Ackee Fruit Toxicity

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Continuing Education Activity

Ingestion of unripe Ackee fruit may result in the metabolic syndrome known as "Jamaican vomiting sickness." Clinical manifestations may include profuse vomiting, altered mental status, and hypoglycemia. Severe cases have been reported to cause seizures, hypothermia, coma, and death. Medical treatment is primarily supportive care with intravenous fluids and dextrose. This activity reviews the evaluation and management of ackee fruit toxicity and highlights the role of the interprofessional team in providing optimal care to patients affected by this condition.

Objectives:

- Explain how hypoglycin A, the major toxin in ackee fruit, leads to glycogen storage depletion and hypoglycemia.
- Describe how the presentation of ackee fruit toxicity differs from that of acute gastrointestinal infections.
- Describe the evaluation and management of patients with ackee fruit toxicity.
- Identify interprofessional team strategies for improving recognition and management of patients with ackee fruit toxicity.

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Introduction

Ingestion of the unripened Ackee fruit (*Blighia sapida)* may result in the metabolic syndrome known as "Jamaican vomiting sickness." Clinical manifestations may include profuse vomiting, altered mental status, and hypoglycemia. Severe cases have been reported to cause seizures, hypothermia, coma, and death. Medical treatment is primarily supportive care with intravenous fluids and dextrose.[1][2]

Etiology

The ackee fruit matures into three sections, each consisting of a large black seed and a fleshy yellow aril. The aril, the edible portion of the fruit, initially contains high levels of the toxin hypoglycin. However, over time, as the fruit ripens, the hypoglycin levels drop to ranges allowing for safe human consumption. Toxicity only occurs with ingestion of the unripe Ackee fruit. Differentiating ripe versus unripe Ackee fruit is necessary to prevent any potential adverse health effects. Ripe Ackee fruit may be identified by a yellow-red to red color and wide-open appearance (seeds easily visible). Unripe fruit is green to yellow and has a closed appearance (seeds are difficult to visualize). Cooking an unripe fruit does not reduce its potential for toxicity. The seeds remain toxic and should not be ingested. Risk factors for Ackee fruit toxicity include eating an unripe Ackee, purchasing tampered Ackee, or the reusing of water that has been previously used to cook unripe ackee.[3][4]

Epidemiology

The Blighia sapida tree is native to West Africa. In 1778, it was imported to Jamaica where it has remained the national fruit of the country and an essential part of the Jamaican diet. The epidemiology of the disease has not been well established in Jamaica, but mortality and morbidity are thought to be underreported. There are more cases of the illness during the Jamaican winter months when the fruit is not yet ripe. Blighia sapida also is found in southern Florida, several Caribbean countries, and Central America. Ackee imported into the United States is regulated by the Food and Drug Administration (FDA) to limit products containing dangerous amounts of the toxin, hypoglycin A. Only a few cases of toxicity have been reported within the United States. Pediatric patients and the malnourished are at the greatest risk for toxicity.[5]

Pathophysiology

The ackee fruit contains both hypoglycin A and hypoglycin B. Hypoglycin A is the toxin primarily responsible for causing Jamaican vomiting sickness. The exact mechanism of hypoglycin A is not known; however, hypoglycemia most likely results from the inhibition of gluconeogenesis. Metabolism of hypoglycin also produces a toxic byproduct, methylene cyclopropyl acetic acid (MCPA-CoA) which inhibits long-chain fatty acid beta-oxidation, resulting in glycogen store depletion. It is impossible to predict what patients will develop symptoms following ingestion as the minimum toxic dose is not currently known. Hypoglycin may be hepatoxic, with liver pathology resembling a Reyes Syndrome-like pattern. Chronic ackee fruit ingestion has been reported to cause cholestatic jaundice among adults.[6]

Toxicokinetics

The onset of hypoglycemia is delayed for a few hours, but once present rapidly progresses. Deaths have been reported within 12 to 48 hours.

History and Physical

Gastrointestinal (GI) symptoms typically develop within six to 48 hours of ingesting of an unripe Ackee fruit, although they may develop more rapidly in severe cases. An apparent period of recovery (approximately 10 hours) may follow the initial GI symptoms. It is important to note that while vomiting is common, an absence of vomiting does not rule out the diagnosis. Diarrhea is usually absent which may help differentiate Jamaican vomiting sickness from other acute GI infections. Seizures may be a predictor of disease severity as they were present in 85% of reported fatal cases. Other neurological manifestations may include altered mental status, tremors, and paresthesias. A thorough history of the patient's recent meals may be necessary to identify recent Ackee fruit ingestion accurately. Toxicity is dose-dependent; therefore, it is important to quantify the amount ingested. If recovery occurs, it happens within one week of ingestion.

Evaluation

Patients with clinical suspicion for Jamaican vomiting sickness should be evaluated in a hospital and admitted for observation. Intravenous access should be obtained to administer fluids and dextrose if needed. Diagnostic evaluation should include blood glucose, serum electrolytes, liver function tests, renal function, lactate, ketones, and blood gasses. Symptomatic patients will require close monitoring of laboratory tests, especially glucose and serum electrolytes. An early ECG may show evidence of electrolyte abnormalities before laboratory diagnostic results. Hypoglycin and its metabolite MCPA may be tested for in blood and urine; however, they may be undetectable secondary to rapid elimination. Other diagnostic tests that help confirm the diagnosis include elevated serum/urine carnitine concentrations and elevated concentrations of urinary dicarboxylic acid.

Treatment / Management

Treatment is primarily supportive as no current antidote for hypoglycin A exists. Patients require close monitoring for hypoglycemia and should be treated with dextrose. Boluses of dextrose may be given initially followed by an infusion which may be titrated to maintain euglycemia. Intravenous fluids should be provided to prevent dehydration and antiemetics from alleviating symptoms. Vomiting and dehydration may lead to electrolyte abnormalities which should be repleted. Benzodiazepines may be used for the treatment of seizures, but again it is prudent to rule out hypoglycemia as the cause of the seizures. GI decontamination, including activated charcoal and gastric lavage, may be considered if there is a concern for potential toxicity if the patient presents within a few hours of ingestion. There is not sufficient evidence to suggest GI decontamination consistently improves patient outcomes. There is no role for syrup of ipecac following acute ingestions. Riboflavin and glycine have been used for treatment as they are thought to antagonize hypoglycin A. Methylene blue has also been proposed as a treatment option for potentially fatal encephalopathy. The true efficacy of these supplements is unknown. A medical toxicologist may assist in providing management and may be reached through the local Poison Control Center. Patients with persistent hypoglycemia, seizures, metabolic acidosis, altered mental status, or poor perfusion will most likely require admission to an intensive care unit (ICU). Education focusing on the health hazards of eating unripe Ackee fruits may decrease the future incidence of the disease.[7]

Differential Diagnosis

- Acute gastritis
- Acute poisoning (general)
- Aspirin poisoning
- Hypoglycemia
- Influenza
- Intestinal obstruction
- Meningitis
- Sulfonylurea poisoning

Enhancing Healthcare Team Outcomes

Health care providers, especially those who work in the emergency department, awareness of the potential toxicity of unripe ackee fruit, especially in the United States, is necessary for early diagnosis and timely management. Public health education on the proper preparation of Ackee fruit may decrease rates of toxicity in areas where it is endemic.

The nursing staff should identify patients exposed in triage and communicate with the clinical emergency department staff so that clinical pharmacy and physician toxicologists can be quickly consulted. Once a working diagnosis is established, the interprofessional team of clinicians and nurses must communicate to assure that appropriate support is rendered quickly. The nurses must monitor the patient for changes in vital signs and immediately report to the clinical team so that adjustments in care can be initiated. [Level V]

The nurses and clinicians including physician assistants, nurse practitioners, and physicians must work towards educating patients and the dangers of consuming Ackee fruit. Making sure children and adults avoid exposure is the safest is the best approach to avoiding morbidity and mortality.

Review Questions

- Access free multiple choice questions on this topic.
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