Gloriosa superba poisoning - A case report

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Abstract

The genus of ten species in plant family colchicaceae is gloriosa. Gloriosasuperba is a species of flowering plant in the family colchicaceae. In Karnataka it is known as *gowri gedde* (Hindi: Kalihari, Gujarathi: Dudhio, Sanskrit: Agnimukhi, Marathi: Indai). This ornamental plant is distributed in both Asian and African continent, it is often known as a poison, a noxious weed and a medicine. In India, Gloriosa superba is commonly distributed in the Western Ghats. *Colchicine* a pseudoalkaloid extracted from the plant, a useful drug with a narrow therapeutic index. This is a case report of an adult male with history of accidental consumption of colchicine and subsequent fatal toxicity.

Keywords: Colchicine, Gloriosa superba, poisoning.

Introduction

Gloriosa superba (G superba) is a wild climbing herb plant with tuberous roots grown all over India. It belongs to the Colchicaceae family and has common names such as flame lily, glory lily, gloriosa lily, climbing lily *etc.* Colchicine is a water-soluble pseudoalkaloid extracted from the plants of Colchium autumnale and G superba. It is a useful drug with a narrow therapeutic index. Gloriosine, superbine and salicylic acid are the other active alkaloids present in the plant. Action of glory lily has shown promising antibacterial, antifungal, anthelminthic and anti-inflammatory potential.[1,2]

Here, we report a case of Gloriosa superba tuber consumption (locally known as 'Gowri gadde') *Figure 1* with its fatal outcome.

Case report

A 50 year old male agriculturist from Davangere district in Karnataka accidentally consumed the tuber of a wild herb locally called 'Gowri gadde' which was seen by his niece. He was taken to the local hospital but was brought back home after two hours of treatment as was found to be apparently well on that day. However, the next morning onwards he had abdominal pain, vomiting, haematuria and loose stools. He was brought to emergency triage by his son in the evening. On examination, he was conscious and vitals were stable. The patient was shifted to the intensive care unit (ICU) for further management. Oxygen supplementation was administered *via* 40% venturi mask.

Figure 1: Gloriosa superba

Gastric lavage was performed. All the investigations were within the normal range but coagulation was deranged. Prothrombin time (PT) was 56.1 s,

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activated partial thromboplastin time (APTT) was 69.9 s, international normalised ratio (INR) was 4.51. Arterial blood gas report revealed metabolic acidosis with good oxygenation. The chest X-ray was normal.

Sodium bicarbonate was administered. Five hours after admission, the patient developed hypotension (80/70 mm Hg) and started desaturating to 79% on 40% oxygen by venturi mask. Subsequently oxygen was increased to 60%. He also received intravenous fluid therapy, furosemide, ranitidine, cefotaxime, dexamethasone.

By 1 am, the next morning, the patient became unresponsive, sustained an acute episode of seizures, blood pressure was not recordable and sustained a cardiopulmonary arrest. Cardiopulmonary resuscitation (CPR) was initiated and 2 doses of adrenaline (1 mg each) were administered. Endotracheal intubation was achieved quickly. He had return of spontaneous circulation (ROSC) within 15 minutes. Mechanical ventilation was initiated with synchronised intermittent mandatory ventilation (SIMV) mode, with set ventilator rate of 15 breaths/min, inspired oxygen concentration (FiO_2) of 100% and then reduced to 60%, tidal volume of 480 ml (8 mL/kg), positive end-expiratory pressure of 5 cm H_2O and pressure support of 12 cm H_2O. Noradrenaline infusion was started at 150 ng/kg/min. His heart rate was 100 beats/min but the blood pressure and oxygen saturation continued to be unrecordable. At 8:45 am, an external jugular venous line was inserted on the right side. Four units of fresh frozen plasma were transfused. Injection Vitamin K 10 mg was administered stat and 8th hourly thereafter. The investigations showed haemoglobin of 11.3 g/dL, urea 41 mg/dL, creatinine 3.3 mg/L, serum sodium and potassium of 157 mmol/L and 3.9 mmol/L respectively. Serial arterial blood gas analysis ABG showed severe metabolic acidosis which was treated using intravenous sodium bicarbonate and N-acetyl cysteine. Patient had a cardiac arrest. Rhythm check revealed asystole and CPR was initiated. Injection adrenaline 1 mg (3 doses) was administered and spontaneous circulation resumed after 20 minutes. An ECHO was performed which showed right ventricular dysfunction (RV) dysfunction and cardiomegaly. During the third cardiac arrest, the patient could not be resuscitated.

Discussion

The poisoning in Gloriosa superba is attributed to cochicine which is the major active alkaloid. The toxic effect of the drug was documented since late third century BC. During sixth century AD, the drug was prescribed to treat joint pain. In 1773, Benjamin Franklin in the USA introduced the drug to treat gout. In 1820, Pellsiter and Caventou isolated colchicines from colchicum for the first time. [3-5]

The drug has antimitotic activity and targets cells with high metabolic rate and turn over which explains the increased susceptibility of the interstitial epithelium, hair follicles and bone marrow.[3]

The lethal dose of colchicine is not known. While some patients have survived ingestion of 60 mg, some have succumbed to 7 mg of colchicine.[4] The early signs of toxicity develop within two hours of ingestion and include vomiting, diarrhoea, numbness and severe effects on the throat leading to dehydration.[5] The poisoning can lead to respiratory, hepatic and renal failure individually and can also progress to a multiorgan failure within 24 to 72 h. The toxic effects of the drug also causes bone marrow depression, disseminated intravascular coagulation, haemolytic anaemia, arrhythmias, neuromuscular disturbances, paralysis eventually leading to coma and death. The late toxic effect includes alopecia and dermatitis.

The management of the victim begins with general treatment for patient with poisoning. There is no specific antidote; there is no added benefit by using colchicines-specific Fab (fragment antigen binding) fragments.[6] Decreasing the systemic availability of the drug and supportive management is the mainstay of therapy.

It is also important to educate the citizens regarding the toxic effects of the plant and probably refrain from using it because of its narrow therapeutic effects.

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