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Case Report

Endogenous Alcohol and Auto-Brewery Syndrome Complicating Liver Transplantation: A Case Report and Literature Review

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Abstract: Introduction: We describe the first reported case of auto-brewery syndrome complicating liver transplantation, wherein a patient was temporarily removed from a liver transplant list not due to ethanol consumption but rather spontaneous ethanolic fermentation within the gastrointestinal tract. Auto-brewery syndrome (ABS) is a rare metabolic condition where gastrointestinal microbiota dysbiosis leads to spontaneous microbial ethanolic fermentation under anaerobic, high carbohydrate conditions. Because no alcohol is directly consumed by the patient, this alcohol is often referred to as "endogenous". Methods: We present a case where a patient awaiting orthotopic liver transplantation was removed from the transplant list due to significantly elevated blood alcohol levels. However, an upper endoscopy revealed Candida esophagitis, and the diagnosis of ABS was made. Results: With antifungal fluconazole treatment, the patient's blood alcohol biomarkers decreased, and the patient underwent a successful liver transplantation. Discerning between patient exogenous alcohol consumption and endogenous alcohol production with ABS remains a significant challenge for clinicians, and this knowledge could have serious implications for a patient awaiting a life-saving liver transplant. Conclusions: This case highlights the importance of listening to the patient and carefully assessing potential liver transplant recipients who consistently deny alcohol consumption, specifically for gut dysbiosis and ABS.

Keywords: auto-brewery syndrome; liver transplant; microbiome; candida; alcohol-associated liver disease

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1. Introduction

Auto-brewery syndrome (ABS) is a condition where patients may appear intoxicated or have detectable blood alcohol without direct alcohol consumption [1]. It is estimated that fewer than 100 ABS cases have been reported in the literature, but ABS likely remains underdiagnosed due to a lack of awareness among the medical community [2]. The pathophysiology of ABS arises due to underlying gastrointestinal fungal or bacterial infections that undergo ethanolic fermentation within the patient. This ethanol is directly absorbed by the patient's gastrointestinal epithelium, causing blood alcohol to rise. Blood alcohol content in ABS patients can range widely based on carbohydrate consumption and fasting states, but blood alcohol levels as high as 400 mg/dL (0.4%) have been reported [3]. Co-morbidities for auto-brewery syndrome include metabolic-associated steatohepatitis, diabetes, and gastrointestinal disorders, but this condition can occur spontaneously in healthy individuals [4,5]. ABS treatment includes low carbohydrate diets along with antifungal medications or antibiotics to treat the underlying gut dysbiosis [6].

Alcohol-associated liver disease (ALD) is a common reason for liver transplantation. Liver transplantation for ALD has increased to 40% over the last decade, now surpassing the numbers for fatty liver disease and viral hepatitis C combined [7]. Due to the contribution of alcohol to liver diseases, a six-month period of alcohol abstinence is typically required for liver transplantation eligibility [8]. Often patients are monitored for alcohol use with the sensitive phosphatidylethanol (PEth) blood biomarker. Herein, we describe a patient with a positive PEth test that was temporarily removed from the liver transplantation list but was found to have endogenous alcohol production and ABS due to Candida esophagitis. This report suggests that evaluation of ABS should be included in candidates for liver transplantation with elevated blood alcohol, despite a detailed history demonstrating adherence to alcohol abstinence.

2. Case Report

A 39-year-old male with alcoholic cirrhosis, ascites, and portal hypertension with a history of variceal bleeding had been abstinent from alcohol for several years. After worsening hepatic decompensation and the development of encephalopathy, he was evaluated and listed for liver transplantation. While undergoing monitoring, his routine blood PEth test was reported positive (300 ng/mL). During physical examination, the patient was alert and did not appear visibly intoxicated. There was no history of underlying neurological conditions. The patient's diet was not particularly high in carbohydrates or excess sugars. With this elevated blood PEth test, there was a concern regarding the patient's sobriety, and the patient was asked if he was consuming alcohol. Although he and his wife adamantly denied alcohol intake, the patient was removed from the transplant list and recommended to undergo substance use rehabilitation. Removal from the liver transplant list was informed based on the current six-month alcohol abstinence guidelines.

However, while off the transplant list, the patient continued to have elevated blood PEth without alcohol consumption. The patient avoided alcohol mouthwash and hand sanitizer with concern that these agents were causing a false positive PEth test, but no change in blood alcohol was documented. Without a history of recent alcohol consumption, auto-brewery syndrome was considered as a possible etiology. To confirm the diagnosis, an upper endoscopy was performed to evaluate gastrointestinal infections contributing to ethanolic fermentation. An upper endoscopy revealed moderately severe Candida esophagitis (Figure 1A) involving the entire esophagus. Brushings were obtained for microbiology and confirmed the *Candida albicans* yeast infection. The patient was diagnosed with auto-brewery syndrome secondary to Candida esophagitis.

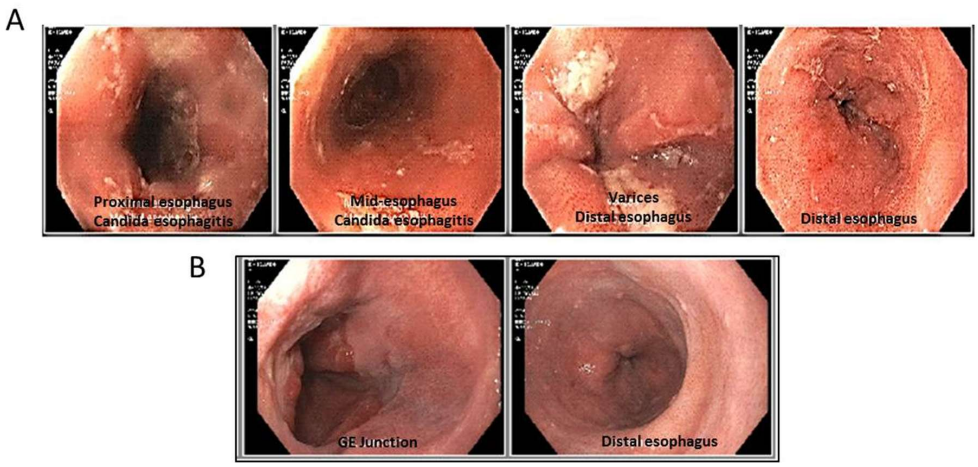

Figure 1. Esophageal endoscopy in patient with auto-brewery syndrome reveals extensive Candida esophagitis that resolved with fluconazole treatment. (**A**) Several images of the esophagus taken during

the initial upper endoscopy demonstrated white, irregular plaque-like lesions coating the entire esophagus with associated erythema and superficial ulceration. Brushings were obtained during the endoscopy for microbiology evaluation and confirmed *Candida albicans* infection. (**B**) Images were taken from the upper endoscopy after fluconazole therapy and normal PEth testing show resolution of the Candida esophagitis.

Antifungal therapy with fluconazole (100 mg daily for two weeks by mouth) was initiated to treat the underlying gut dysbiosis. Shortly after the initiation of fluconazole therapy, the patient's blood PEth dramatically decreased and then returned to baseline values (Figure 2). A repeat endoscopy was performed to evaluate whether the changes in PEth corresponded with management of the Candida infection. The repeat endoscopy after the antifungal therapy confirmed resolution of the Candida esophagitis, thus supporting the diagnosis of auto-brewery syndrome (Figure 1B). Following the normal endoscopy and the normalization of blood PEth levels, the patient was returned to the liver transplant list. The patient subsequently underwent a successful liver transplant and continues to be healthy.

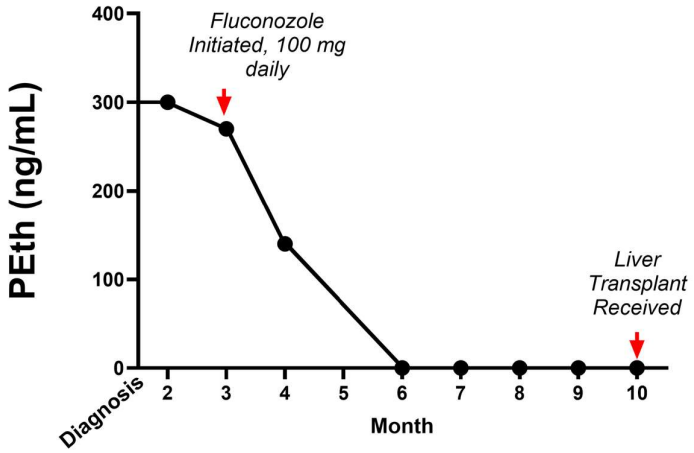

Figure 2. Time course of PEth blood levels and disease. At the time of diagnosis of ABS and Candida esophagitis by endoscopy, blood PEth levels were markedly elevated. Fluconazole treatment was initiated (top red arrow) followed by a rapid decline in blood PEth levels. The blood PEth levels remained undetectable for several months after eradication of Candida esophagitis and the patient underwent successful liver transplantation (lower red arrow).

3. Discussion

Endogenous ethanol production occurs continuously in the human body from the gut microbiota [9]. However, microbiota dysbiosis can lead to pathogenic increases in endogenous ethanolic fermentation, resulting in ABS [10]. While ABS may be an under-reported metabolic condition, the social and medical implications of this condition may translate to poor health outcomes. Due to the lack of medical awareness surrounding ABS, this condition may be misdiagnosed as "exogenous" alcohol consumption as opposed to "endogenous" production. This misdiagnosis carries significant risks when alcohol consumption guides life-saving clinical decisions, such as liver transplantation. Therefore, expanding clinical decisions to include the possibility of ABS may raise medical awareness and improve health outcomes.

We reviewed the literature and report in Table 1 several case reports of ABS including the organisms involved and the sites of infection. At least 66 ABS patients were identified in our literature search; matching previous estimates that total ABS incidence in the literature was fewer than 100 cases. Of the ABS cases reported, fungal infections were the most common etiologic agent, with *Candida* spp. being the often-identified causative

organism. Four of the *Candida* species commonly involved in ABS include *Candida albicans*, *Candida tropicalis*, *Candida parapsilosis*, and *Candida krusei*. These four *Candida* species are considered "Crabtree-negative", meaning these organisms use ethanolic fermentation only when adequate oxygen is not available in their environment [11]. However, *Candida glabrata* is considered "Crabtree-positive" since it uses alcoholic fermentation in response to excessive glucose in the environment [11]. As such, when these *Candida* species contribute to ABS, there may be variability in the dietary and oxygen microenvironments that favor ethanolic fermentation. This difference may explain why excess carbohydrates significantly contribute to ABS, while others may report dietary carbohydrates exerting less of an effect, such as the patient we reported. Therefore, dietary interventions alone may not be useful in helping to confirm the diagnosis of ABS.

Although *Candida* spp. was the most common causative fungus reported in ABS, *Saccharomyces cerevisiae* was also noted as another causative fungal agent in this condition. Bacterial infections from *Klebsiella* spp. also have been implicated in ABS pathophysiology, thus demonstrating that both fungal and bacterial overgrowth may contribute to this condition [12]. Although the luminal gastrointestinal tract is the most common site of infection, several cases involving a urinary source have been reported [11,13]. When the source of alcohol is from the urinary bladder, the host typically has diabetes with glycosuria [13]. The oral cavity can serve as an unusual site for ABS, but a few reports have been noted. Dietary consumption appears to be a major factor in oral ABS, with the organisms undergoing ethanolic fermentation being identified in high carbohydrate foods, such as honey [14] or chocolate [15]. Taken together, ABS is a diverse metabolic condition arising from both fungal and bacterial infections that can occur in many areas of the body. Treatment for ABS must therefore reflect drug bioavailability in the effected tissue, the causative organism, and the species-specific conditions that drive ethanolic fermentation.

Since *Candida* infections remain the most common factor in ABS, we suggest that ABS screening may be indicated in patients with increased risk for candidiasis, such as immunocompromised patients [16]. Patients with hyper IgE syndromes (HIES) have increased risk of candidiasis due to dysfunctional IL-17 signaling, which normally prevents fungal overgrowth [17,18]. Other immune disorders also have a higher incidence of candidiasis, particularly those with *STAT1* gain-of-function mutations (GOF) and autoimmune polyendocrinopathy candidiasis ectodermal dystrophy (APECED) [19,20]. Patients with *STAT1* GOF or APECED may develop liver dysfunctions requiring transplantation, such as nodular regenerative hyperplasia and cirrhosis [21,22]. Therefore, special attention must be given to these patients since *Candida* infections and ABS may complicate liver transplantation.

Table 1. Reports from the literature of auto-brewery syndrome. The location of the infections and organism(s) involved are noted.

Etiology	Organism	Type of Report	Number of Patients	Reference	Note
Gastrointestinal	*K. pneumoniae*, *C.albicans*, *C. glabrata*, *S. cerevisiae*, *C. intermedia*, *C. parapsilosis*, *C. kefyr*.	Review	*N* = 17	[23]	Seven of the subjects had prior antibiotic use
GI dysbiosis	*Candida*	Survey	*N* = 28	[24]	Those with ABS had higher incidence of allergies

Table 1. *Cont.*

Etiology	Organism	Type of Report	Number of Patients	Reference	Note
Metabolic-associated steatohepatitis	*Pichia kudriavzevii*; *C. glabrata*, *C. albicans*, *Galactomyces geotrichum*; *Klebsiella Pneumoniae*	Article	*N* = 10	[25]	Measured fecal ethanol by Mass Spec compared to healthy controls
Gastrointestinal	*C. krusei* *C. parapsilosis*	Case report	*N* = 1	[26]	History of hemicolectomy and constipation
Short bowel syndrome	*C. glabrata* *S.cerevisiae*	Case report	*N* = 1	[27]	13 year old
Crohn's disease	*Candida glabrata*	Case report	*N* = 1	[5]	Bowel obstruction and antibiotics
Gastrointestinal- MASLD	*K. pneumoniae*, *K. quasi pneumoniae*, *K. variicola*	Case study	*N* = 5	[12]	Subjects did not respond to anti-fungal medications
Urinary	*C. glabrata*	Case report	*N* = 1	[13]	Waiting liver TX, Urinary ETOH + plasma neg
Oral	*Geotrichum candidum*	Case report	*N* = 1	[14]	Occurred after eating honey
Oral		Case report	*N* = 1	[15]	Chocolate

In addition to immunocompromising conditions, patients with underlying liver diseases also appear to have an increased risk of microbiota dysbiosis. Importantly, the presence of alcohol consumption in liver diseases guides both diagnosis and treatment management.

Alcohol-associated liver disease (ALD) is associated with alcohol consumption leading to fatty liver disease, fibrosis, and end-stage cirrhosis. In contrast, metabolic dysfunction-associated steatotic liver disease (MASLD) arises from an underlying metabolic or dietary cause, without alcohol consumption. While MASLD does not result from ethanol consumption, MASLD remains a complex interplay between metabolism and gut immunity that can easily be exacerbated by changes in ethanol metabolism. The hallmark of MASLD is fatty acid accumulation and inflammation [28]. Fatty acids, in conjunction with gut-absorbed microbial products, promote oxidative stress and activate pro-inflammatory cytokine expression. Continued inflammation triggers fibrogenesis that exacerbates hepatic dysfunction [29,30].

An accumulating body of evidence suggests an association between gut microbiota dysbiosis and MASLD progression [31,32]. Interestingly, patients with MASLD appear to be at an increased risk for ABS [11,25] due to alterations in ethanol metabolism. In those patients with hepatic dysfunction, ethanol produced by luminal bacteria or fungus cannot be appropriately metabolized due to hepatic fibrosis and inadequate levels of alcohol dehydrogenase (ADH) [33]. It must be noted that not all patients with MASLD develop auto-brewery syndrome. However, it is possible that microbiota changes seen in MASLD may predispose these patients to microbiota dysbiosis that increases endogenous ethanolic fermentation. This idea is supported from studies by Zhu et al. (2013), who found that MASLD patients have enriched ethanol-producing bacterial populations, such as *Escherichia* [34]. Compared to both obese and healthy controls, MASLD patients had significantly elevated serum ethanol concentrations [10]. Lastly, hepatic enzymes responsible for ethanol degradation such as ADH are altered in MASLD, suggesting that the liver may respond to bacterial-derived endogenous ethanol changes during MASLD progression [35].

While direct alcohol consumption may not contribute to MASLD, endogenous alcohol production arising from microbiota dysbiosis appears to play a role in disease progression [36,37]. Alcohol itself is a potent driver of metabolic change, shifting metabolism from

fatty acid degradation towards fatty acid accumulation and steatosis (Figure 3). Steatohepatitis is considered a two-hit phenomenon between lipid metabolism and immunity that can occur in the absence of alcohol consumption. Lipid metabolism is a balance between fatty acid degradation and fatty acid synthesis through lipogenesis (Figure 3). A key regulator of fatty acid degradation is PPARα, while SREBP1c regulates lipogenesis [30]. Steatosis arises from lipogenesis outpacing lipid degradation; however, steatosis alone does not appear to drive inflammation as fatty liver patients can remain asymptomatic. Instead, hepatitis and fibrosis may arise from gut barrier disruption that promotes increased absorption of gut bacterial-derived products such as lipopolysaccharide (LPS) [32] and neutrophil extracellular traps (NETs) [38]. These bacterial-derived products activate resident hepatic immune cells that promote fatty acid peroxidation and oxidative stress, triggering fibrogenesis. Alcohol has the potential to accelerate this pathophysiology by altering both metabolism and immunity. First, alcohol is highly caloric and metabolized into acetyl-CoA within the liver. The acetyl-CoA can be utilized for energy mobilization or sequestered via de novo lipogenesis. However, alcohol is a potent inhibitor of PPARα, further shifting metabolism away from energy mobilization in favor of lipogenesis and steatosis. Second, alcohol disrupts the gut barrier, further increasing absorption of bacterial-derived products and subsequent immune cell activation. Alcohol consumption therefore exacerbates MASLD, giving rise to metabolic dysfunction and alcohol associated liver disease (MetALD). In patients with ABS, however, microbiota dysbiosis causes ethanol fermentation within the gastrointestinal tract without direct ethanol consumption. Therefore, ABS may be superimposed on the patient's current liver disease, leading to worsening liver disease outcomes.

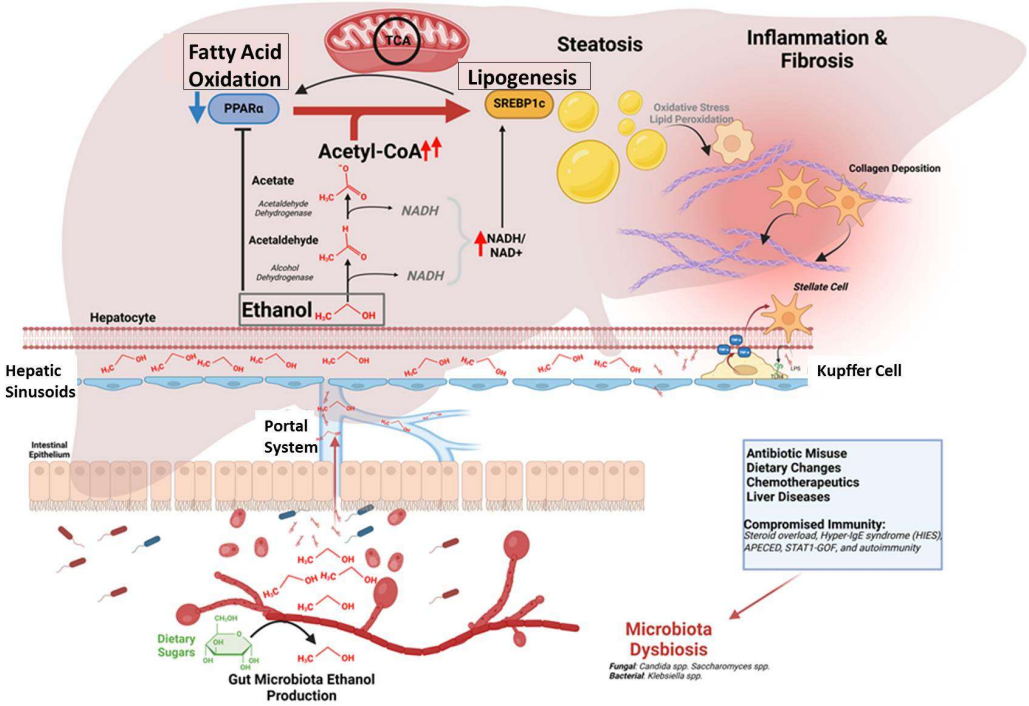

Figure 3. Pathophysiology of alcohol in liver diseases and how auto-brewery syndrome can exacerbate disease outcomes. Coupled with microbiota dysbiosis and oxidative stress, endogenous alcohol may contribute to the lipid oxidation and fibrosis seen in MASLD pathogenesis. As hepatic function decreases in liver diseases, the liver's ability to metabolize ethanol also decreases. This may lead to a positive feedback loop where endogenous ethanol may contribute to worsening disease outcomes and ABS. Key: ALD, alcohol-associated liver disease; MAFLD, metabolic dysfunction-associated steatotic liver disease; PPARα, peroxisome proliferator-activated receptor alpha; SREBP1c, sterol regulatory element binding protein 1c; ABS, auto-brewery syndrome. This figure was created with BioRender.com.

Taken together, endogenous alcohol and auto-brewery syndrome may be important clinical considerations when evaluating patients for a liver transplant. Even in liver diseases such as MASLD, where direct alcohol consumption is not noted, endogenous alcohol production may contribute to disease progression or arise from the associated microbiota dysbiosis. Currently, a six-month period of alcohol abstinence is usually required before transplantation. While this recommendation may improve long-term liver transplantation success, there remains a chance for patients to be removed from the transplant waitlist if endogenous alcohol levels are not recognized as auto-brewery syndrome. As such, clinicians should be aware of ABS, especially in immunocompromised patients that carry a higher risk of microbiota dysbiosis and candidiasis. Even though ABS may be an underappreciated condition, considering ABS during liver transplantation evaluations may prevent unnecessary removal from waitlists.

4. Conclusions

Patients with underlying liver disease are at increased risk of gut dysbiosis, which synergizes with metabolic dysfunction and inflammation to worsen disease outcomes. Therefore, assessing altered microbiota should be an important clinical consideration when evaluating patients for a liver transplant. Microbiota dysbiosis in liver disease patients may increase the risk of auto-brewery syndrome, complicating liver transplant and current alcohol abstinence guidelines. In particular, liver disease patients with an underlying immunodeficiency should be closely monitored for microbiota dysbiosis, rapid disease progression, and auto-brewery syndrome. Patients on liver transplant lists with sudden blood alcohol increases should be evaluated for auto-brewery syndrome and microbiota dysbiosis if there is no indication of direct alcohol consumption. Evaluating auto-brewery syndrome in liver transplant guidelines may avoid unnecessary removal from transplant lists and improve health outcomes.

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Data Availability Statement: The datasets presented in this article are not readily available because of patient confidentiality and the Institutional Review Committee requirement to keep all data de-identified.

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