

Auto-Brewery as an Emerging Syndrome: Three Representative Case Studies

Introduction

Auto-Brewery or Gut Fermentation Syndrome, also known as intestinal candidiasis, is a very rare phenomenon heretofore unstudied and underreported. Since the most recent case study of a man in Texas with the syndrome was published [1], the authors have been contacted by some forty plus patients, family members or physicians who believe the patients have Auto-Brewery Syndrome (ABS). This article details three representative case studies where the physician diagnosed ABS and laboratory findings confirmed pathologic levels of yeast in the intestinal tract. These three patients did not readily respond to conventional medications but responded in varying degrees to holistic treatments. The health histories provided by the patients, as well as follow up on the Texas patient, lead to the conclusion that Auto-Brewery Syndrome may be more of a chronic condition requiring lifestyle changes than an acute infection responding only to medication.

Review of Literature

Since the article “A Case Study of Gut Fermentation Syndrome (Auto-Brewery) with *Saccharomyces cerevisiae* as the Causative Organism” [1] was published in 2012, much has been learned about the human microbiome. The human microbiome is now recognized as an organ with a great range of metabolic activities and the “intestines and colon contain one of the densest known microbial communities on Earth” [2]. Science is beginning to collect data on what defines a normal microbiome, how it varies by traits such as age, location, community and season, and how it affects health and disease [2]. Studies have shown the microbiome can vary according to factors such as method of delivery at birth, being breastfed as an infant, gender, use of antibiotics, and educational level and can vary by body site [3,4]. A recent study of fecal metagenomes from four countries identified three enterotypes or clusters that could not be stratified by nation or continent [5]. Although understanding of how diet affects gut microbes is still limited, studies have shown that dietary variation between winter and summer can cause fluctuations in the composition of the intestinal microbiome [6,7]. Furthermore, we now know there are significant differences in gut microbes between different countries that are apparent in infancy as well as adulthood [7-9].

More importantly, science is beginning to demonstrate the link between the enteric microbiome and human health and disease as evidenced by the following studies. Normally the trillions of bacteria residing in the large intestine exist symbiotically with the host and compete with potentially hazardous microbes to prevent pathogens from gaining a foothold [10]. However, when the microbiome is disturbed, for example through diet, immune dysfunction and disease processes can result such as non-alcoholic fatty liver disease and



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cardiovascular disease [11]. The gut microbiome has been implicated in metabolic disease [12], inflammatory bowel disease [13-16], and obesity [17]. Repeated antibiotic use is one of the most often cited risks for perturbations in the enteric microbiome and resultant infections such as Methicillin-resistant *Staphylococcus aureus* (MRSA) [18], *Clostridium difficile* [19], *Candida albicans* [20], and small intestinal bacterial overgrowth (SIBO) [21]. Additional information is being derived from studies on SIBO and the connection to irritable bowel syndrome [16,22,23].

Opportunistic invasive fungal infections are well known in immuno-compromised patients and neonates [20,24-27]. However, yeast overgrowth in otherwise healthy subjects has rarely been studied. Lewith et al. conducted a study with subjects who had symptoms with no definitive conventional diagnosis; symptoms included fatigue, headaches, poor concentration, GI distress, muscle pain, joint pain or urogenital symptoms [28]. Using the Fungal Related Disease Questionnaire (FRDQ-7) the researchers selected subjects with a score >9 and compared *Candida* IgG concentrations to a control group. The *Candida* antibody concentrations were significantly higher in the non-control group than in the control group ($p < 0.001$).

Typically the by-products of gut fermentation of sugar are acids such as propionic acid (PPA) and short-chain fatty acids (SCFA) and the amount of alcohol fermented in the human intestines has typically been considered so low as to be negligible. However, a recent study demonstrated one *Lactobacillus* species in fecal slurries produced significant amounts of ethanol [29]. Furthermore, it is known that ethanol is elevated in the breath of some patients with end-stage renal disease [30] and bacterial overgrowth in the gut results in elevated breath ethanol [31]. A study by Turner et al., concluded that “the ethanol levels in the exhaled air are clearly increased after consumption of sugars and action on it by either mouth or gut flora/enzymes” [32]. This information adds to the scientific plausibility that the gut microbiomes of patients with Auto-Brewery Syndrome have a pathological level of yeast or fungus which cause their symptoms.

Case Studies

The following three case studies of Auto-Brewery Syndrome (ABS) are representative of the twenty or so patients being followed by the authors and illustrate the wide spectrum of symptoms and sequelae exhibited by many patients. Case 1 is most typical of the patients we have seen since he is a middle-aged man who responded quite well to diet and therapy. Case 2 is a 42 year old woman who responded to treatment but required many lifestyle changes to maintain a mostly symptom-free status. Case 3 is a young male and the most difficult case, in that he has responded for short periods of time to treatment but has had multiple relapses.

Case 1

In September of 2014 a 60 year old male from Illinois presented with a four year history of “spells” where he felt drunk or hung over. The patient has been a recovering alcoholic for 23 years and has not had any alcohol during that time. In 2010 the patient fell and fractured his skull and was hospitalized for three days and shortly after is when he began noticing the “spells”. He reports a history of Hepatitis C, genotype 3, diagnosed in April 2014 and a history of hypertension since 2009 controlled well with lisinopril 40 mg per day and metoprolol 50 mg per day; he is overweight (5’10”, 240 lbs.) and has smoked 2 packs of cigarettes per day for 48 years.

The episodes of drunkenness began to increase in frequency throughout 2013 and the patient became depressed and would not leave the house. He began anti-depressants (bupropion and sertraline) which helped some, but his episodes continued. The patient and his wife thought he had early dementia until he went to the ER feeling dizzy and his blood alcohol concentration (BAC) was 0.17% (g/ dL). The doctors assumed because of his history of alcoholism he had begun to drink again.

In May of 2014 the patient began treatment with sovaldi and ribavirin for his Hepatitis C and described a decrease in his “spells”. Around the same time he was concerned about being pre-diabetic and cut out all sugar from his diet for several months which now explains why his episodes of drunkenness decreased dramatically. Only in retrospect did he connect the fewer episodes to the reduction in sugar. He and his wife noted that the episodes he had in the remainder of 2014 followed food with high-fructose corn syrup rather than refined sugar.

In September of 2014 when the patient presented to these authors with continued episodes of drunkenness, his stool cultures showed low levels of bacteroides, 1+ *C. albicans* and *C. krusei*, low elastase, high lysozymes and elevated sIgA levels. The patient was given nystatin 500,000 units three times a day for three weeks. The patient was counseled on a low carbohydrate diet including the avoidance of cheese as well as coffee which can contain mold. He was cautioned to minimize mold in his house. By November, the patient was feeling better, had quit drinking soda with sugar and was losing weight.

By January of 2015, the patient thought he was improving enough to go back to work. He took a trucking job hauling grain but started getting his symptoms back. His work had him use a Breathalyzer where he blew 0.09% and he was forced to stop hauling. It seems he had been exposed to the mold in the grain elevators which stirred the auto-brewery up again.

By summer of 2015 the low carbohydrate diet was working to decrease the patient’s episodes to fewer than one per month. He had also lost 40 pounds and was working again, although not hauling grain.

Case 2

In December of 2014 a 42 year-old female from Georgia presented with a several year history of what she and her partner termed “episodes”. They had seen numerous doctors of all kinds (internal medicine, psychologists, psychiatrists, neurologists) and no one seemed to be able to identify any cause for the episodes of drunkenness.

The episodes usually began in the late afternoon, around 3:00 PM. According to her partner: “during an episode, she appears highly intoxicated; loses her balance, slurs words, becomes argumentative, is unable to reason or make sense, and then ultimately blacks out for the rest of the night. The loss of coordination has caused her to fall numerous times. She has broken ribs, her nose, and split her eye/head. The only thing that ends an episode is time. It takes the whole night before she becomes normal again. The next morning, she doesn’t remember anything that happened during the episode and is devastated as she hears often embarrassing events of the previous night.”

The patient bought a breathalyzer and began testing before, during and after episodes. Often, she registered far above the legal limit (0.18 to 0.40%) without having consumed any alcohol at all. Upon waking, after having slept all night, she still registered alcohol in her system. The patient had multiple gastrointestinal symptoms including frequent loose stools, vomiting, diarrhea, bloating, gas, and belching.

The patient began keeping a food log and eating a low carbohydrate diet with significant abatement of symptoms. She had been a social drinker before, but starting in the summer of 2014 completely abstained and continues to abstain from alcohol. She reported craving sweets for the first time in her life.

This patient tended to be better on the weekends than during the week. Days that involve a lot of stress, running around, and mental energy almost always end with an episode. Mondays - Wednesdays are her busiest days and were almost guaranteed to result in episodes. On the weekends, when she was relaxed and less stressed, she was less likely to become intoxicated.

Reproduced from patient/partner log:

11/6/14:

Ate 1/2 Cuba sandwich and chicken biscuit in the morning and pizza for lunch. Episode became clear around 3 pm.

4:00 pm blew .24. Passed out shortly after

7 pm woke just long enough to blow .37.

Did not eat dinner.

11/7/14:

6:30 AM woke, no food, blew .21

11:30 am ate fried rice and Mongolia beef. Vomited shortly after

with diarrhea throughout day.

2:30 pm blew .24

3:30 pm blew .3

Passed out

7:30 pm woke up to blow .41!!!

No dinner. Passed out again for the night.

11/8/14:

7 am woke and blew .24 first thing

Ate something...not sure what.

10:30 am blew .30

12 pm [Partner realized:] She is out of it so I'm rushing home.

In December of 2014, the patient's stool culture tested positive for *S. cerevisiae* (brewer's yeast); she started a low carbohydrate diet with some relief. Her partner reported she often went 8 days without an episode of drunkenness.

In January of 2015 the patient had a relapse and began averaging around 0.24% on the breathalyzer in the late afternoons and low or 0.00 in the mornings. She was following a low glycemic index diet and remained on fluconazole. A second stool culture confirmed 3+ *S. cerevisiae* and *S. bulardii* was still present.

Finally in March of 2015, the patient was placed on a very strict diet - high quality meats and non-starchy vegetables only along with supplements and she showed great improvement in symptoms. However, the patient reported she has an "occasional slip" on her diet and her partner states "it's very clear the next day or so because the episode appears".

Due to her inability to follow the recommended dietary changes, it was advised that she work with a nutritionist. Since this implementation, she has reported to be symptom free from her Auto-Brewery episodes.

Case 3

In May of 2013 an otherwise healthy 32 year-old male from Ohio presented with a four year history of gastrointestinal distress. He had experienced bouts of abdominal pain, reflux, and nausea with a 50 pound weight loss and then later a weight gain. He described the "attacks" as occurring once or twice a year for the first two years. However in June of 2011 the patient also began experiencing intermittent diarrhea after a trip to Hawaii. By August of that year the patient had nearly constant abdominal pain with daily diarrhea. He reported having abdominal pain, reflux with considerable belching, nausea, vomiting, weight loss and abdominal pain over a period of several weeks. His wife noted that during his episodes, he appeared to be intoxicated but she did not mention it because they both reported he had not been drinking.

Over the course of the nearly two years from June of 2011 to May of 2013 this patient sought help from various sources. He had multiple esophagogastroduodenoscopies (EGDs) and colonoscopies all of which were negative. The patient's blood work was negative for

inflammatory or malabsorption disorders; his abdominal ultrasound and CT scan were negative, except for circumferential thickening of the left ureter; and his hepatobiliary scan (HIDA) was negative as well. The patient had tried various treatments such as a gluten free diet, medications such as ondansetron (Zofran) and omeprazole (Prilosec), vitamins, as well as extensive rest. The impression in 2012 after his extensive GI workup was: 1. chronic abdominal pain, 2. chronic recurrent nausea and vomiting and 3. Gastro-esophageal reflux disease (GERD). The young man continued to decline and his daily vomiting intruded to the point he was unable to continue working. On a "good day" he could eat one meal and on a "bad day" he lived on Pedialyte and popsicles. He reported his pain at 9 on a scale of 0 to 10 and began to experience depression and insomnia.

When the patient initially presented in May of 2013, his history was reviewed and other than the GI symptoms, his systems were within normal limits. His blood pressure was 104/62, pulse 84, respirations 16. He was 6'2" and 170 pounds. He had no significant medical history other than "at least ten" courses of antibiotics as a child. Additional tests were ordered including stool culture with parasites and food intolerances; and he was referred to a nutritionist.

The stool profile revealed a 4+ yeast/fungi => 100,000 pg DNA/g specimen - taxonomy unavailable; and *Enterobius vermicularis* (most likely an ingested protozoan and not a human parasite). The short chain fatty acids (SCFA) [produced by bacterial fermentation of dietary polysaccharides and fiber], of propionate and valerate were higher than normal. No food intolerances were determined. The nutritionist recommended dietary changes of liquid diet with medical foods which the patient attempted to follow.

Additional stool cultures were conducted in October of 2013, all with negative results for *Giardia lamblia*, *Entamoeba histolytica*, *Cryptosporidium parvum*, *Salmonella*, *Shigella*, *Yersinia*, *Campylobacter* and *E. coli*. By December of 2013, the patient was still experiencing episodes of reflux and daily vomiting, with dry heaving up to 12 times per day. He submitted for biopsies of the duodenum, stomach, and esophagus with no pathologic changes noted and no evidence of *Helicobacter pylori*.

The patient received a DWI after having had two beers six hours previously. The patient's wife had been searching the internet for any clues to her husband's malady and in September of 2013 she ran across mention of "A Case Study of Gut Fermentation Syndrome (Auto-Brewery) with *Saccharomyces cerevisiae* as the Causative Organism" [1]. She converged on the fact that her husband often "seemed intoxicated" during his episodes, tracked down the article and helped connect the dots. She contacted the authors, bought a breathalyzer and began tracking BAC to try and identify triggers to his episodes of intoxication

In November of 2013, the patient was started on nystatin 500,000 IU4/day followed by a course of fluconazole 150 mg. daily. The medications increased his nausea and vomiting and he was unable to continue with them. During the entire year of 2014, the patient continued to struggle with episodes of intoxication, nausea, vomiting, and fatigue. He struggled to follow the prescribed diet to reduce yeast, and his wife continued to try and keep a log of his episodes and BAC, which many days was 0.24 to 0.34%.

During the early months of 2015, the patient was again given a course of fluconazole but his nausea and vomiting were worse than before. By July, he had regressed to “where he was in the beginning” and was very discouraged. They live in a hundred year old house, so he may be exposed to mold in the floorboards, basement, and walls because he seems to be improved when he is out of the house for extended periods of time. As of this writing, the patient has returned to Hawaii to stay with his in-laws to see if he can improve.

Follow-up on the Texas patient

It has now been five years since the Texas patient was treated with fluconazole and nystatin and a year-long low glycemic index diet with probiotics and a resultant weight loss of 60 pounds. He continues to be symptom free and follow a low carbohydrate, high fiber diet, with total abstinence from alcohol, maintaining his weight (5'9" and 160 lbs). Since no one knows how he developed a pathological level of *S. cerevisiae* (brewer's yeast) he has chosen to avoid all sources of brewer's yeast, alcohol, and other known factors that might contribute to yeast overgrowth.

Conclusion

These three representative case studies as well as the follow-up on the original published patient case study from Texas contribute to the notion that Auto-Brewery Syndrome may be treated with antifungals but most often must be managed and prevented by lifestyle changes such as weight control, limiting sugar, corn syrup, and simple carbohydrates in the diet, as well as by eliminating alcohol intake and foods with high yeast and molds such as coffee, peanuts, and corn. Environmental exposure to molds and yeast such as grain silos, house mold, etc. must be considered as well.

Primary providers should not assume that every patient who is drunk and says they haven't been drinking is hiding a drinking problem, but should conduct thorough health histories and keep an open mind to the possibility of Auto-Brewery Syndrome or Gut Fermentation.

Additional study on the human microbiome may shed light on how imbalances of commensal bacteria in the gut allow yeast to colonize to a pathologic level. Much research is needed to determine the mechanism of Auto-Brewery Syndrome in humans as well as the treatment needed to help sufferers.

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