Lepiota subincarnata J.E. Lange Induced Fulminant Hepatic Failure Presenting with Pancreatitis

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Abstract A 43-year-old woman with a medical history significant only for hepatitis B carrier status presented to an emergency department with generalized weakness, dizziness, nausea, and diarrhea 36 h after eating an estimated 170 g of sautéed *Lepiota subincarnata* J.E. Lange (basionym *Lepiota josserandii*). Laboratory evaluation revealed profound metabolic acidosis with mild transaminitis, mild coagulopathy, and renal insufficiency. Marked biochemical evidence of pancreatitis was present prior to significant hepatotoxicity. The patient ultimately required liver transplantation on hospital day 7 and was discharged home on hospital day 12.

Keywords *Lepiota josserandii* · *Lepiota subincarnata* J.E. Lange · Mushroom poisoning · Pancreatitis · Liver failure · Mushroom · Hepatotoxic · Transplant

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Introduction

Ingestion of amatoxin-containing mushrooms typically induces gastrointestinal, hepatic, and renal toxicity. The amatoxin-containing mushrooms include *Amanita*, *Galerina*, and *Lepiota* spp., with ingestion of *Amanita* spp. being the most common etiology of mushroom-induced hepatotoxicity. Cases of *Lepiota* spp.-induced hepatotoxicity are rare, until recently have been mostly confined to reports from Europe, and have not been reported in the Midwest United States [1–5]. We report a case unique for its occurrence in the Midwest United States and early biochemical evidence of pancreatitis prior to the development of significant hepatotoxicity.

Case Report

A 43-year-old female presented to the emergency department via ambulance with complaints of generalized weakness, dizziness, nausea, and diarrhea. Her past medical history was significant only for hepatitis B carrier status. The patient reported eating approximately 6 oz (170 g) of sautéed mushrooms, 36 h prior to presentation, that she had picked from under a pine tree in the front yard of her suburban Chicago home. She had previously eaten “whitish” mushrooms picked from her yard without complication; however, she had never eaten the variety she ate on the day in question. Six hours after ingestion, she began to have abdominal cramping, and 12 h after ingestion she developed nausea, non-bloody vomiting, and diffuse abdominal pain relieved by diarrhea. Over the next 12–24 h, the patient’s pain subsided but profuse diarrhea persisted. Thirty-six hours after ingestion, the patient had two syncopal episodes prompting the neighbors to call emergency medical services.

When paramedics arrived at the patient's home, she reported feeling generalized weakness, dizziness, and nausea. Her vital signs were BP 50/38, HR 144, and RR 20. Her blood glucose was 100 mg/dL, and sinus tachycardia was present on the cardiac monitor. Intravenous fluids were initiated, and the patient was transported to the emergency department. Upon arrival, the patient's vital signs had improved: BP 114/63, HR 123, RR 28, afebrile, SaO₂ 100% on room air. The physical exam was remarkable for dry mucus membranes, tachycardia, tachypnea, and a benign abdominal exam; however, she had multiple episodes of bloody diarrhea.

Initial laboratory values included an arterial pH 6.98, pCO₂ 17.9 mmHg (2.38 kPa), bicarbonate 10 mEq/L (10 µmol/L), lactate 10.9 mmol/L, anion gap 28 mEq/L (28 mmol/L), lipase 702 U/L (11.7 µkat/L), aspartate aminotransferase 125 U/L (20.84 µkat/L), alanine aminotransferase 107 U/L (1.79 µkat/L), alkaline phosphatase 148 U/L (2.47 µkat/L), prothrombin time 17.4, blood urea nitrogen 35 mg/dL (12.5 mmol/L), creatinine 2.9 mg/dL (221 µmol/L), white blood cell count 22,000/µL (22×10^9 /L), and hemoglobin 16.7 g/dL (167 g/L). A presumptive diagnosis of hepatotoxic mushroom ingestion was made, and the patient was transferred to a tertiary care center with liver transplant capabilities. Digital photographs of the cooked mushroom were sent to a mycologist at the Chicago Field Museum via e-mail, a mechanism established and maintained by staff at the Chicago Field Museum and the

Illinois Poison Center for identification of unknown mushroom species, and identified as *Lepiota josserandii* [6]. Mushrooms collected from the location the patient specified were subsequently identified by expert mycologists as *Lepiota subincarnata* J.E. Lange. This is the more accurate scientific name as compared to its basionym *L. josserandii*, which is more commonly found in the medical literature. The digital images in Fig. 1 are of the mushrooms that were submitted for identification prior to collection.

Intensive care unit management included hydration, electrolyte repletion, vitamin K, N-acetylcysteine, fresh frozen plasma, and cryoprecipitate. The patient continued to have hematochezia throughout her hospitalization. On hospital day 4, the coagulopathy had progressed with a prothrombin time of 129 and fibrinogen of 58 mg/dL (1.71 µmol/L), and the lipase peaked at 716 U/L (11.94 µkat/L). On hospital day 6, the patient developed mild hepatic encephalopathy, asterixis, and abdominal pain secondary to ascites. Her laboratory values worsened despite aggressive volume resuscitation and attempted correction of her coagulopathy: lactate 11.8 mmol/L, aspartate aminotransferase 4,206 U/L (70.12 µkat/L), alanine aminotransferase 2,920 U/L (48.68 µkat/L), lactate dehydrogenase 5,793 U/L (96.57 µkat/L), and prothrombin time >140.

On hospital day 7, the patient underwent successful orthotopic liver transplantation without intraoperative

Fig. 1 a–d *L. subincarnata* J.E. Lange at various stages of the life cycle. The images were taken at the location the patient described as having picked the ingested mushrooms. The mushrooms pictured were collected and identified by expert mycologists

complications. Pathological examination of the explanted liver revealed panacinar necrosis consistent with amatoxin. Her postoperative course was complicated by non-oliguric acute renal failure with a peak creatinine of 2.8 mg/dL (213.5 μ mol/L). She was started on immunosuppressives, transferred from the ICU to a surgical floor on hospital day 11 (postoperative day 4), and discharged to home on hospital day 12 (post-operative day 5).

Discussion

Of the amatoxin-containing mushrooms, *Lepiota* spp. were not known to have been associated with poisoning in North America until the mid-1980s. Since that time, one fatal case of *L. josserandii* ingestion (New York) and three non-fatal cases of other *Lepiota* spp. (New York, Northern California) have been reported [7–9]. This case of *L. subincarnata* J.E. Lange toxicity is unique for its occurrence in the Midwest United States and for documentation of significant biochemical evidence of pancreatitis preceding hepatic failure.

Amatoxins are heat-stable bicyclic octapeptides that interfere with RNA polymerase II [10]. The primary sites of toxicity are the gastrointestinal mucosa, liver, and proximal tubules of the kidneys, with hepatotoxicity being the most consequential effect. Elevated pancreatic enzymes in the setting of hepatotoxic mushroom ingestion are rarely documented in the literature, and a clear mechanism for pancreatic injury has not been demonstrated. The only case of *L. josserandii* ingestion demonstrating this phenomenon documented an elevated amylase of 494 s.u./dL (10–194) in the setting of markedly elevated transaminases; a lipase was not reported [7]. The remaining four cases involve *Amanita* spp. ingestions, with only two clearly documenting an association: lipases of 1,339 and 334 with normal to slightly elevated transaminases, respectively, in two patients [11].

This case supports the concept that amatoxin-containing mushrooms, specifically *L. subincarnata* J.E. Lange, may

be pancreatotoxic. However, further study is required before any conclusions can be made in this regard.

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