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PHARMACOTHERAPEUTIC MANAGEMENT OF AUTO-BREWERY SYNDROME: A NARRATIVE REVIEW

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ABSTRACT

Auto-brewery syndrome, also called gut fermentation syndrome, is a rare and underrecognized condition in which gastrointestinal or urinary microorganisms ferment ingested carbohydrates into ethanol, producing intoxication without alcohol consumption. Existing reviews describe its epidemiology, pathophysiology and clinical presentation, but none has been organized around medication management. This narrative review synthesizes published antifungal regimens, dietary adjuncts and emerging therapies for the condition and outlines the pharmacist's role in its stewardship and long-term management. PubMed/MEDLINE, Scopus and Google Scholar were searched from inception to August 2026, and case reports, case series and reviews describing a treatment regimen and outcome were included. Fluconazole remains the empiric first-line antifungal, typically given at 100 to 200 mg daily for 2 to 3 weeks, with resolution reported in most susceptible cases; relapse or suspected azole resistance has prompted successful escalation to nystatin, micafungin, voriconazole or itraconazole in individual reports. A low-carbohydrate, sugar-restricted diet is a near-universal adjunct and, in several cases, was the main driver of relapse when discontinued. Fecal microbiota transplantation has emerged as a promising option in antifungal-refractory disease, with sustained remission reported at 34 months and 16 months of follow-up in

separate cases. The evidence remains confined to case-level reports rather than controlled trials. A structured, pharmacist-informed approach to agent selection, susceptibility-guided escalation and dietary counselling may reduce relapse and support earlier recognition of treatment failure.

KEYWORDS: Auto-brewery syndrome, gut fermentation syndrome, fluconazole, antifungal therapy, fecal microbiota transplantation, pharmacist.

1. INTRODUCTION

Auto-brewery syndrome (ABS) is a rare metabolic condition in which carbohydrates ingested by the patient undergo endogenous fermentation to ethanol within the gastrointestinal or, less commonly, urinary tract, producing symptoms and blood alcohol concentrations indistinguishable from exogenous alcohol intoxication.[1] The phenomenon was first documented in the scientific literature in the late 1940s to 1950s, though it remained a medical curiosity for decades before case reports began accumulating from the early 2000s onward.[2],[3]

ABS carries substantial clinical, social, and medicolegal consequences. Because its presentation mimics alcohol intoxication, patients are frequently misattributed as having alcohol use disorder, and diagnosis is often delayed by years despite repeated emergency presentations.[3],[4] This diagnostic delay is not merely an inconvenience: it carries substantial social, legal, occupational, and medical consequences,[3] and prolonged, unrecognized endogenous ethanol exposure may itself lead to alcohol dependence, compounding the original disorder with a secondary one.[5] Timely and appropriately selected pharmacotherapy is therefore central to limiting both the physiological and psychosocial burden of this disease.

Several narrative and systematic reviews have addressed ABS from complementary angles. Paramsothy et al. provided a broad clinical overview spanning epidemiology, diagnosis, and management.[1] Bayoumy et al. conducted a systematic review of case reports focused on causal microorganisms and treatment evidence.[6] More recently, Stamation produced a mini-review emphasizing the pathophysiological mechanisms of gut dysbiosis and ethanol-producing organisms,[7] while a 2025 review in the Journal of Clinical and Diagnostic Research examined the microbial determinants of ABS in detail.[8] None of these works, however, is organized around pharmacotherapeutic decision-making as its primary axis—that is, around how agent, dose, duration, and dietary adjunct are selected and sequenced across

the published evidence, and where a pharmacist's involvement in that sequencing adds value. This review addresses that gap.

1.1 OBJECTIVES

- To synthesize published antifungal regimens for ABS, including dosing, duration, and outcomes.
- To evaluate the role of dietary carbohydrate restriction and adjunctive probiotic therapy.
- To summarize emerging evidence for fecal microbiota transplantation (FMT) in refractory disease.
- To outline a pharmacist-informed framework for medication selection, monitoring, and patient counselling in ABS.

2. LITERATURE SEARCH METHODOLOGY

This is a narrative review, not a systematic review; no protocol was registered. Reporting was structured in line with the quality criteria of the Scale for the Assessment of Narrative Review Articles (SANRA).[9] PubMed/MEDLINE, Scopus, and Google Scholar were searched from inception to August 2026 using combinations of the terms "auto-brewery syndrome," "gut fermentation syndrome," "endogenous ethanol production," "antifungal," "fluconazole," "nystatin," "micafungin," "low-carbohydrate diet," and "fecal microbiota transplantation." Case reports, case series, and reviews published in English that described a specific treatment regimen and a reported clinical or biochemical outcome were included. Forensic case reports without treatment data and articles available only as conference abstracts were excluded. Given the rarity of the condition and the case-report-dominated literature, no formal quality-appraisal tool was applied and the PRISMA reporting framework for systematic reviews was not followed; findings are synthesized narratively and should be interpreted as hypothesis-generating rather than as a pooled quantitative estimate of treatment effect.

3. PATHOPHYSIOLOGY AND DIAGNOSTIC CONFIRMATION (BRIEF OVERVIEW)

ABS results from overgrowth of ethanol-producing organisms—most commonly *Saccharomyces cerevisiae* and *Candida* species (*C. albicans*, *C. glabrata*, *C. krusei*, *C. intermedia*, *C. parapsilosis*), though bacterial contributors, particularly *Escherichia coli*, *Klebsiella pneumoniae* and other Proteobacteria, have been increasingly implicated in recent

microbiome-based cohort studies.[10] Predisposing factors reported across the literature include recent antibiotic exposure and the resulting dysbiosis, diabetes mellitus, liver disease, inflammatory bowel disease, and other disorders of gastrointestinal anatomy or motility such as short bowel syndrome and chronic intestinal pseudo-obstruction.[3],[5],[11]

Diagnosis is one of exclusion, supported by a monitored oral glucose challenge test with serial blood or breath ethanol measurement, ideally paired with endoscopic culture and antifungal susceptibility testing of gastrointestinal secretions where feasible.[3],[5] Because pharmacotherapy selection increasingly depends on susceptibility data rather than empiric choice alone, diagnostic confirmation and treatment planning are best considered a continuum rather than sequential, separate steps.

4. ANTIFUNGAL PHARMACOTHERAPY

4.1 First-line therapy: fluconazole

Fluconazole is the most consistently reported first-line antifungal, chosen empirically given that *S. cerevisiae* and *C. albicans* are the most frequently implicated organisms,[3] or selected based on culture and susceptibility data where available.[5] Reported regimens generally range from 100 to 200 mg orally per day, most often for 2 to 3 weeks and extended to 6 weeks or longer in relapsing disease, with clinical and biochemical resolution described in multiple independent cases.[3],[5],[12] In one representative case, a 50-year-old woman achieved symptom resolution after a one-month course of fluconazole combined with dietary carbohydrate restriction; symptoms recurred only after she increased her carbohydrate intake and resolved again with a further course of fluconazole and the low-carbohydrate diet—underscoring that dietary nonadherence, rather than antifungal failure, was the likely driver of relapse in that instance.[3]

4.2 Second-line and relapse management

Relapse after fluconazole monotherapy in the absence of dietary nonadherence should prompt endoscopic re-evaluation for culture and susceptibility testing rather than empiric dose escalation alone.[3],[5] Reported second-line and relapse-management options include nystatin, itraconazole, voriconazole, echinocandins (notably micafungin), and, in select cases, amphotericin B.[3],[6] A frequently cited example is a 45-year-old man with a three-year history of undiagnosed ABS and two prior driving-under-the-influence arrests, whose gastric and jejunal cultures grew *Candida intermedia* that persisted despite oral fluconazole at up to 200 mg daily; his condition was treated by switching to intravenous micafungin for eight

weeks, guided by concern for possible azole-resistant *Candida* and the anatomical location of the causative organism in the upper gastrointestinal tract, where alcohol absorption predominantly occurs, and breath and blood alcohol levels became undetectable once micafungin was started.[5]

A separate representative case, in a 61-year-old man with *S. cerevisiae* identified on stool culture, documented complete symptom resolution with a six-week sequential course of fluconazole 100 mg/day for three weeks followed by nystatin 500,000 IU four times daily for three weeks, alongside daily acidophilus tablets and a strict sugar- and carbohydrate-free diet; breath alcohol remained undetectable for 10 weeks and repeat stool cultures were negative—illustrating that sequential antifungal therapy has also been used successfully in the published literature.[12]

4.3 Summary of reported antifungal regimens

The regimens described above are summarized in Table 1.

Table 1: Reported Antifungal Regimens for Auto-Brewery Syndrome.

Agent	Reported dose/duration	Clinical context	Source
Fluconazole (first-line)	100–200 mg/day PO; usually 2–3 weeks, up to 6 weeks	Empiric or susceptibility-guided first-line therapy, combined with a low-carbohydrate diet	[3],[5],[12]
Nystatin	500,000 IU PO q.i.d., 3 weeks	Given after fluconazole with a probiotic and strict diet; also a component of bowel decontamination before FMT	[12],[13]
Micafungin (IV)	Case-reported; 8-week IV course in one azole-refractory case	Used after fluconazole failure with persistent *Candida* culture positivity	[5]
Voriconazole	Case-reported; dose individualized	Reported success in relapse after fluconazole	[3],[6]
Itraconazole / amphotericin B	Case-reported; variable	Reported success in relapse after fluconazole	[3],[6]

Note: Dosing data above are drawn from individual published case reports and should not be interpreted as a standardized protocol; no randomized or comparative-effectiveness data exist for ABS pharmacotherapy at the time of writing. FMT, fecal microbiota transplantation; IV, intravenous; PO, oral; q.i.d., four times daily.

5. DIETARY AND NON-PHARMACOLOGICAL ADJUNCTS

A strict low-carbohydrate, sugar-restricted diet is described as a near-universal adjunct across the treatment literature and, in several cases, appears to be as important to relapse prevention as the antifungal agent itself.[3],[12] Dietitian-prepared low-carbohydrate meal plans have been published as part of multidisciplinary ABS care pathways, reinforcing that dietary counselling is not a peripheral recommendation but a core, ongoing component of management extending well beyond the antifungal treatment course.[3]

Probiotic supplementation, most commonly with *Lactobacillus* or acidophilus preparations, has been used adjunctively in several cases, though its independent contribution to outcomes has not been established through controlled evaluation.[3],[5],[12] Avoidance of unnecessary antibiotic exposure, which can promote the dysbiosis underlying fermentative overgrowth, is also recommended as a longer-term preventive measure; in one reported case, narrow-spectrum antibiotics were advised only after confirmed infection.[3]

6. REFRACTORY DISEASE AND EMERGING THERAPIES: FECAL MICROBIOTA TRANSPLANTATION

For patients with relapsing or antifungal-refractory ABS, FMT has emerged as a promising, though still limited, treatment option. An early published case from University Hospital Ghent described a 47-year-old man whose disease had not responded to antimycotic therapy and a low-carbohydrate diet; after FMT he was able to resume a normal carbohydrate-rich diet, with the benefit persisting through at least 34 months of follow-up.[14] A subsequent case report from Spain described a 40-year-old man with long-standing ABS, persistent hypertriglyceridemia, and raised transaminases, in whom oral bowel decontamination (nystatin, tobramycin, colistin, and vancomycin for 48 hours) preceded capsule-based FMT, with clinical and biochemical improvement reported.[13] A 2026 observational cohort study of 22 patients with ABS and 21 unaffected household partners identified enrichment of ethanol-producing Proteobacteria, including *E. coli* and *K. pneumoniae*, and followed one patient treated with FMT: symptom improvement correlated with shifts in gut microbiome composition and ethanol-producing metabolic pathway activity, and symptom freedom exceeding 16 months followed a second transplant, performed with different antibiotic pretreatment after an initial relapse.[10]

These findings, while still confined to individual cases and small cohorts, provide converging mechanistic and clinical evidence that microbiome-directed therapy may offer a durable

option where antifungal and dietary management alone are insufficient, and represent an area where future controlled study is clearly warranted.

7. UNDERDIAGNOSIS AND ITS IMPLICATIONS FOR PHARMACOTHERAPY

ABS is widely regarded as underdiagnosed, a problem attributable less to any single cause than to the convergence of several factors: low clinical awareness of a condition most physicians have never encountered, symptom overlap with alcohol intoxication that invites misattribution to alcohol use disorder, and the need for a monitored glucose challenge test, which is not routinely available, for confirmation.[4],[11] This underdiagnosis is directly relevant to pharmacotherapy: because patients often present only after years of unexplained episodes, they may already be at risk of alcohol dependence from chronic endogenous ethanol exposure,[5] complicating both the therapeutic relationship and adherence to the dietary restriction that antifungal therapy depends on for durable success. Earlier recognition—supported by structured diagnostic pathways such as that proposed by Zewude et al., involving collateral history, monitored glucose challenge, and culture-guided therapy selection—is therefore not only a diagnostic goal but a precondition for pharmacotherapy to succeed.[3]

8. SPECIAL POPULATIONS

Reported special-population presentations include ABS complicating liver transplant candidacy, where elevated blood alcohol in a patient awaiting transplantation led to temporary removal from the transplant list before ABS secondary to *Candida* esophagitis was diagnosed,[15] and a distinct urinary (bladder fermentation) variant, in which *Candida glabrata* ferments glucose in the urine of patients with poorly controlled diabetes without producing intoxication, so that control of the underlying diabetes forms part of management alongside antifungal therapy.[16] Pediatric cases have also been described, including two children with short bowel syndrome whose symptoms resolved after fluconazole,[17],[18] though the evidence base in this population remains extremely limited and dosing extrapolation should be approached with particular caution.

9. THE PHARMACIST'S ROLE IN THE MANAGEMENT OF AUTO-BREWERY SYNDROME

- Antifungal stewardship: supporting susceptibility-guided agent selection over indefinite empiric escalation, and flagging when a second course of the same agent versus a class switch is warranted.

- Drug interaction and safety screening: azole agents carry significant CYP-mediated interaction potential and can cause hepatotoxicity (fluconazole was stopped in one reported case because of an elevated alanine transaminase level),[3] so a pharmacist's medication reconciliation and liver-enzyme monitoring are particularly valuable given the often-lengthy diagnostic journey and polypharmacy some patients accumulate before diagnosis.
- Adherence counselling: reinforcing that dietary carbohydrate restriction is not adjunctive but co-primary with antifungal therapy, given documented relapse on diet liberalization.
- Destigmatization and communication: given the documented risk of patients being treated as though they have alcohol use disorder, pharmacists are well positioned to reinforce the biochemical basis of the condition during counseling.
- Monitoring for ethanol-related harm: supporting evaluation for hepatic effects of ethanol, as in alcohol use disorder,[3] and screening for cravings or withdrawal-like symptoms as endogenous ethanol production is brought under control.[5]

10. GAPS IN THE EVIDENCE AND FUTURE DIRECTIONS

- No randomized or comparative-effectiveness trials exist; all pharmacotherapy evidence is derived from case reports and small case series.
- No standardized antifungal dosing or duration protocol has been validated.
- No standardized diagnostic algorithm or glucose-challenge protocol exists, which limits comparability between reported cases.[3]
- The independent contribution of probiotic supplementation remains unquantified.
- FMT protocols (decontamination regimen, donor selection, capsule vs. colonoscopic delivery) are not yet standardized; an early-phase single-arm trial of capsule FMT in ABS is registered.[19]
- Pharmacist-led management pathways for ABS have not been formally described or evaluated in the literature.

11. LIMITATIONS OF THIS REVIEW

This review is narrative rather than systematic, and no protocol was registered. Studies were selected without a formal quality-appraisal tool, only English-language publications were included, and the evidence base is dominated by individual case reports, letters, and small case series. Reported doses, durations, and outcomes therefore reflect individual clinical

decisions and are subject to publication bias; they should be interpreted as hypothesis-generating rather than as treatment recommendations.

12. CONCLUSION

Pharmacotherapy for ABS remains guided almost entirely by accumulated case-level experience rather than controlled evidence. Fluconazole combined with strict dietary carbohydrate restriction remains the reasonable first-line approach, with susceptibility-guided escalation to alternative antifungals reserved for relapse, and FMT emerging as a promising option in refractory disease. Given the centrality of agent selection, dietary co-management, and long-term adherence support to successful outcomes, a structured pharmacist role in ABS care represents a logical and currently underutilized extension of existing management pathways.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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