Cerebral Venous Thrombosis Due to Abrin Toxicity: A Case Report

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ABSTRACT
Abrus precatorius (jequirity bean) is a common cause of accidental or intentional poisoning in the tropics. The data on exact incidence of abrus poisoning is largely insufficient in our country, due to lack of reporting. The estimated lethal dose for humans is 0.1-1 µg/kg. The toxic component is the protein abrin that causes widespread endothelial damage. Abrin causes a variety of manifestations like hemorrhagic gastroenteritis with erosions, hemolysis, acute renal damage, dyselectrolytemia, hepatotoxicity with elevated liver enzymes and seizures. Apart from the common manifestation of hemorrhagic gastroenteritis, patients experiencing mental status perturbations have been identified and documented earlier. There have been previous reports of elevated intracranial tension (ICT) in abrus poisoning, however, the exact cause for this phenomenon had not been elucidated. We herein report a case of intentional A. precatorius poisoning in a young girl that caused cerebral venous thrombosis (CVT).

Keywords: Abrus precatorius poisoning, abrin poisoning, cerebral venous thrombosis, increased intracranial tension

Abrus precatorius seeds are often ingested with suicidal intent in India, however, data on exact incidence is lacking. Abrin is a potentially fatal toxalbumin obtained from the seeds of A. precatorius (jequirity bean, gundumani [Tamil]), which is similar in structure and properties to ricin.

We herein report a case of A. precatorius poisoning with cerebral venous sinus thrombosis and intracerebral bleed that has never been documented earlier.

CASE REPORT
An 18-year-old girl, a school dropout, was brought with an alleged history of consumption of approximately 10-15 crushed abrus seeds on July 31, 2013 at 22:00 hours, following a family dispute. She was given local indigenous treatment and taken to Taluk HQ Hospital, Ulundurpet at 08:50 hours on August 1, 2013. There she was instituted emergency care and subsequently transferred to the Govt. Villupuram Medical College, at 19:35 hours, the same day. Upon arrival, the patient was dehydrated and had recurrent diarrhea and vomiting. She was actively resuscitated with oral and intravenous fluid replacement. Her hydration status improved and vital parameters were stabilized. Her blood counts, biochemical analysis and liver function tests (LFTs) were within normal limits. Serum Na+ 158 mEq/l, K+ 2.3 mEq/l, Cl - 126 mEq/l, HCO3- 26 mEq/l.

Patient's vomit improved but diarrhea persisted despite supportive treatment. On Day 4, patient developed bloody diarrhea for which 1 unit of compatible group blood was transfused. Biochemical analysis revealed serum creatinine of 1.1 mg%, Na+ 143 mEq/l, K+ 2.4 mEq/l, Cl - 110 mEq/l and HCO3- 23 mEq/l. Repeat LFTs including prothrombin time/INR (international normalized ratio) were within normal limits. Her blood pressure was 120/86 mmHg and urine output was 2,100 ml/24 hours. On Day 6, the patient experienced one episode of generalized tonic-clonic seizures. She was managed with intravenous loading and maintenance dose of phenytoin. She became progressively drowsy thereafter with persistent depressed mentation. Fundus examination revealed bilateral papilledema. A noncontrast computed tomography (CT) scan of brain revealed a hyperdense lesion with perilesional edema in left occipital region with hyperdense appearance of sagittal sinus (filled delta sign). The features were suggestive of superior sagittal sinus thrombosis with hemorrhage in left occipital lobe. CT contrast study was advised. Anti-edema and anticoagulant treatment with unfractionated heparin commenced.
On further follow-up, despite adequately titrated doses of more than two anticonvulsants, the patient had recurrent seizures on Days 7 through 12. She also developed right-sided hemiparesis with left gaze preference. Patient’s blood glucose and renal values were not much altered. On Day 13, she showed signs of recovery and seizures stopped. ECG revealed a normal sinus rhythm and was within normal limits. Her platelet counts were consistently >2,00,000/mm³ and coagulation profile showed a normal value for prothrombin time and INR, however, activated partial thromboplastin time (aPTT) was prolonged (Control 24.9 test -29.5). D-dimer was 1,270 ng/ml (elevated), familial defective apolipoprotein B-100 (FDB) was within normal limits. Lupus anticoagulant was negative, aticardiolipin titer was normal. On Day 14, a magnetic resonance imaging (MRI) scan of the brain with magnetic resonance venography (MRV) and magnetic resonance arteriography (MRA) were done, which revealed superior sagittal sinus thrombosis and left occipital hemorrhagic infarct (Figs. 1 and 2).

On Day 15, neurosurgical consult was obtained, wherein she was advised decompressive surgery for the hematoma and increased intracranial tension (ICT). Patient was referred to a superspecialty surgical center on the same day.

On further follow-up, she was admitted to a tertiary care center on the same date. The patient underwent a decompressive left hemicraniectomy for elevated ICT and was placed on supported ventilation in intermediate care unit (IMCU). Patient presently has residual right hemiparesis and aphasia and is on follow-up.

**DISCUSSION**

Poisoning from *A. precatorius* is attributed to the protein abrin that acts by inhibiting protein synthesis intracellularly, thereby causing cell death. The estimated human lethal dose is 0.1-1 mg/kg. Many of the features observed in abrin poisoning can be explained by abrin-induced endothelial cell damage, which causes an increase in capillary permeability with consequent fluid and protein leakage and tissue edema (the so-called vascular leak syndrome).

Abrus poisoning results in a variety of clinical manifestations like hemorrhagic gastroenteritis with erosions, hemolysis, acute renal damage, dyselectrolytemia, hepatotoxicity with elevated liver enzymes and seizures. Previous literature also document rare manifestations like increased ICT with papilledema and autoimmune demyelination. Subrahmanyam et al have recommended routine fundus examination in such patients to detect the same. We herein stress the need for brain imaging in such situations. In a series of 131 patients who presented with papilledema and clinically suspected idiopathic intracranial hypertension, 10% had cerebral venous thrombosis (CVT) when MRI/MRV was done.
performed. Imaging of the cerebral venous system has been recommended for all patients with the clinical picture of idiopathic intracranial hypertension, because the distinction between CVT and idiopathic intracranial hypertension has important prognostic and treatment implications, and the yield of imaging is significant.

However, abrin poisoning resulting in CVT has never been documented earlier. We performed a literature review using search terms in PubMed: (‘Abrin toxicity’ OR ‘abrin poisoning’ OR ‘Abrus precatorius poisoning’) and randomized trial: (Abrin poisoning’ OR ‘Abrus precatorius poisoning’) and could not find a previous reporting of CVT in abrus poisoning. Considering this fact and the frequent occurrence of increased ICT in these patients, we hypothesize that undetected CVT is probably the cause for this phenomenon.

The mechanism by which abrus causes CVT is still a matter of speculation and requires indepth chemoanalysis of its components and further histopathologic or immunologic research to elucidate the bioprocess. A possible explanation could be due to widespread vascular endothelial cell damage and a procoagulant state like dehydration. In our case, although dehydration could be implicated as a potential cause, patient was well-hydrated with adequate urine output. Furthermore, it cannot account for occurrence of increased ICT in other patients documented previously.

The management is purely supportive since there is no specific antidote. If early first aid is instituted by way of gastric lavage and absorbents (activated charcoal), the outcome is favorable. Adequate hydration and maintenance of optimal urine output is desirable to avoid acute kidney injury. Delay in institution of treatment worsens prognosis. Also such patients should be ideally placed under close observation for a minimum period of 72 hours, since the toxic features of abrin can manifest late.

**CONCLUSION**

*Abrus precatorius* poisoning is a common occurrence in the Indian subcontinent. The toxic component is abrin that causes cell lysis by inhibiting protein synthesis. Cerebral venous sinus thrombosis has never been reported in abrus poisoning, although the occurrence of increased ICT has been documented. The objective of this case report is to create awareness about this complication and to anticipate it when dealing with such patients.

**REFERENCES**