

Improvement in Liver Function Tests in a Male Patient with Primary Biliary Cholangitis Following Faecal Microbiota Transplantation (FMT): Case Report

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Case Report

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Abstract

Primary biliary cholangitis (PBC) is a chronic disease potentially resulting in liver cirrhosis, liver failure and death. Although PBC predominantly affects women, there is a rising incidence in men who tend to present with worse prognosis and more advanced disease. Although the standard of care is lifelong ursodeoxycholic acid (UDCA), PBC remains progressive in a substantial proportion of patients. Growing evidence suggests that gut dysbiosis contributes to PBC pathogenesis, with faecal microbiota transplantation (FMT) as a potential treatment. This study presents the first report of FMT performed in a 47-year-old male with PBC.

In this study, shotgun metagenomic sequencing was performed on stool samples before and after FMT. Microbial diversity and changes were assessed using alpha diversity indices, multidimensional scaling, relative abundance profiling and log₂ fold-change analysis.

Following FMT and without the use of UDCA, the patient experienced a sustained downtrending of liver function tests (LFTs). The post-FMT microbiome profile increased in diversity and richness substantially, shifting closer toward that of the donor and healthy cohort. There was a marked increase in the number of species shared between the patient and donor from 18 to 110 post-FMT, indicating successful donor engraftment. Amongst the top 10 bacteria detected exclusively post-FMT were *F. prausnitzii* and *A. butyriciproducens*, possessing roles in anti-inflammation and preserving intestinal mucosal integrity.

This report demonstrates promising clinical and microbial shifts with FMT. FMT may serve as an alternative or adjunct to current treatment by directly addressing gut dysbiosis, ultimately reducing pill burden and improving overall quality of life.

1. Introduction

Primary biliary cholangitis (PBC) is a chronic, autoimmune disease involving progressive destruction of the small intrahepatic bile ducts, causing cholestasis and potentially resulting in liver cirrhosis, liver failure and death.¹ Globally, it is the most common form of autoimmune liver disease.²

PBC is typically diagnosed through biochemical and/or histological findings, rather than clinical manifestations.^{3,4,5} Although PBC predominantly affects women, there is a rising incidence in men, as the male-to-female ratio has increased from 1:9 to 1:4 in recent years.¹ Male PBC patients tend to present with relatively worse prognosis, more advanced disease and higher rates of hepatocellular carcinoma (HCC) compared to female patients.⁶

Currently, the standard of care for PBC is lifelong ursodeoxycholic acid (UDCA) which improves transplant-free survival and delays progression to liver cirrhosis.⁷ However, up to 40% of PBC patients are non-responders to this first-line drug.⁸ Even with second-line adjuncts such as obeticholic acid and peroxisome proliferator activated-receptors (PPARs) agonists (e.g. bezafibrate, elafibranor, seladelpar), PBC remains progressive in substantial proportion of patients, with persistent symptoms such as

pruritus, fatigue, and limited disease-modifying options targeting autoimmunity, fibrosis, or dysbiosis.⁹ Therefore, developing therapeutic alternatives to alleviate the symptoms and improves outcomes of PBC would be of significant clinical benefits.

A preclinical mouse model study demonstrated that faecal microbiota transplantation (FMT) from PBC patients produced liver lesions and worsened liver injury in recipient mice, suggesting that gut dysbiosis contributes to PBC pathogenesis.¹⁰ The pathophysiology of PBC is thought to involve gut microbiota dysbiosis, bile acids imbalance and immune-mediated bile duct injury.⁸ This suggests that microbiome-targeted therapies such as FMT could be a potential treatment option, offering the possibility of improved clinical outcomes without reliance on pharmacological therapy.

In this case study, we present the first reported instance of FMT performed in a patient with PBC. The patient's gut microbiome profile and liver function tests were regularly assessed via stool and blood collection pre and post treatment. We hypothesize that the pathophysiology of PBC may be linked to gut dysbiosis, which could be normalised through FMT as an alternative treatment approach.

2. Materials and Methods

2.1. FMT procedure

The patient was fully informed of the study objectives and publication plan and provided explicit written consent. The FMT donor underwent comprehensive screening in accordance with the international consensus.^{11,12,13,14,15,16} A fresh stool sample was collected from the donor, processed under anaerobic conditions and prepared according to standard protocol to produce the FMT product. The patient received a 3-day course of oral vancomycin and standard bowel preparation prior to FMT delivery via colonoscopy. Stool samples for microbiome profiling were collected prior to the FMT and post FMT. The stool samples were collected in DNA/RNA shield fecal collection kit (Zymo research) in 1:4 ratio at ambient temperature.

2.2. Shotgun metagenomic sequencing and data analysis

DNA extraction - Stool samples were collected using Zymo DNA/RNA Shield Stool Collection Tubes and stored at – 80°C until processing. DNA was extracted using QIAmp PowerFecal Pro DNA extraction kits (Qiagen) according to the manufacturer's protocol. Extracted DNA concentration and quality were assessed using Qubit Fluorometer (Thermo Fisher Scientific). DNA samples were stored at – 80°C and sent for library preparation. Shotgun sequencing was carried out on Illumina NovaSeq 2x150PE. Raw sequences were processed with KneadData, MetaPhlAn4 and HUMAnN3 under bioBakery workflow.¹⁷

For analysis, the faecal microbiota of the patient pre- and post-FMT procedure were compared to both the FMT donor and a healthy cohort from AMILI's database, matched by age, gender and ethnicity.

Analysis was conducted in RStudio version 4.3.3 using Phyloseq version 1.42.0¹⁸ and phyloseqCompanion version 1.1¹⁹ and ggplot2 version 4.0.1.²⁰

Raw abundance data were normalised using log transformation, with a pseudocount of 0.0001 added to remove 0 counts. The log2 fold change in bacterial species between pre- and post-FMT was then calculated to determine the magnitude of microbiome changes following FMT as follows:

$$\text{Log2 fold change} = \text{Log2}[\text{Post-FMT}_{\text{relative abundance}} / \text{Pre-FMT}_{\text{relative abundance}}]$$

3. Results

3.1. Case presentation

The patient is a 47-year-old Chinese male who was incidentally diagnosed with PBC through routine diagnostic testing in 2012. He was asymptomatic, with no clinical signs of biliary stasis. No treatment was initiated at diagnosis. The disease was actively monitored via liver function tests (LFTs) over a period of 7 years. With the gradual uptrends in LFTs over this period, the patient was subsequently prescribed with UDCA, following which his liver enzymes normalised. FMT was pursued at a later stage as an alternative treatment strategy to enable deprescription of UDCA and reduce long-term dependency on pharmacological drugs.

3.2. Microbiome profile pre- and post-FMT

At baseline, the patient's microbiome sample exhibited markedly reduced alpha diversity relative to the donor, as reflected across all three indices – Chao 1 (richness), Shannon (diversity) and Pielou (evenness).

Following FMT, both microbial diversity and richness increased substantially, with the patient's profile shifting closer toward that of the donor and the healthy cohort. In contrast, evenness remained largely unchanged post-FMT and continued to diverge from the donor profile (Fig. 1). This suggests FMT's ability to restore the overall taxonomic richness and diversity, it did not alter the underlying dominance structure of the patient's microbiome, potentially reflecting the incomplete engraftment or persistence of dominant taxa associated with PBC leading to disease development and progression.

Principal coordinates analysis (PCoA) was performed to assess overall bacterial community composition. As illustrated in Fig. 2, the patient's pre-FMT microbiome was distinctly separated from both the donor and post-FMT profiles. Following FMT, the patient's microbiome shifted along Axis 1 (77% of variance explained) toward the donor profile, indicating partial convergence in overall community composition. However, complete overlap with the donor was not observed, and separation along Axis 2 (23% of variance explained) persisted, suggesting a partial microbiome engraftment following FMT.

Figure 3 and 4 present the top 10 bacteria species detected exclusively in pre- and post-FMT samples, respectively, highlighting taxa gained or lost following transplantation. Among taxa present only pre-FMT, *Enterocloster bolteae* and *Tyzzlerella nexillis* are notable as opportunistic pathobionts previously associated with intestinal dysbiosis, although their functional role in the patient warrants further investigation. Notably, *Bifidobacterium breve*, a well-characterised beneficial, commensal species, was

also detected only pre-FMT, suggesting that taxonomic loss following FMT was not uniformly directional (Fig. 3).

In contrast, several taxa detected exclusively post-FMT are recognised as short-chain fatty acid (SCFA)-producing species with established anti-inflammatory associations, including *Faecalibacterium prausnitzii*, *Blautia stercoris* and *Agathobaculum butyriciproducens*, suggesting a shift toward a more metabolically beneficial community structure following treatment (Fig. 4).

At the species level, sharing between the patient's microbiome and the donor increased substantially following FMT, from 18 shared species pre-FMT to 110 shared species post FMT (Fig. 5). Notably, 96 of these post-FMT shared species were absent from the patient's pre-FMT microbiome, indicating acquisition of donor-derived taxa following transplantation. This finding is consistent with successful engraftment of donor microbiota as a contributing factor to post-FMT microbiome restructuring.

Among the routine LFTs assessed, alanine aminotransferase (ALT), aspartate, aminotransferase (AST), alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT), bilirubin, total protein and albumin. We focused primarily on ALP and GGT, in keeping with established evidence that PBC patients typically display disproportionate elevation of these two biomarkers reflecting cholestatic liver injury and biliary inflammation, directly related to the severity of PBC. Figures 6 and 7 depict the longitudinal trends of ALP and GGT respectively across the study period, with initiation of UDCA treatment and the timing of the FMT procedure indicated. Both ALP and GGT, declined following UDCA initiation, and this downward trend was sustained following FMT, proceeding at a comparable rate despite tapering of UDCA (Fig. 6, 7). At the most recent assessment, performed 8 months post-FMT, ALP had normalised to 92U/L (normal clinical reference range: 50-116U/L).²¹ GGT remained elevated above the upper limit of normal at 111U/L (normal clinical reference range: <50U/L),²² though it likewise showed an overall decreasing trend following FMT.

4. Discussion

Given the sustained downward trend of LFTs following FMT in the patient, FMT could be a complementary or alternative treatment to UDCA, given its potential to modulate and restore the gut microbiome toward a healthier profile.

Our findings demonstrate that FMT has the potential to serve as a therapeutic option for restoring the overall microbiome of a PBC patient closer to that of healthy individuals. The baseline microbiome of PBC patients typically demonstrates significant gut dysbiosis, characterised by reduced microbial diversity, enrichment of opportunistic pathogens, and depletion of beneficial bacteria.²³ Specifically, depleted beneficial bacteria in PBC commonly include *Oscillospira* and *Sutterella*, both negatively correlated with anti-gp210 antibodies titres, and *Faecalibacterium* a butyrate-producing bacteria associated with reduced inflammation.¹ Enriched opportunistic pathogens typically include the

Enterobacteriaceae family, *Pseudomonas*, *Veillonella*, *Klebsiella*, *Streptococcus*, *Neisseriaceae* and γ -Proteobacteria.^{1,24,25}

Based on the findings, *Faecalibacterium prausnitzii* and *Agathobaculum butyriciproducens* were among the top 10 bacteria detected exclusively post FMT. As both *F. prausnitzii* and *A. butyriciproducens* are major butyrate producers with anti-inflammatory properties and roles in preserving intestinal mucosal integrity,^{26,27} suggesting a strong restorative effect of FMT on gut microbiome.

The relatively high post-FMT abundance of *F. prausnitzii* is notable given its reported hepatoprotective effects through various pathways. Utilising butyrate production, *F. prausnitzii* reduces intestinal inflammation and preserves gut barrier integrity, limiting translocation of inflammatory mediators to the liver.²⁸ Its role on lipid clearance has also been associated with reduced hepatic inflammation and lower levels of hepatic transaminases.²⁹

Furthermore, *A. butyriciproducens* was similarly detected only post-FMT treatment. Similar to *F. prausnitzii*, *A. butyriciproducens* contributes to the gut-liver axis through butyrate production, reducing inflammation.³⁰ *B. stercoris*, meanwhile has been associated with reduced hepatic inflammation via bile acid metabolism, specifically through increased production of beneficial secondary bile acids such as ursocholic acid (UCA).^{31,32,33} The post-FMT emergence and increased abundance of these bacteria is consistent with a shift counteracting the harmful hepatic effects associated with PBC.

Following FMT, the patient's microbiome shifted closer to that of the donor in terms of bacterial richness, diversity, and overall community composition, with a marked increase in the number of species shared between the patient and donor. This is consistent with partial engraftment of donor-derived taxa contributing to microbiome restructuring in the patient, although evenness did not shift toward the donor's profile, indicating that FMT did not fully dominate the patient's microbiota.

FMT may offer advantages over pharmacological therapy alone by directly addressing the gut dysbiosis characteristic of PBC. However, although FMT promoted some degree of microbiome restoration, the patient's microbial community remained dysbiotic relative to that of healthy individuals.^{23,34} Remodelling of the gut microbiota in PBC patients has been separately reported to increase fecal secondary and tertiary bile acids, indicating improved bacterial bile acid metabolism.³⁵ Consistent with this, UDCA treatment at baseline demonstrated significant dysbiosis at baseline in the patient including taxa such as *Ruminococcus sp AF13_28* and *Enterocloster bolteae* are associated with adverse outcomes in PBC. In a recent study, increased *Ruminococcus* abundance was associated with a poorer biochemical response to UDCA treatment, suggesting it may serve as a marker of treatment-resistant disease.³⁴ *E. bolteae* an opportunistic pathobiont, has similarly been shown to exert direct hepatic effects following translocation across a disrupted intestinal barrier, it has been reported to bind hepatocellular carcinoma cells and promote disease progression.³⁶

Although FMT is generally considered a safe procedure, adverse reactions or complications can occur in some patients, including gastrointestinal symptoms, sepsis, perforation, bacteremia, multi-organ failure and death. Patients should therefore be closely monitored before and after the procedure, with consideration on their health status and immune competence.³⁷

This study has several limitations. It represents a single case with limited longitudinal follow-up to date and the bacterial taxa identified may reflect individual microbiome variability rather than reproducible biomarkers of PBC. Moreover, our patient had received UDCA prior to FMT, limiting our ability to directly isolate the effects of FMT from UDCA therapy. Alternatively, this treatment sequence may reflect a role for FMT in sustaining remission established with UDCA. Further studies involving larger cohorts and longer follow-up are needed to evaluate the efficacy of FMT in PBC, as well as its potential role as an alternative or adjunct to UDCA.

5. Conclusion

PBC is a cholestatic liver disease with an obscure etiology. Growing evidence underscores the significance of gut-liver crosstalk in PBC progression, highlighting microbiome as a potential therapeutic target. As the first reported case of FMT in a PBC patient, this study demonstrates promising clinical and microbial shifts with FMT. With further research, FMT could become part of the therapeutic armamentarium for PBC, either as an alternative or adjunct to existing treatments, an approach that may reduce pill dependency and improve patients' overall quality of life.

Declarations

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor in Chief of this journal.

Ethics and Consent to Participate: This case report describes the clinical effects observed in the patient following a fecal microbiota transplant procedure. The procedure was performed as part of the patient's clinical care by Dr David Ong and was not experimental or undertaken for the purpose of this case report. The procedure and clinical management were determined by the treating clinicians based on the patient's clinical needs. The patient was informed of the nature and purpose of the publication with given consent by the patient and all reasonable measures were taken to protect the patient's privacy and confidentiality in accordance with applicable ethical principles. A principal investigator was not appointed for this clinical case study as it is based on the medically indicated treatment.

Data availability

All data generated or analysed during this study are included in this published article (and its supplementary information files).

Conflicts of Interest: Several authors are affiliated with AMILI Pte Ltd, which provided funding for this study. AMILI-affiliated authors contributed to the study in predefined and specifically delineated roles. These contributions did not overlap with the responsibilities of the other study investigators.

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Authors' Contributions:

Conceptualization, D.E.H.O. and J.W.H.P.; Methodology, D.E.H.O. and J.W.H.P.; Investigation, E.Y.T.S., J.X.T.L. and S.C.T.; Resources, J.F.Y.L., D.E.H.O. and J.W.H.P.; Visualization, E.Y.T.S., J.X.T.L. and S.C.T.; Writing – original draft, K.H.X.O., H.X.L., E.Y.T.S.; Writing – review and editing, K.H.X.O., H.X.L., E.Y.T.S., J.X.T.L. and S.C.T.; Funding acquisition, J.F.Y.L.; Supervision, J.W.J.L. and J.F.Y.L.

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Figures

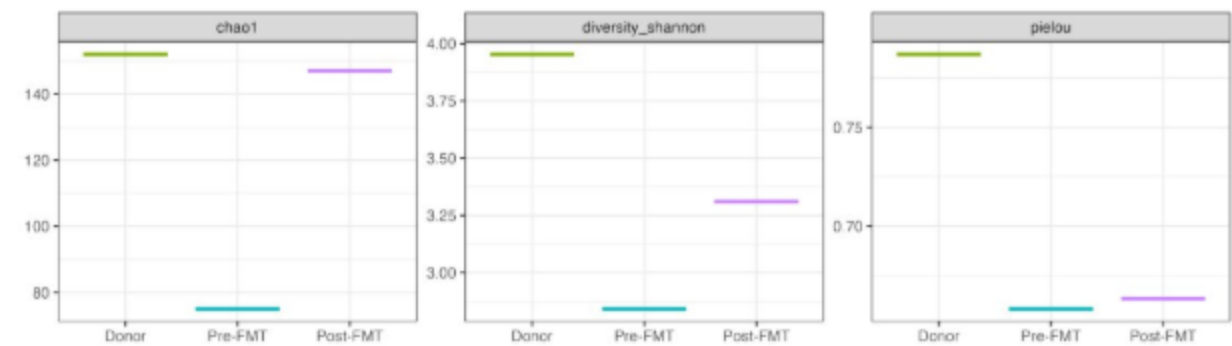


Figure 1

Alpha diversity of the patient’s gut microbiome pre- and post-FMT compared to the donor and healthy individuals, assessed using Chao 1 (richness), Shannon (diversity), and Pielou (evenness) indices.

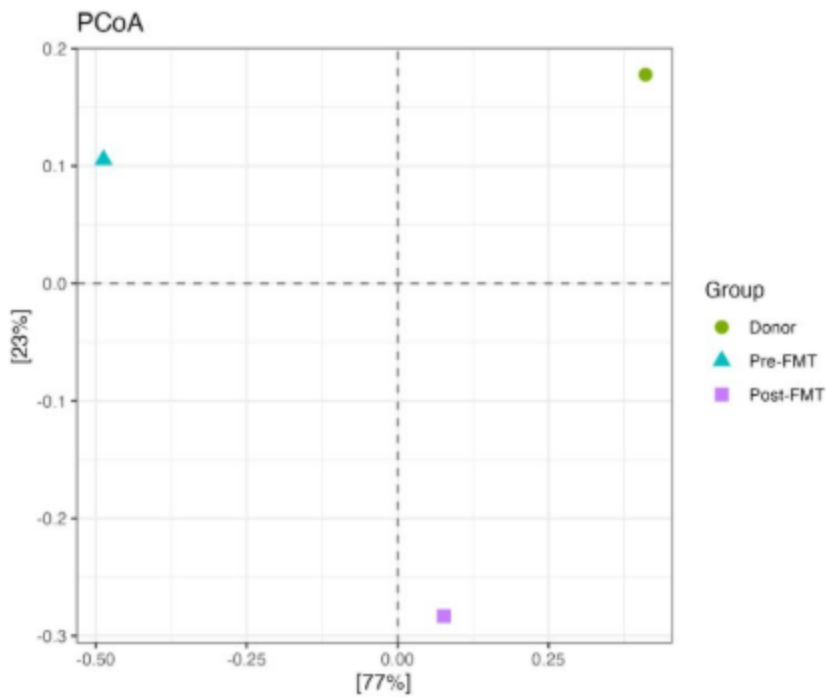


Figure 2

Shift in overall bacterial community composition following FMT. Principle coordinates analysis (PCoA) plot showing the microbiome profiles of the donor, and the patient pre- and post-FMT.

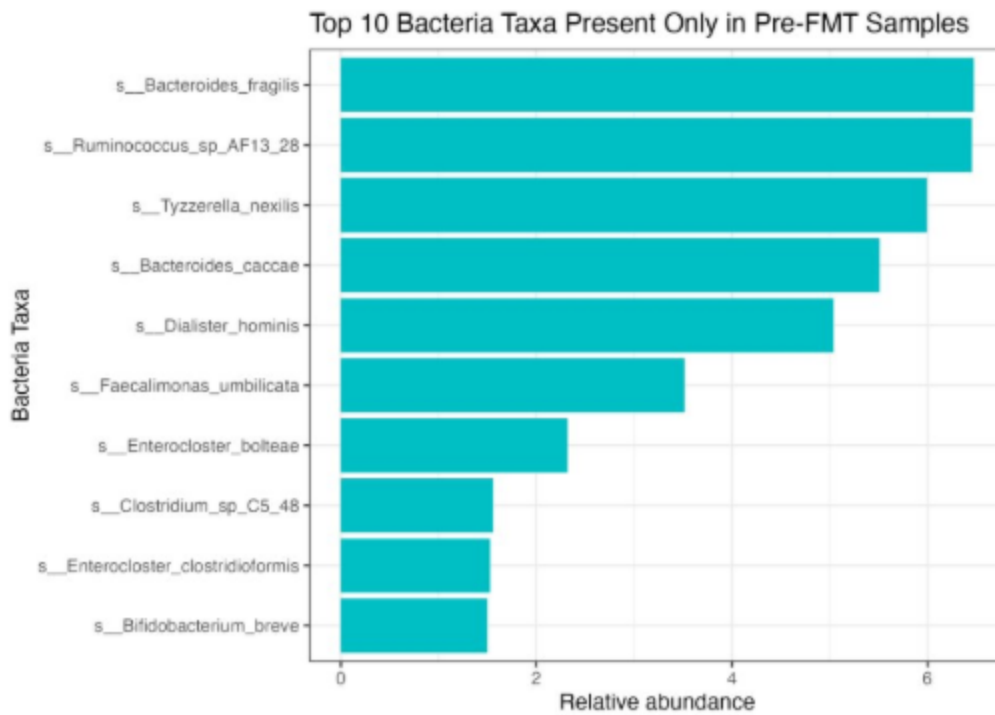


Figure 3

Top 10 bacteria taxa detected exclusively in pre-FMT samples, ranked by relative abundance.

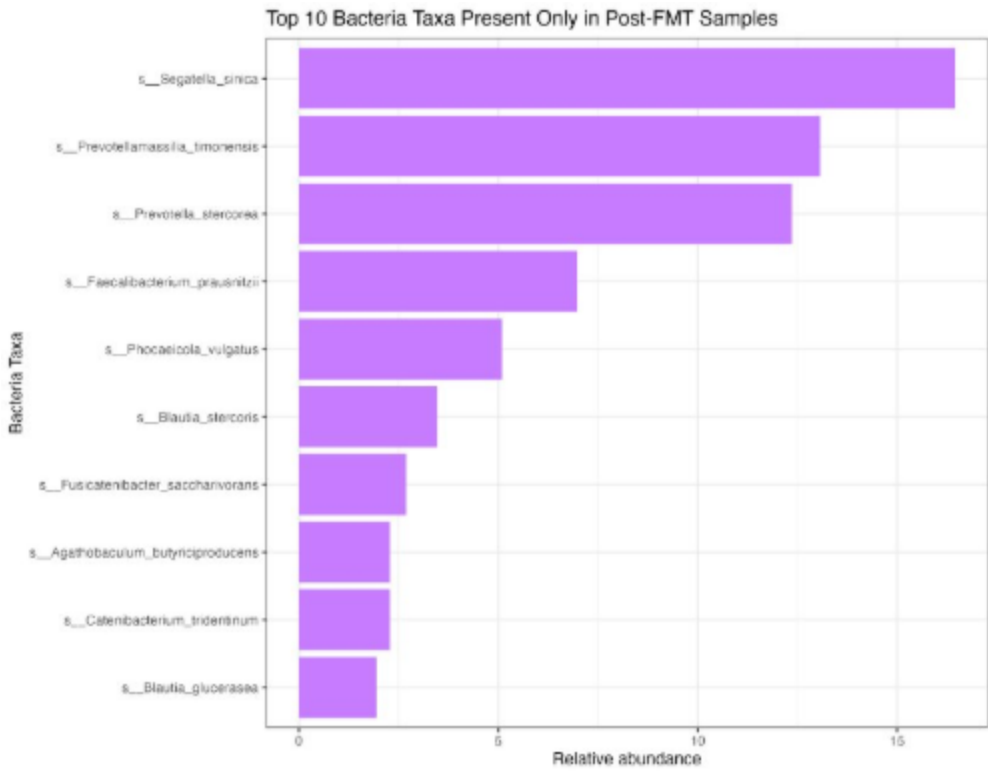


Figure 4

Top 10 bacteria species detected exclusively in post-FMT samples, ranked by relative abundance.

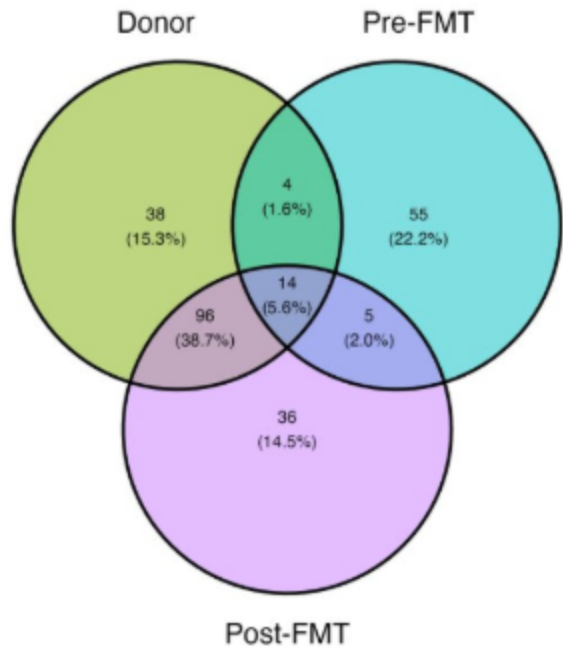


Figure 5

Overlap of bacterial species between the donor and patient's pre- and post-FMT microbiome.

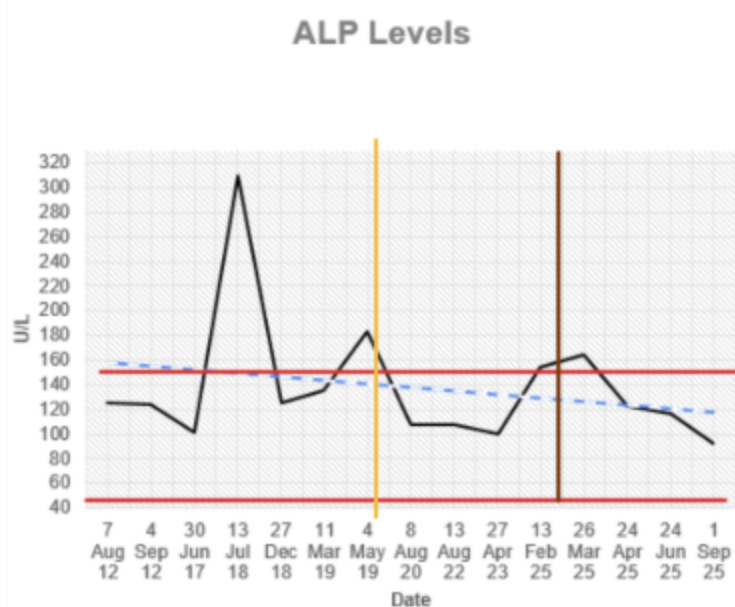


Figure 6

Trend of alkaline phosphatase (ALP) levels pre- and post-FMT. The yellow line indicates initiation of UDCA treatment, the brown line indicates timing of FMT. The red lines denote the upper and lower limits of the normal reference range.

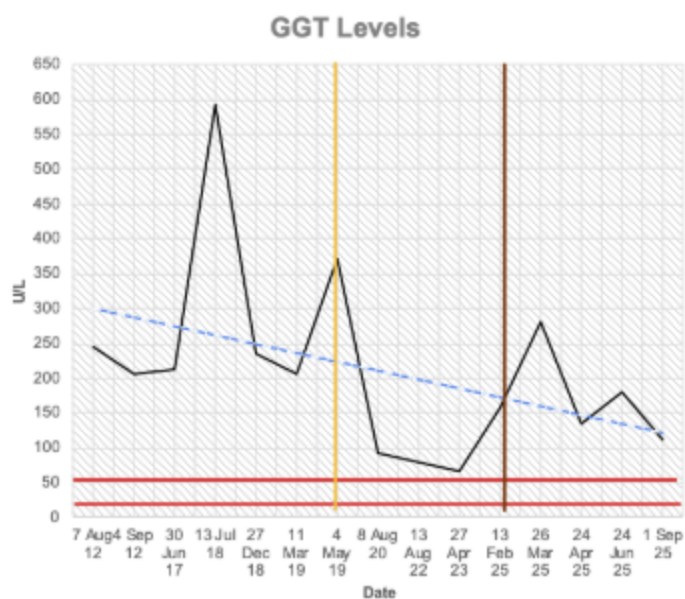


Figure 7

Trend of gamma-glutamyl transferase (GGT) levels pre- and post-FMT. The yellow line indicates initiation of UDCA treatment, the brown line indicates timing of FMT. The red lines denote the upper and lower limits of the normal reference range.