BERRIES:
Jerusalem Cherry & Wild Black Cherry

The Jerusalem Cherry (*Solanum pseudocapsicum*) is a product of a plant that is grown in several New England states for the appearance of its brilliant red berries. The cherry belongs to the botanical class that includes potatoes, tomatoes, eggplant, and the deadly nightshade. The toxic component of the plant, solanine, is primarily found in the leaves and fruits.

The wild black cherry (*Prunus serotina*) is a product of a plant that grows in the wild. The plant is most commonly found along roadsides and in open forest areas. In Roman times the wild black cherry was known as “the cherry of death” because it was used for executions. It appears that the toxic component, which is a cyanide compound, is found in its leaves and also in the pits of the fruit.1,2

Pharmacology

The toxic component of the Jerusalem Cherry is solanine. It is a glycoalkaloid, which, within the alkaloid structure, contains sugar residues. Solanine tastes bitter and can cause irritation of the throat.3 Due to its structural similarity to cardiac glycosides, there can be some weak toxic effects on the myocardium as well. It is poorly absorbed from the gastrointestinal tract and is rapidly eliminated in the urine and feces. Peak levels of solanine in an animal model were found in the spleen, kidney, liver, lung, fat, heart, and brain at 12 hours post-ingestion.4 There is no known therapeutic use for the Jerusalem Cherry. It is purely grown for landscaping needs and as an ornamental plant.

The wild black cherry contains prunasin, which is a cyanide precursor. This product has some therapeutic uses as a purported herbal remedy, acting as an antitussive, astringent, and a sedative agent. None of these actions has been scientifically proven as effective in humans. In folk medicine the wild black cherry was used to treat colds and other lung ailments.

The pharmacologic mechanism of action of prunasin is not well understood. There is some evidence that it can possibly inhibit the cytochrome P450 enzyme system, leading to potential drug—drug interactions. Doses of 5 to 12 drops are mixed in water or tea and taken orally 2 to 3 times a day. The product is not FDA approved for the treatment of any of these ailments. The stem bark of the plant does have a rating from the US government of being “Generally Recognized As Safe” (GRAS) when used in amounts for flavoring; but this is a “grandfathered” rating that is not based on scientific studies. It is considered possibly unsafe when used on a long-term basis and/or in excessive amounts. The fruit itself and the juice that is produced are not the toxic portion of the plants; it is the leaves and the pits that contain the cyanide.4

Adverse Effects

a. Solanine

The adverse effects and presentation seen with solanine toxicity mostly consist of gastro-intestinal symptoms that are usually seen with cholinergic poisoning. Nausea, vomiting, and diarrhea are symptoms that can range in severity and can mimic viral or bacterial gastroenteritis. Cardiac effects that are seen are a positive inotropic effect, tachycardia, and hypotension. Central nervous system depression can range in severity from drowsiness to coma. Neurologic effects can be severe enough to lead to respiratory depression. Death usually results from severe toxicity due to respiratory failure.1,6 There have been case reports of solanine poisoning following potato ingestion in the 1920’s. School age boys in London had eaten rotten “green” potatoes and within 12 to 14 hours developed symptoms of vomiting and diarrhea. Patients were managed by maintaining fluid and electrolyte status. One subject however went on to develop central nervous system depression and died.7,8

b. Prunasin

The symptoms that are seen in poisoning with the wild black cherry resemble those of cyanide poisoning. Early symptoms include headache, dyspnea, hyperventilation, and anxiety. Late presenting symptoms are vomiting, bradycardia, coma, and convulsions. All of these symptoms may progress rapidly and can result in sudden death.8 There have been case reports of fatalities related to wild black cherry ingestion in reference to the “chokecherry” that were reported in the 1930-1940’s. Children were eating the cherries from a cherry tree and then within hours dropped dead suddenly. Upon autopsy they were found to have a significant amount of wild black cherries and seeds in their intestines, and were confirmed as having died of cyanide poisoning.9

Toxicology

Solanine is an inhibitor of cholinesterase activity therefore increasing the amount of circulating choline. The additional amount of acetylcholine at the cholinergic synapses, which are in the central nervous system, parasympathetic, and some sympathetic nerve endings, will cause excitation that will then be followed by a cessation of transmission in the cholinergic synapses.6 Parasympathetic stimulation is responsible for the symptoms of bradycardia and tremors, myoclonus and increased gastrointestinal motility. The activation of the nicotinic receptors can result in tachycardia. Cardiac toxicity can be associated with cholinergic effects but also because of the structural similarity of solanine to cardiac glycosides such as digoxin.1,6

Through case reports of solanine poisoning and animal trials, some estimates of toxicity are possible. For example 2.8 mg/kg of solanine administered to small animals was shown to produce effects of drowsiness, and mild dyspnea. Lethal doses were
estimated in an animal model as 225 mg/kg. It is estimated that there is 1% of solanine in the fruits of solanine-producing plants. Based on these estimates there can be a considerable range of solanine intake and a range of the potential for toxicity.

The toxic principal of the wild black cherry is prunasin. Prunasin is a cyanide precursor, a cyanojenic glycoside, which after ingestion is then hydrolyzed to hydrocyanic acid (HCN). When absorbed cyanide binds readily with trivalent iron molecules within cytochrome oxidase. This interferes with cytochrome oxidase function and cellular respiration using oxidative phosphorylation. Lactic acidosis and hypoxia result. A level of cyanide can be detected after the ingestion of the wild black cherry. However this is not very useful in the diagnosis of cyanide poisoning. There is not sufficient time to wait for such laboratory results before initiating treatment. The lethal dose of cyanide is estimated to be between 200-300 mg.\textsuperscript{5,6} An extraction of the leaves of the wild cherry tree was found to have a cyanide concentration of 0.25%.\textsuperscript{12} Unlike the Jerusalem Cherry, this cherry has a sweet taste. Therefore there is the potential for the ingestion of a significant amount of the cherry, rendering the patient susceptible to severe toxicity.

Conclusion

This has been a brief review of two potentially toxic berries that indigenous to the New England area. Both of these berries are toxic and can be potentially fatal. Prompt identification of the berry and getting the patient to a health care facility are essential in the order to prevent unnecessary morbidity. The wild black cherry has greater potential for being a problem because it does taste sweet. Therefore children may ingest a large amount. While there is an antidote for wild black cherry poisoning, it is very time sensitive. The Jerusalem Cherry tastes very bitter therefore deterring children from ingesting enough to cause a problem. Public education to parents and caregivers of children about what these berries look like, where they grow, and how the early symptoms of toxicity present can also be important in preventing a potentially dangerous poisoning.

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Management of Toxicity

The medical management of Jerusalem Cherry poisoning consists of supportive care and decontamination measures. Supportive care is necessary in order to manage the fluid and electrolyte losses that patients can experience if they develop severe vomiting and diarrhea. Decontamination with activated charcoal can be used to aid in preventing the absorption of solanine from the gut. In some cases, there is enough nervous system depression such that a patient may need airway management.\textsuperscript{4}

Poisoning by the black cherry also depends on adequate supportive care, decontamination measures, and the antidote for cyanide poisoning. Ipecac can be given to patients in the field. Activated charcoal is given at a health care facility as soon as possible to prevent absorption. Cyanide poisoning can be treated by using the Cyanide Antidote Kit. This kit contains 3 products that are used in the management of cyanide toxicity. Amyl nitrite inhalants are used to oxidize hemoglobin to methemoglobin. The methemoglobin competes with cytochrome oxidase for the cyanide ion; this reaction favors the methemoglobin. The inhalants are used for the period it takes to prepare the 10% sodium nitrite solution that should be given intravenously, also to produce methemoglobin. The last product of the kit is 25% sodium thiosulfate; this is used for convert cyanomethemoglobin to thiocyanate which removes cyanide from the body. There is also direct enzymatic conversion by the mitochondrial enzyme rhodanase to thiocyanate, which is non-toxic and excreted in the urine. An alternative used in other countries but not approved for use in the USA is the combination of hydroxycobalamin and sodium thiosulfate. The hydroxycobalamin combines with the cyanide to form cyanocobalamin, which is vitamin B12.

Decontamination of the gut should be done only after the antidote has been administered due to the time sensitivity that is associated with cyanide poisoning. Oxygen therapy can also be used. However even at hyperbaric pressures, oxygen is not very effective. Some studies have suggested that it can be an adjunctive therapy to the protective effect of the thiosulfate.\textsuperscript{11,12,13,14}

References (First author only)

1. Http://www.arboretum.harvard.edu
Hall AH: J Emer Med. 1987; 5: 115-121