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Management of Symptomatic Uncomplicated Diverticular Disease: A Retrospective Observational Study from Diagnosis to Treatment

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Abstract

Symptomatic uncomplicated diverticular disease (SUDD) currently lacks a fully established treatment consensus, while nutraceuticals are emerging as promising strategies to support patients. A retrospective observational study was conducted to investigate real-world diagnostic and therapeutic approaches to SUDD management. The study assessed whether a nutraceutical synbiotic supplement – containing *Lactobacillus rhamnosus* GG, *Hericium erinaceus*, berberine, quercetin, palmitoylethanolamide, and Undaria – could improve stool consistency (Bristol Stool Chart, BSC), abdominal pain intensity (Visual Analogue Scale, VAS), and fecal calprotectin levels. Data were collected at baseline (T0), one month (T1), and 2 months (T2). Colonoscopy was the most frequent diagnostic method (73.2%), followed by abdominal CT (11.6%). At T0, 57.1% of patients were not receiving pharmacotherapy; rifaximin (18.8%) and mesalazine (9.4% alone or combined) were most commonly prescribed agents. Pharmacological treatment declined at T2 (80.4% drug-free), with persistent mesalazine use (7.1%). A significant time-dependent improvement in BSC scores was observed in both constipated and diarrheic patients ($p<0.001$), with stool normalization achieved in 75% of patients at T2. VAS scores decreased progressively across the entire study population ($p<0.001$), independently of pharmacological treatment. Fecal calprotectin levels decreased significantly ($p<0.001$), with more patients achieving negative results. Its baseline correlation with VAS ($p<0.001$) disappeared at T2. These findings suggest that synbiotic anti-inflammatory supplements may support gut function, relieve pain, and normalize stool patterns in SUDD. Controlled clinical trials are warranted to confirm these preliminary outcomes.

Keywords: SUDD management; pharmacological treatment; synbiotic anti-inflammatory supplementation; nutraceuticals

Introduction

Diverticular disease, one of the most common gastrointestinal conditions in Western and industrialized countries, is a chronic disorder that continues to be a worldwide burden on healthcare system [1]. Diverticula, outpouchings in the wall of large bowel, are most frequent anatomical alterations observed in the human colon and define a chronic condition named diverticulosis. In most cases, diverticulosis remains asymptomatic and is often detected incidentally during colonoscopy or abdominal imaging [2]. However, approximately 25% of subjects develop clinical manifestations [1,3]. The term diverticular disease encompasses all conditions in which the presence of colonic diverticula is associated with symptoms. Its severity can range from symptomatic uncomplicated diverticular disease (SUDD) to complicated forms, including acute diverticulitis and diverticular hemorrhage [4].

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SUDD is a chronic condition characterized by persistent abdominal pain, typically localized in the left lower quadrant, along with changes in bowel habit, which may include diarrhea, constipation or alternating patterns. The symptoms occur in absence of macroscopic inflammation and can significantly impact on daily activities and the quality of life (QoL) [5].

The pathogenesis of SUDD remains incompletely understood and appears to be multifactorial. Key contributing factors include low-grade inflammation, alterations in gut microbiota composition, abnormal colonic motility, colonic mucosal ischemia, and neuro-immune interaction (visceral hypersensitivity), all of which may play a role in the onset of symptoms. In particular, inflammation may enhance neuromuscular dysfunction, leading to changes in bowel habits and abdominal pain. Patients with SUDD often show abnormal colonic motility, with prolonged low-frequency contractions in affected segments [4].

Diagnosing SUDD is challenging due to symptom overlapping with irritable bowel syndrome (IBS). However, key distinguishing features include sustained left lower quadrant pain lasting more than 24 hours, not relieved by bowel movements [6]. Diagnosis requires confirmation of diverticulosis through imaging techniques such as colonoscopy, computed tomography (CT) colonography (virtual colonoscopy), barium enema, or abdominal CT scan. Additionally, measuring fecal calprotectin levels may help support the diagnosis by indicating underlying inflammation [7].

The main goals in the management of SUDD are the relief of abdominal symptoms and the prevention of complications, such as acute diverticulitis. Although a standardized therapeutic approach has yet to be defined, several pharmacological and dietary strategies have been proposed for this condition [8].

Given the growing evidence supporting the involvement of intestinal dysbiosis in the pathogenesis of SUDD, several treatments have been developed with the aim of modulating the gut microbiota. Among these, rifaximin represents one of the most extensively studied options. Rifaximin is a poorly absorbed, broad-spectrum oral antibiotic that acts locally in the intestinal lumen, reducing bacterial overgrowth, decreasing microbial metabolic activity, and increasing fecal mass. Its minimal systemic absorption confers a safety profile and low risk of antimicrobial resistance [9].

Cyclic administration of rifaximin (800 mg/day for 7–10 days/month) has been shown to maintain remission in approximately 56% of patients over a 12-month follow-up period; however, a substantial proportion (≈ 44%) remains symptomatic at the end of treatment [10]. In this context, prebiotics, which selectively stimulate the growth of beneficial bacterial species, represent a promising strategy for restoring gut eubiosis [11]. Supporting this rationale, studies have demonstrated that patients with diverticula exhibit a depletion of *Clostridium* cluster IV compared to healthy controls, while symptomatic patients show lower levels of *Clostridium* cluster IX, *Fusobacterium*, and *Lactobacillaceae* than asymptomatic subjects [12]. Moreover, synbiotics – combinations of live microbial species (probiotics) and prebiotics – have been proposed to enhance probiotic survival and activity, providing a synergistic effect on gut microbiota modulation and overall intestinal health [13].

Beyond microbiota-targeted interventions, anti-inflammatory agents such as mesalazine (5-ASA derivatives) have also been evaluated in SUDD. The rationale for mesalazine use is based on the hypothesis that low-grade mucosal inflammation contributes to symptom generation. Continuous administration appears to be more effective than cyclic regimens; however, despite reports of symptom improvement and higher remission rates, overall evidence remains inconsistent, and its clinical utility is still debated [9,10,14]. In addition, natural compounds may represent promising strategies to support gut homeostasis. Among these, berberine and quercetin have gained increasing attention. Recent evidence suggests that berberine can alleviate intestinal inflammation, improve gut mucosal barrier function, and modulate gut microbiota composition [15]. Similarly, quercetin, owing to its well-documented antioxidant and immunomodulatory properties, has been shown to increase tight junction expression and preserve microbial diversity, with significant enrichment of beneficial taxa such as *Lactobacillus* and *Bacteroides* [16,17].

The present retrospective observational study was designed to investigate whether a synbiotic anti-inflammatory supplement could support patients with SUDD. Based on the established pathophysiological role of gut dysbiosis and low-grade mucosal inflammation in SUDD, a multicomponent nutraceutical formulation was evaluated, comprising *Lactobacillus rhamnosus* GG, *Hericium erinaceus*, berberine, quercetin, palmitoylethanolamide (PEA), and Undaria.

Materials and Methods

Study design and setting

A retrospective, multicenter, analysis of real-world data collected during routine clinical practice was performed in patients with SUDD. Data were collected between November 2024 and May 2025. As this was a non-interventional, real-world study, patients continued, where applicable, their usual pharmacological treatments according to the best clinical judgment and practice. Informed consent for this study was waived, given its retrospective, observational, and entirely anonymized design. No individual patient data capable of directly or indirectly identifying study participants were

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collected, retained or processed at any stage of the research. All information was obtained from fully anonymized medical records, with patient identifiers removed and data processed in aggregated form. The study was conducted and reported in accordance with the Declaration of Helsinki and Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines.

Data collection

The following data were extracted from patient records:

(1) Demographics: age and gender

(2) Diagnostic procedure used to confirm diverticulosis (e.g., barium enema, colonoscopy, abdominal CT scan, virtual colonoscopy, abdominal ultrasonography)

(3) Stool consistency, assessed using the bristol stool chart (BSC)

(4) Abdominal pain intensity, measured using a visual analogue scale (VAS) ranging from 0 (no pain) to 10 (unbearable pain)

(5) Pharmacological and nutraceutical treatments administrated

(6) Fecal calprotectin levels, when available

Inclusion criteria

(1) Adult patients with confirmed SUDD diagnosis. SUDD was defined as the presence of diverticulosis confirmed by at least one diagnostic procedure, associated with abdominal symptoms in the absence of acute inflammation or other organic disease.

(2) Patients receiving pharmacological treatments for SUDD, including rifaximin, mesalazine, or other relevant interventions.

(3) Patients not receiving any pharmacological treatment for SUDD.

(4) Patients receiving a synbiotic anti-inflammatory supplement once daily for at least 2 consecutive months. The formulation consisted of one capsule containing *Lactobacillus rhamnosus* GG (1×10^{10} CFU), *Hericium erinaceus* dry extract (100 mg, titrated to 10% polysaccharides, corresponding to 10 mg polysaccharides, PEA (100 mg), and *Undaria pinnatifida* dry extract (100 mg, titrated to 10% fucoxanthin; equivalent to 10 mg fucoxanthin) and one tablet containing berberine extract (*Berberis* spp. dry extract titrated to 85% berberine, 942 mg, corresponding to 800 mg berberine) and quercetin (200 mg).

Exclusion criteria

(1) Patients treated concomitantly with other food supplements potentially affecting the study outcomes, including probiotics, fiber supplements, or anti-inflammatory nutraceuticals.

(2) Patients with concomitant intestinal, renal, pancreatic or endocrine diseases.

(3) Patients with missing data for age, sex, or primary and secondary outcomes at any time point.

Outcomes and Timing

The primary outcome was the longitudinal change in stool consistency, measured by BSC. The secondary outcome was the change in abdominal pain intensity, measured by VAS. Both outcomes were assessed at baseline (T0), after 4 weeks (T1), and after 8 weeks (T2). Because treatment exposure varied across time points, patients were stratified by concomitant pharmacological treatment for descriptive analyses. Exploratory outcomes included fecal calprotectin levels – measured at T0 and T2 in the subset of patients with available data – and their correlation with BSC and VAS. Missing calprotectin data were not imputed; analyses were conducted on available observations only.

Statistical analysis

Categorical variables were presented as frequencies (n) and percentages (%). Continuous variables were reported as mean ± standard deviation (SD), and model-based estimates were accompanied by standard errors (SE) and/or 95% confidence intervals (CI). Normality was assessed using Shapiro-Wilk test; non-parametric tests were used when assumptions were violated.

Longitudinal changes in BSC and VAS scores were analyzed using linear mixed-effects models (LMM) to account for repeated measurements within subjects. Fixed effects included time (T0, T1, T2), pharmacological treatment (yes/no), baseline stool pattern (baseline BSC categorized as diarrheic, normal or constipated), their interactions, and covariates (age, gender). A random intercept for subject ID was included to account for intra-subject variability. Estimated marginal means (EMM) were computed for significant effects and interactions. Pairwise comparisons between timepoints (T0 – T1; T1 – T2; T0 – T2) were performed within each baseline stool pattern × pharmacological treatment stratum, with p-values adjusted using Tukey method to account for multiple comparisons within each stratum-specific family of time contrasts. Fecal calprotectin was analyzed descriptively in the subset of patients with data available at both T0 and T2; paired comparisons were performed using the Wilcoxon signed-rank test. Correlations were explored using Spearman's rank correlation coefficient, with 95% bootstrap confidence intervals (two-tailed tests).

Statistical analyses for LMM and EMM were performed using Jamovi software (version 2.6.44), interfaced with the R (packages: lme4 and emmeans). Additional analyses and graphical representations were conducted using GraphPad Prism software (version 5.0). A p-value < 0.05 was considered statistically significant.

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Results

A total of 224 patients with SUDD were included in the analyses. The STROBE flow chart of patient selection is shown in Figure 1. Baseline patients' characteristics are reported in Table 1.

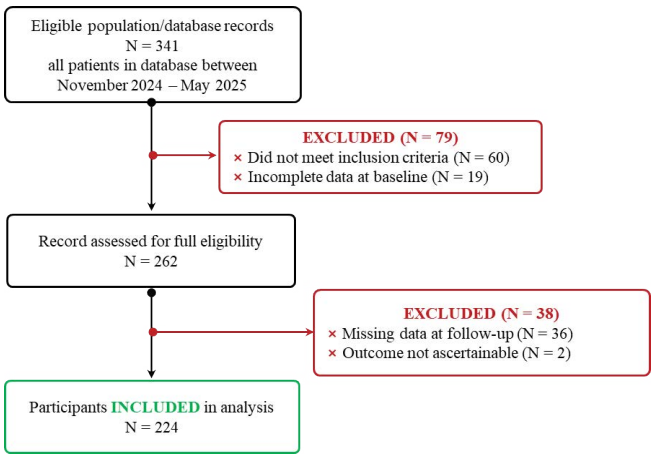

Figure 1: Strengthening the STROBE flow chart illustrating the selection of eligible patients, including reasons for exclusion at each stage.

Table 1: General characteristics of patients at baseline

Characteristics	
Total number	224
Age – mean ± standard deviation	65.3 ± 12.7
Gender	
Females – N (%)	121 (54.0%)
Males – N (%)	103 (46.0%)
Bristol stool chart	
Constipation – N (%)	56 (25.0%)
Normal – N (%)	64 (28.6%)
Diarrhea – N (%)	104 (46.4%)
Visual analogue scale	
Score – mean ± SD	4.2 ± 2.1

Diagnostic approach

Among the 224 patients included in the analysis, colonoscopy was the most frequently employed diagnostic procedure, used alone or in combination with other methods in 83.5% of cases (N=187), followed by abdominal CT scan, employed alone or in combination in 20.1% of cases (N=45). A multimodal diagnostic approach, defined as the concurrent use of two or more procedures, was adopted in 11.2% of patients (N=25). Virtual colonoscopy and barium enema were used less frequently, documented alone or in combination in 4.9% (N=11) and 3.6% (N=8) of cases, respectively. Full details are reported in Table 2.

Table 2: Diagnostic procedure used to confirm diverticulosis

Diagnostic procedure	N observations	%
Barium enema	2	0.9
Barium enema + Colonoscopy	3	1.3
Barium enema + Colonoscopy + Abdominal CT	1	0.4
Barium enema + Colonoscopy + Virtual Colonoscopy	1	0.4
Barium enema + Abdominal CT	1	0.4
Colonoscopy	164	73.2
Colonoscopy + Virtual Colonoscopy	2	0.9
Colonoscopy + Virtual Colonoscopy + Abdominal CT	1	0.4
Colonoscopy + Abdominal CT	15	6.7
Virtual Colonoscopy	6	2.7
Virtual Colonoscopy + Abdominal CT	1	0.4
Abdominal CT	26	11.6

Conventional pharmacological treatments

At baseline, rifaximin monotherapy was the most frequently prescribed treatment, administered to 42 patients (18.8%). Mesalazine was utilized as monotherapy in 21 patients (9.4%) or in combination with rifaximin in an additional 21 patients (9.4%). Combined regimens incorporating other medications – including doxycycline, metronidazole, antispasmodics, bismuth and antidiarrheal agents – were documented in 12 patients (5.3%). Notably, 128 patients (57.1%) did not receive any pharmacological treatment at baseline (Table 3). Treatment patterns remained consistent at the one-month follow-up (T1). By the two-month evaluation (T2), modifications were observed across all treatment categories. Rifaximin monotherapy decreased to 21 patients (9.4%), and mesalazine-rifaximin combination therapy declined substantially to 3 patients (1.3%). Mesalazine monotherapy showed a modest reduction to 17 patients (7.8%), while the use of alternative antibiotics and combined regimens decreased correspondingly. Most notably, the proportion of patients receiving no pharmacological treatment increased to 180 patients (80.4%) at T2, representing a 23-percentage point increase from baseline (Table 3).

Stool consistency assessment and temporal evolution of BSC scores

At baseline, 56 patients (25.0%) presented with constipation, 104 (46.4%) with diarrhea, and 64 (28.6%) with normal stool consistency. Following one month of synbiotic anti-inflammatory supplementation (T1), the distribution shifted to 35 patients (15.6%) with constipation, 52 (23.0%) with diarrhea, and 137 (61.0%) with normal bowel function. At the end of the observation period (T2), 41 patients (18.3%) presented constipation, 15 (6.7%) diarrhea, and 168

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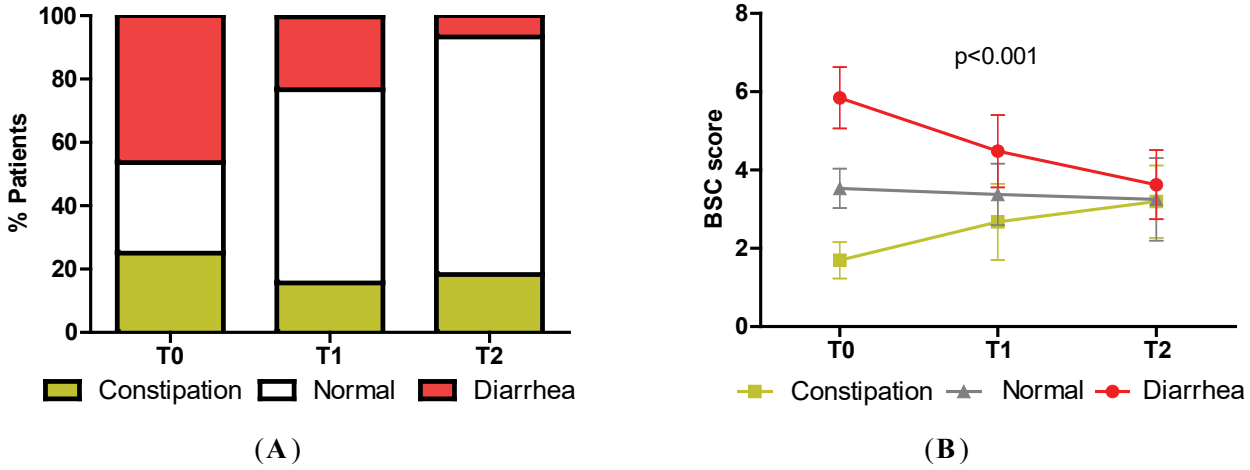

Figure 2: Bowel habit distribution and observed BSC over time. **(A)** Percent of patients with constipation, diarrhea, or normal stool consistency at T0, T1, and T2. **(B)** Observed mean BSC±SD at T0, T1, and T2, stratified by baseline stool phenotype.

(75.0%) normal stool consistency (Figure 2A). BSC scores varied significantly over time, with trajectories depending on baseline stool phenotype ($p<0.001$). Diarrheic and constipated patients showed progressive movement toward normal stool consistency from T0 to T2 ($p<0.001$), whereas patients with normal baseline stool consistency remained stable (Figure 2B). Complete fixed-effect estimates are reported in Supplementary Table S1.

Pharmacological treatment status and BSC trajectories over time

Across the study, EMM of BSC did not differ significantly between patients receiving versus not receiving concomitant pharmacological treatment at any time point, including during synbiotic supplementation phases (T1 and T2, Figure 3). When evaluating changes over time within each subgroup, a significant time effect was observed. Among patients receiving pharmacological treatment, BSC improved from T0 to T1 ($p=0.013$) and from T0 to T2 ($p=0.0014$), with no further significant change between T1 and T2. In patients not receiving pharmacological treatment, a progressive improvement was observed across the entire follow-up (T2 vs T1 vs T0) with all pairwise comparisons reaching statistical significance (p-values <0.001 to <0.0001). EMM outputs and Tukey-adjusted post-hoc contrasts are provided in Supplementary Appendix A (Tables A1-A3).

Stratified analysis according to baseline stool phenotype

The distribution of observed BSC values across time points (T0, T1, T2), stratified by baseline stool phenotype and current treatment status, is shown in Supplementary Figure S1 to visualize the underlying data within each subgroup. As treatment exposure varied across time, these plots are descriptive; all inferential comparisons (including reported

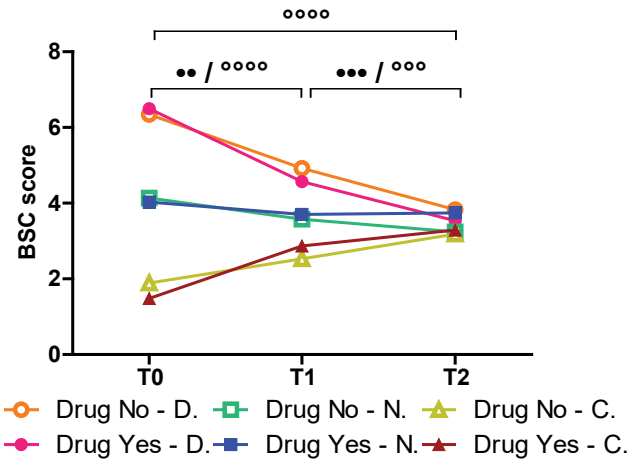

Figure 3: EMM of BSC scores across time (T0, T1, T2) derived from the linear mixed-effects model, stratified by baseline stool phenotype [diarrhea (D), normal (N), constipation (C)] and pharmacological treatment status (Drug: yes/no). Each panel (D, N, C) displays two trajectories (Drug yes vs Drug no). Tukey-adjusted pairwise comparisons among timepoints (T0–T1, T1–T2, T0–T2) were conducted within each baseline phenotype × treatment stratum. Filled symbols indicate Drug yes (• p=0.013; •• p=0.0014); open symbols indicate Drug no (°°° p<0.001; °°°° p<0.0001).

p-values) derive from the LMM/EMM framework with Tukey adjustment, with full outputs provided in Appendix A (Table A4). Consistent with the model-based results, BSC scores shifted over time toward normalization. Values increased in baseline constipation, decreased progressively in baseline diarrhea (T0>T1>T2) (Supplementary Figures S1A and S1B, respectively), and remained broadly stable in baseline normal stools with a decrease in the no-pharmacological treatment subgroup (Figure S1C).

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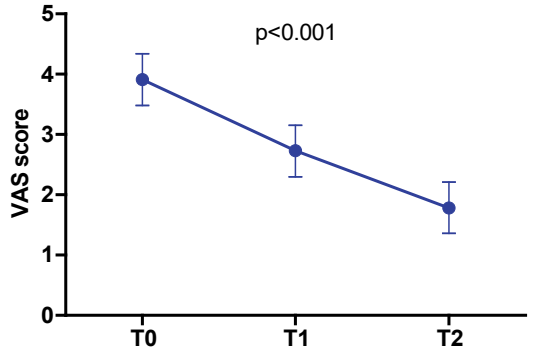
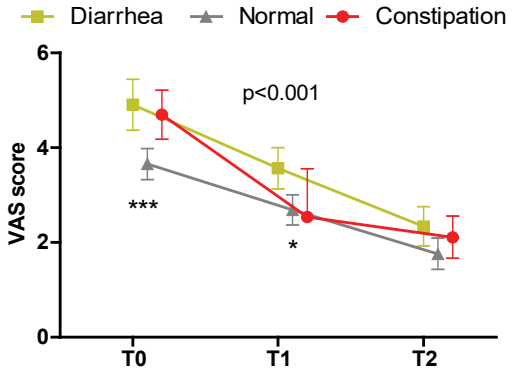
Pain Intensity (VAS)

Pooled and stratified VAS analysis according to baseline stool phenotype

VAS scores decreased significantly over time. EMM from the linear mixed-effects model showed a progressive reduction from T0 to T1 and T2, consistent with a significant overall time effect (p<0.001; Figure 4A). Complete fixed-effect estimates are reported in Supplementary Table S1. When patients were stratified by baseline stool phenotype, VAS decreased over time in each subgroup (all p<0.001). Between-group comparisons indicated higher baseline VAS scores in patients with diarrhea compared with those with normal stool consistency (p<0.001), while the difference between constipation and normal did not reach statistical significance (p=0.08). At T1, these between-group differences were attenuated (diarrhea vs normal, p=0.014; constipation vs normal, p=0.975) and were no longer evident at T2 for any comparison (Figure 4B). Full EMM and post-hoc outputs are provided in Appendix B (Tables B2-B3).



(A)



(B)

Figure 4: VAS scores overtime in the overall cohort and by baseline stool phenotype. (A) EMM of VAS at T0, T1, and T2 for the overall population. (B) VAS EMM trajectories stratified by baseline stool phenotype. Error bars indicate 95% confidence intervals. Asterisks indicate between-group comparisons (Diarrhea vs Normal) at the corresponding timepoint: *p<0.05; ***p<0.001.

Stratified VAS analysis according to pharmacological treatment

When stratified by pharmacological treatment status (Figure 5), within-group pairwise comparisons indicated significant improvements across all timepoint contrasts in both groups. In patients receiving pharmacological treatment, VAS decreased from T0 to T1 and from T0 to T2 (both p<0.0001), and from T1 to T2 (p=0.002). In drug-free patients, VAS decreased from T0 to T1 and from T0 to T2 (both p<0.0001), and from T1 to T2 (p<0.0001).

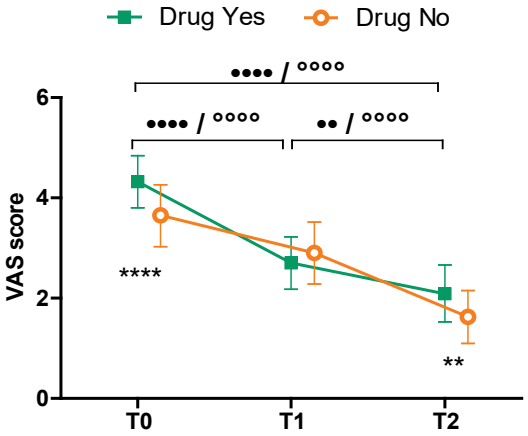


Figure 5: EMM of VAS scores across time (T0, T1, T2) derived from the linear mixed-effects model, stratified by pharmacological treatment status (Drug: yes/no). Filled symbols indicate Drug Yes (•• p<0.01; •••• p<0.0001); open symbols indicate Drug No (°°°° p<0.0001). Asterisks indicate between-group comparisons at the corresponding timepoint: **p<0.01; ****p<0.0001.

Between-group contrasts (Drug yes vs Drug no), averaged over baseline stool phenotype, indicated higher VAS in the pharmacologically treated group at T0 (p=0.0001) and T2 (p=0.003), while no significant difference was observed at T1 (p=0.1858). Full EMM outputs and post-hoc contrast tables are provided in Appendix B (Tables B4-B6).

Stratified analysis according to baseline stool phenotype

The distribution of VAS scores across time points (T0 – T2), stratified by baseline stool phenotype and current treatment status, is shown in Supplementary Figure S2 (descriptive). All inferential comparisons (including reported p-values) derive from the LMM/EMM framework with Tukey adjustment, with full outputs provided in Appendix B (Table B7). VAS scores decreased over the observation period (T0>T1>T2) across all stool baseline phenotypes groups – constipation (Figure S2A), diarrhea (Figures S2B), and normal (Figures S3B) BSC scores – and in both Drug Yes and Drug No subgroups.

Fecal calprotectin levels

Complete fecal calprotectin levels data were available for 24 patients. In this subset, calprotectin levels decreased

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significantly at T2 compared to T0 ($p<0.001$) (Supplementary Figure S3A). The distribution of patients across calprotectin categories changed over the treatment period. At baseline, 3 patients (12.5%) showed calprotectin levels <50 µg/g (negative test), which increased to 9 patients (37.5%) at T2. Patients with moderately elevated calprotectin levels (50-120 µg/g) increased from 12 (50%) at baseline to 14 (58.3%) at T2. Conversely, patients with elevated calprotectin levels >120 µg/g levels (positive) decreased from 9 (37.5%) at baseline to 1 (4.2%) at T2 (Supplementary Figure S3B).

Moreover, correlation analysis revealed a significant positive correlation between calprotectin levels and VAS pain scores at baseline ($r = 0.657$; $p<0.001$, Supplementary Figure S3C), which was no longer significant at 2-month follow-up treatment ($r = 0.235$; $p=0.267$, Supplementary Figure S3D).

Discussion

This real-world, retrospective observational study examined multiple aspects of SUDD management, including diagnostic practices and treatment strategies. Symptom trajectories over time – abdominal pain and stool consistency – were evaluated, and the potential contribution of a synbiotic anti-inflammatory nutraceutical, administered either in combination with pharmacological treatments or as monotherapy, to symptom control and QoL was explored.

Diagnostic approaches

As expected, colonoscopy was the most frequently employed diagnostic tool, used alone in ~73% of cases, in line with current guidelines recommending its use to rule out alternative diagnoses such as colorectal cancer or IBD (National Guideline Centre, UK). However, colonoscopy is not superior to CT colonography or barium enema in detecting diverticula, particularly in the sigmoid colon, highlighting the absence of a gold standard [4]. A multimodal approach – combining colonoscopy with imaging techniques like CT, virtual colonoscopy, or barium enema – was used in 8.8% of cases, often due to incomplete procedures, atypical symptoms, or the need for detailed anatomical clarification in older or complex patients. CT alone was the second most common method (11.6%), supporting its role as a non-invasive and reliable alternative when colonoscopy is not feasible.

Other methods, including barium enema (0.9%) and virtual colonoscopy (2.7%), were used selectively. Overall, this diagnostic heterogeneity reflects real-world practice. While colonoscopy remains central, integrating CT and ultrasound may improve diagnostic precision and enable more personalized management. These findings highlight the need for standardized, evidence-based diagnostic pathways in SUDD [18,19].

Management of SUDD

SUDD management aims to control symptoms, improve QoL, and prevent recurrences or complications. Standard approaches include dietary and lifestyle measures (e.g., high-fiber intake), probiotics, cyclic rifaximin, and anti-inflammatory agents like mesalazine, whereas elective surgery is generally reserved for refractory diseases [5].

In this real-world cohort, baseline prescribing patterns were consistent with these commonly adopted strategies. Rifaximin monotherapy was the most frequently used pharmacological option (18.8%), followed by mesalazine (alone or in combination; 9.4%), while other antibiotics and symptomatic treatments were less commonly prescribed. Notably, more than half of the patients (57.1%) received no pharmacological treatment at baseline, possibly reflecting milder symptoms and/or preference for lifestyle-based management.

These distributions are compatible with guidelines that prioritize rifaximin for symptom relief and prevention of complications in uncomplicated disease, while discouraging routine broad-spectrum antibiotics outside complicated disease [20,21]. These findings are also aligned with previous reports describing rifaximin as a widely used option in clinical practice for symptomatic diverticular disease, both to reduce symptoms and to lower the risk of diverticulosis in primary and secondary prevention setting [22]. Similarly, Cuomo et al. [23] reported that, in Italy, rifaximin is among the most frequently used drugs and is often perceived as a reference treatment for symptomatic diverticular disease.

Mesalazine has been increasingly investigated for its potential to modulate the low-grade inflammatory in SUDD, with randomized trials reporting benefits on symptom control and, in some settings, recurrence-related outcomes [24,25].

Over follow-up, overall pharmacological treatment intensity decreased. By T2, rifaximin use – either alone or combined with mesalazine – decreased markedly, while mesalazine monotherapy showed a smaller reduction. Notably, the proportion of patients receiving no pharmacological treatment increased to 80% at T2. This temporal shift is compatible with overall symptom improvement observed during the study period and occurred in parallel with synbiotic anti-inflammatory supplementation. However, given the retrospective observational design and treatment heterogeneity, this association should be interpreted with caution.

Stool consistency

At baseline, stool consistency was frequently abnormal, with diarrhea predominating in 46.4% of cases and constipation affecting 25% of patients, while 28.6% demonstrated normal bowel habits, consistent with literature reporting diarrhea as a predominant bowel pattern in SUDD [5].

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During follow-up – concurrent with synbiotic anti-inflammatory supplementation from T1 onward – stool consistency progressively shifted toward the normal range, with 75% of patients achieving normal BSC scores by T2. Improvements were time-dependent in both diarrhea and constipated phenotypes, whereas patients with normal stool consistency remained broadly stable. Importantly, the model based analyses indicated no meaningful differences in BSC trajectories between patients receiving versus not receiving concomitant pharmacological treatment at any time point.

This pattern is consistent with a beneficial modulatory contribution of the synbiotic formulation on bowel function, in line with its multi-component design targeting gut microbiota, inflammation, and motility. LGG and *Hericium erinaceus* may provide probiotic/prebiotic support, while berberine may contribute complementary effects relevant to diarrhea-predominant SUDD. LGG is supported by clinical evidence in acute and antibiotic-associated diarrhea [26,27] and has been reported to attenuate intestinal inflammation [28] and enhances barrier integrity via tight-junction regulation [29]. Berberine exerts broad antimicrobial activity, inhibits electrolyte hypersecretion, exerts anti-inflammatory effects, and modulates motility/intestinal transit, supporting relevance for diarrhea-related symptoms [30].

Consistently, in drug-free patients with baseline diarrhea (Drug No), BSC improved over follow-up, and similar improvements were observed among patients who discontinued pharmacological therapy while continuing supplementation, consistent with the time-dependent normalization trend irrespective of drug status.

Quercetin may be particularly useful in constipation through anti-inflammatory and pro-motility effects (including mAChRs-related mechanisms and mucin secretion [31,32] aligning with the observed BSC improvement over time in constipated patients across treatment strata.

However, probiotic selection remains a clinical challenge because efficacy is largely strain- and context-specific. Multi-strain formulations are not consistently superior to single-strain approaches; for example, LGG alone was more protective against necrotizing enterocolitis than multi-strain mixtures containing *B. lactis* [27].

A retrospective study reported that a 12-month probiotic course of *Clostridium butyricum* CBM588 showed similar outcomes to cyclic rifaximin over follow-up (including no acute diverticulosis episodes in either group) and potentially grater patient-reported symptom relief [33].

Overall, these findings support that synbiotic formulations may complement pharmacological therapies and, in some cases, be associated with symptoms improvement even when used without concomitant drugs.

Pain intensity symptom relief across stool patterns

Abdominal pain intensity, assessed using the VAS scale, decreased progressively over time across the overall cohort and within each baseline stool phenotype. Model-based analyses indicated that between-phenotype differences observed at baseline were attenuated at T1 and were no longer evident at T2, when VAS score trajectories converged across phenotypes. Notably, even patients with normal stool consistency at baseline – thus without overt alterations in bowel motility – experienced a substantial reduction in pain. This suggests that pain relief was not solely driven by improvements in bowel habits but may reflect a broader modulation of symptom-generating pathways. Mechanistically, this pattern is consistent with the anti-inflammatory and neuro-modulatory properties of the synbiotic formulation. The product contains, among other components, LGG, berberine, quercetin, PEA, and *H. erinaceus*, compounds with documented effects on gut inflammation, epithelial barrier integrity, and nociceptive signaling. These synergistic mechanisms may underline the observed reduction in pain intensity and normalization of bowel function, particularly in patients with altered stool patterns. In particular, PEA, an endocannabinoid-like compound, exerts well-documented anti-inflammatory and analgesic effects by modulating mast cell activity and interacting with CB2-like receptors and other targets involved in pain transmission [34].

Model-based analyses showed a progressive, time-dependent decrease in VAS scores in both Drug Yes and Drug No groups, with within-group pairwise comparisons indicating significant improvements across all time-point contrasts in each subgroup. Between-group contrast (Drug Yes vs Drug No), average over baseline stool phenotype, indicated higher VAS scores in pharmacologically treated patients at T0 and T2, whereas no significant difference was observed at T1. This pattern is consistent with an overall reduction in pain over follow-up regardless of pharmacological treatment status, while baseline (and residual) differences in pain severity between groups may reflect differences in clinical presentation and treatment allocation. These findings are further supported by the descriptive distribution of VAS scores across time points stratified by baseline stool phenotype and current treatment status. Specifically, a time-dependent reduction in pain was observed within each phenotype (diarrhea, constipation, normal stool consistency) across treatment subgroups, including patients who discontinued pharmacological therapy by T2. Overall, the results suggest that synbiotic supplementation during follow-up may complement standard management strategies for abdominal pain in SUDD; however, causal attribution cannot be established in this observational setting.

Fecal Calprotectin and inflammation

Inflammation may represent a key factor in the

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pathophysiology of SUDD symptoms and the development of complications. Several studies suggest that fecal calprotectin is useful not only in differential diagnosis but also in monitoring the therapeutic response. In fact, fecal calprotectin values tend to decrease in patients responding to treatment, whereas they increase in those who failed to achieve remission [35].

In this cohort, fecal calprotectin data were available for a limited subset of patients (N = 24 with paired T0-T2 measurements). In this exploratory analysis, calprotectin levels decreased from baseline to T2, accompanied by a shift toward fewer positive tests and a corresponding increase in negative results. In parallel, whereas calprotectin levels were positively correlated with VAS scores at baseline, this association was no longer evident at T2. These findings are consistent with prior evidence indicating that fecal calprotectin is typically elevated in SUDD and correlates with both severity of abdominal pain and the extent of diverticulosis [5].

Interestingly, Kvasnovsky et al. reported that, although a multi-strain probiotic improved constipation and diarrhea, it did not significantly affect bloating, abdominal pain, or fecal calprotectin after 3 months of supplementation. This discrepancy may suggest a stronger anti-inflammatory response in the present cohort, potentially related to the multi-component synbiotic formulation. Mechanistically, this pattern is consistent with synergistic anti-inflammatory action of the nutraceutical combination – particularly berberine, quercetin, PEA, and LGG (which may exert anti-inflammatory effects beyond its probiotic activity). Because the proportion of patients receiving mesalazine alone or in combination with other pharmacological treatment remained stable o decrease between T0 and T2, the observed reduction in calprotectin levels may be compatible with an additional contribution of synbiotic supplementation during follow-up.

This study has important limitations inherent to its retrospective observational design. In particular, the absence of a control group and the heterogeneity of treatment exposure limit causal interpretation of the observed association. For these reasons, the work should be regarded as a pilot investigation, and the findings should be interpreted cautiously. Nonetheless, the consistency and the statistical strength of the observed changes across outcomes support the presence of a robust temporal trend during the observation window, which warrants confirmation in adequately powered prospective studies with longer follow-up.

Conclusions

This real-world study provides insight into current clinical management of SUDD, highlighting heterogeneous treatment patterns in routine practice. A general improvement in symptoms was observed across patient subgroups, including those who discontinued pharmacological therapy, suggesting a potential benefit of integrated management approaches. In particular, the synbiotic anti-inflammatory formulation was associated with improvements in stool consistency, abdominal pain, and inflammatory markers. While these findings suggest that nutraceuticals may represent complementary – and in selected patients potentially stand-alone – supportive options, the lack of universally accepted guidelines underscores the need for evidence-based standardization. Given the observational design, absence of randomization, and treatment heterogeneity, causal inference cannot be drawn. Further randomized controlled trials are warranted to confirm these outcomes and to clarify the relative contribution of pharmacological and non-pharmacological approaches in SUDD management.

Author's contributions:

A.R. and L.B.: conceptualization, study design (framework and methodology), critical review.

A.G., C.T., L.N., C.G., F.M., A.T., G.P., P.P., C.N., V.C., L.C. (Lina Corbi), E.L., G.D., R.Q.: data collection. L.C. (Lucia Chico): original draft preparation, statistical analysis, manuscript editing, critical review. G.D.S.: study design, manuscript review.

All authors have read and approved the final version of the manuscript.

Conflicts of interest:

The authors L.B., G.D.S., and L.C. (Lucia Chico) are employed by the company Laboratori Aliveda Srl, Crespina Lorenzana, Pisa, Italy. The remaining authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

AI Use Disclosure:

During the preparation of this manuscript, ChatGPT (OpenAI) was used to assist in the generation of R programming scripts and prompts. The authors independently performed, reviewed, and validated all analyses and take full responsibility for the content of this work.

Supplementary Files Link

https://cdn.fortunejournals.com/supply/management-of-symptomatic-uncomplicated-diverticular-disease-a-ret-10010-supplementary.pdf

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