Poisoning due to White Seed Variety of *Abras Precatorius*

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### Abstract

We report an unusual case of poisoning involving the white seed variety of *Abras precatorius* that caused serious manifestations in a middle-aged male who had consumed the seeds on the advice of a folk medicine practitioner. He recovered after a prolonged duration of hospital treatment without any subsequent complications or sequelae. The case is being reported on account of its rarity. ©

### INTRODUCTION

Poisoning with abrin, the main toxic principle in *Abras precatorius* (jequirity; Rosary pea), a plant that grows wild in most parts of India has been rarely reported in Indian medical literature, even though cases do get reported periodically from around the world. This plant belongs to the family Leguminosae, and is an ornamental, twining, woody vine which grows to a height of 10 to 20 feet when supported by other plants. Leaves are alternate, compound, feather-like (pinnately divided), with small oblong leaflets. Flowers are numerous and appear in the leaf axils along the stems. They are small and occur in clusters 1 to 3 inches long, usually red to purple, or occasionally white. The fruit is a legume (pea-shaped pod) about 3 cm long containing hard ovoid seeds about 1 cm long.

The most common variety of seed is glossy, bright scarlet, with the area around the hilum (point of attachment) being black. Most cases of poisoning involve the ingestion (inadvertently or deliberately) of these attractive red seeds. However, there are other less common varieties of this plant that produce different coloured seeds: for instance, black with a white spot, and white with a black spot.

We report an unusual case of Abrus poisoning in a man who was "prescribed" the white seed variety as an aphrodisiac by a folk medical practitioner, and suffered serious consequences before finally recovering with supportive medical treatment. The case is being reported because of the rarity of such cases in Indian medical literature, as well as the fact that the patient recovered completely even after significant consumption of the toxin.

### CASE REPORT

A 42 years male was referred to a major hospital in Calicut, South India for advanced care by a local hospital. The patient had been admitted at the latter hospital with bloody diarrhoea and altered sensorium which became progressively worse despite treatment. On admission to the referral hospital, he was found to be febrile and comatose, with depressed reflexes and down-going plantars. Pupils were bilaterally constricted. The pulse rate was 82/min, blood pressure 140/70 mm Hg, and respiratory rate 32/min. On enquiry it was learnt that he had consumed some plant seeds on the advice of a practitioner of an indigenous system of medicine, for aphrodisiac purpose. Within a few hours he developed bloody diarrhoea and abdominal pain, with subsequent altered sensorium and seizures. No attempt had been made at the previous hospital to identify the source of the seeds, and the patient was treated symptomatically before being referred out.

The attending physician at the second hospital contacted the Poison Control Center at Cochin, a neighbouring city, which confirmed that the seeds were those of *Abras precatorius*, albeit a less common variety, being mostly white with a black spot ("eye") near the hilum (Fig. 1). Meanwhile, the patient was admitted to the intensive care unit and placed on ventilator, since he had developed breathing difficulty. Chest X ray was however essentially normal, and auscultation revealed nothing grossly abnormal. Cardiac monitoring did not reveal evidence of significant rhythm disturbances. A lumbar puncture disclosed evidence of haemorrhage with increased protein and lymphocytes. There was no indication of neck stiffness, and Kernig’s sign was negative. Fundoscopy did not reveal any abnormality. The blood picture was essentially normal (Hb: 14.2 gm/dL; TC: 21600 cells/cu mm; DC: N79, L15, M6; Platelets:

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2.67 L/cu mm; HCT: 42%), while urine analysis revealed mild proteinuria and occasional RBC. Liver function tests demonstrated nothing significant. AST was slightly elevated (60 units/dL). Serum creatinine was 1.6 mg/dL. Other biochemical investigations were within normal limits (with mild electrolyte changes), while a routine toxicology screening did not turn up anything. Investigations for common infections such as malaria, tuberculosis, and viral fevers were negative. Tests were also done to rule out HIV infection and microbial food poisoning.

Previous medical history was unremarkable, and the patient had been in reasonably good health until the time of the present incident. He was not an alcoholic, but smoked 80 to 100 cigarettes per day.

During the course of his stay at the referral hospital (2 weeks), the patient had two episodes of seizures in the first few days, and had depressed sensorium with respiratory compromise. He was placed on broad spectrum antimicrobials (meropenem, ceftriaxone, acyclovir), anticonvulsants (fosphenytoin, phenobarbitone, valproate), and supportive therapy. Over the next few days he became progressively afebrile and did not suffer further attacks of seizures. His sensorium rapidly improved and he was taken off the ventilator, and discharged without any sequelae, with instructions to report back in the event of recurrence of symptoms.

**DISCUSSION**

The main active principle in *Abrus precatorius* is abrin which is a toxalbumin very similar to ricin found in castor seed. It is a lectin composed of two polypeptide chains (A and B) connected by a disulfide bridge. This basic structure of two peptide chains linked by a single disulfide chain is similar to that of botulinum toxin, tetanus toxin, cholera toxin, diphtheria toxin, and insulin.4

Like castor, the seeds of abrus are harmless when ingested whole, since the hard outer shell resists digestion. However, chewing or crushing of the seed before swallowing will enable the toxins to be released. Abrin is a powerful gastrointestinal toxin, and one of its polypeptide chains (B) binds to the intestinal cell membrane, while the other chain (A) enters the cytoplasm. Once in the cell, the A chain acts on the 60S ribosomal subunit, preventing binding of elongation factor 2, thus inhibiting protein synthesis and leading to cell demise.5 The usual fatal dose is reported to be just 2 to 3 seeds for an average adult.6 In the case being reported however, the patient claimed to have taken “several” seeds, though he did not recollect the exact number. It is not clear as to whether the variety of the seed involved in our case (white with black eye) has a different toxic profile as compared to the usual reported variety (red with black eye), though the literature does not document any differences. While abrus poisoning generally causes severe vomiting and abdominal pain, the patient in this case did not provide any history of vomiting, though he suffered moderate abdominal pain in the initial stages. Bloody diarrhoea is characteristic of abrus poisoning and was very much evident in this case. Convulsions are also frequently associated with this toxic plant, and the patient in our case did suffer several episodes of seizures. Altered sensorium with depression of central nervous system also appears to be characteristic features. But while several other reports in the past have documented cardiac arrhythmias in severe poisoning, in our case ECG was essentially normal throughout. Miosis was noted by us, but is not said to be a constant feature, and a given case may demonstrate either dilated or constricted or even normal pupils.

Laboratory investigations in our case were consistent with abrus poisoning with evidence of haemolysis, and blood in the stools and urine. However there was no evidence of renal or hepatic damage, though some of the parameters were slightly abnormal.

Toxicological analysis is generally not helpful in cases of abrus poisoning, but thin layer chromatography in this case utilizing the seed extract did correspond to the chromatographic result on the patient’s serum, confirming that the patient had consumed abrus seeds.

Treatment of abrus poisoning comprises timely decontamination (by stomach wash) and institution of supportive measures. There is no antidote, though some investigators mention an antiserum (“anti-abrin” or “jequirityl”) that used to be available in the past. There is no evidence to suggest that such an antiserum was ever available in India, or that it was ever tried. Since the cause of death in most reported cases appears to be renal failure, haemodialysis is advocated in severe poisoning with associated renal compromise. It was not deemed necessary in our case, and treatment was restricted to
stabilization of vital functions, and institution of supportive measures. After a total hospital stay of more than 3 weeks, the patient was discharged without any sequelae, and subsequent follow up over a month has been uneventful.

**REFERENCES**


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**Obituary**

Dr. DR Shah, Managing Trustee of APICON-86 breathed his last on 2nd February 2005. During a professional career spanning over 50 years he served at various places in Rajasthan and abroad. He retired as a Senior Professor and Head of Deptt. of Medicine, RNT Medical College, Udaipur. He was closely associated with Association of Physicians of India for last 40 years and contributed immensely towards its activities. He was a renowned Clinician with more than a hundred papers to his credit. During the past 15 years he has Organized two CME Programs every year at Udaipur through the corpus he created after Organizing the API Conference of 1986. He was a long standing member of National Advisory Board of JAPI.

GS Sainani,
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**Book Review**

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