A fatal poisoning from an amatoxin containing *Lepiota*

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Abstract

The mushroom *Lepiota josserandii* Bon and Boif. has been identified as the cause of an unintentional, fatal intoxication in New York. The course of the symptoms, beginning with a 9 h latent period, was similar to what would be expected in a case of *Amanita phalloides*-type intoxication. Despite supportive medical care the victim expired 110 h after ingestion. Thin layer chromatography detected the presence of alpha- and gamma-amanitin and radioimmunoassay confirmed a level of 3.5 mg/gm dry weight of amatoxins in mushrooms from the same location.

Introduction

*Lepiota* is seldom mentioned in American literature as a deadly poisonous mushroom and until now there was no record of a fatality or life threatening intoxication due to this fungus in North America. This appears to have been either good luck or bad reporting as there are some very toxic, small lepiotas in North America as there are in Europe.

Materials and methods

An upstate New York fatality has been traced to *Lepiota josserandii* Bon and Boif. (Fig. 1), a member of the *L. helveola* Bres. complex. The victim was a middle-aged, Finnish-born engineer who was a knowledgeable amateur mycologist with a reputation for being cautious. The mushrooms were gathered under scots pine (*Pinus sylvestris* L.). He identified them incorrectly as *Lepiota excoriata* (Fr.) Quéè and sauteed about 30 of the tiny fruiting bodies and ate them at about 3 p.m. Sunday, October 9, 1983. The taste of the mushrooms indicated nothing out of the ordinary. There were no symptoms until 9 h after ingestion, when nausea, vomiting and diarrhea ensued, but this was not accompanied by abdominal pain or cramping. These symptoms continued into the second day, but they eventually subsided. The victim complained of muscle cramps in his fingers and toes. On the third day he became weak, dehydrated and short of breath.

He was admitted to the hospital about 30 h after ingestion. His physical examination was remarkable for the following. His respiratory rate was 40 breaths per minute, systolic blood pressure 60 mm Hg, abdomen soft and the liver was noted to be markedly enlarged and diffusely tender. His distal extremities were cold and cyanotic. His rectal temperature was normal, his lungs clear and he was awake and responsive. A cardiac exam suggested mitral valve regurgitation.

Initial laboratory findings revealed a hematocrit of 60% (40–49), sodium of 120 meq/L (135–145), potassium of 6.7 meq/L (3.5–5.2), bicarbonate of 7.5 meq/L (21–30), glucose of 39 mg/dl (65–115) urea nitrogen of 82 mg/dl (10–20), arterial pH of...
7.16 (7.37–7.45), and a platelet count of 30 000 mm$^{-3}$ (130 000–350 000). The Prothrombin time and activated partial thromboplastin time were elevated beyond routine measurement capabilities. These findings suggested severe metabolic acidosis, hemoconcentration, coagulopathy and renal and hepatic failure.

The patient was immediately transferred to the intensive care unit where invasive hemodynamic monitoring and ventilatory support were instituted. The patient received large amounts of intravenous fluids, calcium chloride, sodium bicarbonate, and glucose to correct electrolyte imbalance, hypoglycemia and hypotension. He received intravenous doses of methyl-prednisolone, clindamycin, gentamicin and vancomycin. Twelve hours after admission, intravenous dopamine was required for blood pressure support. The patient received fresh frozen plasma and cryoprecipitate in large doses, with correction of the prothrombin time and activated partial thromboplastin time to twice the normal values. Transfusion of red blood cells and platelets was required.

Results

Laboratory tests revealed both impaired hepatic function and evidence of hepatocellular necrosis. Total serum bilirubin rose from 4.7 mg/dl (0.2–1.2) on admission to 6.1 mg/dl, and conjugated serum bilirubin remained stable at approximately 3.0 mg/dl (0–0.4). Serum glutamyl oxgloacetate transaminase rose from 1825 units/L to 2135 units (0–4.1). Lactate dehydrogenase rose from 1630 units/L to 2230 units (60–200). Alkaline phosphatase remained normal. The serum amylose was elevated at 494 s.u./dl (30–194) and fibrinogen was depressed at 68 mg/dl.

Within 30 h after admission (100 h after ingestion), the patient became comatose. Charcoal hemoperfusion was begun as treatment for fulminant hepatic failure. Despite all supportive efforts, the patient expired 110 h after mushroom ingestion. An autopsy performed 2 h after death demonstrated hepatic necrosis consistent with amatoxin mushroom poisoning.

Several other procedures were considered and rejected. Liver transplant was rejected because of the poor condition of the patient and thioctic acid was not advised due to the severity of the toxication and its advanced stage on admission.

The mushroom was identified from the available literature by one of the authors (JHH). This identification was confirmed by Walter Sundberg who pointed out that the Lepiota helveola complex needs further study. Lepiota josserandii is known from the west coast of the U.S. but only a few col-