Dr. Nick Connors

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Therapies Old and New

The treatment of cyclopeptide mushroom toxicity includes fluid and electrolyte replacement as needed, given large volumes of gastrointestinal losses due to vomiting and diarrhea. If ingestion is suspected earlier before significant vomiting develops, multidose activated charcoal can be administered and is associated with improved outcomes. In the past, high dose penicillin was recommended with the theoretical mechanism of preventing the transport of alpha-amanitin into hepatocytes and therefore preventing toxic effects. This has not been firmly demonstrated, nor has a significant clinical benefit; penicillin is therefore a second-line agent. Similarly, thioctic acid has been described as a beneficial treatment in case series, though further investigation failed to validate associations with patient recovery. Cimetidine has been helpful in animals, though there is no human data to support its use. Silymarin is an extract of milk thistle (*Silybum marianum*) made up mostly of silibynin, which can inhibit the entry of alpha-amanitin into hepatocytes due to inhibition of the organic anion transporter (OAT). In vitro and animal studies have shown the benefit of silymarin and silibynin administration, and retrospective studies in humans have corroborated these findings. N-acetylcysteine is beneficial for liver failure of any etiology. In cyclopeptide mushroom toxicity, there is no specific benefit of N-acetylcysteine, but it is a sensible additional intervention in cases of acute liver injury. Polymyxin B can compete with alpha-amanitin at binding sites of RNA polymerase II to prevent inactivation and treatment resulted in better outcomes in an animal study. Human data is lacking, though it would be reasonable to administer polymyxin B to a patient who developed hepatotoxicity from cyclopeptide mushroom ingestion.