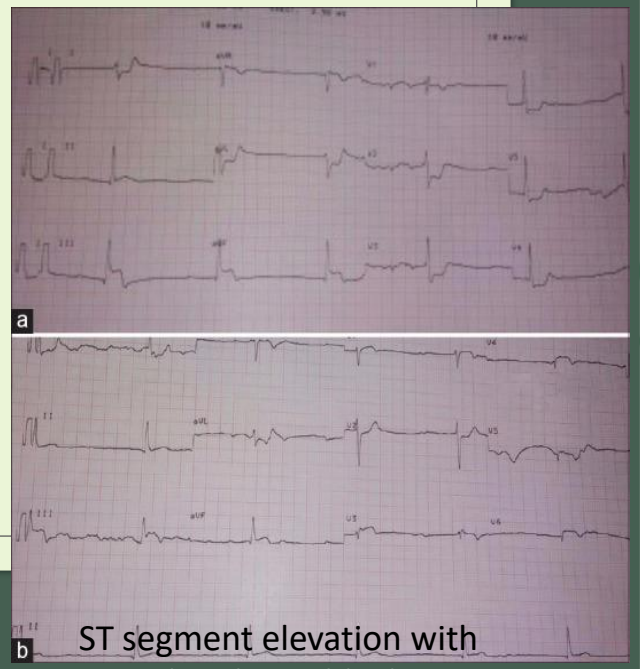
- Suicidal poisoning is very common.
- Now common in : Anuradhapura, Pollonaruwa and Kurunegala Districts.
- Accidental : Children and adults
- Homicidal : Very rare

YELLOW OLEANDER POISONING SIGNS & SYMPTOMS

- General symptoms and signs;
  - Nausea, vomiting, diarrhea, abdominal pain and tenderness
  - Restlessness
- Cardiac manifestations: Cardiac toxicity
  - Hypotension
  - Cardiac dysarrhythmias
  - Sick sinus syndrome
  - Varying degrees of heart block
  - Atrial flutter/fibrillation
  - Bidirectional ventricular tachycardia
  - Ventricular fibrillation



Normal sinus rhythm



ST segment elevation with Complete heart block

- Causes disruption of cardiac electrical activity by inhibiting the Na-K-ATPase exchanger in myocardial cells.

Management:

- Initial resuscitation.
- Assessment of severity of toxicity.
- Continuous hemodynamic and cardiac monitoring.
- Measurement of serum creatinine; electrolytes such as sodium, potassium, calcium, magnesium, and serum cardiac glycoside levels.
- Gastric decontamination is still practiced.
- Digoxin-specific Fab fragments are an effective treatment of acute intoxication.
- Supportive treatments such as atropine, cardiac pacing, and antiarrhythmic agents.

CASE 01 – *Thevetia peruviana* poisoning

A 37-year-old man with no known comorbidities presented to Emergency Department with alleged history of consumption of yellow oleander seed. He had consumed a single crushed yellow oleander seed mixed with water. Initially, he was asymptomatic for 4 h, following which, he developed vomiting and dizziness.

He presented 16 h after ingestion. On arrival, he was conscious, oriented, with a pulse rate of 63 beats/min, blood pressure of 140/80 mmHg, and SpO_2 of 98% at room air. Initial ECG showed normal sinus rhythm. He was kept under observation with continuous cardiac monitoring.

After 2 h of observation, he developed profound lightheadedness and vomiting with a PR of 40/min and BP of 60/40 mmHg. With IV crystalloids resuscitation, BP improved to 90/60 mm of Hg. Repeat ECG showed ST-segment elevation in leads II, III, aVF, V5R, V6R with complete heart block. The patient was immediately transferred to cardiac cath laboratory for primary coronary intervention (PCI). Since the patient had hemodynamically unstable bradycardia, temporary pacemaker implantation (TPI) was done before angiography. His angiography revealed single vessel disease involving the right coronary artery (RCA) (total occlusion of mid-RCA due to thrombus); hence PCI to RCA was performed. Post procedure, patient's cardiac rhythm improved and TPI was removed. His ECG on day 2 did not show any regional wall motion abnormality. Rest of the in-hospital course was uneventful. He was discharged on day 5 and advised to follow-up after a month.

Anandhi D, Prakash Raju KNJ, Basha MH, Pandit VR. Acute myocardial infarction in yellow oleander poisoning. *J Postgrad Med*. 2018;64(2):123-126. doi:10.4103/jpgm.JPGM_141_17

CASE 02 – *Thevetia peruviana* poisoning

A 17-year-old previously healthy school girl was transferred from a local hospital for further management of yellow oleander poisoning. She had ingested four Kaneru seeds and was admitted to the local hospital around one hour after the ingestion. She found the plant from her neighbouring home garden.

She was given gastric lavage and a single dose of activated charcoal at the local hospital. On admission to the medical unit she had no complaints; BP- 110/70 mmHg; PR- 94 beats/min. Bilateral lung fields were clear. Other examination findings showed no abnormality. On admission ECG showed T wave inversions on Lead II, III, aVF and V1-V6.

She was immediately given multiple dose activated charcoal and continuous cardiac monitoring was carried out with serial ECGs. But troponin I or 2D-echo could not be done at this stage.

Following day, she was clinically normal and her serum K+ level was 3.7mmol/L and Na+ level was 137mmol/L. But same ECG changes were present throughout the hospital stay. She was discharged on the 4th day of the ingestion after having psychiatric assessment and closely followed up ECGs with cardiac assessment.

Two months after the ingestion, her ECG showed disappearance of T inversions of Lead II, V5 and V6. However, her ECG was completely normal only after about four months of the poisoning.

Rathnayaka, R.M.M.K.N. and Ranathunga, P.E.A.N., 2018. Extensive T wave inversions on electrocardiogram following *Kaneru* (*Thevetia peruviana*) poisoning. *Sri Lankan Journal of Anaesthesiology*, 26(2), pp.147–149.

KANERU (LORIER BOL/ROSEBAY/ROSE LAUREL) කනේරු

Botanical Name: *Nerium oleander*




Most poisonous:

Pods

Seed

All parts of tree

Toxin: Cardiac Glycocides

- Oleandrin
- Oleandrigenin
- Neriine

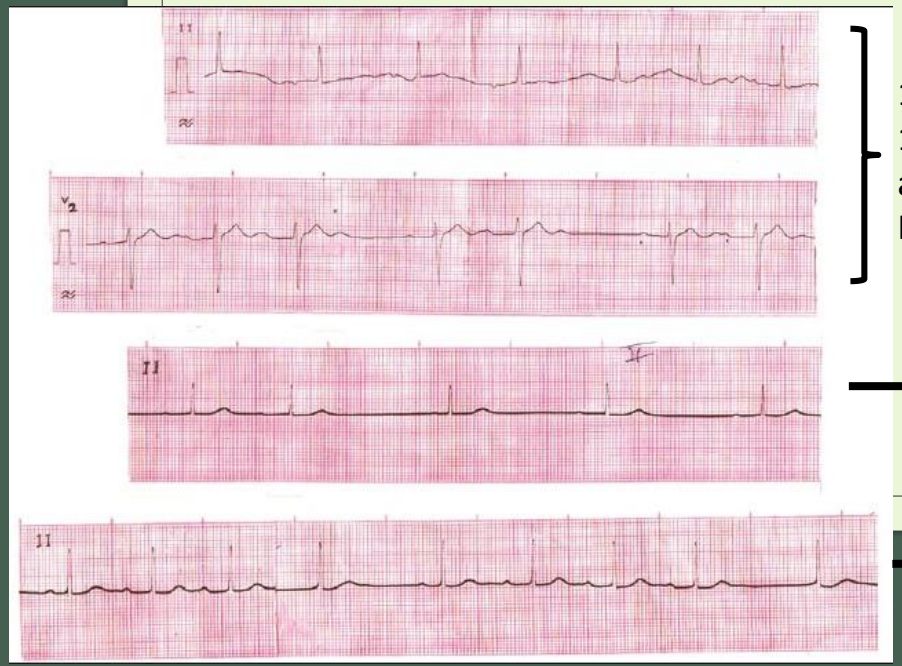
CIRCUMSTANCES OF KANERU POISONING

- Suicidal poisoning is very common.
- Accidental : Children

  Adults- when used in medicinal preparations
- Homicidal : Very rare

KANERU POISONING SIGNS & SYMPTOMS

- General symptoms and signs;
  - Nausea, vomiting, diarrhea, abdominal pain and tenderness
  - Restlessness
- Cardiac manifestations: Cardiac toxicity
  - Hypotension
  - Hyperkalaemia
  - Cardiac dysarrhythmias
  - Varying degrees of heart block



15 hours after the ingestion;
1st degree AV block with inverted P wave and prolonged PR interval and,
In lead V2 intermittent 2:1 AV block

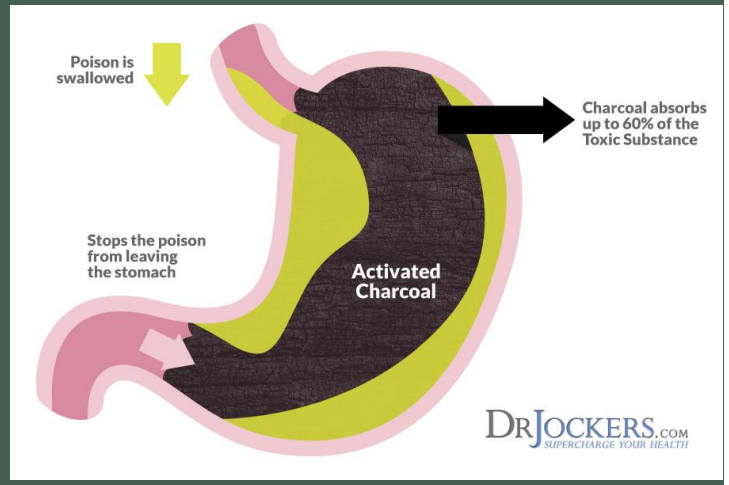
→ After IV atropine administration;
First degree AV block in lead II

After IV atropine administration;
→ First degree AV block Type I Second-Degree AV block with atypical wenckebach periodicity with junctional rhythm in lead II

- Causes disruption of cardiac electrical activity by inhibiting the Na-K-ATPase exchanger in myocardial cells.

Management:

- Initial resuscitation.
- Continuous hemodynamic and cardiac monitoring.
- Measurement of serum creatinine, electrolytes and serum cardiac glycoside levels.
- Gastric decontamination with activated charcoal is still practiced to disrupt the enterohepatic circulation.



- Digoxin-specific Fab fragments are an effective treatment of acute intoxication.
- Supportive treatments such as atropine, isoproterenol, cardiac pacing, and antiarrhythmic agents.

CASE 01 – *Nerium oleander* poisoning

A 18-year-old female presented to the emergency room with nausea, vomiting, lightheadedness, and abdominal pain 8 hours after drinking a cup of oleander tea. She had been advised to prepare oleander tea as part of weight loss regime.

On presentation, her blood pressure was 90/50 mmHg in the right arm and 85/50 mmHg in the left arm, with an irregular pulse of 40/min. The lungs were clear. A 12-lead electrocardiogram (ECG) on admission showed Mobitz type II atrioventricular block with right bundle branch block (RBBB), left anterior hemiblock, and T-wave inversions in leads V4 through V6. Transthoracic echocardiography showed a left ventricular ejection fraction (LVEF) of 65%, with mild tricuspid regurgitation, and no evidence of pulmonary hypertension or atrial septal defect. Liver, renal, thyroid function tests were in normal range, and serum digoxin level was 4.1 ng/mL at admission.

A temporary transvenous pacemaker was inserted in order to improve hemodynamic status. IV atropine sulfate was failed to resolve the bradycardia. She was treated with digoxin-specific Fab antibody fragments 3 hours after admission. After 15 minutes of infusion, a 12-lead ECG showed first degree heart block, and a serum digoxin level of 2.08 ng/mL. A 12- lead ECG revealed sinus rhythm with RBBB at the end of infusion. Serum digoxin level was 0.64 ng/mL 4 hours after infusion. After 72 hours, the patient was discharged without any symptoms.

Tatlısu MA, Çekirdekçi Eİ, Akyüz Ş, Nurkalem Z. A case of Mobitz type II atrioventricular block due to Nerium oleander poisoning successfully managed with digoxin-specific Fab antibody fragments. Turk Kardiyol Dern Ars. 2015 Oct;43(7):648-50. doi: 10.5543/tkda.2015.65848. PMID: 26536992.

CASE 02 – *Nerium oleander* poisoning

A 71 year old white male, who had worked as a laboratory technician in the past, was found dead at home. During the scene survey, a steel pan medium size, closed with its cover and sealed with packaging tape, was found near the body. A small piece of white sellotape was on the cover, with the following hand-written note on it: "*Poison. Wash pan and funnel carefully or throw everything away*". Elongated dark green leaves weighing 256.8 g, along with small pieces of stem and a plastic funnel were found within the pan. A second steel pan, smaller than the first one, and a plastic bottle were also present at the scene, containing respectively 400 mL and 100 mL of a yellow fluid infusion. On the bottle, a small piece of white sellotape was present with a second hand-written note: "*Poison*". An empty glass was found close to the bottle, both of which were on the bedside table close to the dead body.

No relevant autopsy findings were observed except for multi-organ congestion, which is common in fatal poisoning. Visceral congestion was observed in the liver, brain, and lungs. The lungs also showed some histologically evident hemorrhagic pulmonary edema. No other findings of injury or disease were observed. A few milliliters of brown fluid were present in the stomach.

Cont.

CASE 02 – *Cont.*

Samples of peripheral blood, vitreous humor, urine, liver, and gastric content were collected for toxicological analysis. In all samples, toxic levels of oleandrin were detected. Qualitative results for oleandrigenin, neritaloside, and odoroside were obtained. Oleandrigenin was present in all tissue samples whereas neritaloside and odoroside were absent in the blood and vitreous humor but present in urine, liver, gastric content, and in the leaf brew.

Items found at the death scene: (a) the steel pan containing 256.8g of dark green leaves, small pieces of stem, and a plastic funnel; (b) the second smaller steel pan containing 400mL of yellow fluid infusion

Carfora, A., Petrella, R., Borriello, R. *et al.* Fatal poisoning by ingestion of a self-prepared oleander leaf infusion. *Forensic Sci Med Pathol* **17,** 120–125 (2021). https://doi.org/10.1007/s12024-020-00338-w

NIYAGALA (GLORY LILY) නියඟලා

Botanical Name: *Gloriosa superba*

Seeds

Tubers

Flowers

Most poisonous:

Tubers

All parts of plant

Fatal dose: 10g of tubers

Toxin: Alkaloid

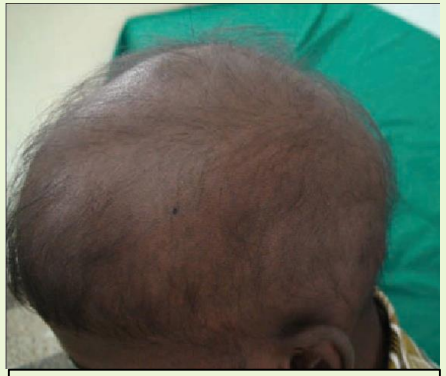
- Colchicine
- Gloriosine
- Superbine

CIRCUMSTANCES OF NIYAGALA POISONING

- Commonly used in medical treatments;
  - Native practitioners use tubers for the treatment of joint pains, bruises, sprains, colic, chronic ulcers, haemorrhoids, cancer, impotence, nocturnal seminal emissions, leprosy, inducing labour pains and abortion.
  - Used in rheumatological and immunological conditions in modern western medicine.
- Accidental poisoning : common (Mistaken as sweet potato/ Hulankeeriya etc.)
- Suicidal poisoning : very common.
- Homicidal : Very rare

- Severe gastroenteritis – Nausea/vomiting/severe diarrhea/Abdominal pain and tenderness
- Dehydration/Hypotension/shock
- Heptotoxicity/Renal toxicity/cardiac toxicity
- Adult respiratory distress syndrome (ARDS)/respiratory failure
- Toxic encephalopathy/ progressive paralysis CNS and PNS
- Multiple organ failure

- Electrolyte imbalance
- Metabolic acidosis
- Hypocalcemia
- Pancytopenia /thrombocytopenia
- Clotting defects/bleeding
- Rhabdomyolysis
- Generalized massive alopecia



Massive alopecia due to Glorisa Superba poisoning

Autopsy findings: Hemorrhagic features in stomach, intestines, heart, lung, kidney etc.

NIYAGALA POISONING SIGNS & SYMPTOMS

- Colchicine inhibits the polymerization of microtubules and formation of mitotic spindle in cell division.
- Therefore, the rapidly dividing cells of the intestinal mucosa are severely affected.

- Typical toxicity follows 3 sequential phases;

PHASE 01
0 – 24 hours
After consumption

- Gastrointestinal symptoms predominates

PHASE 02
2 – 7 days

- Multiple organ system dysfunction
- Death can also occur

PHASE 03
1 – 3 weeks

- Recovery
- Significant alopecia

Management:

- Mainly supportive
  - Symptomatic treatment with intravenous fluid.
  - Continuous hemodynamic and cardiac monitoring.
  - Gastric lavage.

CASE 01 – *Glorisa superba* poisoning

A 27-year-old man was admitted to the General Hospital Chilaw, Sri Lanka, with acute onset severe epigastric pain and vomiting. There was no history suggestive of food poisoning or the consumption of poison.

A few hours before admission, the patient had consumed boiled coriander tea, which is a traditional medicine in Sri Lanka, prepared for him by his sister-in-law as a treatment for common cold. Symptoms developed about 2 h after drinking the coriander tea and he was rushed to the hospital. When other family members examined the contents of the teapot, they noticed, in addition to the coriander, the tea cup contained different seeds, which they identified as Gloriosa seeds. Gloriosa seeds were known to be present in the house because the patient worked on a farm that cultivated Gloriosa, and occasionally brought the seeds home. Seeking confirmation, the wife of the victim tasted a small amount of the remaining tea and also developed nausea and vomiting.

On admission to the hospital, the patient was having profuse watery diarrhea, burning abdominal pain, reduced urine output, and dysuria. On examination, he was febrile with BP- 100/70 mmHg, PR- 100 bpm, and diffusely tender abdomen.

After gastric lavage, the patient was started symptomatic treatment.

Cont.

Boiled seeds from the pot

Gloriosa seeds from patients pocket

CASE 01 – *Cont.*

On day two after poisoning, he complained of pleuritic-type chest pain. His RR was 40/min with bilateral diffuse crepitation. Blood gas analysis revealed hypoxia with respiratory alkalosis and he was transferred to the intensive care unit (ICU) for intubation and ventilation. Chest X-rays showed mild diffuse inflammatory shadows, and blood counts revealed a neutrophilic and lmphocytic leukocytosis, CRP-96 and ESR-60 mm/1st hour.

On day three after admission, the patient had thrombocytopenia with normal white blood cell count, mild renal impairment. ECG and 2D echocardiogram were normal. In addition to ventilator support, he was treated with broad-spectrum antibiotics (cefotaxime and metronidazole), parenteral nutrition, proton pump inhibitors, and intravenous fluids. The patient required ventilator support for 3 days and required high-dependency care for a further 6 days. His total hospital stay was 15 days. He also developed generalized alopecia by day 10 of hospital admission. By that time, the patient had fully recovered from acute toxicity and was hemodynamically stable.

Because there was reasonable suspicion of homicidal poisoning, the Judicial Medical Consultant and the hospital police post were informed to initiate medico legal investigation. The patient also admitted that he had taken Gloriosa seeds from his workplace where Gloriosa plants were grown for harvest and export of seeds. Toxicological analysis by the government analyst also confirmed the presence of colchicine in the gastric lavage samples.

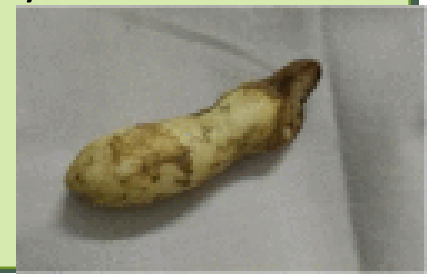
Kande Vidanalage CJ, Ekanayeka R, Wijewardane DK. Case report: a rare case of attempted homicide with Gloriosa superba seeds. *BMC Pharmacol Toxicol*. 2016;17(1):26. Published 2016 Jun 21. doi:10.1186/s40360-016-0069-6

CASE 02 – *Glorisa superba* poisoning

An 18- year-old woman presented to the emergency department with an alleged history of consumption of G. superba tubers. She presented with generalized abdominal pain associated with multiple episodes of non-bilious vomiting and loose stools. She was referred from a peripheral clinic following first aid, which included gastric lavage and fluid resuscitation. On clinical examination, she was found to be pale, tachycardiac (110 beats/min) with signs of moderate dehydration. Physical examination revealed abdominal tenderness with the rest of the systemic examination being within normal limits.

In the first 12hours of hospital admission, she developed progressive oliguria and respiratory distress requiring intensive care and non-invasive ventilation. Laboratory investigations revealed a haemoglobin level of 110g/L with leukocytosis and thrombocytopaenia, mild elevation in coagulation parameters, elevated cardiac enzymes, and rhabdomyolysis with creatine phosphokinase levels of 30 860/µL, which increased to 56 960/µL over a period of 12hours. She had acute oliguric kidney injury, hypoalbuminemia with transaminitis and metabolic acidosis. Chest radiography showed features of acute respiratory distress syndrome.

Cont.



CASE 02 – *Cont.*

She was admitted in the medical intensive care unit for ventilatory and haemodynamic supports. She was initiated on non-invasive ventilation, and was subsequently initiated on mechanical ventilation. She continued to be anuric, hence was maintained on haemodialysis. She received blood products for disseminated intravascular coagulation and a session of plasmapheresis. She was initiated on intralipid rescue therapy at 100mL/ day for two consecutive days. She developed pancytopenia secondary to bone marrow aplasia for which was unresponsive to granulocyte colony stimulating factor.

She continued to deteriorate, and despite all measures she progressed to develop cardiovascular collapse with refractory hypotension and succumbed to the death in 8 days.

Gunasekaran K, Mathew DE, Sudarsan TI, Iyyadurai R. Fatal colchicine intoxication by ingestion of *Gloriosa superba* tubers. BMJ Case Rep. 2019 May 16;12(5):e228718. doi: 10.1136/bcr-2018-228718. PMID: 31101749; PMCID: PMC6536255.

WETA ENDERU

(BARBADOS NUT/ PURGING NUT/ PHYSIC NUT) වැට එඩරු

Botanical Name: *Jatropha curcas*

Most poisonous:

Seeds

Fatal dose: 2 seeds ca

Toxin:

- Toxalbumin (Curcin)

WETA ENDERU POISONING SIGNS & SYMPTOMS

- Mainly Gastrointestinal symptoms;
  - Nausea, vomiting, diarrhoea
  - Epigastric and abdominal pain
  - Dehydration, electrolyte imbalance, cramps and shock
- Other features of poisoning;
  - Hypoglycaemia,
  - Retinal haemorrhages
  - Haematuria
  - Convulsions, and shock
  - CNS and cardiovascular depression
- Hepatic necrosis and acute renal failure have been reported

CIRCUMSTANCES OF WETA ENDERU POISONING

- The seeds are used in traditional medicine as; abortificients, anthelmintics and purgatives.
- Trees are now grown commercially to obtain bio- diesel.
- Poisoning is mainly seen in children as accidental.

CASE 01 – *Jatropha curcas* poisoning

Four children aged between 2 and 6 years were playing close to an abandoned yard where the plant grew. A fruit-bearing branch of J. curcas was hanging over the fence and the children collected the fruit and ate the seeds.

Shortly after ingesting the fruit, they developed abdominal pain and were rushed to hospital. The lag time before the onset of gastrointestinal (GI) complications varied between 90 and 120 minutes. The GI symptoms that developed included abdominal pain, nausea, vomiting and diarrhoea, as well as a burning sensation in the throat.

The patients were treated with intravenous hydration and anti emetic medication and the symptoms resolved within 12 - 14 hours. Three of the 4 children were observed for 24 hours and discharged the following day. The 2-year-old child was hospitalised for 7 days owing to prolonged diarrhoea.

Moshobane, M C; Wium, C; Mokgola, L V (2017). *Acute poisoning in children from Jatropha curcas seeds. South African Journal of Child Health, 11(3), 149–.* doi:10.7196/SAJCH.2017.v11i3.1401

CIRCUMSTANCES OF BEHETH ENDERU POISONING

- Commonly used in traditional medical treatments;
  - Oil is used for medical purposes: laxative, treatment of infection and inflammation, in treatment of cancer.
- Industrial raw material for lubricants, paints, coats, cosmetic products.

- Press cake of the seeds after detoxification used as additive in organic fertilizer, or animal feed (animal poisonings: dog/ horse/ sheep/cattle).
- Accidental ingestion of seeds by children.
- Occasional suicide ingestion of seed extracts /oil (oral/ parental/ IM).
- As a weapon of bio-terrorism (aerosol/ IM).

BEHETH ENDARU/ THEL ENDARU

(CASTOR PLANT) බෙහෙත් එඩරු

Botanical Name: *Ricinus communis*

Most poisonous:

Seeds

Plant

Fatal dose: 1 seeds

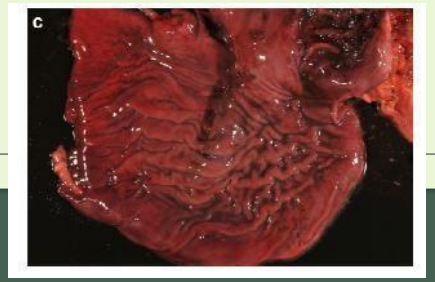
Toxin:

- Alkaloid Ricinin
- Toxalbumin Ricin

BEHETH ENDERU POISONING SIGNS & SYMPTOMS

- Symptoms seen after 3 to 20 hours.
- Abdominal pain
- Emesis and diarrhea with or without blood
- Muscular pain
- Cramps in the limbs
- Circulatory collapse
- Dyspnoea
- Dehydration
- Late features: liver and kidney dysfunction
- Injected ricin: pain at the injection site, muscular necrotizing

Autopsy findings: Haemorrhagic necrosis in intestines and heart, oedema in lungs



- Toxicity results from the inhibition of protein synthesis.

CASE 01 – *Ricinus communis* poisoning

A 51 year-old Omani man, neither diabetic nor hypertensive and not on any regular medication, was brought to the Accident & Emergency Department of Nizwa Hospital, Oman, by his son. He was in a confused state after ingesting three hours previously one green fruit of castor bean, as stated by the son. He had vomited once at home. There were no other complaints.

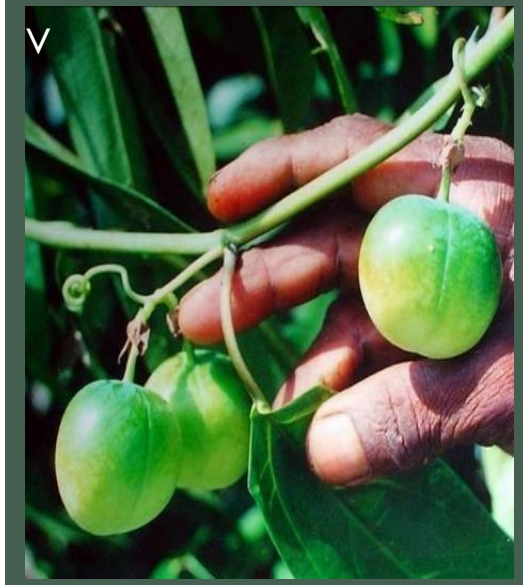
On examination the patient was confused, disoriented as to time and place, and was afebrile. His pulse was 108/min regular, BP was 110/68 mmHg and the RR was 22/min. Examinations of the chest, cardiovascular system and abdomen were unremarkable. He had dryness of mouth and the pupils were bilaterally dilated with sluggish reaction to light. The patient was admitted to the medical ward and treated symptomatically and with activated charcoal.

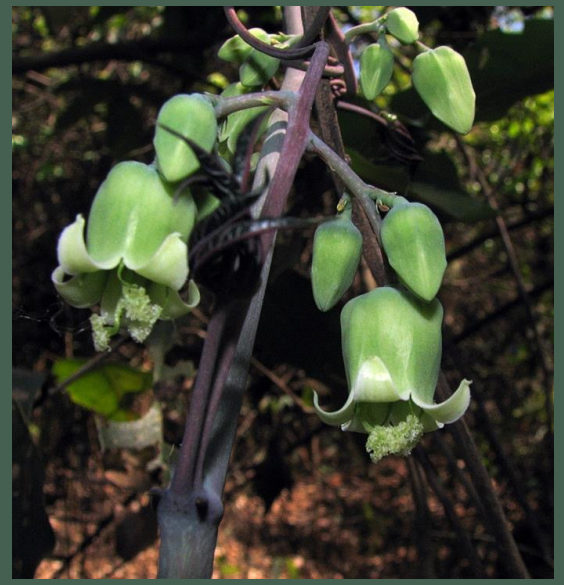
His initial laboratory results showed normal results for renal function tests, electrolytes, liver function tests, coagulation profile, cardiac enzymes, full haemogram and routine urine tests. An initial ECG showed normal sinus rhythm with T wave inversion in leads II, III and AVF. A repeat ECG after 6 hours showed sinus bradycardia with T inversion in inferior leads as well. CT of the brain was normal. He stayed in hospital for 2 days which were uneventful. His pulse rate returned gradually to normal and his conscious level improved and he was discharged in a good condition with the advice to evaluate his cardiac status with anECG and treadmill test at a later date.

Al-Tamimi FA, Hegazi AE. A case of castor bean poisoning. *Sultan Qaboos Univ Med J*. 2008;8(1):83-87.

HONDALA හොඬල

Botanical Name: *Adenia palmata*





Most poisonous:
Fruits

Toxin:

- Cyanogenic glycoside
- Toxalbumin

HONDALA POISONING

SIGNS & SYMPTOMS

- Clinical manifestations occur in 3 phases;

PHASE 01

- Vomiting
- Fever
- Restlessness
- Dizziness
- Disorientation
- Abdominal pain
- Diarrhoea

PHASE 02

- Necrotising enteritis leading to diarrhoea with blood and mucus
- Abdominal colic
- Abdominal tenderness over the right iliac fossa

PHASE 03

2 – 3 weeks after ingestion

- Myocarditis
- Tender hepatomegaly
- Retinopathy

CIRCUMSTANCES OF HONDALA POISONING

- The fruit closely resembles to passion fruit.
- Poisoning is mainly seen in children as accidental.

OLINDA

(CRAB EYES/ROSARY BEAN/JEQUIRITY BEAN) ඔලිඳ

Botanical Name: *Abrus precatorius*




Most poisonous:
Seeds

Fatal dose: 1 seed

Toxin:

- Toxalbumin Abrin

OLINDA POISONING SIGNS & SYMPTOMS

- Nausea, vomiting, diarrhea, haematemesis and melaena
- Abdominal cramps
- Hepatotoxicity
- Haemaglutination
- Haemolysis of red cells
- Hypovolaemic shock and direct toxic effect on kidneys lead to acute renal failure
- Retinal haemorrhages
- Anaemia
- Hypoglycaemia
- Drowsiness and convulsions

Circumstances of Olinda Poisoning

- Suicidal poisoning
- Accidental poisoning: Children

- Toxicity results from the inhibition of protein synthesis causing cell death.

CASE 01 – *Abrus precatorius* poisoning

A 17 year-old woman presented to our hospital with history of eating ten crushed seeds of *Abrus precatorius* following a family quarrel. Within 4 hours of consuming the seeds, she developed multiple episodes of vomiting and presented to our emergency department.

Her vital signs were stable with a pulse rate 100 beats/ minute and blood pressure 110/70 mm Hg. Physical examination was unremarkable. Within 6 hours of consuming the seeds she was treated with gastric lavage followed by activated charcoal, and intravenous hydration. Gastric lavage showed crushed remnants of the seeds that were identified by their distinctive red outer covering. Further toxicological analysis of the gastric contents was not done.

In hospital she developed loose watery stools on day 2, six to eight episodes per day, associated with epigastric pain and tenderness that continued until day 3. On day 4 her diarrhea turned bloody and the abdominal pain worsened. Hydration was maintained and adjusted to maintain a normal central venous pressure. Intravenous proton pump inhibitors were added to the treatment regimen.

Cont.

CASE 01 – *Cont.*

She appeared to doing well except for diarrhea, when on day 6 she developed altered sensorium in the form of drowsiness and decreased speech output. There was no fever, headache, vomiting, seizures, or ear discharge. Her vitals were stable and she was maintaining adequate hydration.

Central nervous system examination revealed grade 2 sensorium with Glasgow coma score of 10. Both pupils were 3 mm and reactive to light. She was moving her limbs in response to pain and deep tendon reflexes were exaggerated with bilateral extensor plantar response. Detailed motor and sensory examination could not be carried out in view of altered sensorium. There was no neck stiffness.

Her biochemical parameters were normal. Liver enzymes, bilirubin, and prothrombin time were normal. An ophthalmologic examination at this point revealed bilateral established papilledema. CT of the brain showed diffuse cerebral edema and features suggestive of raised ICP. Treatment consisted of mannitol, dexamethasone and acetazolamide to decrease cerebral edema. Her sensorium remained poor with bowel and bladder incontinence and impaired speech output.

Cont.

CASE 01 – *Cont.*

The biochemical parameters that had remained stable during the first week of hospital stay started to rise during this time. She developed renal failure, hyponatremia, and hypokalemia over a one-week period. She was maintaining a normal urine output and central venous pressure. Ultrasound revealed enlarged kidneys with increased cortical echoes and preserved corticomedullary differentiation. Her hyponatremia and hypokalemia responded to conservative measures and her serum urea and creatinine returned to baseline (without dialysis) over a 10-day period.

By the end of the second week her sensorium started improving and became normal by the third week of hospital stay. Repeat fundus examination showed resolving papilledema. Mannitol and dexamethasone was tapered gradually and she was discharged after a month-long hospital stay.

Follow-up visit one month later showed no evidence of renal or CNS sequelae.

Dharanipragada Subrahmanyan, Jomal Mathew Dks & Mithun Raj (2008) An unusual manifestation of Abrus precatorius poisoning: A report of two cases, Clinical Toxicology, 46:2, 173-175, DOI: 10.1080/15563650601185134.

CASE 02 – *Abrus precatorius* poisoning

A 16-year-old girl (perfectly healthy before, 40 kg body weight) was admitted to the department of gastroenterology owing to intentional ingestion of 10 crushed seeds of *A precatorius* (approximate weight 5 g). Following ingestion of the seeds, the girl developed multiple symptoms of nausea and vomiting. Two hours after ingestion, she emerged abdominal pain (persistent pain, obviously in upper and middle abdomen) and diarrhea (watery diarrhea, approximately 2 times/h). After admission, the girl started defecating liquid black stools which turned into bloody later; her urine presented in red color and urine output decreased (less than 30 mL/h).

On physical examination, she looked unwell, with a temperature of 37.0°C, heart rate of 112 beats/min, respiratory rate of 30 breaths/min, blood pressure of 85/48 mm Hg, and oxygen saturation of 90%. The girl had a somnolent and anxious mental status. Her whole abdomen had tenderness, especially in the upper and middle abdomen, but without abdominal muscle tension or rebound tenderness. The bowel sound was active (about 10 times/min).

Laboratory tests showed neutrophilic leukocytosis, increased creatine kinase (CK).The urine and stool routine tests revealed: white and red cells and glucose in urine, and positive fecal occult blood test.

Cont.

CASE 02 – *Cont.*

Other tests, including liver and renal function, coagulation function, serum electrolytes, and creatine kinase MB isoenzyme (CK-MB), were normal.

Vital signs were closely monitored and treated the patient with gastric lavage, purgation, gastric acid suppression by PPI, liver protection, blood volume and electrolytes resuscitation, and hemostasis. CRRT was administered for 72 hours and HP.

The patient's condition started improving after the combined therapy of CRRT and HP, including that heart rate fluctuated at 70 to 90 beats/min, blood pressure was stable and in the range 100–120/55–75 mm Hg, oxygen saturation maintained over 98%. Her unwell mental status was improved to the point at which she became conscious and relaxed. The symptoms of vomiting, abdominal pain, bloody stools, gross hematuria, dizziness, and fatigue were ameliorated gradually. Urine output increased to 60 mL/h after 3 days. On 5th day after admission, the blood routine, urine routine, stool routine and occult blood test, liver and renal function, coagulation function, serum electrolytes, and myocardial enzyme series for the patient, returned normal.

The girl eventually made an excellent recovery with no sequelae at her 3-month follow-up.

Huang, Jiliang MD[a]; Zhang, Wenbin MD[b]; Li, Xin MD[a]; Feng, Shufen MD[a]; Ye, Gang MD, PhD[a]; Wei, Hongcheng MD[a]; Gong, Xiaobing MD, PhD[a,*] Acute abrin poisoning treated with continuous renal replacement therapy and hemoperfusion successfully, Medicine: July 2017 - Volume 96 - Issue 27 - p e7423 doi: 10.1097/MD.0000000000007423

KAPUM KIRIYA (CORAL BUSH) කැපුම්කිරිය

Botanical Name: *Jatropha multifida*




Most poisonous:
Seeds

Fatal dose: 1 seed

Toxin:

- Toxalbumin (Jatropin)

KAPUMKIRIYA POISONING SIGNS & SYMPTOMS

- Nausea, protracted vomiting
- Diarrhea
- Abdominal pain
- Hemorrhagic gastroenteritis
- Dehydration
- Shock
- Known to mimic organo-phosphate poisoning - vomiting, diarrhea and constricted pupils

CIRCUMSTANCES OF KAPUMKIRIYA POISONING

- It is called ‘kapum kiriya’ because the latex of the plant is applied to wounds and ulcers.
- In Africa the leave juice is used for oral candidiasis
- Accidental poisoning: Children

CASE 01 – *Jatropha multifidi* poisoning

A 3 ½ year old girl from Kaduwela was found by her mother to be ingesting the seed of the Kapum Kiriya (*Jatropha multifida*) plant which was growing near the fence. The mother immediately removed the remaining particles of the seed from the child's mouth. The child spontaneously vomited several times and became drowsy. The parents brought the child to the Lady Ridgeway hospital, Colombo 2.5 hours later. On admission, the child was drowsy and dehydrated. Gastric lavage was given, activated charcoal administered and intravenous fluid therapy started. The child recovered uneventfully and was discharged from the ward after 24 hours. Management was facilitated as the mother brought the flower with a part of the plant.

Guruge, K., Seneviratne, A. and Badureliya, C., 2008. A case of Jatropha multifida poisoning. *Sri Lanka Journal of Child Health*, 36(4), p.148. DOI: http://doi.org/10.4038/sljch.v36i4.78

CASE 02–*Jatropha multifidi* poisoning

Two unrelated healthy boys, 9.5 and 8.5 years of age, were admitted to the pediatric section of the Emergency Department (ED) approximately 90 min after ingesting a large amount of fruit, probably more than 10 each, of a yet unidentified ornamental tree growing in the backyard. One hour after ingestion, both experienced intractable vomiting, colicky abdominal pain, and watery diarrhea.

On admission to the ED, they were found to be obtunded, mildly disoriented, continued to vomit repeatedly and had diarrhea. They seemed clinically dehydrated, with sunken eyes, dry mucous membranes and pallor. The vital signs of the older boy were: oral temperature 36.2°C, blood pressure (BP) 135/86 mmHg, pulse rate 93 beats/min, respiratory rate 18/min, and SpO2 98%. The vital signs for the younger boy were: oral temperature 36.4°C, BP 112/60 mmHg, pulse rate 118 beats/min, respiratory rate 18 breaths/min, and SpO2 98%. Physical examinations of the two patients were unremarkable for the heart and lungs. Both abdomens were soft, diffusely tender with no signs of acute abdomen. Neurologic examinations disclosed no pathologic findings. The pupils were equal and reactive, motor function and sensorium were intact, the reflexes were normal, and no fasciculations were observed. ECGs of both patients were normal. Complete blood counts were normal.

Cont.

CASE 02 – *Cont.*

Both patients continued to have watery diarrhea while in the ED. Samples of the fruit, leaf, and flower were brought in by the families, and the plant was identified as Jatropha multifidi. Treatment included aggressive volume resuscitation with intravenous (i.v.) bolus of saline (20 cc/kg), and urine alkalization. Ipecac syrup and activated charcoal were not given.

After initial stabilization, the children were transferred to the Pediatric ICU for further monitoring and treatment. Treatment included rehydration, fluid, and electrolyte replacement. Vomiting and diarrhea ceased after 36 h, and they were transferred to the Pediatric Department. The recovery was uneventful, except for a subclinical rise of liver function tests, which climaxed on the fourth day.

The boys were discharged in good health on the fifth day of hospitalization, and a follow-up 3 weeks later revealed no residual effects of the intoxication with normalization of liver enzymes. One nurse, who also handled the fruit, developed signs of localized erythema immediately after the contact, with no additional sequel.

Yotam Levin; Yaniv Sherer; Haim Bibi; Menachem Schlesinger; Emile Hay (2000). Rare jatropha multifida intoxication in two children. , 19(2), 0–175. doi:10.1016/s0736-4679(00)00207-9

CASE 03 – *Jatropha multifidi* poisoning

Four previously healthy siblings (ages 3, 4, 5 and 7 years) presented to the pediatric emergency department approximately 3 hours after eating the fruit of a garden plant (later identified as *J. multifida*). Shortly after ingestion of the plant, all four patients began suffering from profuse watery diarrhea and protracted vomiting.

Upon admission to the emergency department, two of the siblings were mildly obtunded. The consciousness of the other two was intact. All four siblings were dehydrated.

On admission, all four children had constricted and reactive pupils that lasted for approximately 3 hours after arrival at the hospital. Primary laboratory workup included a complete blood count, which showed leucocytosis in all four. Electrolytes and plasma acetylcholinesterase activity levels were normal.

The patients were all treated with intravenous rehydration solutions. All four patients demonstrated full recovery within 24 hours of ingestion. Aspartate aminotransferase levels were measured in two of the children and were normal. Repeat blood counts on day 2 were normal. Plasma acetylcholinesterase activity levels were normal in all four children

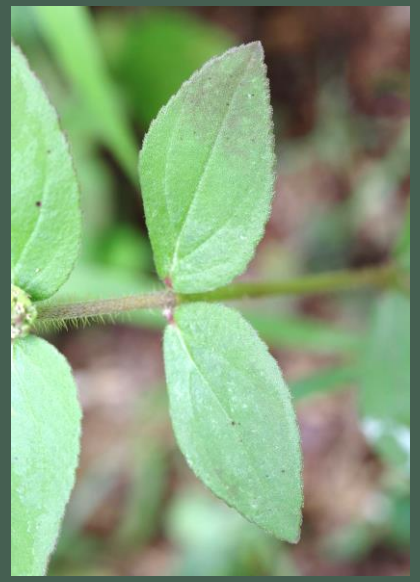
Koltin, Dror; Uziel, Yosef; Schneidermann, Dina; Kotzki, Shula; Wolach, Baruch; Fainmesser, Pinchas (2006). *A Case of Jatropha multifida Poisoning Resembling Organophosphate Intoxication. Clinical Toxicology, 44(3), 337–338.* doi:10.1080/15563650600584584

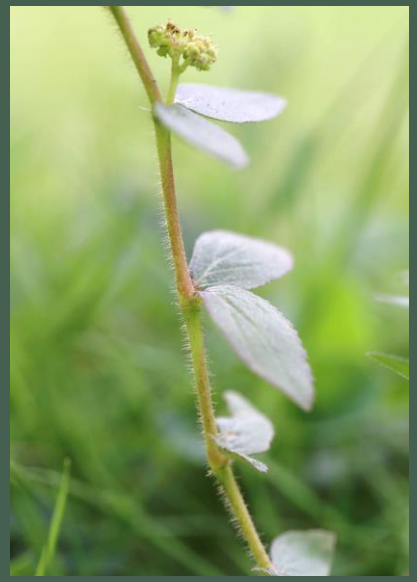
KEPUNKIRIYA/BUDADAKIRIYA/DADAKIRIYA

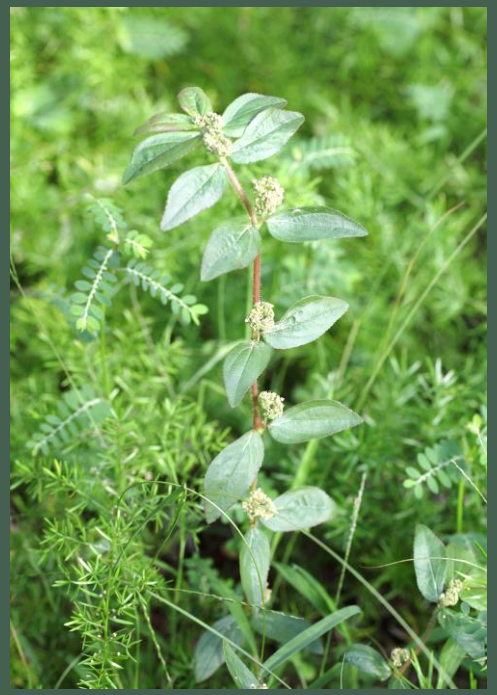
(AUSTRALIAN ASTHMA WEED/ THAWA THAWA)

කැපුම්කීරිය/බුදද කීරිය/දද කීරිය

Botanical Name: *Euphorbia hirta*







Most poisonous:
Whole Plant

Toxin:

- Alkaloid xanthoramine
- Gallic acid
- Phenolic substance

KAPUMKIRIYA/THAWA THAWA POISONING
SIGNS & SYMPTOMS

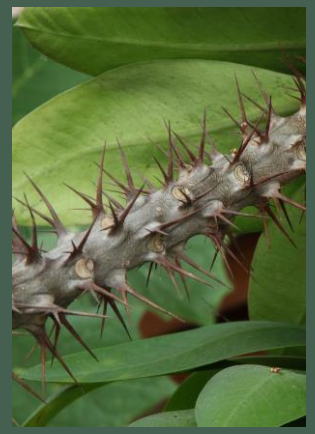
- Drowsiness
- *Skin contact:* irritant contact dermatitis
- *Eye contact:* keratoconjunctivitis
- *Ingestion:* Nausea, vomiting, diarrhoea
- Should observe for cyanide poisoning features

CIRCUMSTANCES OF KAPUMKIRIYA/ THAWA THAWA POISONING

- Accidental poisoning is common.

CHRIST THORN/ CROWN OF THORN

Botanical Name: *Euphorbia milii*



Most poisonous:
Plant sap

Toxin:
- Milliamines,
- Euphorbol
- Euphorbin.

CROWN OF THORN POISONING SIGNS & SYMPTOMS

- Highly toxic and an irritant to the skin and eye
- *Skin contact:* irritant contact dermatitis
- *Eye contact:*
  - Severe burning sensation with blurring of vision
  - Lacrimation
  - Severe reduction in visual acuity
  - Kerato-conjunctivitis
  - Mild to severe corneal edema
  - Epithelial defects
  - Anterior uveitis
  - Secondary elevated intraocular pressure

CIRCUMSTANCES OF CROWN OF THORN POISONING

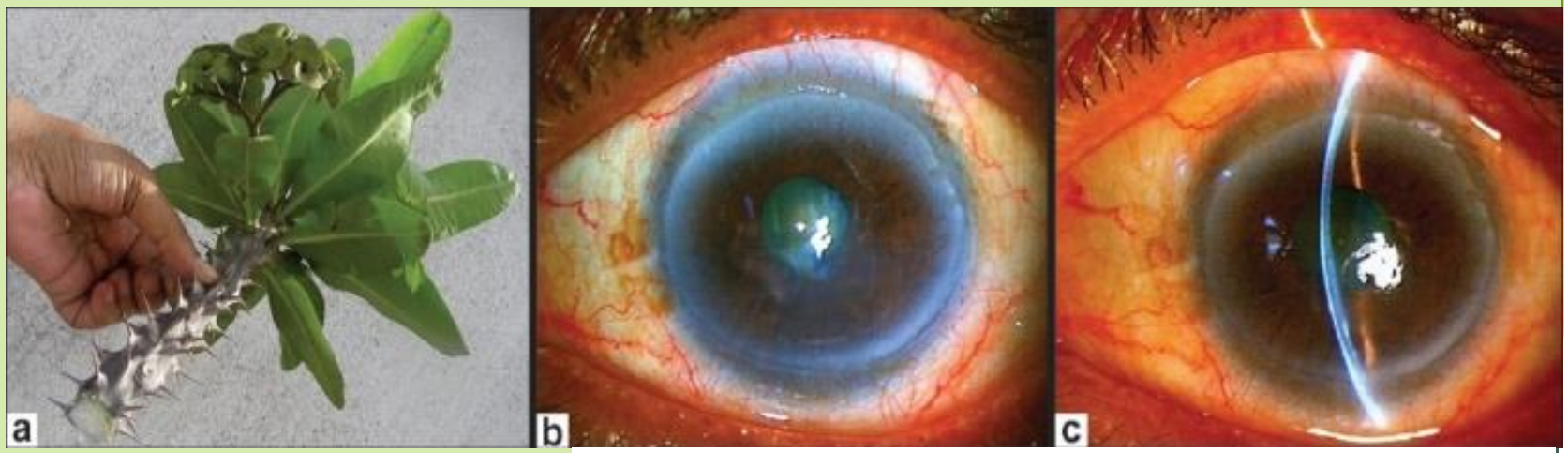
- Accidental poisoning is common.

CASE 01 – *Euphorbia milii* poisoning

A 54-year-old woman was pruning her Euphorbia milii (crown-of-thorns) houseplant, when she felt a stinging sensation as a drop of sap entered her left eye. She did not wash her eyes immediately. Fifteen minutes later she felt severe pain, blepharospasm and dimness of vision in the left eye. She presented three hours later and irrigation was done immediately with copious Ringer's lactate solution.

On examination, BCVA in the RE was 20/20 and in the LE 20/120. There was conjunctival hyperemia, punctate localized corneal epithelial lesions, and moderate corneal edema with Descemet's folds. There was anterior uveitis with moderate anterior chamber reaction with 2+ cells and 2+ flare. The IOP was 14 mm Hg. The right eye was within normal limits.

The patient was treated and followed up. The punctate epitheliopathy had completely resolved by Day three. By Day 15 all signs and symptoms had resolved and the patient regained BCVA of 20/20.



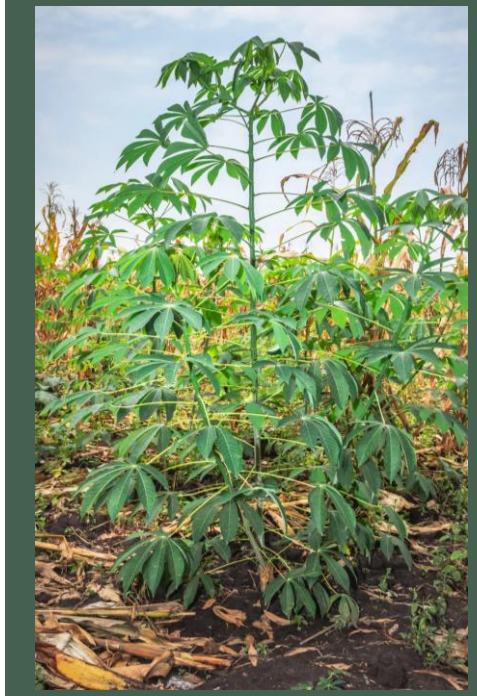
[b & c]- Punctate epithelial lesions, corneal edema, Descemet's folds and moderate anterior uveitis – one day after exposure to E. milii sap

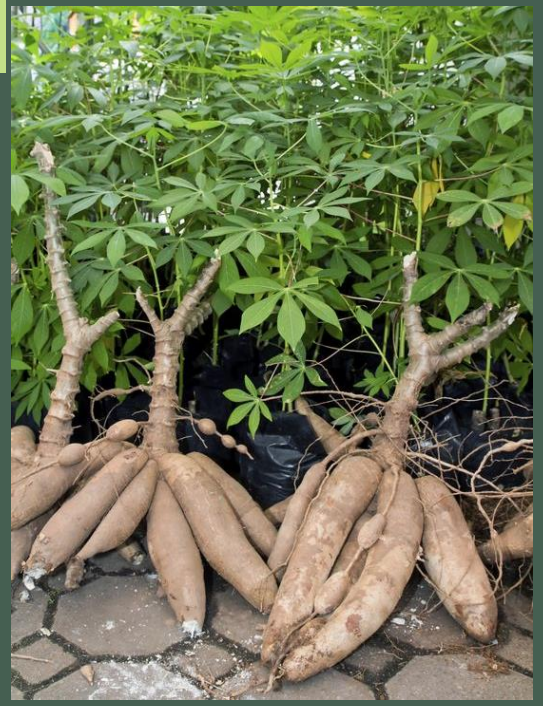
Basak SK, Bakshi PK, Basu S, Basak S. Keratouveitis caused by Euphorbia plant sap. Indian J Ophthalmol. 2009;57(4):311-313. doi:10.4103/0301-4738.53060

MANYOKKA (TAPIOCA/CASSAVA/MANIOC)

මඤ්ඤොක්කා

Botanical Name: *Manihot utilissima*





Most poisonous:

Root covering

Toxin:

- Cyanogenic glycosides

MANIOC POISONING SIGNS & SYMPTOMS

- Acute cyanide intoxication features occur.
- Rapid respiration
- Drop in blood pressure
- Rapid pulse
- Dizziness and headache
- Mental confusion,
- Twitching and convulsions
- Abdominal pain, nausea, vomiting, diarrhea
- Dehydration and shock
- Collapse
- Drowsiness

CIRCUMSTANCES OF MANIOC POISONING

- Accidental poisoning most commonly due to improper cooking.

CASE 01 – *Manihot utilissima* poisoning

In September 2017, an outbreak of suspected cyanide poisoning, involving 98 cases with two deaths, occurred in western Uganda. Epidemiologic and laboratory investigation identified consumption of a cassava flour dish made from wild cultivars of cassava with high cyanogenic content as the cause of the outbreak.

On September 5, 2017, a funeral was held in Kasese District in western Uganda. Following the funeral, 33 persons with symptoms that included diarrhea, vomiting, and abdominal pains were admitted to Bwera Hospital in Kasese District. Symptoms occurred a few hours after meals during which a cassava flour dish was served at the funeral.

On September 8, the Uganda Ministry of Health received notification from the Kasese District health team regarding this outbreak of suspected food poisoning. An investigation to determine the cause of the outbreak and recommend control measures revealed that the outbreak resulted from consumption of a cassava dish made by combining hot water with cassava flour. The implicated batch of cassava flour was traced back to a single wholesaler and found to contain high cyanogenic content. Informed by the investigation findings, police confiscated all cassava flour from retailers identified as the patients' source of the flour.

Alitubeera PH, Eyu P, Kwesiga B, Ario AR, Zhu B. Outbreak of Cyanide Poisoning Caused by Consumption of Cassava Flour — Kasese District, Uganda, September 2017. MMWR Morb Mortal Wkly Rep 2019;68:308–311. DOI: http://dx.doi.org/10.15585/mmwr.mm6813a3

CASE 02– *Manihot utilissima* poisoning

NSI, a six year old Malay girl, was admitted to Hospital Universiti Sains Malaysia (HUSM) on 11.12.1988 at 8.30 a.m. with complaints of one episode of vomiting and diarrhoea at 5.00 a.m. that morning, after eating three to five blocks of tapioca at 3.30 p.m. the previous day. The tapioca had been grown near her house and was dug out eight days before being eaten. Her mother had peeled the tubers which were then chopped into blocks and boiled for half an hour.

On admission, NSI appeared in good condition. Her vital signs were as follows:- heart rate 110/min, blood pressure 100/60mmHg, respiratory rate 30/min, temperature 37°C. She was not drowsy nor acidotic. Her pupils were equal and reactive, and the rest of the examination was normal. Investigation revealed that her blood contained 4 µg cyanide/ml; her gastric aspirate did not have cyanide or thiocyanate, and her urine was free of thiocyanate. Her haemoglobin was 12.2 g/dl, total white count 20.7 x 10^3/L, platelet count 541 x 10^3/L, blood urea 4.4 mmol/L, serum sodium 136 mmol/L, serum potassium 4.4 mmol/L, serum calcium 2.4 mmol/L, and blood glucose 7.1 mmol/L. Her liver function tests, urine and stool for microscopic examination and culture were normal.

Cont.

CASE 02 – *Cont.*

Analysis of the uncooked tapioca tuber revealed 181 mg/kg (181 ppm) of hydrocyanic acid (HCN); it was negative for aflatoxin. Her blood pressure, pulse and respiratory rates, intake/output were closely monitored; they were all normal. Supportive treatment given were stomach washout, oxygen and intravenous dextrose-saline. She did not have further episodes of diarrhoea and vomiting. Her pupils remained equal and reactive to light. She made an uneventful recovery and was discharged well.

Her younger sister, NZ (1.5 years old), also ate the tapioca at the same time. At 10.00 p.m. on that same night, she developed abdominal cramp and nausea followed by diarrhoea and vomiting. She then became drowsy, and was sent to HUSM, but died on the way. An elder sister, WSI (8 years old), and their mother who also ate the tapioca only had vomiting and had no symptoms respectively.

An investigation of food poisoning outbreak was carried out. Analysis of food specific attack rate incriminated tapioca as the culprit. Food poisoning was limited to this family; there were no other reports in the area.

Ariffin WA, Choo KE, Karnaneedi S. Cassava (ubi kayu) poisoning in children. Med J Malaysia. 1992 Sep;47(3):231-4. PMID: 1491651.

HABARALA (ELEPHANT'S EAR PLANT) හබරල

Botanical Name: *Alocasia macrorrhiza*





Most poisonous:

Stems

Leaves

Toxin:

- Calcium oxalate crystals
- Sapotoxin

HABARALA POISONING SIGNS & SYMPTOMS

- Profuse salivation, irritation, burning sensation and pain of the oral cavity
- Oedema of the tongue, lips, cheeks, salivary glands and throat lead to disphagia and aphonia.
- Ingestion of large amount lead to necrotic oesophagitis and haemorrhagic gastritis.
- Death may result from laryngeal oedema.
- Bradycardia, tremos, muscle twitching and trismus, convulsions and acute renal failure also can occur

CIRCUMSTANCES OF HABARALA POISONING

- Accidental poisoning mostly in children.

CASE 01 – *Alocasia macrorrhiza* poisoning

A 44-year-old man took the tuber of *Alocasia macrorrhiza* by mistake. Several minutes after ingestion of the tuber, he presented with numbness of lip, sore throat, nausea, vomiting, salivation, dyspnea, and dysphonia. Twenty minutes after poisoning, he was hospitalized and diagnosed as having acute laryngeal edema. He received diphenhydramine 20 mg and dexamethasone 5 mg via IV push, followed by an IV infusion of dexamethasone 10 mg. Meanwhile, oxygen inhalation, liver protective treatment, and other symptomatic treatment were given.

Five hours after poisoning, his symptoms gradually resolved and, 50 days later, he recovered.

Xie Lijing;Wang Yingwei; Long Xin;Sun Chengye. One case of acute Alocasia macrorrhiza poisoning and management (2011). Adverse Drug Reaction Journal. 13(4): P. 240-3

CASE 02 – *Alocasia macrorrhiza* poisoning

A 1½-year-old male child was brought by a grandfather with complaints of excessive cry, irritability immediately after eating leaves of a household plant known locally as Arbi/Hathikan. He developed redness of buccal mucosa and tongue after 2-3 min of eating leaves and excessive drooling of secretion from the mouth after half an hour. Out of curiosity, father also tasted leaves of the plant following which inflamed painful blister on the tip of the tongue and perioral numbness developed immediately. It was not relieved by home remedies (sweetened fluids) and lasted for 8-10 h.

On examination at the time of admission (after 1 h of eating leaves), child was very irritable with an inconsolable cry and had salivary drooling. Lips and tongue were swollen and reddened. He had dysphagia. On 2nd day, leaves and tuber were brought by parents, which after consultation with botany department, were from a decorative home plant called A. macrorrhiza.

Patient was put on intravenous fluids, ranitidine, antacids, local antiseptic and anesthetic mouth gel. Healing occurred by 3rd day and patient was discharged.

He was asymptomatic on follow-up after 1 week.

Joshi A, Karnawat BS, Narayan JP, Sharma V. Alocasia macrorrhiza: A Decorative but Dangerous Plant. Int J Sci Stud 2015;3(1):221-223

CASE 03 – *Alocasia macrorrhiza* poisoning

A 28-year-old man presented with severe numbness, paraesthesiae and pain over his tongue, oral cavity and throat, and repeated vomiting immediately after eating the root tuber of a plant he had picked from the hillside. He claimed to be a herbalist, who had been trying to find out more about the properties and toxicity of Chinese medicinal plants. Only a portion of the tuber (about 30g) was ingested. The plant was verified by the patient to be identical to the one he had ingested. Experts from the Agricultural and Fisheries Department later confirmed its identity as *A. macrorrhiza*.

When he arrived in the emergency department one hour later, he was fully alert. His other complaints included nausea, mild abdominal pain, and numbness and pain over the right side of his face. His BP was 142/86 mmHg and PR 95 beats minm. His throat looked slightly inflamed. No focal neurological defects such as motor weakness or loss of tendon reflexes were detected. He was observed in our general medical ward for the next 3 days. Apart from a leucocytosis of 15.2 x 109 L-1, his blood biochemistry, electrocardiogram and chest X-ray were all normal. The next morning he still complained of mild throat pain and numbness as well as pain over his tongue.He was empirically given amitryptyline 10 mg bid for a total duration of 5 days. On discharge, he had only minimal numbness affecting his tongue.

Chan, T.; Chan, L.; Tam, L.; Critchley, J. (1995). *Neurotoxicity following the ingestion of a Chinese medicinal plant, Alocasia macrorrhiza. Human & Experimental Toxicology, 14(9), 727–728.* doi:10.1177/096032719501400905

CASE 04 – *Alocasia cucullata* (Nai Habarala) poisoning

A 7-year-old boy and his 4 year-old sister were admitted to a local hospital 6 hr after ingestion of the Nai Habarala fruit. The girl, who had consumed the major portion of the fruit, was vomiting, pyrexial and restless . She had no cyanosis but had deteriorated and succumbed within 20 hr of ingestion of the fruit. The boy, who had similar symptoms to a lesser degree, was transferred to our hospital for secondary care. On admission (i.e . 24 hr from ingestion), he was conscious but was intermittently restless. Gastric lavage did not reveal any fruit. He became confused, drowsy and more restless at 72 hr from poisoning. The pupils were widely dilated but reacted well to light. The fundi were normal . He developed bilateral ptosis, generalised muscle weakness and had hypotonia and diminished reflexes.

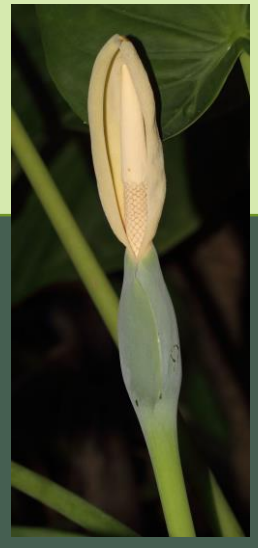
His BP was 110/70 mmHg, PR was 130/min, and RR was 36/min . He was not cyanosed. There was mild jaundice, but no hepatosplenomegaly. Despite full supportive care, he steadily deteriorated requiring intubation and positive pressure ventilation at 96 hr from poisoning. The arterial blood gases on air at the time of intubation showed a compensated metabolic acidosis and a hypoxia. Low platelet count, Na+; increased clotting time, prothrombin time, SGOT, SGPT); ECG, sinus tachycardia, right bundle branch block, T inversion in V3 V4; chest X ray, enlarged heart, lung fields `streaky'. The renal function remained normal throughout.

Cont.

CASE 04 – *Cont.*

Gradually he became deeply unconscious. At 120 hr from poisoning, he developed a supraventricular tachycardia whilst on IPPV and developed a cardiogenic shock.

There were no striking abnormalities at post-mortem. The brain was slightly congested. The lungs had a few areas of aspiration collapse . The heart appeared normal except for a few flame-shaped subendocardial haemorrhages. The kidneys and the gastrointestinal tract appeared normal. Significant histopathological changes were found only in the liver, appearances suggested a severe zonal necrosis and an extensive microvesicular fatty change.



C.D.A. Goonasekera; V.W.J.K. Vasanthathilake; N. Ratnatunga; C.A.S. Seneviratne (1993). Is Nai Habarala (Alocasia cucullata) a poisonous plant?. , 31(6), 0–816. doi:10.1016/0041-0101(93)90388-y

RATHU HABARALA/MAL HABARALA

(CALADIUM/ANGEL'S WINGS) රතු හබරල/මල් හබරල

Botanical Name: *Caladium andreanum*
Caladium bicolor

Most poisonous:

Plant juice (sap)

Stems

All parts

Toxin:

- Calcium oxalate crystals

KETALA (KOTAKIMBULA/ DUMB CANE) කෙටල

Botanical Name: *Dieffenbachia amoena*

Most poisonous:
Plant juice (oil)
Stems

Toxin:
- Calcium oxalate crystals

KETALA POISONING SIGNS & SYMPTOMS

- Skin contact with the plant oil;
  - Localized swelling
  - Redness
  - Burning, and pain
- Chewed plant
  - Life-threatening systemic symptoms, such as respiratory failure.

CIRCUMSTANCES OF KETALA POISONING

- Stem of the plant is similar to a sugar cane.
- Accidental poisoning mostly in children.
- Suicide

CASE 01 – *Dieffenbachia amoena* poisoning

An eight-year-old female, having no previously known diseases, was taken to the emergency department with sudden numbness in the tongue, lower lip swelling and redness, slurred speech, and difficulty breathing.

Half an hour after arrival, the vital signs of the patient were stable (heart rate: 112 bpm, blood pressure: 100/65 mmHg, respiratory rate: 28/min, oxygen saturation: 96%). All physical examination findings were normal except for swelling and redness of the lip and eye, and tongue numbness. The child was breathing with an open mouth and had slurred speech.

According to the mother, the patient was playing with soil from a dieffenbachia plant with a pen, and then she placed this pen in her mouth. This occurred five minutes before the symptoms began. The patient complained of tongue numbness, and given this complaint, plant poisoning was considered.

Treatment with steroids and antihistamines (methyl prednisolone 1 mg/kg, pheniramine 1 mg/kg) was administered. Because of the sudden onset of complaints and respiratory distress, chest radiography was conducted to exclude the possibility of a foreign body aspiration. However, the results of this procedure were normal.

Cont.

CASE 01 – *Cont.*

The patient was monitored, and her symptoms improved within two hours after treatment. The initial plan was to closely monitor the child in the emergency department for 24 hours to ensure that airway obstruction or dyspnea did not develop due to the toxic effects of Dieffenbachia.

However, after 24-hours of observation, the child made a full clinical improvement.

The patient was discharged after she and her family were informed about plant poisoning.

Berksoy, Emel & Topalakçı, Ebru & Bekem, Ozlem & çelik, Tanju. (2018). Accidental Poisoning of a Child by Dieffenbachia. Turkish Journal of Pediatric Emergency and Intensive Care Medicine. 5. 86-88. 10.4274/cayd.86547.

CASE 02 – *Dieffenbachia amoena* poisoning

A girl aged 12 1/2 years ate a leaf of common house plant Dieffenbachia in a suicidal attempt. She was admitted to emergency department 6 hours later. A nasogastric tube was placed and saline gastric lavage was begun. Many small fragments of Dieffenbachia leaves were evacuated. Esophagitis grade 2 of the whole esophagus was revealed by emergent endoscopy. Nasogastric tube was left in place and antibiotics and systemic steroids were administered.

Fourteen days later, she was discharged. She was able to swallow liquids and enteral nutrition was provided via nasogastric tube.

Five weeks after the injury, a follow-up endoscopy showed some improvement with receding esophagitis. Two days later, melena appeared. The next morning, the girl fainted and was readmitted to hospital. By that time, she reported hematemesis as well.

Blood pressure on admission was 90/60 mm Hg and hemoglobin level 111 g/L. Rigid esophagoscopy failed to show any source of bleeding and the patient was transferred to department of pediatric surgery for suspected gastric bleeding. Her blood pressure dropped to 60/30 mm Hg and she was profoundly anemic (hemoglobin level 49 g/L).

Cont.

CASE 02 – *Cont.*

After volume resuscitation, a new upper GI endoscopy of esophagus, stomach, and duodenum revealed arterial bleeding from the esophagus at the level of upper esophageal sphincter.

Infiltration with Adrenalin (epinephrine) and attempted closure with metallic clips were fruitless and the surgical exploration of the cervical esophagus followed. The source of bleeding was not identified from left-sided collar incision. During repeated endoscopy, a great amount of blood in stomach was found. A gastrotomy of the massively dilated stomach filled with clotted and fresh blood was performed. No source of gastric bleeding was found but there was a steady flow of bright blood from the esophagus. A Sengstaken-Blakemore tube was inserted and urgent right-sided thoracotomy performed. There was a 3-mm fistula between the thoracic esophagus and the aorta at the branching of the brachiocephalic trunk.

The aorta was partially occluded by clamping, the fistula resected, and both defects were closed. The blood pressure promptly returned to normal values. The girl was disconnected from ventilator on first postoperative day. One week later on echocardiography, there was a small narrowing on the aorta at the origin of the brachiocephalic trunk.

She was discharged from the hospital after 3 weeks and remained asymptomatic with normal swallowing and no sequelae 7 years after the incident.

Jiří Šnajdauf; Vladimír Mixa; Michal Rygl; Martin Vyhnánek; Jiří Morávek; Zdeněk Kabelka (2005). *Aortoesophageal fistula — an unusual complication of esophagitis caused by Dieffenbachia ingestion.* , *40* (6), 0–. doi: 10.1016 / j.jpedsurg.2005.03.036

DUMKOLA (TOBACCO) දුම්කොළ

Botanical Name: *Nicotiana tabacum*






Most poisonous:

Leaves

Stems

Roots

Flowers

Toxin:

- Alcoloid

TOBACCO POISONING SIGNS & SYMPTOMS

- Mild:
  - Salivation,
  - Nausea, vomiting, diarrhoea
  - Dizziness, drowsiness
  - Headache
  - Hand tremor
- Serious:
  - Mental confusion,
  - Circulatory collapse (shallow rapid pulse, 'cold sweating')
  - Convulsions
  - Loss of consciousness
  - Cardiac arrest, respiratory paralysis

CIRCUMSTANCES OF TOBACCO POISONING

- Accidental poisoning in children and adults.

CASE 01 – *Nicotiana tabacum* poisoning

Green-tobacco sickness is an occupational illness of tobacco harvesters. The disease is self-limited and of short duration, but recurs frequently in susceptible workers. Symptoms characteristically begin with headache and dizziness during the afternoon of the harvest, and progress to abdominal pain, protracted vomiting, and prostration by evening. The illness lasts only 12-24 hours but recurs frequently in susceptible workers after repeated harvest exposures. As many as twelve recurrences have been reported in 8 weeks by some workers. The etiology is not known, but nicotine has been suspected as a causative agent.

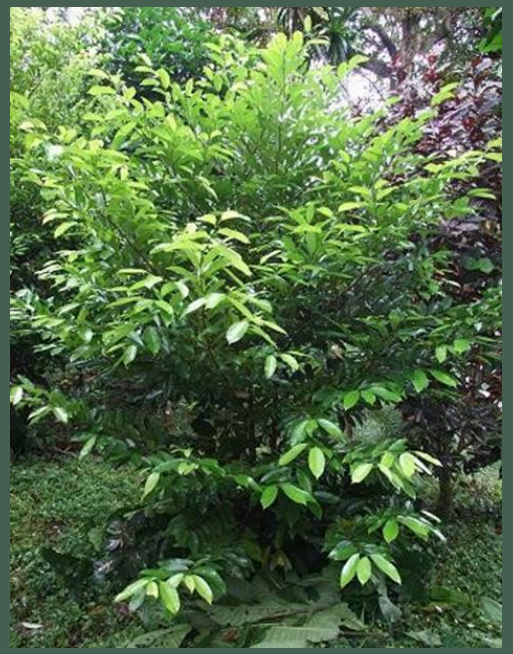
Thirty-two workers on four North Carolina tobacco farms were studied during harvesting. None of these workers smoked or chewed tobacco. Urinary cotinine (the major metabolite of nicotine) levels were monitored over a 24-hour period to evaluate nicotine absorption.

There was a tenfold rise in mean excretion of cotinine among workers who had greatest contact with the tobacco. Less cotinine was found in urine of workers who had less exposure. Levels of cotinine exceeded those found in novice smokers who smoked 3 cigarettes in succession. Absorption of nicotine from tobacco leaf is the likely cause of tobacco sickness.

Gehlbach, s (1975). Nicotine absorption by workers harvesting green tobacco. , 305(7905), 478–480. Doi:10.1016/s0140-6736(75)92829-9

SADIKKA (NUT MEG) සාදික්කා

Botanical Name: *Myristica fragrans*



Most poisonous:
Seeds (Nut meg)
Aril (Mace)

Fatal dose: Adults – 3 seeds
Children – 2 seeds

Toxin:
- Myristicin
- Elemicin

SADIKKA POISONING SIGNS & SYMPTOMS

- Symptoms usually seen within 3-6 hours after ingestion
- Resembles anticholinergic intoxication.
  - Profuse sweating, flushed face
  - Dry mouth
  - Burning epigastric pain
  - Tachycardia, chest pain, palpitations
  - Restlessness, dizziness
  - Giddiness, extreme drowsiness
  - Hallucinations, delirium
  - Stupor
  - Nausea, vomiting, abdominal pain

CIRCUMSTANCES OF SADIKKA POISONING

- Used as a spice.
- Has many medicinal properties.
- Accidental poisoning mostly in children.
- Abuse of the spice as means of inducing hallucinatory effects.

CASE 01 – *Myristica fragrans* poisoning

A 22-year-old male university student was brought to the emergency department because of an altered state of consciousness and hallucinations. We learned that the patient had crushed six cloves of nutmeg and mixed these with fruit juice and drunk this approximately 8 h before admitting to emergency service.

Nausea, dry mouth, and facial flushing commenced approximately 6 h after consumption, followed by an altered state of consciousness. He had no history of smoking and alcohol or drug use or disease.

Blood pressure at the time of presentation was 140/90 mmHg, PR 75 beats/min, RR 20 breaths/min, body temperature 36.5°C, and oxygen saturation at room temperature 98%. At initial examination, the GCS was 13, orientation and cooperation were limited, and agitation and incoherent speech were observed. Both pupils were mydriatic, and light reflex was bilateral positive. Other system examination findings were normal. Electrocardiogram was in a normal sinus rhythm. Complete blood count, kidney and liver function tests, serum electrolytes, arterial blood gas values, and cardiac markers were normal.

The patient was admitted to the intensive care unit with a diagnosis of nutmeg intoxication. Symptomatic treatment and intravenous fluid therapy were started. Intravenous midazolam was administered for sedation. Consciousness returned to normal status after 12th hour of nutmeg intake and all symptoms resolved entirely. Vital signs were stable throughout hospitalization, and no impairment was observed in laboratory values.

The patient was discharged following a 48-h follow-up.

Günaydın M. , Tatlı Ö. , Altuntaş G. , Uslu Z. , Özşahin F. , Beşlioğlu N. Nutmeg Intoxication Associated with Consumption as a Stupefacient. Journal of Emergency Medicine Case Reports. 2017; 8(3): 64-65

CASE 02 – *Myristica fragrans* poisoning

A previously well 18 year old student presented with complaints of palpitations, drowsiness, nausea, dizziness, thirst, and dry mouth. She was very anxious, restless, and agitated and described being ‘‘in a trance state’’. She specifically felt ‘‘like Jack in the box wanting to get out’’ but did not have hallucinations. She did not complain of urinary or abdominal discomfort and gave no history of seizures or migraine. She had an unremarkable medical and psychiatric history and denied any suicidal ideation. The patient refused to give any information regarding recreational drug use.

On examination the patient was agitated but alert, flushed but apyrexial, with a RR of 20/minute and saturation of 96% on room air. She was tachycardic at 102/ minute with a BP of 105/68. Cardiopulmonary examination was unremarkable. The abdomen was soft and non-tender. Cranial nerves were normal, while peripheral nervous system examination showed brisk, symmetrical deep tendon reflexes. There was no neck stiffness. Pupils were dilated to size 4 mm and were symmetrically brisk to light and accommodation.

A 12 lead ECG showed a fast sinus arrhythmia (rate 95– 110/minute) with no ischaemic or hypertrophic changes. Serum urea, electrolytes, liver transaminases, full blood count, and urgent catecholamines were normal. Serum and urine toxicology screens were negative.

Cont.

CASE 02 – *Cont.*

In view of the complexity of her condition she was admitted and five hours later admitted to having taken a large dose of nutmeg while trying to ‘‘get high’’.

Some 50 g of commercially available grated nutmeg were blended into a milkshake, the patient drinking three quarters of the amount. A feeling of elation was experienced by at least two people, but in our patient this was followed by her presenting symptoms 30 minutes after ingestion.

The patient was kept for observation, offered reassurance, and rehydration. After symptoms had resolved she was allowed to return home 10 hours after presentation, 16 hours after ingestion.

Demetriades AK, Wallman PD, McGuiness A, Gavalas MC. Low cost, high risk: accidental nutmeg intoxication. Emerg Med J. 2005;22:223–225. doi: 10.1136/emj.2002.004168.

Acknowledgement

- Photographs included in this presentation are taken from the Google images and relevant references of the case reports.
- We are thankful for the personnel involved in taking these photographs and all the credits of the images belong to the relevant parties.

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THANK YOU!