CASE REPORT

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A Deadly Alternative

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Abstract

Solanum nigrum is a species in the genus Solanum. It is also known as black night shade. It has been widely used as food in various parts of the plant world. In South India, its leaves and berries are routinely consumed as food (Mannathakalli keerai, Red Makoi). Senna occidentalis is a species in the family Fabaceae. In India, the weed is widely prevalent (known locally as Bana chakunda). Both S. occidentalis and S. nigrum are used commonly in the alternative system of medicine for various chronic conditions. Both of them can cause liver failure in high doses. We are reporting a case of Solanum nigrum and Senna occidentalis overdose presenting as subacute liver failure, tachyarrythmia and ultimately leading to death.

Key words: Solanum nigrum; Senna occidentalis; alternative medicines; hepatotoxicity; liver failure; tachyarrhythmia.

Case report

A 34-year-old male, resident of New Delhi, carpenter by occupation, presented to the medical emergency with symptoms of fever and jaundice for 3 weeks, and epigastric pain and vomiting for 7 days. He was apparently well 3 weeks back when he developed progressive jaundice, epigastric pain, fever and vomiting. There was no history of alcohol intake or recent travel. There was no past history of jaundice or blood transfusion also. On examination, he was conscious and oriented. Icterus was present. Abdominal examination revealed hepatomegaly (2 cm below costal margin) but spleen was not palpable and there was no free fluid on percussion. Respiratory, cardiovascular and nervous system examination was unremarkable. Initial investigations revealed leucopenia, thrombocytopenia and deranged liver function tests. Viral markers, serologies for infection and autoimmume profile were negative. All the investigations have been summarised in Tables I and II. On further inquiry, later in the course of hospital stay, his relatives gave a history of consumption of large amount of Ayurvedic medications (containing *Solanium nigrum* and *Senna occidentalis*) for atleast 3 weeks prior to his illness (Fig. 1). He was taking this to promote better digestion.

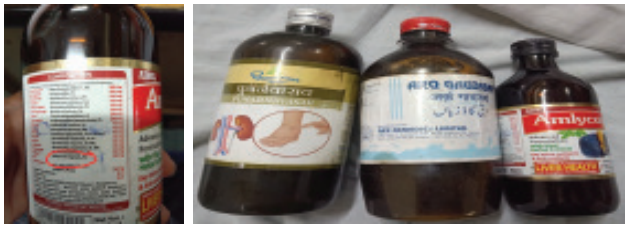

Fig. 1: *Bottles showing the contents of the Ayurvedic preparations being consumed by the patient.*

Even with supportive management, the patient's liver and kidney functions gradually deteriorated. He had an episode of ventricular tachycardia on day 7 of hospital admission and despite best efforts, succumbed to his illness.

Table I: Routine investigations of the patient.

Parameters	Day 1	Day 3	Day 5	Day 7
Haemoglobin (gm/dl)	11.2	9.4	9.3	9.7
Total leukocyte count (per ul)	2,700	2,170	1,290	1,400
Platelet count (per mm^3)	56,000	70,000	68,000	89,000
Blood urea (mg/dl)	73	48	48	89
Serum creatinine (mg/dl)	1.1	0.4	1.1	2.0
Sodium (Na^+) (meq/l)	136	134	132	131
Potassium (K^+) (meq/l)	4.1	4.2	3.8	4.2
Total bilirubin (mg/dl)	6.5	9.4	12.3	15.7
Direct bilirubin (mg/dl)	2.7	6.0	7.2	6.6
SGOT (U/L)	552	166	445	800
SGPT (U/L)	212	266	510	853
ALP (U/L)	173	55	215	108
Total protein (gm/dl)	6.1	–	–	5.3
Serum albumin (gm/dl)	2.3	–	–	2.2
INR	1.6	1.5	1.2	1.3

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Table II: Further investigations done to look for cause of liver failure.

Urine routine examination: Normal
Blood and urine cultures: No growth.
HIV: Negative. Hbs Ag: Negative, HCV Ab: Negative
Hepatitis A antibody: Negative; Hepatitis E antibody: Negative
Peripheral smear for malarial parasite: Negative; Dengue serology: Negative
Leptospira and Rickettsia serology: Negative
Slit lamp examination: No KF ring; Serum ceruloplasmin: Normal.
Autoimmune profile (AMA, ANA, cANCA, pANCA,SLA): Negative.
Ultrasound of abdomen: Liver -16.5 cm, grade I fatty liver, No intrahepatic biliary dilatation.
Gall Bladder: Normal; Common Bile Duct/Portal Vein: Not dilated; Spleen: 13 cm Pancreas: Head appears normal; UB: Distended
Kidney: Normal size/echotexture, CMD maintained, no calculus. Mild free fluid seen
Chest X-ray: Normal study
ECG: Normal.

A post mortem liver biopsy was done (Fig. 2) which showed disarray in hepatic architecture. Hepatocytes showed steatosis (60 - 70%).There were numerous apoptotic bodies with areas of spotty necrosis. Portal areas showed mild lymphoplasmacytic inflammation with neutrophils. Bile duct injury was seen. Mild interface activity was seen. Lobules showed marked lymphoplasmacytic infiltrate. No significant fibrosis was seen. A final diagnosis of poisoning and death due to overuse of Ayurvedic preparation containing the hepatotoxic and cardiotoxic compounds - *Solanum nigrum* and *Senna occidentalis* was proposed, based on the temporal progression after a definite history of ingestion of these compounds in large doses, and absence of a credible alternative diagnosis.

Discussion

Solanum nigrum (black night shade berry) is widely used in the alternative system of medicine as antioxidant, antipyretic, anti-inflammatory and antitumorigenic (Figure 3). The active compound is Solanine. The estimated toxic dose of Solanine is 20 to 30 mg and estimated fatal dose is 400 to 500 mg. In mild to moderate toxic doses, Solanine is irritant to the throat and presents mainly with gastrointestinal symptoms. Fever may be an early sign of poisoning. Drowsiness and fatigue may develop. Blurred vision can be present due to mydriasis from it's anticholinergic effects. Most clinical effects are seen within 2 to 24 hours of ingestion. In fatal doses, Solanine may cause liver failure, neurological involvement, arrhythmias and respiratory failure. In severe cases, neurologic events may include: drowsiness, apathy, confusion, weakness, and hallucinations, that may be followed by unconsciousness. Tachycardia and other anticholinergic effects have been seen. Both tachypnea and respiratory failure may occur, but

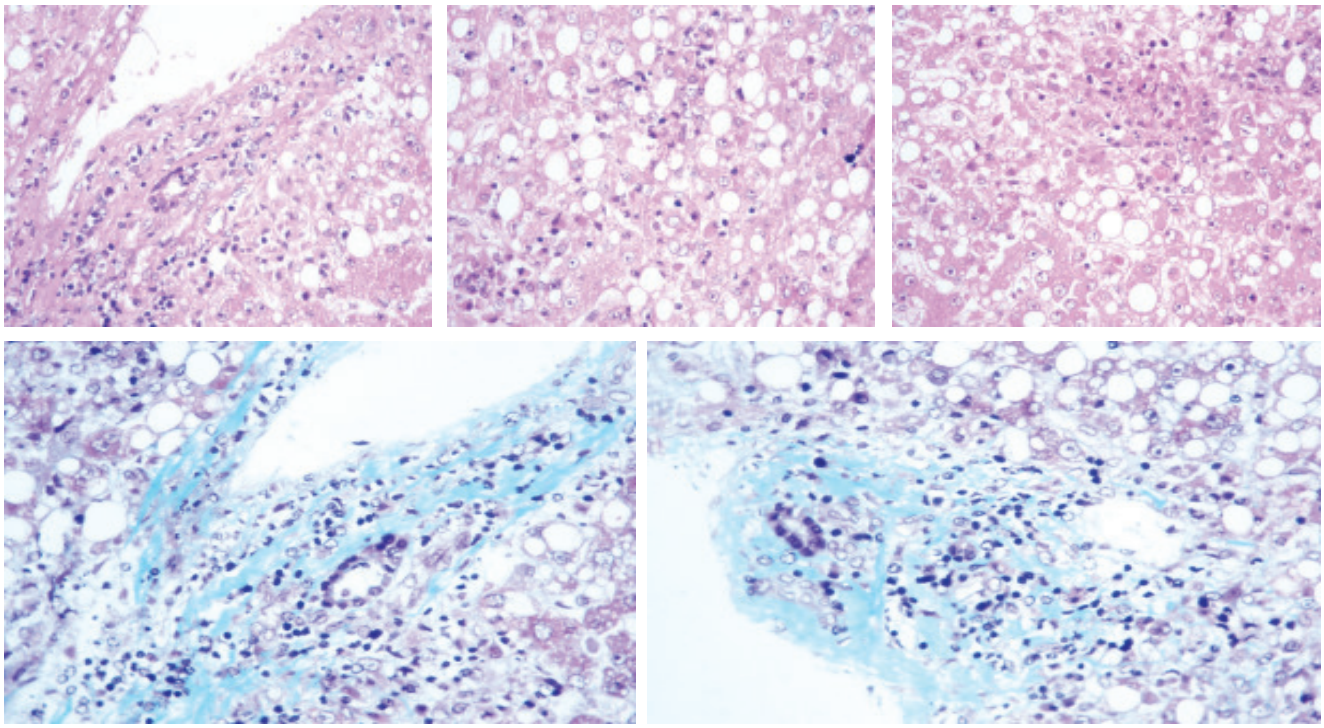

Fig. 2: *Post-mortem liver biopsy of the patient showing marked steatosis and inflammatory infiltration.*

Fig. 3: *Solanum nigrum and Senna occidentalis plants.*

are uncommon. No specific antidote is available for Solanine poisoning. Treatment is only supportive.[1]

An outbreak of Solanine poisoning was reported among 78 schoolboys in South London. They became ill after eating potato at lunch on the second day of the autumn term. Seventeen of the boys required admission to hospital. The gastrointestinal, circulatory, and neurological findings and the results of laboratory investigations were in keeping with Solanine poisoning. The amount of Solanine in the potato waste recovered after the meal was quite excessive.[2]

Senna occidentalis is also used in alternative systems of medicine for chronic illnesses like arthritis, malignancy, chronic pain, diabetes, fungal infections of skin (Figure 3). The active compound is anthraquinone. A case series of an outbreak of posioning from Uttar Pradesh was reported in 2007. In this case series *Senna occidentalis* (also known as *Cassia occidentalis*) poisoning in children affected mainly three systems–hepatic, skeletal muscles and brain. Almost all children had definitive history of exposure to Senna consumption.[3]

More case reports of *Senna occidentalis* poisoning in the literature also suggest that it mainly affects liver, kidney, brain and skeletal muscles[4-8]. No specific antidote is available for senna poisoning. Treatment is again supportive.

Our case presented with subacute liver failure which may be caused by both *Senna* or *Solanum* consumption. All other possible causes of liver failure were looked for and ruled out in our patient. Finally, the temporal association of ingestion of the toxin was established before making diagnosis of *Senna* and *Solanum* poisoning. Our patient died because of refractory tachyarrythmias which are commonly reported with *Solanum nigrum* toxicity.[1]

Many such cases of *Senna* and *Solanum* poisoning may remain undiagnosed because of lack of awareness among doctors. Both compounds are used commonly in alternative systems of medicine and widely prescribed for various chronic conditions. They are easily available over the counter in chemist shops and even general stores in the market. This case report should serve as an eye-opener and readers may take away 2 important learning points: 1) Always ask for use of alternative drugs among all patients and check the ingredients of these drugs, and 2) Always warn patients that, like any other drugs of any system of medicine, these so-called "*natural and harmless*" drugs are not harmless and benign – they can turn out to be a deadly alternative – as in our case.

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