An Arrow Poison (Abrus Precatorius) Causing Fatal Poisoning in a Child

Article · March 2016

DOI: 10.7860/JCDR/2016/18234.7439

5 authors, including:

M. M. Patil
BLDE University
12 PUBLICATIONS **55** CITATIONS
SEE PROFILE

Shankar Patil
BLDE University
17 PUBLICATIONS **141** CITATIONS
SEE PROFILE

DOI: 10.7860/JCDR/2016/18234.7439

Case Report

Paediatrics Section

An Arrow Poison (Abrus Precatorius) Causing Fatal Poisoning in a Child

MM PATIL[1], SV PATIL[2], AS AKKI[3], BHAVANA LAKHKAR[4], SHARAN BADIGER[5]

ABSTRACT

Ingestion of whole seeds of Abrus precatorius often does not produce serious illness. It generally presents initially with gastrointestinal manifestations. Haemolysis, acute renal damage, hepatotoxicity and seizures are the other manifestations. Herewith we report a child with accidental ingestion of abrus precatorius seeds who presented with altered sensorium and convulsions. The case is being reported on account of its rarity in its initial neurological manifestation and has not been reported earlier in paediatric age group.

Keywords: Childhood poisoning, Neurotoxicity, Rosary pea seeds

CASE REPORT

A two-year-old boy born by non consanguineous marriage, second in birth order with normal developmental milestones brought with altered sensorium of one hour duration. On enquiry it was learnt that parents have seen the child passing Rosary pea seeds in the stool two day prior to the admission which was neglected by the parents. There was no history of fever, vomiting or pain abdomen or rashes prior to the convulsions. There was history suggestive of pica since the last two to three months.

On admission the pulse rate was 120/min, blood pressure was 100/70 mmHg and respiratory rate was 30/min. He was in altered sensorium, with depressed reflexes and flexor plantars. Both pupils were normal in size, regular and reactive to light. There was bilateral papilloedema. Within few minutes of admission, the child had generalised tonic clonic seizures. The convulsions were treated initially with intravenous dextrose and calcium gluconate. However, convulsions required midazolam and loading doses of both phenytoin and phenobarbitone to get controlled. As the respiration was irregular and there was deterioration in Glasgow Coma Scale (GCS), the baby was intubated and put on ventilator support. Antioedema measures like mannitol, dexamethasone were started. The child was placed on broad spectrum antimicrobials and supportive therapy.

In the next twenty four hours, he developed upper gastrointestinal bleeding without any skin or mucosal bleeds. The complete haemogram revealed microcytic hypochromic anaemia with haemoglobin of 7.2 gm/dl and mild thrombocytosis (platelet count 5.5 lakhs/mm^3). Blood sugar was within normal range throughout the hospitalization. The renal and liver function tests were normal at admission [Table/Fig-1]. The Bleeding Time, Prothrombin Time, Activated Partial Thromboplastin Time, performed at 24 hours was normal. The child was given cold saline stomach wash and intravenous ranitidine. Hypocalcaemia and hypokalaemia were corrected with intravenous calcium gluconate and potassium chloride respectively. Cardiac monitoring did not reveal evidence of significant rhythm disturbances. ABG revealed slight respiratory alkalosis. Chest X-ray was normal. CT scan brain revealed bilateral diffuse cerebral oedema. The child was transfused with whole blood as the child was anaemic and had active gastrointestinal bleeding. The child improved over next twenty four hours and GCS became 12/15. The baby was weaned and extubated over next twelve hours. The child was conscious with normal cardio respiratory function. On fourth day the child went into Acute Renal Failure (ARF) in spite of appropriate fluid therapy. Ultrasound of abdomen was normal. ARF was treated according to standard protocol. The child was shifted to the ward and started on oral fluids and feeds. He was conscious, tolerating feeds. On the eleventh day, the child's general condition suddenly deteriorated. The child was reintubated and ventilated. The child could not be revived.

DISCUSSION

Abrus precatorius, (Rosary pea) a plant that grows wild in most parts of India. All parts of the plant are toxic, but highest concentrations are found in the seeds [1]. The seeds exist in different colours such as black, orange, and most commonly, glossy red [2]. A black band is found at the end of the seed. The colours may attract the child.

Mode of action -The seeds contain Abrin, which inhibits ribosomal function, halting protein synthesis and leading to cellular death. Abrin is known to be the most toxic plant substance. It has two polypeptide chains (A-chain and B–chain) linked by disulfide bond. A-chain removes adenine residue from "28 S" ribosomal RNA thereby stopping protein synthesis. The B-chain facilitates entry of molecule in the cells by acting on cell membrane where A-chain causes toxic effect [3]. It was used as arrow poison in olden days due to this property.

The fatal dose of abrin in human beings is 0.1-1 µg/kg [4]. The crushing of the seeds releases toxin from within the protective outer hard coat of the seeds. This will increase the severity of toxicity and decreases the time to onset of symptoms [5]. The classical delay in onset of toxicity as seen in our case is because of ingestion of intact whole seeds which does not occur when the seeds have been crushed and swallowed.

Following ingestion of the seeds, initial symptoms may occur in less than six hours but usually are delayed for one to three days.

Parameters	Day 1	Day 2	Day 4	Day 5	Day 7	Day 9
Serum Calcium(mg/dl)	8.9	7.6	8.5	8.7	7.0	9.7
Blood Urea(mg/dl)	16	-	101	120	95	98
Serum Creatinine(mg/dl)	0.7	-	2.2	1.9	1.4	1.0
Serum sodium(mEq/L)	127	-	139	142	140	141
Serum Potassium(mEq/L)	2.4	-	5.0	4.5	3.6	2.7
Serum Chloride(mEq/L)	88	-	101	103	101	101

[Table/Fig-1]: Trend of biochemical values during hospital stay.

Haemorrhagic gastroenteritis with erosions is the most common manifestation of toxicity. This will present with vomiting and watery diarrhoea at first, later with bloody diarrhoea and malena [6].

CNS toxicity includes headache, dilated pupils, hallucinations, drowsiness, tetany, and seizures. Demyelination is a rare complication of Abrus precatorius poisoning [7]. Our patient presented with evidence of neurotoxicity – altered sensorium and seizures as the initial manifestation. It could be a manifestation of severe poisoning with Abrus precatorius. In the case being reported the parents have seen the child passing several seeds in the faeces two days prior to the admission, though they have not seen him consuming the seeds. The presence of pica and attractive nature of seeds might have contributed for the ingestion of large number of seeds. Some seeds might have been crushed before being swallowed, that might have resulted in severe poisoning manifesting with neurotoxicity. Abrin-induced cytotoxicity and consequent vasogenic oedema resulting from endothelial cell damage could be the possible mechanism for papilloedema. The altered sensorium and seizures in the patient responded well to cerebral oedema therapies (mannitol, dexamethasone) and anticonvulsant therapy with complete resolution of papilloedema.

Suggested laboratory investigation and treatment- Based upon the toxin's action, suggested investigations include complete haemogram, peripheral smear for haemolysis, renal and liver function tests, serum electrolytes, and coagulation profile. Immediate treatment is mainly supportive and symptomatic with intravenous fluids, correction of electrolytes imbalance and blood transfusion if required. Gastric lavage should be done with caution due to necrotizing action of abrin. While there is no antitoxin available, symptomatic treatment is the mainstay of management.

CONCLUSION

Ingestion of intact seeds of Abrus precatorius might be the probable reason for the delayed and unusual initial neurologic manifestation rather than classical initial haemorrhagic gastroenteritis that will occur when the seeds are crushed and swallowed.

REFERENCES

[1] Shih RD, Goldfrank LR. Plants. In: Goldfrank LR, Flomenbaum NE, Lewin NA, Weisman RS, Howland MA, Hoffman RS, eds. Goldfrank's Toxicologic Emergencies. 6th edition. East Norwalk, Connecticut: *Appleton and Lange*; 1998.
[2] Jang DH, Hofmann RS, Nelson LS. Attempted suicide, by mail order: Abrus precatorius. *J Med Toxicol*. 2010;6(4):427-30.
[3] Olsnes S, Refsnes K, Pihl A. Mechanism of action of the toxic lectins abrin and ricin. *Nature*. 1974;249(458):627–31.
[4] Dickers KJ, Bradberry SM, Rice P, Griffiths GD, Vale JA. Abrin poisoning. *Toxicol* Rev. 2003;22:137–42.
[5] Reedman Lisa, Shih RD, Hung Oliver. Survival after an Intentional Ingestion of Crushed Abrus Seeds. *Western Journal of Emergency Medicine*. 2008;9(3):157-59.
[6] Pillay VV, Bhagyanathan PV, Krishnaprasad R, Rajesh RR, Vishnupriya N. Poisoning due to white seed variety of Abrus precatorius. *J Assoc Physicians India*. 2005;53:317–19.
[7] Ratnakar S, AbdoulHamide S, Deepak A, SrikantNarayana B. Acute demyelinating encephalitis due to Abrus precatorius poisoning – complete recovery after steroid therapy. *Clinical Toxicology*. 2008;46:1071–73.

PARTICULARS OF CONTRIBUTORS:
1. Associate Professor, Department of Paediatrics, Shri B M Patil Medical College, Vijayapur, Karnataka, India.
2. Professor and HOD, Department of Paediatrics, Shri B M Patil Medical College, Vijayapur, Karnataka, India.
3. Professor, Department of Paediatrics, Shri B M Patil Medical College, Vijayapur, Karnataka, India.
4. Professor, Department of Paediatrics, Shri B M Patil Medical College, Vijayapur, Karnataka, India.
5. Professor, Department of Medicine, Shri B M Patil Medical College, Vijayapur, Karnataka, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. MM Patil,
Associate Professor, Department of Paediatrics, Shri B M Patil Medical College,
Vijayapur -586103, Karnataka, India.
E-mail: mmp076@gmail.com

FINANCIAL OR OTHER COMPETING INTERESTS: None.

Date of Submission: **Dec 07, 2015**
Date of Peer Review: **Jan 05, 2016**
Date of Acceptance: **Jan 07, 2016**
Date of Publishing: **Mar 01, 2016**