Toxic encephalopathy due to colchicine—*Gloriosa superba* poisoning

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ABSTRACT

*Gloriosa superba*, a flowering plant widespread in South and Southeast Asia, is implicated in many cases of self-poisoning. Colchicine is concentrated in the seeds and tubers and this mediates its toxicity. We describe a 28-year-old woman who developed delayed encephalopathy after eating *G superba* tubers. MR scan of the brain showed bilateral symmetrical T2 basal ganglia hyperintensities in the caudate and lentiform nuclei. The delay in onset of encephalopathy is attributable to a direct-effect colchicine, probably mediated through its effect on microtubular transport.

BACKGROUND

*Gloriosa superba*—the climbing lily—is a flowering plant (figure 1) found in equatorial countries, such as India and Sri Lanka.1 It is one of the commonest self-ingested plant poisons in Sri Lanka.2 Poisoning can follow ingestion of seeds and tubers (figure 2), which contain the alkaloid compound colchicine.3 Colchicine poisoning causes vomiting and diarrhoea, hepatic and renal failure and bone marrow toxicity, with pancytopenia and disseminated intravascular coagulation.4 5 It can also alter neuromuscular function6 and cause myotoxicity.7 However, there are few data on the neurological manifestations of *Gloriosa* poisoning.

CASE REPORT

A 28-year-old woman, previously healthy, presented with abdominal pain, diarrhoea and profuse vomiting. Six hours before, she had eaten *G superba* tubers when attempting to end her life after a domestic dispute. On admission, she was stable; we gave activated charcoal and treated...
her symptomatically. After initially improving, on the 5th day she developed generalised tonic-clonic seizures. She was hypocalcaemic, with serum ionised calcium of 0.9 mmol/L (1.1–1.4). We gave intravenous calcium and anticonvulsants and achieved seizure control. On day 7, her level of consciousness declined to a Glasgow coma scale score of 6 out of 15. There were no focal neurological signs. Her full blood count, arterial blood gas tensions, urinalysis, erythrocyte sedimentation rate, serum C-reactive protein, urea, electrolytes, glucose, ammonia, calcium and magnesium were normal. Her serum transaminases were transiently mildly elevated: serum aspartate aminotransferase 278 U/L (1–31) and alanine aminotransferase 216 U/L (5–35). CT scan of head, lumbar puncture, ultrasound scan of pelvis and abdomen were normal. Serology for Epstein-Barr virus, cytomegalovirus, Japanese encephalitis (JE) virus, and herpes simplex virus were negative. EEG showed bilateral diffuse slow waves, suggestive of encephalopathy.

MR scan of her brain (figure 3) showed bilateral T2 hyperintensities in the basal ganglia. We managed her supportively in the intensive care unit. By day 15, her Glasgow coma scale score had improved to 11/15 and remained static up to the point of the current report. She also developed alopecia.

**DISCUSSION**

This case highlights a delayed toxic encephalopathy following *Gloriosa* poisoning. Based on the unequivocal history of ingestion of *Gloriosa* tubers, the extracranial nervous system clinical features supporting *Gloriosa* toxicity, the temporal profile of the onset of encephalopathy and the exclusion of other neoplastic, immune, infective and metabolic causes for encephalopathy, we conclude that her encephalopathy resulted from colchicine toxicity following *Gloriosa* tuber ingestion. However, we had no facilities to assess serum levels of colchicine. Patients with colchicine poisoning occasionally develop confusion and seizures, but all such cases have concomitant renal or liver dysfunction. By contrast, our patient had no significant metabolic derangement. The other unusual feature of her encephalopathy was the delayed onset, reminiscent of that following anoxic encephalopathy and carbon monoxide poisoning.

MR brain scan findings of bilateral T2 hyperintensities in the caudate and lentiform nuclei of the basal ganglia in this patient and preparation of the paper for submission.

**Figure 3** MRI evidence of bilateral T2 hyperintensities in the basal ganglia—lentiform and caudate nuclei.

**Contributors** KG, RG, PW and MC were involved in the management of the patient and preparation of the paper for submission.
Competing interests None.

Patient consent Obtained.

Ethics approval Ethics review committee, National Hospital of Sri Lanka.

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