

MELIA AZEDARACH POISONING

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Poisoning in man due to plants had been known for centuries, in some cases occurring by accident and in others by intent. Perhaps the most important and illustrious historical example of poisoning is that of Socrates, who, when the time came for him to die, fearlessly took the cup of hemlock from the hands of his weeping jailor and then walked about, carrying on normal conversation, until the poison took effect.

Plant poisoning is a common problem because of its universal and perennial occurrence. More important still, it is little realised that many plants, although quite harmless to man and animal under one set of conditions, can, under another, prove to be most toxic and noxious. It is for this reason that a case of poisoning by a relatively common plant *Melia azedarach* Linn. is reported. (Fig. 1)



Fig. 1. *Melia azedarach*.

This plant is commonly mistaken for the relatively harmless *Melia indica*, a medicinal plant which finds wide use among the local Indians. (Figs. 2a, 2b, 2c) It is little surprise that such a mistake should arise as even among the botanists much confusion existed till of late as to the uses and toxic properties of both. (In fact, the patient's relatives brought the *Melia indica* when asked to produce a sample of the poisonous plant and gave it to us as the *Melia azedarach*.)

However, no local case of poisoning arising from the mistaken use of *Melia azedarach* has been reported and it is hoped by this article to induce the local Chinese, Indian and Malay communities to exercise greater care in the practice of herbal medicine.

CASE REPORT

MV, an 18 year old Indian female, complained of dysmenorrhoea. She was given a concoction prepared from the bark of a tree which was ground up, mixed with water and squeezed dry. She received this at 8 a.m. and about 15 minutes later, complained of dizziness. She then fainted at 8.30 a.m., half an hour after the ingestion. She was seen at 9.30 a.m. by the attending medical officer at the Maxwell Road Outpatients' Dispensary. On examination, the patient was found to be comatose. All reflexes were absent. The pupils were dilated and fixed. Her pulse was 100/min. and B.P. 110/80. Later at 9.40 a.m. the patient collapsed and her B.P. was not recordable. She was also gasping. After resuscitation was started, she was admitted.

In hospital, the patient was still in deep coma, not responding at all. The pupils remained dilated and fixed. She was flaccid in all limbs. The deep tendon reflexes were absent but the planter response was bilaterally flexor. Fundi: normal. Patient was not breathing on her own but had good circulation. B.P. 140/80 and pulse 100/min., regular and of good volume. The abdomen was very distended.

The patient remained in a state of coma from the time of admission till death, three days later. Her respiration was all the time maintained by a bird's respirator. At 8 p.m. on the day of admission, she was noticed to have slight increase in muscular tone and slight twitching movement of all limbs. Pupils were slightly smaller but still unreactive. From then on, her progress was one of decline. Despite hydrocortisone and pressor agents her B.P. could not be maintained. Body temperature was subnormal and she was never breathing on her own. She was not given any antidote or drugs with possible antagonistic actions until the plant from which the extract was prepared was identified. This was found to be the plant *Melia azedarach* (Dr. W.L. Chew) and as some of its products have been reported to cause symptoms like those of atropine poisoning, neostigmine and edrophonium were tried, but without success. Three days later the patient died.

The relevant laboratory investigations were as follows: Hb. 10.4gm.%, TW 14,400 (P93.

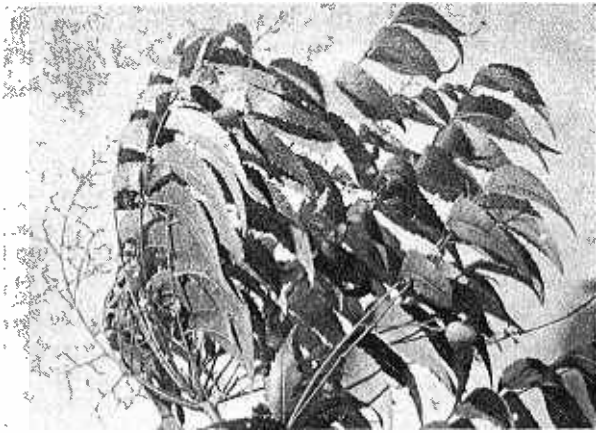


Fig. 2(a). *Melia indica* showing leaves, fruit and flowers.



Fig. 2(b). Flowers of *Melia indica*.



Fig. 2(c). Fruit and leaves—*Melia indica*.

L6, M1, EO); the urine had 20-30 wbc, 10-15 rbc, + albumin and hyaline and granular casts; blood urea 16 mgm. %; alkali reserve 41 vol. %, pH 6.98, pCO₂ 26.9 mm. Hg.; serum potassium 2.6 mEq/L, sodium 140 mEq/L and chloride 108 mEq/L.

At postmortem examination (Dr. S.K. Ting) the following were found: *Brain*: this was oedematous. The gyri were flattened and sulci obliterated. There was marked coning and uncal herniation. The brain substance was soft with necrosis of the midbrain and haemorrhages in the left caudate nucleus. (Fig. 3) *Lungs*: Both were congested and oedematous, with 400 ml. blood in the left pleural cavity and 100 ml. in the right. *Gastrointestinal tract*: The abdominal cavity contained 200 ml. blood-stained fluid. The stomach was distended with gas and blood-stained fluid. There were two areas of ulceration on the anterior wall with bulging of the wall at the weakened parts due to the distension. (Fig. 4) The jejunal mucosa showed patches of haemorrhages and whitish discoloration. The ileum and large bowel were congested. (Fig. 5) *Liver*: This showed areas of yellow discoloration. (Fig. 6) *Kidneys*: Both were congested.

DISCUSSION

From the above, it can be seen that the bark of the plant *Melia azedarach* contains a toxic substance which takes effect soon after ingestion. The symptoms which appeared in rapid succession were severe dizziness with fainting, followed by collapse, mainly of the respiratory system. The cardiac function was temporarily excited with subsequent suppression. Abdominal distension was a prominent feature. The dilated and fixed pupils and the transient excitatory phase also suggested atropine-like reactions though antidotes against atropine were ineffective.

These and other symptoms have been described in animal and man who has been poisoned by the plant. Confusion arises, however, because of a wide symptom complex as various parts of the plant cause different effects in the same species and the same part causes different effects on different species. All parts of the plant have been described to cause poisoning but no case of poisoning from ingestion of the bark products has been reported. Symptoms referable to the gastrointestinal tract include nausea, vomiting, diarrhoea and abdominal distension.

The main pathological changes were in the gastrointestinal tract, with inflammation and

hyperaemia as the outstanding features. There was some degree of softening and fatty degeneration of the liver. Cerebral haemorrhage and softening were also seen.

MELIA AZEDARACH LINN.

As reported by Chopra (1), this plant is found mainly in warm climates, especially in India, but it grows also in other parts of Asia, South Africa and Australia. (3). Different parts of this plant have been used as poison and as medicine. As the former, it was mainly employed as a stupeficient narcotic in fishing but as medicine its use has varied around the world. In South Africa, the Europeans apply it to eczematous skin lesions; in the Mauritius Islands, the root bark is used as an anthelmintic; whilst in Algeria its use as a tonic and anti-pyretic has been reported. (3). Nath (2) described the use of an aqueous extract of the heartwood to relieve asthma, and the Indians (1) have found antispasmodic, emetic and emmenagogic actions.

Watt (3) quoted the first incidence of poisoning by the tree from Sim's report (1907) of a trial at Port St. John's in 1899 for the murder of an African woman who died after using a decoction of the bark as an enema. In another incidence at Lourenco Marques, an African was reported ill from drinking a decoction of the leaves, the symptoms being severe stomatitis, marked oliguria and sanguinous vomiting. In Southern Rhodesia, children were reported poisoned by taking the fruits. A peculiar finding was malignant ulcers of the nose which developed when the children forced the fruit in. (This might be due to the mechanical irritation).

Carratala (4) in 1939 described the death of a three year old child some days after eating fruits from the Paradise Tree, as the *Melia azedarach* is sometimes called.

ANIMAL EXPERIMENTS

Steyn and others, as reported by Watt (3), have done extensive animal research using swine, sheep, goat, poultry and cow as subjects. Of all, the swine is the most susceptible and two main groups of symptoms were seen. The first, gastrointestinal, consisted of nausea, vomiting, violent colic, tympanites, diarrhoea and thirst and was caused by ingestion of the seeds. The second, respiratory and neurological, was seen as coma, depressed respiration, marked dyspnoea, laboured and irregular breathing, gasping, cyanosis and tachycardia; all these being caused by taking the drupe. Thus it can be seen that

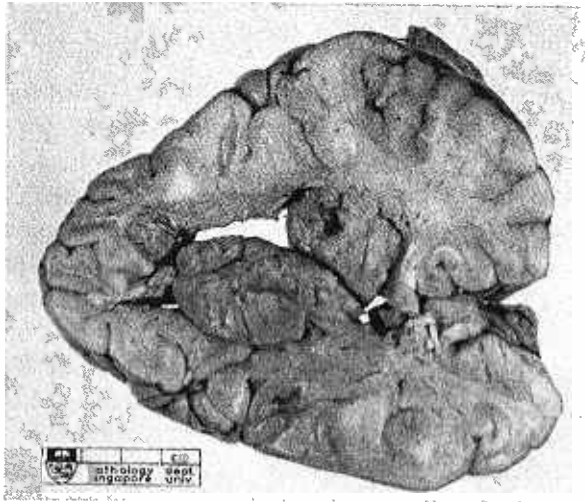


Fig. 3.

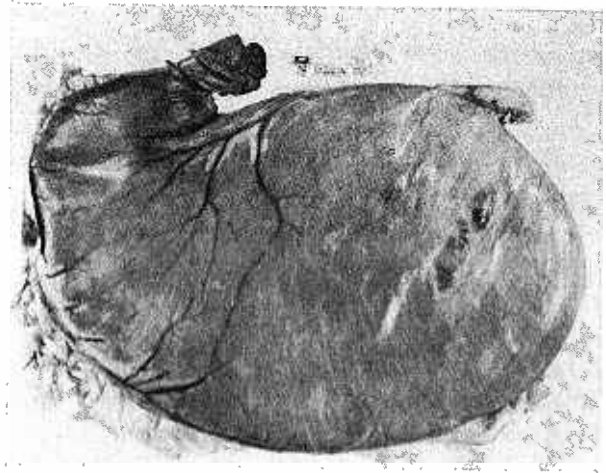


Fig. 4.

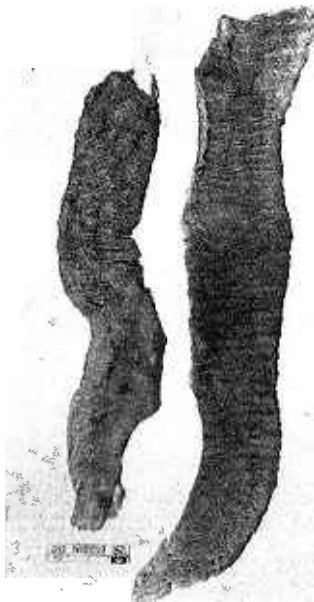


Fig. 5.

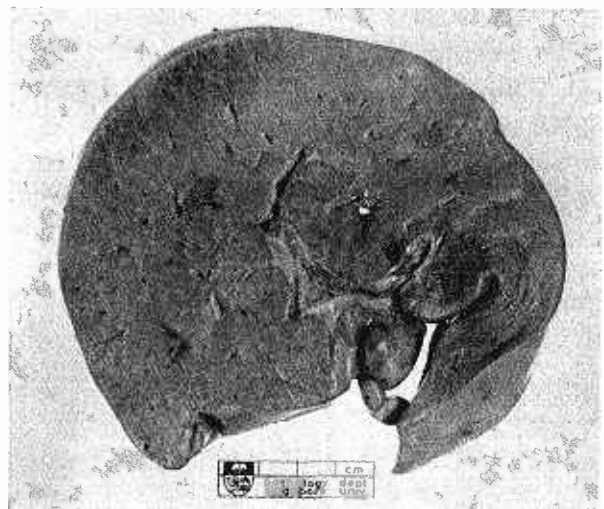


Fig. 6.

different parts of the same plant cause different reactions on the same species.

Carratala in 1939, after the report of poisoning in a child, prepared an aqueous extract of the fruit and found that subcutaneous injection of 1 ml. into the rabbit caused dyspnoea, tremor, convulsions and death. When given orally, it produced gastrointestinal symptoms.

The toxic principle would appear to be an alkaloid azaridine (4), tannin, bakacactone, and certain oils and proteins. (3).

TREATMENT

There is no specific treatment or antidote and the management is chiefly symptomatic though de Wildeman has been quoted by Watt (3) as having achieved an effective remedy with sugary drinks when gastrointestinal symptoms supervened.

SUMMARY

A case report of poisoning by *Melia azedarach* in an Indian female is made, stressing the importance it bears on local herbal medical practice and the dangers of indiscriminate prescribing. The symptomatology and pathology are discussed. A short account of the *Melia*

azedarach is given including a historical review of human poisoning and animal experimentation and toxicology. Treatment is at best symptomatic.

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