



Case Report

# When the 100 red seeds struck the red button – A case report on survival following intentional ingestion of *Abrus precatorius*

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## Abstract

**Background:** *Abrus precatorius* is among the most lethal natural poisons, with its seeds commonly known as rosary peas, jequirity beans, or crab's eye seeds. The primary toxin, abrin, is an extremely potent ribosome-inactivating protein capable of causing multiorgan dysfunction even at very low doses. Because no specific antidote exists, early recognition and prompt supportive management are essential. Here we discuss a case of intentional ingestion of 100 crushed seeds as per the patient which he got it through online platform and how we managed the patient in a multidisciplinary approach.

**Key words:** *Abrus precatorius*; Crab's eye seeds; *Ricinus communis*

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## 1. Introduction

*Abrus precatorius* commonly known as rosary pea, jequirity bean, crabs eye, is a vine native to India and other tropical and subtropical areas of the world. The plant contains abrin, a highly potent toxalbumin that is structurally and functionally similar to ricin, the well-known protein toxin derived from *Ricinus communis* (castor beans). Notably, abrin is considered even more toxic than ricin, with an exceptionally low lethal dose and rapid onset of cellular injury. The seeds in the crushed form are more toxic, with no antidote and is taken orally for suicidal purpose. An estimated human fatal dose of crushed seeds of *Abrus* is 0.1-1 µg/kg and has caused death after ingesting 1-2 crushed seeds. Most of the patients are from rural areas since this plant is predominantly seen in villages.



*Image Source: Forest & Kim Starr Photographs*

**2. Case Presentation**

A 33-year-old male was presented to, then admitted to, the casualty with intentional ingestion of 100 crushed seeds of *Abrus precatorius* on 21/10/2025. Following ingestion of seeds, he felt multiple symptoms of nausea and abdominal pain (persistent pain around the umbilical and epigastric regions). Gastric lavage was given in the casualty, and he was shifted to HDU care for further management.

Approximately 12 hours after ingestion, he started to have bloody diarrhoea approximately 2 times in 3 hours. There were no symptoms of vomiting, decreased urine output, or fall in BP in the last 4 hours.

**3. On Examination**

On physical clinical examination, the patient was drowsy with vitals of: PR: 80/min BP:110/80 mmHg SpO2:98%RR: 20/min. His abdomen had tenderness over the epigastric and umbilical regions but without abdominal muscle tension or rebound tenderness.

**Lab Investigations**

Lab reports showed;

TC	7850
Hb	14.1
Platelet	3,24,000 lakhs/Cumm
PT-INR	0.96
Stool occult blood test	Positive.

### Liver Function Test

Total bilirubin	1.2 mg/dL
Direct Bilirubin	0.3mg/dL
Indirect bilirubin	0.9 mg/dL
SGOT	27U/l
SGPT	1U/L
Urea	17.2 mg/dL
Creatinine	0.8 mg/dL
Serum sodium	140 mmol/L
Serum potassium	3.3 mmol/L

### 4. Management

USG Abdomen and pelvis showed edematous caecum, ascending colon, and right transverse colon. Central line was put on Right IJV, for administering large volumes of fluid. Repeat lab report after 24 hours showed a rise in PT -INR value as well as fall in albumin level.

Bloody diarrhea continued. He was started on hemoperfusion HP through an emergency indwelling dual-lumen catheter placed in the right femoral vein and total 3 cycle were done. N-acetyl cysteine infusion was given continuously for 3 days with intravenous pantoprazole infusion. Eventually the blood parameters as well as patient's symptoms also improved.

Psychiatry opinion was sought and he was evaluated to have Acute Stress Reaction. No pharmacotherapy was initiated at present and psychological counseling sessions were given. He was shifted to ward on the 6th day of admission and discharged on 9th day. Total protein, serum albumin took nearly 2 weeks to fall into normal range.

### 5. Discussion

Abrin is a highly potent toxalbumin derived from the seeds of *Abrus precatorius*. It is a ribosome-inactivating protein with an approximate molecular weight of 65 kDa and constitutes only a minute fraction of the total seed mass. Structurally, abrin is composed of 2 polypeptide chains—A and B—linked by a disulfide bond. The A chain, roughly 30 kDa, possesses N-glycosidase activity and irreversibly damages the 60S ribosomal subunit, inhibiting elongation during protein synthesis.

This inhibition prevents cellular protein formation and ultimately results in apoptotic or necrotic cell death. The B chain, about 35 kDa, facilitates toxin entry by binding to specific carbohydrates on cell surfaces and enabling internalization of the A chain into the cytosol. Whole, unchewed seeds of *Abrus precatorius* typically pass through the gastrointestinal tract intact and cause minimal harm. However, crushed or chewed seeds release abrin, significantly increasing its bioavailability and toxic potential. Despite the fact that abrin is partially degraded in the digestive tract and demonstrates limited absorption, even small amounts of the intact toxin can cause clinically significant poisoning.

Clinical manifestation includes gastrointestinal symptoms predominantly which is vomiting, nausea, diarrhea, hematochezia, hypovolemic shock, hepatic dysfunction, renal impairment, coagulopathy and also increased intracranial hypertension which can lead to papilledema. Severe cases can rapidly deteriorate, most commonly due to dehydration which is the main factor for mortality and timely intervention is very crucial.

In our case the deranged parameter was PT INR which went increasing from 0.96 to 1.47, total protein decreased from 7.5 to 4 and albumin 4.5 to 2.1. Liver function was primarily affected. Creatinine and urine output were maintained throughout in his case.

The conventional treatments including gastric lavage, purgation, gastric acid suppression by PPI, liver protection, blood volume and electrolytes resuscitation, and hemostasis were performed immediately after admission. To eliminate such a highly toxic agent with a molecular weight of 65 KD, we considered to perform 3 cycles of hemoperfusion. CRRT was not considered since it can't effectively filter high molecular weight toxins like abrin eventhough it can filter creatinine and other solutes. And patient was hemodynamically stable as well, we first went with hemoperfusion.

Hemoperfusion is an extracorporeal technique that filters blood to remove toxic metabolites or exogenous toxins through adsorption and is commonly used in the management of acute poisonings.

Its toxin-removal efficiency is generally superior to that of conventional hemodialysis, as hemoperfusion can effectively adsorb both protein-bound and lipid-bound toxins. In cases of abrin poisoning, hemoperfusion may help reduce circulating toxin levels, thereby mitigating the severity of toxicity and shortening the clinical course.

However, a notable limitation of continuous renal replacement therapy (CRRT) is its relatively slower clearance of many toxins when compared with standard intermittent hemodialysis. Additionally, CRRT typically requires the use of heparin for anticoagulation, which may increase the risk of bleeding and contribute to electrolyte disturbances.

From this case, we derived several important clinical insights which includes

- Early gastric lavage and purgation can help remove residual toxin from the gastrointestinal tract, thereby reducing further absorption.
- Prompt establishment of venous access and continuous monitoring of vital signs are critical and were key factors in the successful management of this patient.
- Maintaining adequate circulating blood volume and ensuring sufficient perfusion of vital organs—including the heart, brain, liver, and kidneys—is essential.
- Early administration of proton pump inhibitors (PPIs) is beneficial in addressing gastrointestinal bleeding. PPIs increase gastric pH, promote platelet aggregation, facilitate fibrin clot formation, and help prevent clot lysis.
- Effective removal of circulating toxins should be prioritized. The use of hemoperfusion highly benefits by enhancing continuous toxin clearance, reducing toxin rebound, and improving overall clinical outcomes. It is beneficial over conventional hemodialysis.

- Even though CRRT is not being used in this case, it can be combined with hemoperfusion especially in hemodynamically unstable patient as well as in severe deranged parameters of Liver, renal systems.
- Liver protectives such as N-Acetyl cysteine infusion can also help for its antioxidant and anti-inflammatory property.

## 6. Conclusion

This case highlights that survival from severe abrin poisoning is possible with timely recognition, aggressive supportive care, and early initiation of extracorporeal blood purification techniques. The use of hemoperfusion contributed to effective toxin removal and stabilization of organ function. Prompt gastrointestinal decontamination, hemodynamic support, and vigilant monitoring were critical to the patient's recovery. This experience underscores the importance of a multidisciplinary approach in managing rare but potentially fatal toxic exposures.

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