Dr. Sophie Gosselin

Dr Sophie Gosselin completed her training in Emergency Medicine at McGil University and medical toxicology fellowship with the Centre Antipoison du Québec and the Newcastle Mater Misericordiae Hospital in NSW, Australia, the Royal Infirmary in Edinburgh and St-Thomas Hospital in London, UK in 2003-2005. She is currently a consultant to the Centre antipoison du Québec, an academic emergency physician with McGill University as well as associate chief of the Emergency Department for the CISSS Montérégie Centre, in Québec. She is a co-chair of the EXTRIP workgroup and the chair of the international Clinical Toxicology Recommendations Collaborative. She is a fellow of the American Academy of Clinical Toxicology for which she was the Scientific Committee Chair and Trustee from 2014-2019. Her scientific publications focus on briging together clinical expertise and evidence-based data to produce collaborative knowledge translation tools and clinical guidelines in medical toxicology.

Clinical Presentation, Diagnosis and GI Decontamination

Sophie Gosselin, MD

The gold-standard for assessment of most poisonings requires confirmation of the toxin in a bodily fluid. Unfortunately timely toxin identification is rarely available in the clinical arena. Identification of uneaten or regurgitated mushroom parts or spores by a trained mycologist is useful, in real life, very often either the myologist is absent or suitable mushroom leftovers are absent. Fortunately, patients who have ingested cyclopeptide containing mushrooms present with such characteristic signs and symptoms of toxicity that a well-defined "toxidrome" can be used to identify cases with sufficient diagnostic accuracy to allow clinical research to proceed without biological confirmation of exposure. As a result, most descriptions of patients with cyclopeptide poisoning are based on clinical rather than biological or mycological evidence of ingestion.

The hallmark of cyclopeptide poisoning is a long latency between ingestion and onset of symptoms; typically no earlier than 5-6 hours, but possibly as late as a day or more. Gastrointestinal symptoms are the predominant early findings with virtually all patients having one or more of either vomiting, diarrhea or abdominal pain; vomiting being the most common. The diarrhea is usually non-bloody and may result in significant fluid and electrolyte imbalances. The gastrointestinal symptoms typically resolve as the patient enters what is often referred to as a quiescent phase. Often, on the second day liver function tests are abnormal with increases in aminotransferases and abnormal coagulation parameters. These abnormalities are often transient in mildly poisoned patients. However, in severe cyclopeptide mushroom poisoning, hepatic abnormalities can worsen over the next several days with progression to fulminant hepatic failure. The same time, some patients will develop acute kidney injury and may progress to acute kidney failure. Both acute tubular necrosis from volume loss and hepatorenal injury are described.

The early treatments to prevent toxicity focus on good clinical care. Prevention of the complications associated with severe vomiting with fluid resuscitation and correction of electrolyte imbalances is key. Activated charcoal is a sensible therapy for gastrointestinal decontamination with antiemetics. Vomiting and diarrhea are so common in the early stages that gastric lavage, emesis and or catharsis are not helpful. In one clinical review, the use of activated charcoal was common, though far from universal. Limited evidence supports a role for activated charcoal unless otherwise contraindicated either for decontamination and enhanced elimination because amatoxins are excreted in the bile where they likely undergo enterohepatic recirculation. Moreover, in-vitro data demonstrate adsorption of amatoxins to activated charcoal. For all these reasons, given the low cost, minimal risks in the patient with adequate airway protection and potential benefits it is reasonable to give multiple dose oral activated charcoal over the first 24h. This could be extended to 72 hours post ingestion according to clinical judgement if persistent hepatic dysfunction as amatoxin were described to be in circulation for this time period.