**Abrus precatorius, Gaertn — An Ayurvedic Potent Phytomedicine**

Sirs,

The case report of poisoning, due to white seeds of *Abrus precatorius, Gaertn*, reported in the journal (JAPI 2005;53:317-19) is a reminder that Ayurvedic remedies just like allopathic drugs, can be toxic with their improper use. The report mentions a “folk medical practitioner” in the introduction and refers to a practitioner of an indigenous system of medicine “in the case report. This is confusing. We, as a nation, have officially adopted pluralistic medical systems, with their respective colleges of medical education, professional councils and research centres. We must not have a prejudice to label all the non-allopathic systems as folk medicine or quackery. When we made the presentations to the Lord Walton Committee of the upper house of the British Parliament, a cogent and scientific plea was made on behalf of evidence-based Ayurveda.1 Lord Walton and the members of the committee were open-minded enough to consider what Lasagna, the father of Clinical Pharmacology considered as different kind of evidence.2

The plant is considered poisonous, particularly the seeds. Hence a process of detoxification is considered absolutely necessary to remove toxicity of seeds. The process involves boiling of pounded seeds in kanji or cow’s milk for 3 hours.3 Heat-denatured abrin looses its haemagglutinating toxicity. The research group, at IIT Kharagpur has done some outstanding research on retention of the immunomodulatory activity of heat-denatured or trypic-digested abrin, while loosing toxicity.4,5 Once again an ancient insight on *Abrus precatorius* and its usage has been validated by state - of - the - art relevant science. This has been the path of major advances in pharmacology, by studying the mechanisms of action of poisons eg ergot, calabar beans, curare etc.6 Recently, in “Science”, RA Mashelkar, Director General of the Council of Scientific and Industrial Research, has re-emphasized this path of Reverse Pharmacology in drug research.7 One of us (Ashok DB Vaidya) had earlier the privilege to deliver Sir Ram Nath Chopra at Indian Pharmacology Society, on the subject of Reverse Pharmacology of medicinal plants which brings the clinical observations and experiential data centre-stage for further research.8 Patwardhan et al have shown how this path can be very productive for biomedical research on natural products.9

The Ayurvedic and pharmacological properties of different parts of Abrus precatorius have been well described. Table 1 lists selected uses and activities, confirmed by research in modern pharmacology.

A physician may wonder how such diverse activities have been reported in different parts of a single plant. The chemical constituents of Abrus precatorius have been identified. For example, the leaves, which are widely consumed with pan (betel-leaf) contain 10% glycyrrhizin. Several triterpenol saponins, flavonol glycosides, isoflavaquinones, abrine (alkaloid), pentosans, anthocyanins, etc have been isolated and studied pharmacologically.

The poisoning by the seeds of *Abrus precatorius* has been reviewed and reported often in literature. Death has been reported with twenty seeds bended with water. The symptoms included vomiting of blood, severe pain in the eyes and burning of ears. Death ensued in two days.10 Death in children has been reported from ingestion of one or two seeds.11 The human fatal dose of abrin - the toxalbumin is 0.1-1.0 microgram/kg. Recently Duckers et al were concerned about the use of abrin as potential chemical warfare agent.12 As far as the management of Abrus poisoning is considered, it is essentially symptomatic and supportive. The use of strong broad-spectrum antibodies like meropenem, ceftriaxone and acyclovir, reported by Pillay et al, in the case report has no evidence-based rationale. It is of interest to note that eminent Ayurvedic physician V.M. Gogte has mentioned in his textbook that the antidotes are the juice of Amaranthus (Tandulja) and sugar.13 This needs to be investigated in an experimental toxicity study.

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**REFERENCES**


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**Table 1: Putative mechanisms of actions of parts of *Abrus precatorius***

<table>
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<th>Ayurvedic Uses</th>
<th>Mechanisms of Action</th>
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<tr>
<td>Antipruritic</td>
<td>Urticaria, eczema</td>
<td>Mast-cell membrane stabilizer¹²</td>
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<tr>
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<td>Stomatitis, conjunctivitis</td>
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<td>Hair-growth promotive</td>
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<td>Analgesic</td>
<td>Migraine</td>
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<td>Antitumour</td>
<td>Lymphomas/Leukemia</td>
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<tr>
<td>Antipyretic</td>
<td>Malaria</td>
<td>Antiplasmodial¹⁴</td>
</tr>
<tr>
<td>Abortifacient</td>
<td>Dysmenorrhoea</td>
<td>Antiprostaglandin activity¹⁵</td>
</tr>
</tbody>
</table>
Sir,

It is not clear as to what message is intended to be conveyed in the letter by Vaidya ADB et al regarding our case report on poisoning due to white seed variety of Abrus precatorius. We reported the case highlighting the relatively less common variety of seed (white with black "eye") compared to the almost ubiquitous red seed with black "eye" reported in most cases of abrus poisoning. Our intention was only to alert physicians to this rare variety of abrus poisoning. Regarding its toxic profile, we only wondered as to whether three could be significant differences between the two types. Apparently the toxic profile is essentially similar (as demonstrated in our case).

What however, Vaidya et al appear to have completely misunderstood in our case report is that the patient did not consume any "Ayurvedic" preparation made out of abrus, but instead consumed some seeds of this plant (on the advice of a practitioner of "folk medicine" or "herbal medicine"), which had not been subjected to any process of "detoxification". Isn't "folk medicine" an indigenous form of medicine? One must not hasten to infer pejorative allusions where none is intended. All the authors of the case report work in a place where Ayurveda is held in high esteem.

Regarding the use of antibiotics in our case, this was tried as an empiric measure before it could be conclusively established that the underlying cause was plant toxin-related and not microbial in nature.

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Inferior Vena Cava Obstruction and Hepatocellular Carcinoma: An Uncommon Association in India

Sir,

An association between hepatocellular carcinoma and membranous inferior vena cava obstruction has been reported, but is uncommon in India. We report a 21-year-old man with inferior vena cava (IVC) obstruction that was treated with IVC stenting. He presented four months later with upper gastrointestinal bleeding and was found to have hepatocellular carcinoma. He had portal vein thrombosis so transarterial chemotherapy (TAC) was offered but he refused and was lost to follow up after a month.

Membranous obstruction of IVC (MOVC) is a common cause of Budd Chiari syndrome (BCS) in Asia and South Africa. Its reported incidence in India is 30%-46%.1 MOVC is a recognized risk factor for hepatocellular carcinoma (HCC) especially in countries like South Africa and Japan.

There is only one earlier report of its association with HCC from India.2

A 21-year-old man, non-addict, presented in April 2003 with history of haematemesis 1 year back. He had dilated, tortuous veins over the lower limbs, flanks and back with firm hepatomegaly, splenomegaly, but no ascites. Investigations revealed normal liver enzymes and proteins, and deranged prothrombin time (19/13 s). Viral markers were negative. Serum ceruloplasmin and alpha-fetoprotein levels were normal. Endoscopy showed 3 small esophageal varices.

USG with colour Doppler showed coarse liver, splenomegaly, no ascites. There was complete cutoff of splenomegaly, no ascites. There was complete cutoff of
the terminal IVC with dilated proximal IVC and multiple collaterals. IVC gram showed complete IVC membrane with high proximal pressure and multiple collaterals. Hepatic veins were normal. Balloon membranoplasty and self-expanding metal stent placement was done in Sept 03.

He was readmitted in Jan 04 with bleeding esophageal varices and was started on endoscopic variceal sclerotherapy (EVS) schedule. Haemogram and liver function tests at this time were normal. Colour Doppler showed patent stent with normal flow in IVC with portal vein thrombosis and two small lesions (< 2 cm) in segment VIII of the liver. CT-abdomen showed the same. Alfa-fetoprotein level was 1156 ng/mL (n- 0-30). USG-guided biopsy from the lesion showed HCC. In view of associated portal vein thrombosis he was offered TAC but he refused and lost to follow up after a month.

Budd Chiari syndrome is defined as obstruction of the hepatic venous outflow which includes obliteratorive vena caval lesions such as a membrane or occlusive long-segment lesions. Membranes may be complete, partial, thick or thin, and may have a central hole or fenestrae. The genesis of these membranes is still debated. Recent reports suggest it to be an acquired lesion.

The reported incidence of HCC complicating MOVC is 4.6%-47.5%. In one South African study 20% of patients with HCC were found to have underlying MOVC. The mechanism of carcinogenesis proposed is prolonged and persistent injury due to congestion and the time lag described ranges from years to decades.

The uncommon association in India, with only one earlier case report raises questions about the risk of HCC with MOVC. The short span between presentation and development of HCC in our patient as well as in the patient reported by Karia et al may suggest other complicating factors.

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REFERENCES

Hypertension and Colicky Pain as The Presenting Features in ‘Acute Intermittent Porphyria’

Sir,

The article “Acute Intermittent Porphyria in a Kumhar Community of Western Rajasthan” by Sachdev et al which appeared in February 2005 issue of JAPI was interesting. Acute Intermittent Porphyria (AIP) is an inborn error of metabolism with extremely varied clinical picture, diverse and apparently unrelated manifestations chiefly pertaining to the gastrointestinal and neuropsychiatric system. Around 177 cases have been reported from all over India till today out of which 128 were from Rajasthan. Hypertension as a presenting feature in AIP is an uncommon occurrence. Sachdev et al and Gauri et al did not come across any case of hypertension in their epidemiological studies of AIP. Bhargava et al and Mundhara et al have reported presence of hypertension along with other manifestations in few cases of AIP. We would like to report a case of AIP which we came across recently who presented for treatment of hypertension without other clinical manifestations of AIP.

A 26 year old female was referred for control of hypertension and persistent tachycardia from 2 months. She had no past history of hypertension, diabetes mellitus or any other major illness. She had 3 full term normal deliveries. She was detected to have hypertension after she had undergone tubectomy 2 months ago. She gave history of intermittent colicky pain in abdomen which used to subside without treatment since last 2 months. There was no history of passage of red coloured urine.

On examination she was found to have pulse rate of 104/min and BP was 150/104 mm of Hg. There were no other positive findings on clinical examination. Her investigations revealed a normal haemogram. Liver function tests, renal function tests and serum electrolytes were normal. Chest X-ray, ECG and USG abdomen and pelvis and the fundus examination were normal. Routine

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urine examination did not reveal any abnormality except for the red colour of the sample. Freshly voided sample of urine was found to be colourless and after exposure to sunlight for few hours was noticed to change its colour. The diagnosis of AIP was put forth and Urinary porphobilinogen was estimated which was found to be raised 25.5 mg/dl (normal 0-2 mg/dl). She was treated with Atenolol 25 mg daily to which she responded.

Her pulse rate and BP came to normal. On follow up she did not require any antihypertensive agents and was doing well without any symptoms. In this case AIP was precipitated by the surgical procedure she had undergone 2 months ago. The other factors which may precipitate an attack of AIP include starvation or low caloric diet, infections and use of certain commonly used drugs like oral contraceptives, phenobarbitone, sulfonamide antibiotics and valproic acid.

The case report emphasizes the need of general awareness and importance of correct diagnosis so that patients counselling can be done and attacks can be prevented which at times may progress to life threatening neurological damage.

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REFERENCES


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