Case Report

Acute Toxic Encephalopathy caused by Gloriosa Superba Poisoning - A Case Report

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Abstract

Background: A flowering plant seen widely in Southeast Asia and India is gloriosa superba. The poisoning most commonly caused by it is self-poisoning. All part of the plant is toxic. The toxic substance in the seeds and tubers of this plant is colchicine.
Case report: In this report, a case of self-poisoning of gloriosa superba poisoning by ingestion of the tuber with an intention of deliberate self-harm is reported. A 29 year old female, had presented with features of Leukocytosis, Pancytopenia, Metabolic disturbance, Alopecia and Acute Toxic encephalopathy.
Conclusion: The patient was managed with multi dose activated charcoal MDAC, N -acetyl cysteine (NAC), antiemetics, proton pump inhibitors (PPIs), IV Fluids, Inj. Potassium chloride and Magnesium sulphate, G-CSF (granulocyte colony stimulating factor) injection, plasmapheresis, 3%Nacl and antiepileptic medications, patient clinically improved.

Key words

Gloriosa superba, Poisoning, Colchicine, toxic encephalopathy.

Introduction

The suicide rates are high in India and southern states have higher suicide rates than the northern states [1]. Due to the easily available and accessibility to the pesticides and plant poisons, they remain the most commonly consumed poisons among rural areas. Gloriosa superba is an ornamental, tropical plant found in many parts of south India [2, 3]. Poisoning can occur by ingestion of seeds or tubers, which have the toxic principle alkaloid compound colchicine [4]. The most common symptoms of colchicine poisoning are vomiting, diarrhoea. It can cause renal, hepatic failures and bone marrow suppression which can lead to thrombocytopenia and disseminated intravascular coagulation [5, 6]. Colchicine can also affect the neuromuscular system and cause myotoxicity [7, 8]. However, there is a lacuna on the data on the neurological manifestations of the gloriosa superba poisoning in south India.

Case report

A 29 years old female with no previous known co-morbidities was brought to Emergency Department by her husband with alleged history of self-consumption of Gloriosa Superba Tuber (one tuber grinded and mixed in water). The patient was brought to hospital after four hours of consumption. Following consumption, the patient had history of abdominal pain, nausea and non-bilious vomiting. There was no history of LOC, seizure, headache, giddiness, blurring of vision and muscle weakness, chest pain, palpitation, breathing difficulty and cough and fever, loose stools, dysuria and decreased urine output. On clinical examination, the patient was conscious, vitals stable. Patient was admitted in ICU.

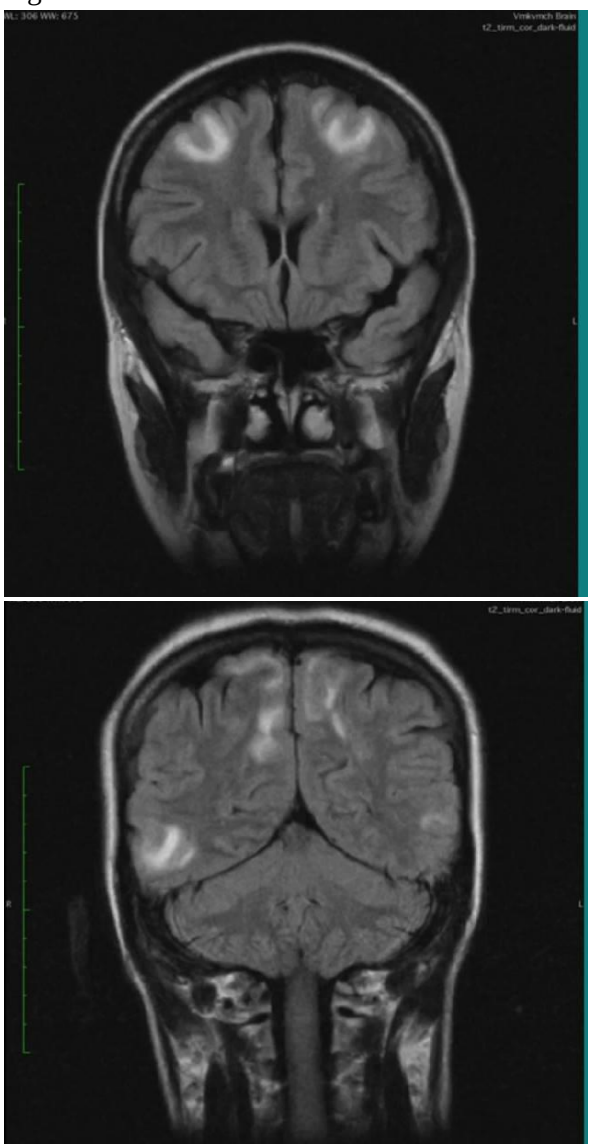
All routine investigations were sent and all blood routines were within normal limits except leukocytosis. Gastric lavage was done and started on MDAC, NAC, antiemetics, PPIs, IV Fluids and other supportive care. IV antibiotics and intralipid emulsion were also started. On day 1, plasmapheresis was done. Patient was clinically stable. On day 2, Patient had hypokalemia (1.9mEq/L) and hypomagnesemia (1.3 mEq/L) and was treated with IV Inj. KCl and Magnesium sulphate. Blood picture on day 3 showed thrombocytopenia and anaemia. Patient was clinically stable and there were no signs of bleeding tendencies. Patient was put on continuous monitoring and vitals were observed throughout the day. On day 5, patient had melena. Stool occult blood was negative and all coagulation profile was within normal limits. Peripheral smear on day 6 revealed pancytopenia and G-CSF was given. Psychiatrist opinion was sought and diagnosed as adjustmental disorder and started on SSRIs (Selective Serotonin Reuptake Inhibitors). On day 7, patient had complaints of hair loss (alopecia) and fever with chills and rigor. Management of fever was done and echo showed multiple ectopic foci during study. On day 8, patient had GTCS and treated with antiepileptics. Serum electrolyte showed hyponatremia, In view of symptomatic hyponatremia treated with 3% saline. In spite of correcting hyponatremia patient developed another episode of GTCS, while other causes has been ruled out and subjected to MRI brain showed, Acute Toxic Leukoencephalopathy started on IV antioxidants and nutritive supports. On day 10, patient condition improved, all blood pictures were within normal limits, no further episodes of seizure. Patient was kept in admission for 2 days for observation and was discharged on day 12. The follow-up was done through telephonic conversations, which revealed the patient was comfortable and developed no other symptoms.

Discussion

The initial presentation of the patient revealed gloriosa superba toxicity affecting mainly the gastrointestinal system. Toxic encephalopathy manifestations started after few days. This case highlights a delayed toxic encephalopathy following Gloriosa poisoning. Based on the confirm history of consumption of gloriosa superba and after ruling out all other possible causes of encephalopathy, we conclude that the

delayed toxic encephalopathy was caused by colchicine poisoning only. The limitation of this case report is that we lacked the facilities to study the serum levels of colchicine. With the previous case reports available in literature, patients those who had poisoning had confusion, seizures, but all such cases have concomitant renal or liver dysfunction [9, 10]. In contrast to this, our patient did not have major metabolic derangements. MRI brain showed symmetrical subcortical white matter oedema in bilateral frontal, parietal, posterior temporal and occipital region of brain (**Figure - 1** and **Figure – 2**).

Figure – 1, 2: MRI Cuts showing Symmetrical subcortical white matter oedema in bilateral frontal, parietal, posterior temporal and occipital region of brain.



The previous reported neurological manifestations include ascending polyneuropathy and toxic encephalopathy [11, 12]. In Sri Lanka, a cohort study among 4556 poisoning patients, 2.5% were due to plant poisons and among them 44% were Gloriosa superba poisoning [13]. Toxicity typically follows three phases of symptoms, Phase I occurs within 24 hours and predominantly is gastrointestinal, phase II symptoms lasts for 2 to 7 days and include multiple organ system dysfunction and fatality can occur during this phase. Phase III occurs after a week of consumption, where the patient slowly recovers and there can be features of significant alopecia [2].

The treatment of poisoning is mostly symptomatic and various other methods have been tried to manage symptoms like plasma exchange, intravenous lipid emulsion. This study has shown that the colchicine toxicity can present with delayed toxic encephalopathy and appropriate diagnosis and timely treatment with supportive care can lead the patient towards recovery.

Ethical statement: We obtained written informed consent from the patient in order to publish her clinical information and the photographic materials without divulging his identity.

Consent: Written informed consent was obtained from the patient for publication of this Case report and any accompanying images.

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