Gut Fermentation Syndrome: Unraveling the Enigma of Auto-Brewery Syndrome.

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Background

- A disorder known as “auto-brewery syndrome” or “gut fermentation syndrome” occurs when bacteria or fungus in the urinary, oral, or gastrointestinal tract ferment endogenously, producing ethanol. Patients with auto-brewery syndrome frequently report eating a diet heavy in sugar and carbohydrates and exhibit many of the symptoms and indicators of alcohol intoxication while disputing alcohol consumption.
- Research on Nonalcoholic Fatty Liver Disease indicates that endogenous alcohol synthesis may have bacterial roots; these bacteria may also be the causative agents in GFS. Probiotics, low-carb diets, antifungal medications, and antibiotics are currently used as therapies for GFS. Fecal microbiota transplantation may have a part in the management of GFS.

Objective

- This case report discusses a case of gut fermentation syndrome complicated with NASH cirrhosis, esophageal varices, and portal hypertension. It further investigates the clinical presentation, diagnostic journey, complications, and therapeutic management of a patient diagnosed with auto-brewery syndrome. Finally, the case report further discusses the medico-legal implications commonly associated with gut fermentation syndrome.

Methods

- A 50-year-old male with PMHx significant for NASH cirrhosis, esophageal varices, portal hypertension, GI bleeds, and gut fermentation syndrome had presented to the emergency department with complaints of dizziness, shortness of breath, and fatigue for 4-5 days. Of note, as the patient has a history of GI bleeds for which he follows a gastroenterologist, the patient mentioned his symptoms may be due to his chronic anemia. Initial complete blood count revealed Hemoglobin: 6.4 g/dl (critically low) and hematocrit: 21.2% (critically low). Liver function tests revealed T-Bili: 2.3 (high), AST: 49 (high), ALT: 33 (within normal limits), and Alkaline phosphatase: 91 (within normal limits). PT: 17.1 seconds (high), INR: 1.5 and PTT: 37.4 seconds (high). Ammonia level: 68 (high). Blood alcohol level 98 mg/dl (critically high). Ultrasound of the abdomen was significant for a cirrhotic liver, splenomegaly, and a small amount of ascites in the right lower quadrant. MRI of the abdomen without contrast revealed a cirrhotic-appearing liver with portal venous hypertension, splenomegaly, and ascites. The patient was treated with Octreotide, Pantoprazole, Furosemide, Spironolactone, Sucralfate, Nadolol, and Lactulose. He was transfused 2 units of packed red blood cells and observed overnight for any acute changes. On discharge, the patient had improvement in Hemoglobin to 8.7 g/dl and no longer felt symptomatic from his anemia. He was instructed to follow up with his gastroenterologist outpatient for a need for possible endoscopy, and his transplant hepatologist for further management regarding his NASH cirrhosis.

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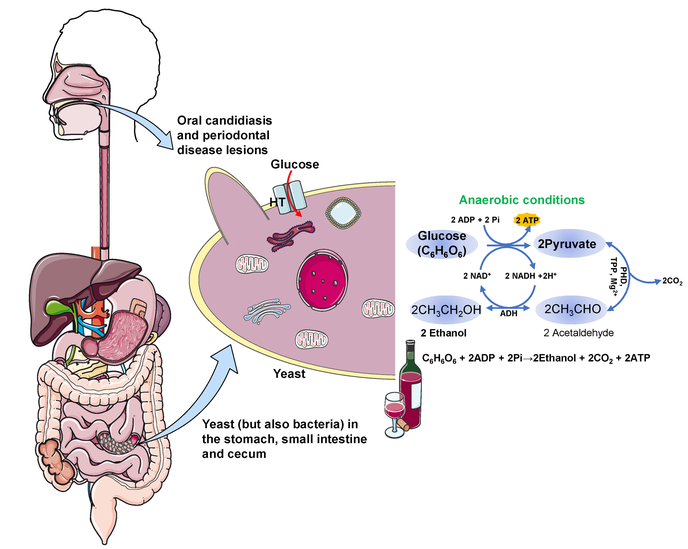
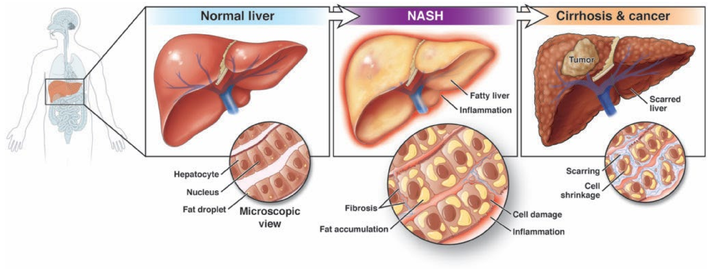
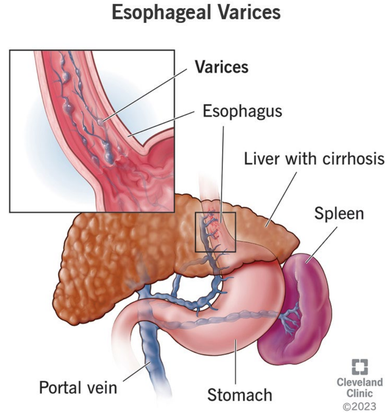
Results







Discussion

- Gut fermentation syndrome is a condition in which the body produces ethanol with the consumption of carbohydrates through fermentation by microorganisms. The body will have increased levels of ethanol without the consumption of any alcohol. Patients with underlying pathologies that disturb the microbiota of the gut are more susceptible to this condition. One such example is prior antibiotic use. Diagnosing gut fermentation syndrome requires laboratory testing. A glucose challenge test where an increase in BAL after consumption of glucose is confirmatory. Gut fermentation occurs through the fermentation of carbohydrates through microorganisms. Fungi and bacteria are most commonly implicated including Saccharomyces and Candida species. After obtaining these results, treatment with antifungal medications such as Micafungin, Fluconazole, Voriconazole, Trichomycin B, and Nystatin can be used. Further management includes limitations in carbohydrate consumption along with an increase in protein consumption.
- A difficult complication of gut fermentation syndrome can be the development of NASH cirrhosis. Dysfunction of the liver-gut axis results from disruption to the gut microbiome and disruption to the mucosal permeability. This leads to metabolic byproducts of bacteria and fungi to reach the liver via the portal system. These metabolic byproducts such as lipopolysaccharide (LPS) and peptidoglycan cause an inflammatory response to the Kupffer cells in the liver eventually leading to liver injury eventually causing fibrosis overtime.

Conclusion

- This case contributes to the understanding of Gut Fermentation Syndrome, highlighting the importance of considering this rare disorder in patients with unexplained alcohol-related symptoms. Increased awareness among healthcare professionals is crucial for accurate diagnosis and effective management of GFS, ultimately improving the quality of life for affected individuals. To understand how imbalances of commensal bacteria in the gut allow yeast to grow on a pathogenic level, more research is especially needed on the human microbiome.

References

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