

Case Report

Death due to poisoning with *Gloriosa superba* ('Niyagala') seeds

R M M K Namal Rathnayaka¹, K Jeganathan¹, H D M S K Dissanayake¹, P E A Nishanthi Ranathunga²

¹Provincial General Hospital, Ratnapura, Sri Lanka, ²Base Hospital, Kahawatte, Sri Lanka

Keywords: Niyagala; *Gloriosa superba*; plant poisoning; colchicine

Corresponding Author: R M M K Namal Rathnayake, E-mail <namalrath10@yahoo.com >

Received: June 2016, Accepted revised version July 2016, Published: August 2016

Competing Interests: Authors have declared that no competing interests exist

Introduction

Plant poisoning may be accidental or intentional. Accidental poisoning is normally a problem of young children¹. Ingestion of yellow oleander seeds (*Thevetia peruviana*) has recently become a popular method of self-harm in northern Sri Lanka². Here we report a case of death due to poisoning with 'Niyagala' ('glory lily', *Gloriosa superba*) seeds.

Case report

A 35 year old female patient was transferred to Provincial General Hospital, Ratnapura, from Base Hospital, Kahawatte with a history of loose stools following self ingestion of about 50 'Niyagala' seeds (figure 1) more than 10 hours previously.



Figure 1: *Gloriosa superba* ('Niyagala') seeds and fruits

The patient who had no previous illnesses was a mother of one child. On admission she had moderate watery diarrhoea but no vomiting or abdominal pain. On examination she was afebrile, blood pressure (BP) 87/56mmHg, pulse rate 96/min, respiratory rate 30/min, SpO₂ 96% on room air, normal bilateral lung fields on auscultation and mild lower abdominal tenderness. As this was a late presentation, gastrointestinal decontamination with gastric lavage and activated charcoal was not performed. Initial investigations revealed white blood cell count $27.1 \times 10^9/L$, neutrophils $14.092 \times 10^9/L$, lymphocytes $11.924 \times 10^9/L$, platelets $202 \times 10^9/L$, haemoglobin 141g/L, serum sodium 147mmol/L, serum potassium 2.8mmol/L, serum creatinine 92 μ mol/L, INR 1.52, normal urinalysis and ECG. She was resuscitated with

intravenous fluids (0.9% sodium chloride solution and Hartmann's solution) and potassium was replaced with potassium chloride tablets.

The following day, she developed shortness of breath associated with a few bilateral crepitations and acute liver dysfunction (AST 292.1U/L and ALT 50.7U/L). Arterial blood gas (ABG) analysis on room air was suggestive of metabolic acidosis (pH 7.283, pCO₂ 28mmHg, pO₂ 68.6mmHg, serum HCO₃ 13.4mmol/L, base excess -13.5mmol/L) for which intravenous 8.4% NaHCO₃ 50 ml was administered with fluid bolus of 500 ml Hartmann's solution. She was on O₂ inhalation via face mask (10L/min). There was no renal impairment (normal urine output, serum creatinine 98.7µmol/L) and she maintained normal O₂ saturation and blood pressure. Repeat serum K⁺ was 3.9mmol/L. Her vital functions were monitored. However, she died on day two following hospital admission (42hrs after ingestion) due to sudden onset severe respiratory failure in spite of resuscitation with Ambu ventilation and attempts at intubation.

Post mortem examination showed mild enlargement of the liver with yellow discoloration, reduced cortico-medullary demarcation in both kidneys, petechial haemorrhages on the outer surface of kidneys and mild gastric erosions.

Discussion

Deliberate self-harm using 'Niyagala' usually occurs due to ingestion of tubers (roots)^{3,4,5}. However in this case the victim had ingested seeds, which is not the common practice.

'Glory Lily' is a plant that grows wild in all regions of Sri Lanka. All parts of this plant are poisonous of which tubers may be the most poisonous. They have been found to contain several alkaloids such as colchicine, gloriocine, superbine etc., among which colchicine is the most important alkaloid⁶ which results in multi-organ toxicity. The commonest clinical presentation of poisoning is severe gastroenteritis associated with nausea and vomiting. Other features include muscle weakness, respiratory failure, bone marrow depression, convulsions, polyneuropathy and coagulation defects⁷. Generalized alopecia⁴ and dermatitis⁵ are late manifestations that develop about 1-2 weeks after poisoning. Cardiac toxicity (ST elevation in ECG) following 'Niyagala' poisoning has also been documented³.

This patient had watery diarrhoea, hypokalaemia, moderately elevated liver enzymes and clotting defects (elevated international normalised ratio (INR) 2.76) and died due to sudden onset of severe respiratory failure. On day 2 of admission (around 35 hours after ingestion) the patient developed shortness of breath and mild dyspnoea (respiratory rate of 40/min), few bilateral crepitations in lungs and ABG analysis showed lowering of pO₂ (68.6mmHg). But at this stage we were unable to do a chest x-ray as the patient deteriorated. Autopsy examination revealed that both lungs were normal in appearance. Therefore, sudden onset of respiratory failure may be due the action of colchicine on intercostal muscles. Angunawela et al. suggested that respiratory failure is due to the paralysis of intercostal muscles rather than the direct depression of the respiratory centre by colchicines⁵.

The patient may have ingested a lethal dose of colchicine which varies from 6-20mg. She had ingested about 50 seeds (weight-10g). It is estimated that 10g of fresh tuber contains about 6mg of colchicines⁷ and in severe poisoning there may be respiratory depression⁸. The LD50 of colchicine for rats was 5 mg/kg⁶. This case report points out the importance monitoring respiratory function and the need for early respiratory support for a patient with 'Niyagala' poisoning.

References

1. Lucas G N. Plant poisoning. A hospital-based study in Sri Lanka. the Indian Journal of Pediatrics .July 1997, Volume 64, Issue 4, pp 495-502
2. Eddleston M, Ariaratnam C.A, et al. Epidemic of self-poisoning with seeds of the yellow oleander tree (*Thevetia peruviana*) in northern Sri Lanka. Tropical Medicine and International Health volume 4 no 4 pp 266–273 April 1999.
<http://dx.doi.org/10.1046/j.1365-3156.1999.00397.x>
PMid:10357862
3. Mendis S. Colchicine cardiotoxicity following ingestion of *Gloriosa superba* tubers. Postgrad Med J. 1989 Oct; 65(768):752-5.
<http://dx.doi.org/10.1136/pgmj.65.768.752>
PMid:2694138 PMCID:PMC2429836
4. Gooneratne, B.W.M. Massive generalized alopecia after poisoning by *Gloriosa superba*. British Medical Journal 1966, 1: 1023-1024.
<http://dx.doi.org/10.1136/bmj.1.5494.1023>
PMid:5909848 PMCID:PMC1844473
5. Angunawela R.M., Fernando H.A.(1971) Acute ascending polyneuropathy and dermatitis following poisoning by tubers of *Gloriosa superba* Ceylon Medical Journal 16(4):233-235
PMid:5154236
6. Dunuwille, R., Balasubramaniam, K. Bibile, S.W. Analysis of alkaloids in *Gloriosa superba*. Ceylon Journal of Medical Science 1968, 17: 1.
7. Fernando, R. Widyaratna D. (1989). *Gloriosa superba*. INCHEM. International Programme on Chemical Safety (IPCS).
8. Fernando R. Management of Poisoning.2007, National Poisons Information Centre, National Hospital of Sri Lanka, Colombo.p286-288.