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Case Report

Acute Aflatoxin-Related Hepatitis Presenting as Unexplained Multi-Organ Failure in the Emergency Department

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Abstract

Acute aflatoxin-related hepatitis is rare and may go unrecognized due to nonspecific early symptoms and unreported dietary exposure. We describe a 63-year-old man who presented in critical condition with abdominal pain, vomiting and profound metabolic acidosis, alongside markedly elevated liver enzymes and signs of emerging multi-organ failure. Initial evaluation did not reveal any toxic ingestion, delaying diagnostic orientation. Only after admission, a more detailed history uncovered the consumption of spoiled lentils days before symptom onset, raising suspicion for acute aflatoxicosis. Supportive management, including glutathione therapy, led to full clinical recovery. This case underscores the need for careful dietary history in unexplained fulminant hepatic failure and highlights aflatoxin exposure as a potential but often overlooked cause, particularly when spoiled or imported foods are involved.

Keywords: Acute aflatoxicosis; Aflatoxin B1; Fulminant hepatic failure; Mycotoxin poisoning; Oxidative liver injury; Hepatotoxicity; Dietary exposure; Aspergillus contamination

Introduction

Acute aflatoxicosis is a rare but severe toxic hepatitis resulting from ingestion of foods contaminated with aflatoxins, potent mycotoxins produced by *Aspergillus flavus* and *Aspergillus parasiticus*. While chronic low-level exposure is widely recognized for its carcinogenic potential and association with hepatocellular carcinoma, acute high-dose ingestion can cause abrupt and profound hepatocellular injury [1]. Clinically, patients may present with nonspecific gastrointestinal symptoms followed by jaundice, coagulopathy, encephalopathy, and, in the most severe cases, fulminant hepatic failure [1]. Most documented outbreaks originate from regions where food preservation and safety monitoring are limited; however, acute aflatoxicosis is not restricted to these settings [2-5]. Cases have been described even in highly regulated environments when individuals consume imported or improperly stored foods. In one report, a previously healthy young adult developed fulminant hepatic failure and rhabdomyolysis after prolonged consumption of canned food later found to contain elevated levels of aflatoxin B1, highlighting how exposure may occur unnoticed and lead to life-threatening liver injury [6]. A recent systematic review further underscored the clinical relevance of this condition, reporting mortality rates ranging from 16% to 76% across outbreaks and noting that early symptoms are often subtle and easily misattributed. The review also emphasized substantial variability in diagnostic methods and inconsistent reporting of exposure levels, which contribute to delays in recognition and hinder accurate estimation of global burden [1]. Because acute aflatoxicosis is rarely encountered in everyday clinical practice, particularly outside endemic regions, its presentation may not be immediately recognized. Awareness

of this entity and careful exploration of dietary history remain key elements for timely diagnosis. Here, we describe a rare case of acute aflatoxin-induced hepatitis characterized by an unusual clinical course, contributing to the growing recognition of this underdiagnosed toxicological emergency.

Case Presentation

A 63-year-old man with no known comorbidities presented to our Emergency Department at 3:00 AM with a four-day history of progressive abdominal pain, nausea, and worsening general condition. On arrival, he appeared severely unwell and mildly confused (Glasgow Coma Scale 13). Vital signs were: heart rate 100 beats/min, respiratory rate 16 breaths/min, blood pressure 150/70 mmHg, temperature 36°C, and marked hypoglycemia (glucose 27 mg/dL). The patient was anuric, and urinary catheterization produced only a minimal amount of urine.

Initial arterial blood gas analysis revealed severe metabolic acidosis (pH 7.21, HCO_3^- 8.8 mmol/L) associated with profound hyperlactatemia (16.99 mmol/L). Laboratory testing demonstrated marked systemic injury: white blood cell count 40.23 $\times 10^3/\mu L$ with 90.3% neutrophils, AST > 6000 U/L, ALT > 3300 U/L, LDH > 4500 U/L, total bilirubin 2.63 mg/dL (direct 1.45 mg/dL), alkaline phosphatase 141 U/L, INR 2.67, ammonia 228 µg/dL, CPK 342 U/L, and creatinine 1.9 mg/dL. Procalcitonin was elevated at 4.58 ng/mL.

Given the severity of presentation and the absence of clear etiological clues, an extensive infectious workup was initiated, including serology for hepatotropic viruses, leptospirosis, blood cultures, and procalcitonin measurement. Since the initial suspicion included possible severe sepsis of undetermined origin, empirical broad-spectrum antibiotic therapy with vancomycin and piperacillin/tazobactam (Tazocin) was promptly started.

A contrast-enhanced CT scan of the thorax and abdomen showed no abnormalities in the chest. Abdominal imaging revealed a small amount of intra-abdominal free fluid, diffuse bowel distension, and hyperemia of the intestinal loops, without signs of mechanical obstruction or other surgical pathology.

After fluid resuscitation, a repeat arterial blood gas revealed partial improvement (pH 7.32, HCO_3^- 12.5 mmol/L, lactate 13 mmol/L), and the patient's urine output began to increase, suggesting a prerenal component to the acute kidney injury. However, laboratory tests repeated six hours later showed persistent severe organ dysfunction: WBC 36.48 $\times 10^3/\mu L$ (neutrophils 91.7%, lymphocytes 3.7%), unchanged levels of transaminases, ammonia still elevated at 228 µg/dL, creatinine rising to 2.18 mg/dL, and marked progression of CPK to 1313 U/L.

During the initial hours in the Emergency Department, the patient's history remained non-contributory: he denied recent travel, dietary changes, or ingestion of suspicious foods. Only **after several hours,** following a more detailed interview with family members, it emerged that he had consumed **spoiled lentils 5–6 days earlier,** with symptom onset occurring shortly afterward. This delayed but crucial information raised strong suspicion **of acute aflatoxin-related hepatitis.**

The Transplant Surgery Team was contacted due to the severity of hepatic dysfunction, and the patient was transferred to the hepatology unit. Supportive management including metabolic correction, intravenous fluids, and glutathione administration was continued. Over the following days, the patient showed progressive and sustained clinical improvement, with normalization of liver enzymes, coagulation parameters, renal function, and acid–base balance. His mental status returned to baseline, diuresis normalized, and he ultimately made a full recovery. The patient was discharged home in good condition.

Discussion

Acute aflatoxicosis is an exceptionally rare diagnosis in developed countries, where strict food quality controls limit significant human exposure [2-5]. Nevertheless, the condition remains clinically relevant because its early manifestations typically abdominal pain, vomiting, fatigue, and nonspecific metabolic derangements overlap with numerous causes of acute liver injury [7]. As shown in our case, the absence of an immediately apparent toxic ingestion may delay recognition. Only a targeted dietary history revealed the ingestion of spoiled lentils, prompting suspicion of aflatoxin exposure, a detail that would otherwise have remained unnoticed.

Aflatoxin B1 (AFB1) is the most potent hepatotoxic aflatoxin and exerts its acute toxic effects through multiple synergistic mechanisms. After absorption, AFB1 undergoes bioactivation predominantly via CYP3A4 and CYP1A2 into the highly reactive AFB1-8,9-epoxide, which forms DNA adducts and induces direct genomic injury. In parallel, AFB1 disrupts mitochondrial function by impairing oxidative phosphorylation, reducing ATP synthesis, increasing membrane permeability, and promoting mitochondrial swelling. This mitochondrial dysfunction triggers intrinsic apoptotic pathways mediated by p53 up-regulation, decreased Bcl-2 expression, and Bax translocation to the mitochondrial membrane. These mechanisms collectively explain the profound hepatocellular necrosis reflected in the extremely elevated transaminase levels observed in our patient [7, 9].

Oxidative stress plays a central role in the pathophysiology of acute aflatoxicosis. AFB1 rapidly depletes intracellular glutathione (GSH), generating reactive oxygen species that further damage mitochondrial DNA, proteins, and lipids [9, 10]. The rationale for administering glutathione in this case is supported by the known vulnerability of hepatocytes to GSH depletion and by experimental data showing that restoration of GSH reserves

enhances detoxification of the epoxide metabolites and mitigates oxidative injury [8, 9]. The patient's progressive improvement in liver biochemistry over the subsequent days is consistent with interruption of this oxidative cascade.

Although aflatoxicosis is most commonly reported in large outbreaks in low-income regions where contaminated grains constitute dietary staples sporadic cases in developed settings have been described following ingestion of imported or improperly stored foods. Mortality in acute aflatoxicosis varies widely, with reported rates exceeding 50% during major outbreaks [11]. The favorable outcome in this case likely reflects early supportive management, absence of concomitant hepatic comorbidities, and relatively rapid recognition once the dietary exposure was identified.

Diagnostic confirmation relies on a combination of clinical suspicion, exclusion of alternative etiologies of fulminant hepatic failure, and, when available, laboratory testing for aflatoxin biomarkers such as serum AFB1-lysine adducts. Imaging and routine laboratory parameters are nonspecific, underscoring the indispensable role of a meticulous dietary history. The detection of aflatoxin in the patient's serum in our case provided definitive confirmation, reinforcing the clinical interpretation derived from anamnesis [1].

This case underscores the need for clinicians to maintain a high index of suspicion for aflatoxin exposure when confronted with unexplained acute liver failure, particularly when symptoms follow the ingestion of spoiled, home-stored, or imported food items. Awareness of the molecular mechanisms underlying AFB1 toxicity may aid in understanding the rapid clinical deterioration observed in such patients and support timely initiation of hepatoprotective supportive therapies [1, 11]. As global food distribution networks expand and climate-related factors increasingly influence fungal contamination patterns, isolated cases of aflatoxicosis may become more frequent even in regions previously considered low-risk [7]. Early recognition remains essential to optimize outcomes.

The paucity of reports on acute aflatoxicosis in the medical literature likely reflects both the exceptional rarity of the condition in high-income countries and the substantial diagnostic challenges associated with it. Because the early manifestations are nonspecific and biochemical findings lack discriminatory value, many cases may remain undiagnosed or erroneously attributed to alternative etiologies of fulminant hepatic failure. Furthermore, confirmation requires targeted dietary inquiry and specialized biomarker testing, which are seldom pursued unless exposure is suspected. These factors contribute to significant underreporting and justify the clinical importance of detailed single-case documentation.

Conclusion

his case illustrates that acute aflatoxicosis, though exceedingly rare in developed countries, must be considered in cases of unexplained fulminant hepatic failure. Early diagnosis relies on a high index of suspicion and a meticulous dietary history, as routine laboratory and imaging findings lack specificity. Serum biomarker detection can provide decisive confirmation when exposure is suspected. Prompt supportive management particularly strategies aimed at mitigating oxidative injury can lead to full recovery even in severe presentations. Clinicians should remain alert to the possibility of aflatoxin ingestion, especially when illness follows the consumption of spoiled or imported foods.

Declarations

Contributors

All authors contributed to the clinical assessment, data collection, literature review and preparation of the manuscript. All authors have read and approved the final version.

Competing interests

None declared.

Patient consent for publication

Informed consent for publication was obtained from the patient; a signed consent form is available upon request.

Ethics approval and consent to participate

Not applicable. This case report did not require institutional review board approval according to local guidelines, as it describes a single anonymized clinical case.

Availability of data and materials

Not applicable.

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