

Pyrrrolizidine Alkaloids

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1. Name of Toxin:

Pyrrrolizidine Alkaloids

2. Name of Acute Disease:

Pyrrrolizidine Alkaloids Poisoning

Pyrrrolizidine alkaloid intoxication is caused by consumption of plant material containing these alkaloids. The plants may be consumed as food, for medicinal purposes, or as contaminants of other agricultural crops. Cereal crops and forage crops are sometimes contaminated with pyrrrolizidine-producing weeds, and the alkaloids find their way into flour and other foods, including milk from cows feeding on these plants. Many plants from the Boraginaceae, Compositae, and Leguminosae families contain well over 100 hepatotoxic pyrrrolizidine alkaloids.

3. Normal Course of Disease

Most cases of pyrrrolizidine alkaloid toxicity result in moderate to severe liver damage. Gastrointestinal symptoms are usually the first sign of intoxication, and consist predominantly of abdominal pain with vomiting and the development of ascites. Death may ensue from 2 weeks to more than 2 years after poisoning, but patients may recover almost completely if the alkaloid intake is discontinued and the liver damage has not been too severe.

4. Diagnosis of Human Illness:

Evidence of toxicity may not become apparent until sometime after the alkaloid is ingested. The acute illness has been compared to the Budd-Chiari syndrome (thrombosis of hepatic veins, leading to liver enlargement, portal hypertension, and ascites). Early clinical signs include nausea and acute upper gastric pain, acute abdominal distension with prominent dilated veins on the abdominal wall, fever, and biochemical evidence of liver dysfunction. Fever and jaundice may be present. In some cases the lungs are affected; pulmonary edema and pleural effusions have been observed. Lung damage may be prominent and has been fatal. Chronic illness from ingestion of small amounts of the alkaloids over a long

period proceeds through fibrosis of the liver to cirrhosis, which is indistinguishable from cirrhosis of other etiology.

5. Associated Foods:

The plants most frequently implicated in pyrrolizidine poisoning are members of the Boraginaceae, Compositae, and Leguminosae families. Consumption of the alkaloid-containing plants as food, contaminants of food, or as medicinals has occurred.

6. Relative Frequency of Disease:

Reports of acute poisoning in the United States among humans are relatively rare. Most result from the use of medicinal preparations as home remedies. However, intoxications of range animals sometimes occur in areas under drought stress, where plants containing alkaloids are common. Milk from dairy animals can become contaminated with the alkaloids, and alkaloids have been found in the honey collected by bees foraging on toxic plants. Mass human poisonings have occurred in other countries when cereal crops used to prepare food were contaminated with seeds containing pyrrolizidine alkaloid.

7. Target Population:

All humans are believed to be susceptible to the hepatotoxic pyrrolizidine alkaloids. Home remedies and consumption of herbal teas in large quantities can be a risk factor and are the most likely causes of alkaloid poisonings in the United States.

8. Analysis in Foods:

The pyrrolizidine alkaloids can be isolated from the suspect commodity by any of several standard alkaloid extraction procedures. The toxins are identified by thin layer chromatography. The pyrrolizidine ring is first oxidized to a pyrrole followed by spraying with Ehrlich reagent, which gives a characteristic purple spot. Gas-liquid chromatographic and mass spectral methods also are available for identifying the alkaloids.

9. Selected Outbreaks:

There have been relatively few reports of human poisonings in the United States. Worldwide, however, a number of cases have been documented. Most of the intoxications in the USA involved the consumption of herbal preparations either as a tea or as a medicine. The first patient diagnosed in the USA was a female who had used a medicinal tea for 6 months while in Ecuador. She developed typical hepatic veno-occlusive disease, with voluminous ascites, centrilobular congestion of the liver, and increased portal vein pressure. Interestingly, the patient

completely recovered within one year after ceasing to consume the tea. Another herbal tea poisoning occurred when *Senecio longilobus* was mistaken for a harmless plant (called "gordolobo yerba" by Mexican Americans) and used to make herbal cough medicine. Two infants were given this medication for several days. The 2-month-old boy was ill for 2 weeks before being admitted to the hospital and died 6 days later. His condition was first diagnosed as [Reye's syndrome](#), but was changed when jaundice, ascites, and liver necrosis were observed. The second child, a 6-month-old female, had acute hepatocellular disease, ascites, portal hypertension, and a right pleural effusion. The patient improved with treatment; however, after 6 months, a liver biopsy revealed extensive hepatic fibrosis, progressing to cirrhosis over 6 months. Another case of hepatic veno-occlusive disease was described in a 47-year-old nonalcoholic woman who had consumed large quantities of comfrey (*Symphytum* species) tea and pills for more than one year. Liver damage was still present 20 months after the comfrey consumption ceased.

For more information on recent outbreaks see the [Morbidity and Mortality Weekly Reports](#) from CDC.

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mow@cfsan.fda.gov

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