

ABSTRACT # 1

Atypical Presentation of Hodgkin's Lymphoma as Subacute Non-thrombotic Budd-Chiari Syndrome

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Background: Budd-Chiari Syndrome (BCS) is characterized by hepatic venous outflow obstruction, commonly occurring due to venous thrombosis predisposed by hypercoagulability, or rarely due to external venous compression or direct invasion by malignancy. **Clinical case:** A 37-year-old male presented with a five-week history of abdominal distension, jaundice, and lower limb edema; associated with night sweats and unintentional weight loss. Physical examination was significant for hepatomegaly and ascites. Investigations revealed deranged liver functions with AST 1,640 U/L, ALT 632 U/L, ALP 375 U/L, total bilirubin 74.9 $\mu\text{mol/l}$, INR 2.1, and albumin 21 g/L. Abdominal ultrasound showed ascites however it had suboptimal venous evaluation. CT chest-abdomen-pelvis with contrast demonstrated heterogenous central liver enhancement, intrahepatic IVC compression without thromboses, and collateralization by recanalized umbilical vein and lower esophageal varices. Furthermore, right axillary adenopathy and multiple splenic lesions were seen. Venous angiography was performed which showed intrahepatic IVC narrowing from external compression with brisk flow and no intraluminal pathology. Right axillary lymph node biopsy revealed Nodular lymphocyte-predominant Hodgkin lymphoma (NLPHL), and a bone marrow biopsy showed no infiltration. Transjugular liver biopsy was taken which showed prominent sinusoidal dilatation, however no lymphoproliferative involvement. Treatment was commenced with high-dose IV dexamethasone and Mini-CHOP chemotherapy. Unfortunately, patient developed tumor lysis syndrome after the second cycle leading to his demise. **Conclusions:** This case illustrates NLPHL presenting with non-thrombotic subacute BCS. Secondary BCS can occur secondary to intrahepatic IVC compression without thrombosis or vascular invasion. Unfortunately, prognosis is poor for lymphoma-associated BCS, as most reported cases were fatal.

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ABSTRACT # 3

Endoscopic Staged Management of Large Pancreatic Walled-Off Necrosis in a Pediatric Patient

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Introduction / Background

Walled-off pancreatic necrosis (WON) is a late complication of necrotizing pancreatitis, typically developing four or more weeks after disease onset. In pediatric patients, large WON is uncommon but associated with significant morbidity due to infection, mass effect, and prolonged systemic inflammation. While surgical necrosectomy was historically the standard of care, minimally invasive endoscopic ultrasound (EUS)-guided approaches have emerged as effective alternatives with lower morbidity.

Objectives

To describe the successful staged endoscopic management of a large pancreatic walled-off necrosis in a pediatric patient and highlight the role of serial endoscopic necrosectomy guided by clinical and laboratory response.

Clinical Case

A 12-year-old boy with obesity and fatty liver disease presented with severe acute pancreatitis complicated by ascites and pleural effusion, requiring pediatric intensive care admission. Following initial recovery, he developed persistent abdominal pain and early satiety. Magnetic resonance imaging revealed a large multiloculated pancreatic collection measuring approximately 13.7 × 13.6 × 15.5 cm, consistent with walled-off necrosis.

EUS-guided cystogastrostomy was performed using a 15-mm lumen-apposing metal stent (LAMS), draining purulent material that cultured methicillin-resistant *Staphylococcus aureus* and *Streptococcus mitis/oralis*. The patient underwent staged endoscopic necrosectomies due to recurrent fever and partial stent obstruction. Serial laboratory monitoring demonstrated a peak in inflammatory markers during febrile episodes, followed by gradual normalization after repeated necrosectomy. The LAMS was successfully removed six weeks later, with near-complete resolution of the necrotic cavity and no requirement for surgical intervention.

Conclusion(s)

Staged EUS-guided drainage and necrosectomy represents an effective and safe strategy for managing large pediatric pancreatic walled-off necrosis. Recurrent fever following initial drainage may reflect ongoing necrotic burden rather than treatment failure. Careful clinical, laboratory, and endoscopic reassessment allows successful non-surgical resolution in specialized centers.

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Echoes of a Smaller Stomach: Roux-en-Y Gastric Bypass Reversal for Refractory Complications — A Case Report

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ECHOES OF A SMALLER STOMACH ,ROUX-EN-Y GASTRIC BYPASS REVERSAL FOR REFRACTORY COMPLICATIONS: A CASE REPORT



ROUX-EN-Y GASTRIC BYPASS (RYGB) IS AN EFFECTIVE BARIATRIC PROCEDURE FOR WEIGHT LOSS AND METABOLIC BENEFIT. RARELY, PATIENTS DEVELOP SEVERE REFRACTORY COMPLICATIONS SUCH AS MALNUTRITION, PERSISTENT VOMITING, MARGINAL ULCERATION, DUMPING SYNDROME, OR HYPOLYCEMIA. WE REPORT A CASE OF SUCCESSFUL RYGB REVERSAL PERFORMED FOR PERSISTENT SYMPTOMS UNRESPONSIVE TO MEDICAL MANAGEMENT.

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INTRODUCTION

ROUX-EN-Y GASTRIC BYPASS (RYGB) IS A WIDELY PERFORMED METABOLIC/BARIATRIC PROCEDURE THAT PRODUCES DURABLE WEIGHT LOSS AND METABOLIC BENEFIT COMPARED WITH INTENSIVE MEDICAL THERAPY IN SELECTED PATIENTS, SUPPORTED BY LONG-TERM OUTCOME DATA. [1] HOWEVER, ITS COMBINED RESTRICTIVE-MALABSORPTIVE PHYSIOLOGY PREDISPOSES PATIENTS TO LATE MACRO- AND MICRONUTRIENT DEFICIENCIES, NECESSITATING LIFELONG SURVEILLANCE AND SUPPLEMENTATION. [2] DESPITE ESTABLISHED GUIDELINES, ADHERENCE IS OFTEN POOR AND LOSS TO FOLLOW-UP IS COMMON, COMPROMISING OUTCOMES. [8] NEUROLOGICAL COMPLICATIONS ARE VARIED, WITH PERIPHERAL NEUROPATHY FREQUENTLY LINKED TO THIAMINE OR VITAMIN B12 DEFICIENCY. [3] SEVERE PROTEIN-CALORIE MALNUTRITION CAN OCCUR YEARS LATER, PRESENTING WITH EDEMA AND SYSTEMIC MANIFESTATIONS. [4-6]

OBJECTIVE

TO REPORT SEVERE LATE NUTRITIONAL DEFICIENCY AFTER RYGB, EVALUATE DIURETIC-RESISTANT EDEMA WITH NEUROPATHY, STRESS LIFELONG FOLLOW-UP/SUPPLEMENTATION, AND DISCUSS ENDOSCOPIC REVERSAL. [2-3,6-8]

CLINICAL HISTORY AND EXAMINATION

A 44-YEAR-OLD FEMALE, POST-RYGB (2016), PRESENTED WITH 1-YEAR BILATERAL LOWER-LIMB SWELLING NON-RESPONSIVE TO DIURETICS, PARESTHESIA AND PROXIMAL LOWER-LIMB WEAKNESS, AND RECURRENT ORAL ULCERS. EXAMINATION REVEALED MARKED BILATERAL Pitting EDEMA UP TO DISTAL THIGHS, ORAL ULCERS, MILD HEPATOMEGALY WITHOUT ASCITES, AND LOWER LIMB SENSORY IMPAIRMENT. [2-6]

DIAGNOSTIC EVALUATION

GIVEN PRIOR RYGB, LATE MALABSORPTION AND NUTRITIONAL DEFICIENCY WERE SUSPECTED. WORK-UP INCLUDED NEUROLOGY EVALUATION TO EXCLUDE AUTOIMMUNE/DEMYELINATING NEUROPATHY, AND GASTROENTEROLOGY EVALUATION WITH ENDOSCOPIC ASSESSMENT. LABORATORY ASSESSMENT FOCUSED ON PROTEIN-CALORIE MALNUTRITION AND MICRONUTRIENT DEFICIENCIES, AS PER BARIATRIC NUTRITIONAL MONITORING RECOMMENDATIONS. [2-6]

Issues Due to Bariatric Surgery (Post-RYGB)

Issue	Bariatric Link
Protein-calorie malnutrition (albumin <3.0g/dL)	Hypocalcemia, protein-calorie malnutrition from malabsorption post-RYGB
Severe protein-calorie malnutrition	Anorexia, malabsorption physiology, inadequate intake and increased demands
Paresthesia / sensory neuropathy	Micronutrient deficiency (thiamine/B1, vitamin B12) after RYGB
Proximal lower limb weakness	Protein deficiency + micronutrient deficiency post-RYGB
Recurrent oral ulcers	Micronutrient deficiency (vitamin B12) due to malabsorption
Deficiency state anemia (Hb 8.0g/dL)	Iron/B12/folate malabsorption and reduced intake after RYGB
Mild hepatomegaly (normal transaminases)	Iron/B12/folate malabsorption and reduced intake after RYGB
Deficiency state anemia (Hb 8.0g/dL)	Iron/B12/folate malabsorption and reduced intake after RYGB
Mild hepatomegaly (normal transaminases)	Micronutrient deficiency (iron, folate, vitamin B12) after RYGB

TABLE 1: SUMMARIZES THE MAJOR CLINICAL PROBLEMS OBSERVED IN THE PATIENT FOLLOWING ROUX-EN-Y GASTRIC BYPASS (RYGB) AND OUTLINES THE MOST LIKELY BARIATRIC SURGERY-RELATED MECHANISMS LEADING TO PEM AND MICRONUTRIENT DEFICIENCIES, MANIFESTING AS EDEMA, NEUROPATHY, WEAKNESS, ORAL ULCERS, AND ANEMIA.



SHE UNDERWENT NUTRITIONAL REHABILITATION WITH HIGH-PROTEIN SUPPLEMENTATION AND TARGETED MICRONUTRIENT REPLACEMENT BASED ON DETECTED DEFICIENCIES. LIFELONG ADHERENCE TO BARIATRIC SUPPLEMENTS WAS REINFORCED. A MULTIDISCIPLINARY FOLLOW-UP PLAN WITH STRUCTURED NUTRITIONAL SURVEILLANCE WAS ADVISED TO PREVENT RECURRENCE. IN REFRACTORY SEVERE MALNUTRITION, ENDOSCOPIC REVERSAL USING LAMS WAS CONSIDERED TO IMPROVE NUTRITIONAL OUTCOMES.

Representative Improvement in Parameters Pre- and Post-LAMS

Parameter	Pre-LAMS	Post-LAMS	Interpretation
Serum Albumin (g/dL)	1.8	3.4	Marked improvement in protein status
Total Protein (g/dL)	4.2	6.6	Nutritional recovery
Hemoglobin (g/dL)	8.9	11.8	Anemia improved
Vitamin B12 (pg/mL)	160	520	Deficiency corrected
Thiamine /Vit B1 (nmol/L)	45	95	Neuropathy-related deficiency improved
Folate (ng/mL)	2.8	8.3	Deficiency corrected
Vitamin D (25-OH) (ng/mL)	9	28	Improved stores
Calcium (mg/dL)	7.8	9.0	Improved absorption
Magnesium (mg/dL)	1.3	1.9	Improved
Zinc (µg/dL)	45	78	Improved mucosal healing
Edema status	Severe	Resolved	Clinical response
Neuropathy symptoms	Severe	Improved	Functional recovery

TABLE 2: COMPARISON OF KEY NUTRITIONAL, HEMATOLOGICAL AND CLINICAL PARAMETERS BEFORE AND AFTER ENDOSCOPIC REVERSAL USING LAMS, DEMONSTRATING IMPROVEMENT IN PROTEIN STATUS, MICRONUTRIENT DEFICIENCIES, EDEMA, AND NEUROPATHY SYMPTOMS.

CONCLUSION

POST-RYGB PATIENTS CAN DEVELOP SEVERE LATE NUTRITIONAL COMPLICATIONS MANIFESTING AS DIURETIC-RESISTANT EDEMA, NEUROPATHY, WEAKNESS AND ORAL ULCERS. PROTEIN-CALORIE MALNUTRITION WITH MICRONUTRIENT DEFICIENCY SHOULD BE PROMPTLY SUSPECTED AND CORRECTED. LONG-TERM OUTCOMES DEPEND ON LIFELONG SUPPLEMENTATION AND STRUCTURED FOLLOW-UP. IN REFRACTORY MALNUTRITION, ENDOSCOPIC REVERSAL WITH LAMS MAY HELP. [2-8]

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FMT in Ulcerative Colitis: Targeting Gut Dysbiosis

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1. Introduction / Background

Ulcerative colitis (UC) is a chronic inflammatory bowel disease characterized by relapsing inflammation of the colonic mucosa, leading to significant morbidity and reduced quality of life.

Emerging evidence implicates gut microbiota dysbiosis as a central factor in the pathogenesis of UC. Fecal microbiota transplantation (FMT), a procedure involving the transfer of stool from a healthy donor to a recipient to restore microbial balance, has been approved by the U.S. Food and Drug Administration (FDA) for recurrent *Clostridioides difficile* infection. Its potential role in UC management has gained increasing attention due to promising clinical outcomes reported in recent studies.

2. Objectives

The primary objective of this study was to evaluate the efficacy and safety of fecal microbiota transplantation as a therapeutic intervention for patients with ulcerative colitis. Secondary objectives included assessing the impact of FMT on gut microbiota composition and determining clinical response and remission rates following treatment.

3. Methods

A comprehensive literature review was conducted using authentic electronic databases including PubMed, Google Scholar, ClinicalTrials.gov, FDA.gov, and ProQuest. Relevant clinical trials, cohort studies, case studies, systematic reviews, and meta-analyses focusing on FMT in ulcerative colitis were identified and analyzed. A total of 22 studies were included, comprising 9 randomized controlled trials, 10 cohort studies, and 3 case studies, involving 506 UC patients.

Data regarding study design, patient outcomes, FMT administration routes, efficacy, and adverse events were systematically extracted and qualitatively analyzed.

4. Results

Analysis of the included studies demonstrated a favorable clinical outcome following FMT in patients with ulcerative colitis. The average clinical response rate across studies was 65.5%, while clinical remission was achieved in 45.4% of patients. Improvements in gut microbiota diversity and composition were consistently reported post-FMT. Most adverse events were mild and transient, including gastrointestinal discomfort and bloating, with serious adverse effects being rare.

5. Discussion

The findings suggest that FMT is a promising therapeutic approach for UC, particularly in patients with refractory disease. Restoration of gut microbial diversity appears to play a key role in reducing intestinal inflammation and inducing remission. However, variability in study protocols, donor selection, dosing frequency, and routes of administration limits the ability to draw definitive conclusions. Standardization of FMT procedures and long-term follow-up studies are essential to better establish its clinical utility.

6. Conclusion

Fecal microbiota transplantation demonstrates encouraging efficacy and safety profiles in the treatment of ulcerative colitis, with substantial clinical response and remission rates. These findings support the potential role of FMT as an adjunctive or alternative therapeutic strategy in UC management. Further large-scale, well-designed clinical trials are warranted to optimize treatment protocols and confirm long-term benefits.

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ABSTRACT # 6

Anticancer Potential of *Lactobacillus rhamnosus*: A Systematic Review of In Vitro Evidence in Colorectal Cancer Cells

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Background: Experimental evidence suggests *Lactobacillus rhamnosus* / *Lactocaseibacillus rhamnosus* (LR) and their derivatives (postbiotics, cell-free supernatants, exopolysaccharides, outer membrane vesicles, and engineered products) exert anticancer effects against colorectal cancer (CRC) cell models.

Methods: We conducted a structured, narrative systematic review following the user-specified flow. Eligibility criteria, study selection, data extraction, and planned synthesis methods are reported. Because the available summaries did not provide sufficient extractable numerical data for many endpoints (e.g., means, SDs, sample sizes per experiment) a quantitative meta-analysis could not be completed and a narrative synthesis was performed.

Results: Across the 26 studies, LR and derivatives consistently reduced CRC cell proliferation, induced apoptosis, and produced cell-cycle arrest (commonly G0/G1 or G2). Mechanisms reported included: upregulation of pro-apoptotic mediators (Bax, caspases-3/9, p53) and downregulation of anti-apoptotic proteins (Bcl-2, Bcl-xl), suppression of Wnt/ β -catenin signaling, impairment of mitochondrial respiration, inhibition of invasion/migration (MMP-9 reduction, ANXA9 suppression), reactive oxygen species (ROS) accumulation, and immunomodulation (increased IFN- γ , TNF- α , IL-17A in some contexts; increased IL-10 in others). Several studies reported selective cytotoxicity (cancer cells affected, minimal harm to normal epithelial/fibroblast/HEK/HUVEC cells). A few in vivo models (AOM/DSS, xenografts) demonstrated reduced tumor incidence and improved histopathology when LR preparations were administered orally. Notable potent formulations included EPS, certain microparticle formulations (M2), Ag-LNPs with LR lysate, and isolated proteins (p8) showing reproducible antiproliferative and antimigratory effects.

Conclusions: Preclinical literature supports multi-modal anticancer effects of LR and LR-derived bioactive preparations on CRC models. Heterogeneity in interventions and incomplete numeric reporting limits pooled quantitative synthesis. Future work should provide standardized, fully reported experimental data and expand translational studies.

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ABSTRACT # 7

Inflammation of the liver is called Hepatitis. Various factors, including viral infections, alcohol, toxins, certain medications, or autoimmune diseases, can cause it. A virus causes viral hepatitis - the commonly known types are HVB and HVC, which can lead to acute or chronic diseases.

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Introduction:

Inflammation of the liver is called Hepatitis. Various factors, including viral infections, alcohol, toxins, certain medications, or autoimmune diseases, can cause it. A virus causes viral hepatitis - the commonly known types are HVB and HVC, which can lead to acute or chronic diseases.

Aims and Objectives:

The study aims to assess the knowledge and attitudes of RAKMHSU students towards hepatitis B and C infections.

Materials and methods

A cross-sectional survey was conducted among RAKMHSU students aged 18–24 through voluntary participation. A structured questionnaire measured knowledge and attitudes towards Hepatitis B and C. Data collection was anonymous to ensure confidentiality, and descriptive statistics were used to analyze awareness levels and perceptions among students from all four colleges.

Result

A total of 304 students of RAKMHSU participated in this study, notably from RAKCOMS (74.3%), with a majority of participants from Year 1 (32.9%) or Year 4 (25.3%).

Inter-college comparisons (Chi-square test) revealed significant differences in the knowledge of disease ($p=0.00256$) and perception of infected individuals ($p=0.00066$), indicating variation across colleges.

Senior year-level students were associated with better knowledge of disease ($p=0.000000258$), treatments ($p=0.0010$), preventive measures ($p=0.00007$) and attitude ($p=0.0228$).

Discussion

Knowledge and attitudes toward hepatitis B and C among medical and health science students are important for effective prevention and patient care. Differences in awareness may be influenced by variation in academic exposure and training across disciplines. Identifying gaps in understanding helps highlight the need for focused education to improve preventive practices and reduce stigma toward infected individuals.

Conclusion

Knowledge and attitudes about the disease vary more by year of study than by college. Tailoring health education to the academic level can enhance understanding and improve attitudes.

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Outcomes after intraoperative hypovolemic phlebotomy in patients undergoing liver surgery: a meta-analysis.

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Background: The risk of blood loss in liver surgery remains as a recent concern. Recent investigations have shown that intraoperative hypovolemic phlebotomy (HP) is more effective at reducing blood loss and transfusions during liver surgery. In this meta-analysis, we seek to assess the effects of HP on patients who have undergone liver surgery.

Methods: Electronic databases: PubMed, Embase, and Web of Science, were searched from inception to January 2025, with an update in November 2025 without any automated filters or language restrictions. Our eligibility criteria included randomized controlled trials (RCTs) or observational studies comparing HP to standard care in patients undergoing liver surgery. Random effects model was used for analysis pooling the mean differences (MD), and risk ratios (RRs) with corresponding 95% confidence intervals (CIs).

Results: A total of 13 studies, with 4,577 patients, were analyzed. The use of HP was associated with a lower perioperative RBC transfusion (RR: 0.55, 95% CI: [0.46 to 0.67], $p < 0.0001$, $I^2 = 18.50\%$). Subgroup analyses showed consistent results across different subgroups according to type of surgery: liver resection subgroup (RR: 0.57, 95% CI: [0.43 to 0.75], $I^2 = 27.87\%$) and liver transplantation subgroup (RR: 0.52, 95% CI: [0.42 to 0.63], $I^2 = 18.74\%$) ($P_{\text{interaction}} = 0.85$), and study design: RCTs subgroup (RR: 0.65, 95% CI: [0.44 to 0.96], $I^2 = 14.15\%$) and the observational cohort studies subgroup (RR: 0.52, 95% CI: [0.40 to 0.67], $I^2 = 34.34\%$) ($P_{\text{interaction}} = 0.34$). Similarly, the use of HP significantly reduced the incidence of intra-operative RBC transfusion (RR: 0.46, 95% CI: [0.31 to 0.68], $p < 0.0001$), and post-operative RBC transfusion (RR: 0.60, 95% CI: [0.44 to 0.82], $p < 0.0001$) compared to the control group. However, there was no statistical significance between the HP and control group in the plasma transfusion, platelet transfusion, or albumin transfusion, central venous pressure, hospital stay, and safety outcomes. GRADE assessment showed that perioperative RBC transfusions were at high certainty of evidence in the RCTs subgroup and at low certainty of evidence in the observational subgroup.

Conclusion: Our analysis showed higher benefits of applying intraoperative HP for patients undergoing liver surgery through the reduction of the incidence of blood transfusion and blood loss within an acceptable safety profile.

Keywords: hypovolemic phlebotomy, HP, liver surgeries, liver resection, liver transplantation blood loss, blood transfusions.

ABSTRACT # 10

Efficacy of using oral zinc supplementation beside the phototherapy for infants with Hyperbilirubinemia: a meta-analysis of randomized controlled trials.

Authors:

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Running title.

Zinc sulfate in neonatal hyperbilirubinemia.

Abstract

Introduction: Previous investigations on the impact of oral zinc supplementation treatment on newborns' serum bilirubin levels have produced conflicting results. The goal of this meta-analysis is to clarify the efficacy of zinc supplementation in infants with hyperbilirubinemia through combining the results from published randomized controlled trials (RCTs).

Methodology: In this systematic review, PubMed, Embase, Cochrane CENTRAL, Scopus, and Web of Science were searched in August 2024 for the RCTs that investigate the use of zinc supplementation in infants with hyperbilirubinemia. Quality assessment was done by two authors with adherence to ROB-2 domains. Mean difference (MD) with 95% confidence interval (95% CI) was used to estimate the overall effect of the outcomes. Der-Simonian Laird's random effects model was used to pool the results.

Results: A total of 1,345 infants, who were pooled from the 13 RCTs, were included. Zinc sulfate significantly reduced bilirubin levels at 24 hours (Mean difference (MD): -1.05, 95% Confidence Interval (CI) [-1.72; -0.38], $p=0.01$, $I^2=91.35\%$, $p<0.001$), 48 hours (MD: -1.23, 95% CI [-2.14; -0.31], $p=0.01$, $I^2=89.07\%$, $p<0.001$). In the subgroup analysis, full-term and normal birth weight infants, using lower doses (1 mg/d – 5 mg/d), were the group that benefited from the intervention. On the other hand, no significant difference in preterm and low-birth-weight infants was found. There were no significant differences in TSB at 72 hours, 4 days, phototherapy duration, and hospital stay. GRADE assessment showed that most of the outcomes ranged from low and very low certainty of evidence.

Conclusion: This meta-analysis underscores the effect of zinc sulfate in the reduction of total serum bilirubin levels in infants, particularly within the first 24 to 48 hours of treatment, and who are full-term and normal birth weight infants.

ABSTRACT # 11

Knowledge, attitudes, and practices of the general UAE population toward weight management medications

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Introduction:

Obesity is a major driver of metabolic associated steatotic liver disease and obesity related gastrointestinal disorders, placing weight management at the center of modern gastroenterology and bariatric practice. GLP 1 based weight management medications (WMMs) are effective but frequently cause gastrointestinal symptoms and, more rarely, pancreatitis and gallbladder disease, making patient understanding crucial for safe use within GI pathways. This study evaluates knowledge, attitudes, practices, and usage patterns of WMMs among adults in the UAE, to identify gaps that directly impact GI, hepatic, and pancreatobiliary care.

Methodology:

A cross-sectional online survey targeting UAE's adults ran for four weeks (February–March 2024). The 65-question survey included 22 items on knowledge and 8 on attitudes toward WMMs. Descriptive statistics and chi-square tests were performed using SPSS. Mean knowledge (10) and attitude (14) scores were set as cut-off points; participants scoring above these were considered to have adequate knowledge and positive attitudes. Doctors, endocrinologists, pharmacists, and medical students were excluded.

Results:

Among 394 participants, 29.8% were overweight, and 26.4% were obese. Although 70.9% demonstrated awareness and assumed knowledge, only 57.1% had adequate knowledge. Females had 2.17 times higher knowledge than males (95% CI: 1.43-3.29, $p < 0.001$). Knowledge was 2.79 times higher in participants with thyroid dysfunction (95% CI: 1.10-7.09, $p = 0.025$). WMM users for weight-loss were 3.91 times more knowledgeable (95% CI: 1.11-13.7, $p = 0.028$) compared to diabetes users. BMI significantly influenced attitudes ($p = 0.042$). Doctor-provided information improved knowledge by 2.654 times vs. social media (95% CI: 1.033-6.819, $p = 0.037$) and attitudes by 2.601 times vs. family/friends (95% CI: 1.024-6.605, $p = 0.041$).

Conclusion:

In a population with substantial overweight and obesity, public understanding of WMMs is incomplete and unevenly distributed, despite growing reliance on these drugs in GI and bariatric pathways. Females, individuals with thyroid dysfunction, and those using WMMs for weight loss show higher knowledge, while BMI strongly shapes receptivity to pharmacologic options. Gastroenterologists and bariatric specialists must prioritize clinician-led education to guide safe WMM use alongside endoscopic and surgical options, ultimately improving management of obesity-driven GI and liver disease.

ABSTRACT # 13

Metabolic and Stress Pathway Co-activation Identifies Dual-Blockade Targets in Cancer

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Background & Rationale

Cancer cells live in a stressful environment. Even without treatment, they face low oxygen, lack of nutrients, and protein-folding problems because of their rapid growth. When therapy such as chemotherapy or radiation is added, the stress becomes even stronger.

To survive, cancer cells switch on four major protective pathways:

- ER stress response (UPR)
- Autophagy
- Nucleotide synthesis
- Glycolysis feeder

When these pathways are activated together, tumors become highly adaptable. Though, this dependence may create weakness if only blocking two pathways at the same time thus making it much harder for cancer cells to survive.

Aim

To find cancers that switch on ER stress, autophagy, nucleotide synthesis, and glycolysis feeder at the same time, and to propose dual-drug strategies that block two of these survival routes together.

Methods

1. Differential Expression Analysis

- Platform: UALCAN (<http://ualcan.path.uab.edu>)
- Cancers: All TCGA cancer types available in UALCAN
- Gene sets to test:
 - o ER stress/UPR: HSPA5, HSP90B1, CALR, PDIA4, ERO1A, EIF2AK3(PERK), ATF4, DDIT3, XBP1, ATF6
 - o Autophagy: ULK1, BECN1, ATG5, ATG7, ATG12, MAP1LC3B, WIPI1, SQSTM1, LAMP1, CTSD
 - o Nucleotide synthesis: CAD, DHODH, UMPS, PPAT, PAICS, IMPDH1/2, GMPS, RRM1, RRM2, TYMS, TK1, DCK, HPRT1
 - o Glycolysis feeder: SLC2A1, HK2, PFKFB3, PFKFB4, LDHA, PKM, HK1

2. Co-Activation Identification

- Generate heatmaps of $\log_2$ fold changes across cancers.
- Define “high co-activation” cancers as those with concurrent upregulation of ER stress, autophagy, glycolysis feeder, and nucleotide genes.

3. Drug–Gene Interaction Mapping

- Tool: DGIdb (<https://dgidb.org>)
- Filter for FDA-approved or clinical-stage drugs with interaction score ≥ 2 targeting the upregulated genes.

4. Gene–Disease Associations

- Tool: Open Targets Platform (<https://platform.opentargets.org>)
- Extract Association Score and Europe PMC score for prioritized genes in each cancer type.

5. Pathway Enrichment

- Tool: Enrichr (<https://maayanlab.cloud/Enrichr/>)
- Libraries: KEGG 2021 Human and GO Biological Process 2025.
- Identify enriched terms with adjusted $p < 0.05$.

6. Protein–Protein Interaction Networks

- Tool: STRING (<https://string-db.org>), confidence > 0.4 .
- Visualization: Cytoscape (two modules: ER/Autophagy $\leftrightarrow$ Nucleotide).
- Annotate hubs that are druggable.

Results:

Through specific filtration method from the 36 Cancer types only 6 Cancer types were chosen: Breast invasive carcinoma, Head and neck squamous cell carcinoma, colon Adenocarcinoma, stomach adenocarcinoma, Cholangiocarcinoma, lung squamous cell carcinoma. All mentioned genes got significant difference between normal and cancer cells. By identifying cancers that activate these systems at the same time, we can point to dual-drug strategies that target two defence pathways at once. The findings could guide drug repurposing with FDA-approved compounds and open new directions for overcoming therapy resistance in cancer.

Intracholecystic tubulopapillary neoplasm (ICPN)—Premalignant or Malignant?

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1 Introduction—Intracholecystic tubulopapillary neoplasm (ICPN) is a pre-invasive neoplastic lesion characterized by papillary growth in the gallbladder. ICPN can also be considered a counterpart of intraductal papillary mucinous neoplasm (IPMN) in pancreatic duct epithelium [1]. ICPN has four subtypes: gastric, biliary, intestinal, and oncocytic. Biliary and intestinal subtypes are more associated with invasion as compared to other subtypes [2]. Most of the early cases were diagnosed after routine laparoscopic cholecystectomies, and the disease entity was considered to be benign. In the 2019 WHO classification, it was proposed as a preinvasive neoplasm of the gallbladder. A case report by Suda et al. has shown the aggressive nature of these preinvasive neoplasms, especially those associated with an invasive component, to the extent of the presence of liver metastasis in a patient [3]. In light of recent events, a simple laparoscopic cholecystectomy could not justify the appropriate treatment option in this entity if it is suspected preoperatively [3,4]. Here, we present 2 cases diagnosed with ICPN, one of which rapidly progressed to invasive carcinoma.

2 Objectives—To diagnose and determine adequate treatment modality for this pathology

3 Clinical case—A 39-year-old lady presented with severe pain in the right upper abdomen 4 months back associated with vomiting. She was diagnosed with cholangitis and choledocholithiasis with mild pancreatitis. Computed Tomography (CT) of the abdomen revealed choledocholithiasis with a thickened and edematous gallbladder that is overdistended. Multiple hyperdensities (likely blood clots) were noted filling the gallbladder lumen, consistent with calculi and sludge. Endoscopic retrograde cholangiogram and stenting were done 4 months back. Laparoscopic cholecystectomy was planned, but the patient refused at that time. The patient had a repeat attack of cholangitis 3 months later with deranged liver function tests. Magnetic Resonance Imaging (MRI) The abdomen showed Irregular intraluminal soft tissue is seen filling most of the gallbladder with focal loss of fat plane between the gallbladder wall and segment 5 of the liver anteriorly. Staging laparoscopy showed a gallbladder tumor with a liver deposit in the adjoining segment 4b. The patient underwent open radical cholecystectomy (gallbladder with 2.5 cm liver wedges in segments 4b and 5) with hepatoduodenal, periportal, and retropancreatic lymphadenectomy. Histopathology revealed Moderately differentiated adenocarcinoma of the gallbladder arising in the background of intracholecystic papillary neoplasm with high-grade dysplasia involving the neck, body, and fundus of the gallbladder with direct extension of the tumor into the adjoining liver (pT3). Cystic margin—free of tumor, retropancreatic, hepatic artery, and hepatoduodenal lymph node free of tumor. Lympho-vascular and peri-neural invasion present. Liver resection margins were free.

A young male presented with pain in the right hypochondrium and epigastrium. An abdominal ultrasound showed a 2.3 x 1.6 cm mass lesion in the gallbladder. Computed tomography (CT) of the abdomen showed an irregular polypoidal intraluminal soft tissue density of 21 x 17 mm in the fundus of the gallbladder. Endoscopic ultrasound (EUS) evaluation was suggestive of a gallbladder mass. On suspicion of carcinoma of the gallbladder, the patient underwent radical cholecystectomy with a 2 cm liver wedge with standard hepatoduodenal ligament, periportal, and retropancreatic lymphadenectomy with uneventful recovery.

Postoperative histopathology revealed an ICPN with predominant intestinal morphology and low-grade dysplasia. All 16 lymph nodes were negative for malignancy. Dysplasia was present in the neck region but not in the cystic duct margin.

4 Conclusion— ICPN being a preinvasive gallbladder neoplasm, is less invasive and has a better prognosis than gallbladder carcinoma. The presence of an invasive component at the time of preoperative detection cannot be determined. Invasive carcinomas may be associated with lymph nodal metastasis. Standardized guidelines are required for preoperative evaluation, investigations, and surgical planning in patients diagnosed with ICPN.

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Patterns of Presentation and Management of Anorectal Disorders: A Retrospective Review from a UAE Tertiary Center

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Abstract

Background: Anorectal disorders, including hemorrhoids, anal fissures, and anal fistulas, are common conditions that can significantly impact quality of life. Data on their prevalence, clinical presentation, and management in the UAE are limited. This study aimed to evaluate the distribution, symptoms, and treatment patterns of anorectal disorders in a tertiary care setting in the UAE.

Methods: A retrospective review of medical records was conducted for patients diagnosed with anorectal disorders at Thumbay Group of Hospitals, UAE, between January 2024 and January 2025. Sociodemographic data, clinical presentation, and management modalities were extracted using a structured proforma. Descriptive statistics were performed using SPSS v29.

Results: A total of 176 patients were included, with a predominance of males (86.9%) and individuals aged ≥ 35 years (55.1%). Most patients were of Asian nationality (77.8%), and 69.4% were overweight or obese. Hemorrhoids were the most common disorder, with grade III (40.8%) and grade IV (14.8%) predominating, followed by anal fissures (27.3%) and anal fistulas (6.3%). The most frequent presenting complaint was a combination of pain and bleeding (53.4%), followed by bleeding alone (35.2%) and pain alone (11.4%). Surgical intervention was more common than conservative management, with hemorrhoidectomy performed in 42.6% of cases, conservative treatment in 39.2%, fissurectomy in 14.2%, and fistulectomy in 4%.

Conclusion: Hemorrhoids and anal fissures are the most prevalent anorectal disorders in this UAE tertiary care population, frequently presenting with pain and bleeding. Surgical intervention predominates, likely due to delayed presentation and advanced disease. Early detection, conservative management, and targeted public health strategies could reduce progression to severe disease and minimize the need for surgery. Training primary care providers to recognize anorectal disorders promptly may further improve patient outcomes.

Keywords: Anorectal disorders, Hemorrhoids, Anal fissure, Anal fistula, Surgical management, Conservative management

An Unusual GI manifestation of unicentric Castleman disease.

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Introduction

Castleman disease (CD) is a rare, benign lymphoproliferative disorder mostly manifesting as lymphadenopathy with or without the presence of systemic symptoms.¹ Its prevalence is 16-19 cases per million, with the majority of cases being unicentric in nature, i.e. they involve one lymph node, as opposed to multicentric CD (MCD) which involves more than one lymph node and is more commonly associated with systemic signs and symptoms.² Unicentric CD (UCD) is most commonly detected asymptotically in individuals in their 30s-40s, with a slight female preponderance. According to Talat et al, the mean size of affected lymph nodes was 5.5cm, while the most commonly affected locations were the chest (29%), followed by the neck (23%), abdomen (21%) and retroperitoneum (17%).³ Most commonly patients manifest with symptoms due to local compression such as abdominal pain, nausea and vomiting, early satiety and post-prandial pressure.^{3,4} The most common histologic subtype is the hyaline vascular subtype, comprising approximately 90% of cases while the plasmacytic and mixed subtypes are less common and are more frequently associated with systemic signs.⁵ Complete surgical resection of the mass is the treatment of choice in UCD and is associated with an excellent prognosis and a 10-year survival rate of 95%.^{3,5}

Here we present the case of a 24-year-old woman with mixed histology UCD presenting with chronic abdominal pain.

Objectives

- Highlight the signs and symptoms, diagnosis and management of unicentric Castleman disease.
- Outline the main differential diagnoses when considering unicentric Castleman disease.
- Compare and contrast unicentric Castleman versus multicentric Castleman disease.

Clinical Case

A 24-year-old female presented with a 3-month history of epigastric abdominal pain radiating to the back with no associated night sweats or weight loss. Upper gastrointestinal endoscopy showed external compression over the second part of the duodenum. Contrast-enhanced computed tomography scan of the abdomen revealed an 8.1x 5.2 x 4.5 cm oval-shaped enhancing mass in the lesser sac, compressing the stomach and abutting the pancreatic body and left adrenal gland, raising suspicion of either a gastrointestinal stromal tumor, a pancreatic neuroendocrine tumor, an adrenal tumor or a paraganglioma. Blood tests showed an elevated C-reactive protein at 169mg/L (normal 0-5mg/L), a hemoglobin of 9.2g/dL (normal 12-15g/dL) and an albumin of 22g/L (normal 39-49g/L). Urine and plasma metanephrines as well as tumor markers CEA, CA-125 and CA-19-9 were normal; however, urinary 5-HIAA, urinary dopamine and serum chromogranin A (188.1mg/ml, normal up to 101.8mg/ml) were elevated; the latter was attributed to a concurrent proton pump inhibitor therapy.

Endoscopic ultrasound demonstrated a 9.5 x 5.4cm well-circumscribed, hyperechoic homogenous mass arising from the body and tail of the pancreas measuring, an FNA sample showed sheets of reactive lymphoid and

plasma cells/lympho-plasmacytic infiltrate with attenuated follicles, suspicious for Castleman disease, and more specifically the mixed histology subtype.

The patient underwent an uneventful laparoscopic resection of a 9cm retroperitoneal mass that was abutting, but not infiltrating the pancreas, left adrenal and stomach. The pathologic assessment confirmed mixed subtype UCD. The patient's post-operative course was not complicated, and the albumin as well as CRP gradually improved. Subsequent serum protein electrophoresis revealed a polyclonal gammopathy. At 6 months follow-up the patient remains well and symptom-free with no evidence of recurrence on imaging.

Conclusions

UCD is a rare entity that is both underreported and underrecognized in the MENA region. Most patients are asymptomatic, but when symptoms do arise they are most commonly due to local organ compression. While systemic manifestations and lab abnormalities such as hypoalbuminemia, elevated inflammatory markers and gammopathy are more commonly associated with MCD, they can still be seen in UCD, particularly the plasmacytic and mixed subtypes.

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Endoscopic Submucosal Dissection with Complete Endoscopic Hand Suturing for Solitary Rectal Ulcer Syndrome: First Reported Case Worldwide

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Background: Solitary rectal ulcer syndrome (SRUS) is a rare, non-malignant mucosal disorder typically affecting young individuals. While conservative medical therapies resolve ulcers in approximately 57% of patients, many cases remain refractory, demanding an innovative, minimally invasive alternatives to avoid the morbidity of traditional treatment.

Objectives: To evaluate the safety and efficacy of combining Endoscopic Submucosal Dissection (ESD) with Complete Endoscopic Hand Suturing (EHS) as a novel, organ-preserving treatment for large, refractory SRUS lesions.

Clinical Case: A 38-year-old male with a 5-year history of rectal bleeding and chronic constipation presented with a large (6x4 cm) ulcerated lesion near the dentate line. Following failed medical management, the patient underwent ESD using the HYBRIDknife® flex (ERBE) for precision dissection of dense submucosal fibrosis. The resulting large mucosal defect was completely closed using the SutuArt endoscopic hand-suturing system (Olympus) to promote primary intention healing.

The ESD procedure allowed for the precise separation of the lesion from the muscularis propria while sparing muscle fibers. EHS provided excellent tissue approximation and complete closure of the rectal defect. The patient was discharged the following morning without complications such as pain, bleeding, or incontinence. A 4-week follow-up endoscopy confirmed complete mucosal healing and healthy granulation tissue, with a total resolution of the preoperative symptoms.

Conclusion: This represents the first reported case worldwide combining ESD and EHS for SRUS. This dual-technique approach offers a promising, sphincter-preserving solution for complex rectal lesions, effectively addressing the challenges of submucosal fibrosis and large mucosal defects that often limit standard endoscopic interventions.

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Endoscopic versus Surgical Myotomy for Achalasia: A Systematic Review and Meta-analysis of POEM and Laparoscopic Heller Myotomy

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ABSTRACT

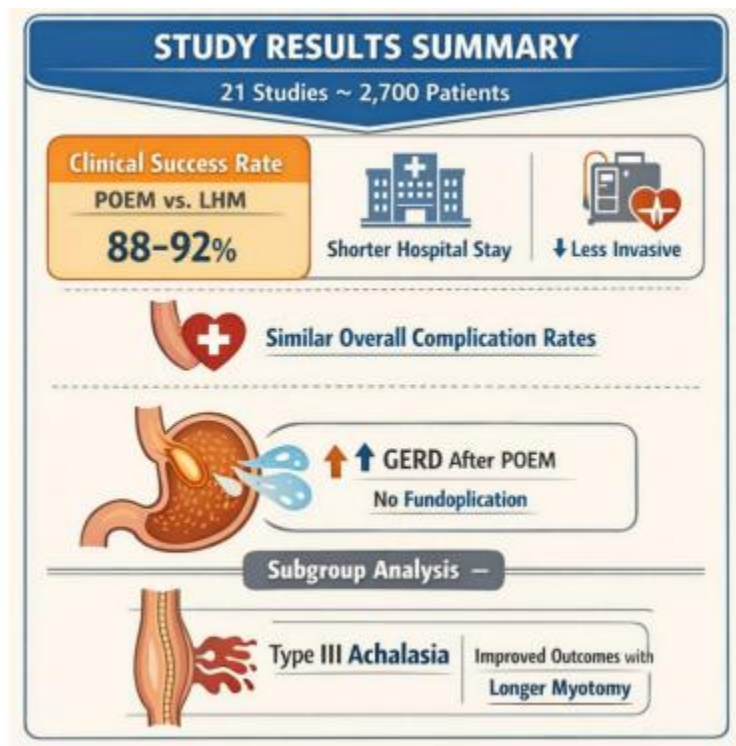
Introduction: Achalasia is a chronic esophageal motility disorder causing progressive dysphagia, regurgitation, and significant impairment in quality of life. The disease is characterized by impaired relaxation of the lower esophageal sphincter and absent coordinated peristalsis. For decades, Laparoscopic Heller Myotomy (LHM) with partial fundoplication has been considered the definitive surgical treatment. The emergence of Peroral Endoscopic Myotomy

(POEM) has transformed minimally invasive management by enabling tailored myotomy through an endoscopic submucosal tunnel without external incisions. Although POEM has gained rapid international adoption and demonstrated promising clinical outcomes, uncertainty remains regarding its comparative efficacy, safety profile, and risk of postoperative gastroesophageal reflux relative to LHM.

Objectives: To systematically compare clinical success, perioperative outcomes, and postoperative complications between POEM and LHM in adult patients with achalasia.

Methods: A systematic review and meta-analysis were conducted according to PRISMA guidelines. Databases (PubMed, Embase, Scopus, Cochrane Library) were searched for studies published between 2006–2026. Eligible studies included randomized controlled trials and observational cohort studies reporting clinical success (Eckardt score ≤ 3), operative time, hospital length of stay, adverse events, and postoperative reflux. Two independent reviewers performed study selection, data extraction, and quality assessment. Pooled outcomes were calculated using a random effects meta-analysis model.

Results: Twenty-one studies including approximately 2,700 patients met inclusion criteria. Clinical success was high and comparable between POEM and LHM (88–92%). POEM demonstrated shorter hospital stay and reduced invasiveness, while overall complication rates were similar. Postoperative gastroesophageal reflux occurred more frequently following POEM, likely due to the absence of fundoplication. Subgroup analyses suggested that POEM may provide improved outcomes in type III achalasia due to longer myotomy length.



Discussion: Both procedures provide durable symptomatic relief. POEM offers procedural flexibility, faster recovery, and benefits in complex or spastic achalasia phenotypes. LHM with fundoplication demonstrates lower rates of postoperative reflux, an important long-term consideration. These findings support POEM as an effective minimally invasive alternative to LHM, while highlighting the need for reflux surveillance.

Conclusion: POEM and LHM demonstrate comparable efficacy and safety. POEM offers minimally invasive advantages for complex phenotypes, whereas LHM provides superior reflux control. Further long-term randomized trials are required to refine patient-specific treatment strategies.

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The Gut Feeling That Was Right: Acute Stanford Type A Aortic Dissection Presenting as Acalculous Cholecystitis

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Background: Aortic dissection is a time-critical vascular emergency that classically presents with sudden tearing chest or back pain. However, atypical presentations are not uncommon and may lead to misdiagnosis and delayed treatment. Atypical presentations reported in previous literature include syncope, focal neurological deficits, limb weakness, and abdominal discomfort. We report a case of aortic dissection presenting as acalculous cholecystitis.

Case Presentation: A 53-year-old male, presented with acute upper abdominal discomfort associated with mild dyspnea. Initial investigations and abdominal ultrasound were suggestive of acalculous cholecystitis and mild acute kidney injury, with otherwise unremarkable ECG and first Troponin. Serial troponin elevation prompted further assessment with transthoracic echocardiography revealing an intimal flap in the ascending aorta with moderate pericardial effusion. CT angiography confirmed a Stanford type A aortic dissection extending from the ascending aorta to the level of the bilateral renal arteries, involving the origins of the celiac and superior mesenteric arteries. The patient was transferred emergently for surgical repair.

Conclusion: Atypical presentations of acute aortic dissection remain an important cause of diagnostic delay. This case demonstrates one of the rare, atypical presentations and highlights the importance of maintaining a high index of suspicion for aortic dissection in patients with acalculous cholecystitis associated with serial troponin elevations.

Refractory Hyperbilirubinemia Post-Endoscopy: Navigating the Overlap of Intrinsic Duct Stones and Extrinsic Compression

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1. Introduction / Background

Mirizzi Syndrome (MS) is an unusual and rare manifestation of gallstone disease (0.3% to 3% incidence) where a calculus impacted in the cystic duct or gallbladder neck causes extrinsic compression of the common hepatic duct. It is famously described as a "trap in the surgery of gallstones" because the intense inflammation and distorted anatomy in Calot's triangle significantly increase the risk of iatrogenic bile duct injury (BDI) during standard cholecystectomy.

2. Objectives

The objective of this report is to describe a complex "double obstruction" (intraductal choledocholithiasis and external MS compression) that resulted in misleading clinical signals and fluctuating bilirubin levels post-intervention, necessitating a staged multidisciplinary management strategy.

3. Clinical Case

Diagnostic Evaluation: A 41-year-old male presented with a 4-day history of symptomatic obstructive jaundice, including pruritus, dark urine, and pale stools. Initial labs showed a cholestatic pattern (Total Bilirubin 7.696 mg/dL) with elevated inflammatory markers. An initial ERCP identified and extracted a common bile duct (CBD) stone and placed a biliary stent. While the patient initially improved, symptoms recurred five days later with a rebound in bilirubin (7.238 mg/dL). Subsequent MRCP and a second ERCP revealed the "double obstruction": the CBD was clear, but a large stone was impacted in Hartmann's pouch, causing severe extrinsic compression and stricture of the common hepatic duct (Mirizzi Syndrome).

Treatment: A multidisciplinary team (Gastroenterology and General Surgery) moved from an immediate combined procedure to a staged approach. The plan included an elective cholecystectomy utilizing a "fundus-first" approach—a critical principle to avoid Calot's triangle and prevent BDI—followed by a delayed ERCP for stent retrieval six weeks post-surgery.

Follow-up: At the last assessment, the patient's condition had stabilized with a resolution of pain and jaundice. He was counselled on lifestyle modifications, including smoking cessation and weight management, and scheduled for his definitive procedure.

4. Conclusion(s)

This case demonstrates that fluctuating biochemical markers after an apparently successful ERCP should raise a high index of suspicion for Mirizzi Syndrome. Accurate preoperative mapping via MRCP is paramount to identifying extrinsic compression, as it mandates a shift in surgical technique to a fundus-first approach to ensure biliary ductal integrity. Furthermore, clinicians must remain vigilant for concomitant gallbladder carcinoma, which has a high coincidence (up to 27%) with MS, necessitating the availability of intraoperative frozen sections.

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Cystic lymphangioma of the gallbladder after gastric bypass surgery

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1. Introduction / Background Cystic lymphangioma of the gallbladder is an exceedingly rare, benign malformation of the lymphatic system[1-3], Lymphangioma may involve any organ in the body except the brain [1]. Skin and the soft tissue of the head and neck are the most common sites (95%) [2]. Gastrointestinal tract lymphangiomas are relatively uncommon, lymphangiomas arising from the gallbladder are extremely rare, representing only 0.8-1% of all intra-abdominal lymphangiomas [3]. Though often asymptomatic, they may present with non-specific abdominal symptoms or be discovered incidentally [4]. While the primary etiology is considered congenital, secondary factors such as previous surgery or inflammation may contribute to their development in adults[1,4].

2. Objectives The objective of this report is to document a rare clinical occurrence of gallbladder lymphangioma in an adult woman with a history of gastric bypass surgery and to highlight the efficacy of laparoscopic cholecystectomy as a definitive treatment.

3. Clinical Case

Patient Presentation: A 34-year-old female presented with a three-month history of colicky right hypochondrial pain radiating to the right shoulder, aggravated by fatty meals. Her medical history included morbid obesity, diabetes, and hypertension, all of which resolved following a gastric bypass surgery in 2008.

Diagnostic Evaluation: Physical examination revealed tenderness in the right hypochondrium. Laboratory investigations were within normal limits. Abdominal ultrasound identified multiple calculi and a partially septated gallbladder with normal wall thickness.

Treatment: The patient underwent an uncomplicated laparoscopic cholecystectomy. Intraoperatively, a 5cmx3cm subserosal cystic lesion was identified on the gallbladder.

Follow-up & Histopathology: Histopathological examination confirmed a cystic structure with multiple spaces lined by flattened cells and proteinaceous fluid containing lymphocytes, consistent with cystic lymphangioma. The postoperative recovery was uneventful. The long-term prognosis for this condition is excellent following complete surgical resection.

4. Conclusion(s) Cystic lymphangioma of the gallbladder is a rare but important differential diagnosis for cystic lesions of the biliary system. While preoperative diagnosis can be complex and may be aided by MRI or MRCP, complete surgical excision remains the treatment of choice [5]. Open cholecystectomy may be the treatment of choice in most cases of lymphangioma of the gallbladder [1, 2, 6, 7, 8, 10, 11] laparoscopic cholecystectomy can also be considered a successful treatment option [5,9] This would apply even if the lymphangioma was more than 15cm in diameter [5], or if it is a complicated hemorrhagic cystic lymphangioma of the gallbladder mimicking a subhepatic abscess [9]. In this study, lymphangioma of the gallbladder was an accidental finding. We were able to excise it with the gallbladder laparoscopically

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Inflammatory Bowel Disease and Cardiac Arrhythmias: Inflammatory Mechanisms, Clinical Associations, and Therapeutic Implications

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1. Introduction/Background

Metabolic dysfunction-associated steatotic liver disease (MASLD), previously termed non-alcoholic fatty liver disease, is the most common chronic liver disease worldwide. Increasing rates of obesity and metabolic syndrome have contributed to a rising prevalence among younger individuals. Many patients remain asymptomatic and are frequently diagnosed incidentally during imaging performed for unrelated clinical complaints.

2. Objectives

To highlight the importance of recognizing incidental hepatic steatosis in young adults and to emphasize the role of early lifestyle intervention in preventing disease progression.

3. Clinical Case-Diagnostic Evaluation, Treatment and Follow-up

A young adult presented with mild, non-specific abdominal discomfort. Routine laboratory investigations were largely unremarkable. Abdominal ultrasonography performed during evaluation revealed increased hepatic echogenicity suggestive of fatty liver. The patient reported minimal alcohol intake but had metabolic risk factors including overweight status and a sedentary lifestyle. Further metabolic assessment demonstrated features of metabolic dysfunction. Management focused on lifestyle modification, including dietary counseling, weight reduction, and increased physical activity. On follow-up, the patient demonstrated improvement in symptoms with stabilization of metabolic parameters.

4. Conclusion(s)

Incidental identification of MASLD provides an important opportunity for early intervention. Increased clinical awareness and timely lifestyle modification may help prevent progression to advanced liver disease.

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The Liver–Heart Axis in MASH: Current Evidence on Cardiovascular Risk, Lipid Lowering Plaque Regression, and Emerging MASH Directed Therapies

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Background: Patients with metabolic dysfunction-associated steatotic liver disease (MASLD) and metabolic dysfunction-associated steatohepatitis (MASH) have substantially elevated cardiovascular risk, and cardiovascular events exceed liver-related mortality. Contemporary lipid-lowering trials show that pharmacologic regression of coronary atherosclerotic plaque is associated with meaningful reductions in major adverse cardiovascular events (MACEs). A 2023 meta-regression of 23 lipid-lowering studies (7407 patients) found that each 1% reduction in percent atheroma volume (PAV) was associated with a 17–25% reduction in MACE odds, establishing plaque regression as a plausible surrogate of risk reduction. Resmetirom, GLP-1 receptor agonists, and other MASH-directed agents improve hepatic steatosis and atherogenic lipids, but their combined effects with statins on coronary plaque remain undefined.

Objectives: To summarize evidence on (1) cardiovascular risk and plaque biology in MASLD/MASH and (2) the impact of resmetirom and GLP-1–based therapies, with or without statins, on atherosclerotic burden, and to determine whether any data exist on coronary plaque change by CT angiography (CCTA) in this setting.

Methods: We systematically reviewed trials and observational studies on MASLD/MASH pharmacotherapy, coronary plaque imaging, and lipid-lowering–induced plaque regression.

Results: High-intensity statins and other lipid-lowering therapies consistently induce regression of coronary plaque volume and improve fibrous-cap thickness, with greater effects at lower achieved LDL-cholesterol levels. Resmetirom and GLP-1 agonists significantly reduce liver fat and improve lipids and cardiometabolic profiles in MASLD/MASH trials, but no published studies report CCTA-based plaque regression specifically attributable to resmetirom or GLP-1 therapies, alone or in combination with statins, in MASH cohorts.

Discussion: Current data strongly support intensive lipid lowering and MASH-directed pharmacotherapy for cardiometabolic risk reduction, but the liver–heart mechanistic link remains indirect.

Conclusion: There are no current data on statin-potentiated CCTA plaque regression with resmetirom or GLP-1 agents in MASH; this is a critical, unmet research need suitable for future prospective imaging trials.

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Incidental Detection of Metabolic Dysfunction-Associated Steatotic Liver Disease in a Young Adult: Importance of Early Recognition

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1.Introduction/Background

Metabolic dysfunction-associated steatotic liver disease (MASLD), previously termed non-alcoholic fatty liver disease, is the most common chronic liver disease worldwide. Increasing rates of obesity and metabolic syndrome have contributed to a rising prevalence among younger individuals. Many patients remain asymptomatic and are frequently diagnosed incidentally during imaging performed for unrelated clinical complaints.

2. Objectives

To highlight the importance of recognizing incidental hepatic steatosis in young adults and to emphasize the role of early lifestyle intervention in preventing disease progression.

3. Clinical Case-Diagnostic Evaluation, Treatment and Follow-up

A young adult presented with mild, non-specific abdominal discomfort. Routine laboratory investigations were largely unremarkable. Abdominal ultrasonography performed during evaluation revealed increased hepatic echogenicity suggestive of fatty liver. The patient reported minimal alcohol intake but had metabolic risk factors including overweight status and a sedentary lifestyle. Further metabolic assessment demonstrated features of metabolic dysfunction. Management focused on lifestyle modification, including dietary counseling, weight reduction, and increased physical activity. On follow-up, the patient demonstrated improvement in symptoms with stabilization of metabolic parameters.

4. Conclusion(s)

Incidental identification of MASLD provides an important opportunity for early intervention. Increased clinical awareness and timely lifestyle modification can help prevent progression to advanced liver disease.

5. References

- Rinella ME et al. New nomenclature for fatty liver disease. *Hepatology*. 2023.
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Choledocholithiasis with Normal Liver Enzymes: A Diagnostic Challenge in Persistent Right Upper Quadrant Pain

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1.Introduction/Background

Choledocholithiasis is a common hepatobiliary condition that typically presents with right upper quadrant abdominal pain, jaundice, and abnormal liver function tests. However, atypical presentations may occur in which biochemical markers remain within normal limits, potentially delaying diagnosis and appropriate management. Early recognition of such cases is important to prevent complications including cholangitis and acute pancreatitis.

2. Objectives

To highlight the diagnostic challenge of suspected choledocholithiasis in patients presenting with persistent right upper quadrant pain despite normal liver enzyme levels.

3. Clinical Case-Diagnostic Evaluation, Treatment and Follow-up

A young adult presented with intermittent right upper quadrant abdominal pain without associated fever or jaundice. Physical examination demonstrated localized tenderness in the right upper quadrant. Initial laboratory investigations, including liver function tests, were within normal limits. Abdominal ultrasonography demonstrated mild gallbladder distention without clear evidence of common bile duct stones. Due to persistent symptoms and continued clinical suspicion, magnetic resonance cholangiopancreatography (MRCP) was performed and revealed a small obstructing stone in the common bile duct. The patient subsequently underwent endoscopic retrograde cholangiopancreatography (ERCP) with successful stone extraction. The patient recovered well and remained asymptomatic during follow-up.

4. Conclusion(s)

Choledocholithiasis may occasionally present with normal liver enzyme levels, posing a diagnostic challenge. Persistent clinical suspicion and timely use of advanced imaging modalities such as magnetic resonance cholangiopancreatography (MRCP) are essential to ensure accurate diagnosis and appropriate management.

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Neuroendocrine Transformation in Recurrent Gallbladder Adenocarcinoma with Response to Chemoimmunotherapy

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Introduction:

Gallbladder carcinoma is the most common biliary tree cholangiocarcinoma, which is an aggressive cancer with a poor prognosis. It is more common in women. 90% of gallbladder cancers are adenocarcinomas and transformation into high-grade neuroendocrine carcinoma during recurrence is rare and aggressive. For localized disease, surgical resection is still the only possible effective treatment. Systemic chemotherapy has long been the accepted first-line treatment for advanced or metastatic biliary tract cancers.

Chemoimmunotherapy has become the new standard of care for advanced biliary tract cancers after the TOPAZ-1 trial, a randomized phase III study, showed that adding Durvalumab to gemcitabine and cisplatin significantly improved overall survival compared with chemotherapy alone.

Objectives:

Describing a rare presentation of neuroendocrine transformation in recurrent gallbladder adenocarcinoma and to highlight the clinical response to chemoimmunotherapy in the era of immunotherapy-based treatment strategies for advanced biliary tract cancers.

Clinical case:

A 41-year-old female with hypertension and hyperlipidemia presented with jaundice, dark urine, pruritus, insomnia, and intermittent right upper quadrant discomfort. Labs showed a cholestatic pattern with direct hyperbilirubinemia, elevated alkaline phosphatase, and mildly raised transaminases. Ultrasound and Magnetic resonance cholangiopancreatography revealed gallbladder microlithiasis, intrahepatic biliary dilatation, and a 3–4 cm mid-common bile duct mass causing upstream obstruction. Endoscopic Retrograde Cholangiopancreatography confirmed malignant stricture.

Subsequently, she underwent radical en-bloc bile duct resection with D2 lymphadenectomy, cholecystectomy, and Roux-en-Y hepaticojejunostomy. Histopathology showed intestinal-type moderately differentiated adenocarcinoma (pT3N0Mx) with perineural invasion. She completed adjuvant gemcitabine and capecitabine.

Five months later, CT scan revealed 6.0x4.0x4.6 cm subhepatic and 11x8.7x11 cm abdominopelvic masses causing mass effects. Her biopsy showed metastatic high-grade neuroendocrine carcinoma. She also developed primary pulmonary thromboembolism, for which she was started on rivaroxaban, later changed to aspirin.

She completed six cycles of palliative chemoimmunotherapy with cisplatin, etoposide, and durvalumab, resulting in a marked radiologic response. She is currently on durvalumab.

Recent CT scans compared to the previous scan revealed stable disease with minimal new lesions, not meeting the criteria for progressive disease. The relapse was followed by a disease-free interval of approximately six months.

Conclusion:

This case highlights the aggressive nature and rare neuroendocrine transformation of gallbladder adenocarcinoma. Although advanced biliary malignancies have historically been associated with poor prognosis, outcomes have improved in recent years with the introduction of chemoimmunotherapy, as demonstrated in the TOPAZ-1 trial.

In our patient, a personalized treatment approach was adopted. Given the short disease interval of five months, re-challenging with cisplatin and gemcitabine was avoided. Considering the neuroendocrine transformation, treatment with cisplatin, etoposide, and durvalumab was initiated, resulting in an excellent clinical response.

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Epidemiology and Diagnostic Patterns of Helicobacter pylori Infection in the UAE: A Retrospective Study from the Northern Emirates

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CLINICAL RESEARCH & STUDY (A RETROSPECTIVE STUDY)

1. INTRODUCTION - Helicobacter pylori is a clinically significant gastric pathogen associated with chronic gastritis, peptic ulcer disease, and gastric carcinoma. Its transmission and persistence is linked by dietary habits, food hygiene, and environmental and living conditions. Delayed or missed diagnosis may permit persistent infection and progression to significant gastrointestinal complications. Understanding the regional epidemiology of infection is therefore essential for effective clinical management and prevention strategies. Nevertheless, epidemiological data on H. pylori infection in the United Arab Emirates remain limited, highlighting the need for regional studies to guide screening and preventive approaches.

2. OBJECTIVE- To determine the prevalence of H. pylori infection among residents of the Northern Emirates, UAE, and to compare the diagnostic performance of non-invasive tests.

3. METHODS - A retrospective analysis of approximately 8,000 patient records (2018-2024) was conducted in the Northern Emirates, UAE. Diagnostic modalities included urea breath test (UBT), stool antigen testing, and serological assays (IgG and IgA). Demographic and clinical data were extracted from hospital records and analyzed using SPSS software. Statistical analysis included chi-square testing and logistic regression to evaluate infection prevalence and diagnostic performance.

4. RESULTS - The overall serological prevalence of H. pylori infection was 24%. The urea breath test showed the highest sensitivity (93%) for detecting active infection, whereas stool antigen positivity was observed in 7.8% of cases. The most frequent symptoms among infected patients were abdominal pain, dyspepsia, and bloating. Data analysis demonstrated a declining trend in active infection during the study period, indicating reduced transmission within the region.

5. DISCUSSION - The findings highlight the clinical value of non invasive diagnostic methods for detecting H. pylori infection. The superior performance of the urea breath test supports current guideline recommendations favoring its use for identifying active infection and monitoring treatment outcomes. The difference between seroprevalence and active infection rates may reflect improvements in public health measures and healthcare access within the region.

6. CONCLUSION - This study provides important epidemiological insight into H. pylori infection in the Northern Emirates and underscores the importance of selecting appropriate non-invasive diagnostic tools for accurate detection and effective clinical management.

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ABSTRACT # 32

Ehlers–Danlos Syndrome Causing Gastroparesis, Gastrointestinal Dysmotility, and Gut Failure: A Case Series

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Introduction / Background:

Ehlers-Danlos syndrome (EDS) is a rare connective tissue disease that manifests as joint hypermobility, skin hyper-elasticity, and blood vessel fragility, resulting in significant morbidity and mortality. A majority of patients also experience various gastrointestinal (GI) complications, including both structural problems such as hiatus hernias, visceroptosis, rectoceles, and rectal prolapse, as well as functional problems including disordered gut motility. Despite a significantly increased prevalence in EDS patients compared to the general population, GI dysmotility remains underdiagnosed. It requires an extensive investigational approach and a high index of clinical suspicion for diagnosis. Moreover, the therapeutic approach can be challenging given the limited options and the disabling nature of the disease.

Objectives:

This case series aims to present eight cases of GI dysmotility in patients diagnosed with EDS, demonstrating the varied presentations, disease progression, and potential management options, while highlighting the importance of multidisciplinary care.

Clinical Case:

The reported patients were diagnosed with EDS hypermobility type (type III), with a mean age of 27 years and a mean BMI of 15.4 kg/m². Four patients were diagnosed with postural tachycardia syndrome (POTS), and four patients were diagnosed with superior mesenteric artery (SMA) syndrome. Seven out of eight patients were diagnosed with delayed gastric emptying on a background of abdominal pain and weight loss, with one patient (case 8) exhibiting normal gastric emptying. Diagnostic evaluations included gastric emptying studies, small bowel follow-through, colonic transit time studies, and upper endoscopy. An ongoing challenge among all patients was malnutrition, which had a significant impact on their quality of life. Seven patients required at least one form of nutritional support, including enteral or parenteral feeding. The disease course was severe enough in six patients to require surgical intervention. Follow-up ranged from several months to years, with variable outcomes including partial tolerance to oral intake to continued dependence on parental or enteral nutritional support.

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7	Case 8
EDS type	EDS-HT (Type 3)	EDS-HT (Type 3)	EDS-HT (Type 3)	EDS-HT (Type 3)	Type IV	EDS-HT (Type 3)	EDS-HT (Type 3)	EDS-HT (Type 3)
Diagnosis	Clinical	Genetic testing	Clinical	Clinical	Genetic testing	Clinical	Genetic testing	Clinical
POTS	Absent	Present	Absent	Present	absent	absent	Present	Present
Pneumothorax	Absent	Absent	Present	Absent	absent	Absent	Absent	Absent
vWF deficiency	Absent	Absent	Present	Absent	absent	Absent	Absent	Absent
SMA syndromediagnosis	Present	Present	Absent	Absent	absent	Absent	present	Present
Scoliosis	Absent	Present	Absent	Absent	absent	Absent	Absent	Absent
Diverticulosis	Absent	Absent	Present (small and large bowel)	Absent	absent	Absent	Absent	Absent
Age (years)	25	22	24	23	47	30	27	19
Gender	Female	Female	Male	Female	male	Female	Female	Female
BMI (kg/m2)	13.66	15.29	15.95	15.8	15.8	14	16	16.7
Predominant symptoms	Abdominal pain, vomiting, weight loss, muscle weakness, <u>neurological symptoms</u>	Poor oral intake, constipation, abdominal pain, weight loss, muscle weakness, <u>neurological symptoms</u>	Weight loss, abdominal pain, vomiting	Constipation, dysphagia, early satiety, abdominal pain, weight loss, bloating, nausea, vomiting	Constipation	Early satiety Vomiting Bloating constipation	Vomiting Bloating constipation	Nausea, vomiting, bloating, constipation, weight loss
Gastric emptying	Delayed	Delayed	Delayed	Delayed	Not done	Delayed	Delayed	Normal
Colonic transit time	Not done	Borderline abnormal	Not done	Delayed	Not done	Delayed	Not done	Not done
Small bowel follow-through	Not done	Visceroptosis	Markedly delayed SB transit	Normal	Not done	Rapid transit	Normal	Visceroptosis
Mechanical obstruction	No	No	Yes (Narrowing in duodenum)	No	Recurrent SBO	no	Intussusception	none
Tube feeding	PEG J tube	PEG J tube	PEG J tube	NJ tube, PEG tube	none	NJ	NJ	NJ then J tube
Tolerance to tube feeding	Maximum tolerance to half daily requirement	Maximum tolerance to half daily requirement	No	No	no	no	no	yes
Parenteral nutrition	Yes (stopped due to recurrent line sepsis and blood clots)		Yes	Yes	no	yes	yes	no
IVIG administration	Yes	Yes	None	Yes	no	no	no	no
Surgical intervention	SMA surgery; duodenojejunostomy	SMA surgery; duodenojejunostomy	Subtotal colectomy, proximal jejunum resection, duodenal jejunostomy tube ileostomy	Subtotal colectomy, ileorectal anastomosis, pyloroplasty, and gastric reduction	Ex. lap , lysis of adhesions, excision of fistula and repair of abdominal wall defect with mesh, to treat bowel perforation	recent examination under anesthesia and injection of anal muscles for abnormal results of anorectal manometry	POP Distal Gastrectomy with Roux en Y Fundoplication and Diagnostic Laparoscopy with Adhesiolysis	none
Outcome	Barely maintains weight on oral intake with intermittent GI symptoms	Barely maintains weight on oral intake with intermittent GI symptoms	Weaned off TPN Continued for few months on tube feeding Recurrent intolerance to tube feeding Ended up on TPN Not candidate for small bowel transplant	Weaned off TPN and tube feeding Significant improvement Maintaining weight Intermittent symptoms of bloating and diarrhea	Underweight but maintains weight, symptomatic treatment of constipation	TPN dependent awaiting surgery pyloroplasty	TPN dependent	On Tube Feeding

Conclusion:

We aim to highlight the importance of evaluating for gut failure in patients with EDS and vice versa especially in the presence of autonomic dysfunction features - such as POTS- and malnutrition. It is crucial for gastroenterologists to understand how these manifestations are linked to their underlying etiology in order to tailor treatments, prevent iatrogenic complications, and address related issues. Additionally, to gain a comprehensive understanding of the clinical picture of EDS, a multidisciplinary team approach is recommended to enhance patient care and ensure a more holistic management strategy.

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Predicting Liver Failure Before It Happens: AI and iPSC Organoids for Drug-Induced Liver Injury

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INTRODUCTION

Drug-induced liver injury (DILI) remains one of the most difficult complications in hepatology and a leading cause of drug withdrawal during development. Many medications that appear safe in traditional preclinical models later cause severe liver toxicity in humans, highlighting the limitations of current animal and cell-based testing systems.

A major scientific breakthrough has been the development of induced pluripotent stem cells (iPSCs), which allow adult human cells to be reprogrammed into stem cells capable of differentiating into various tissues, including liver cells. Using this technology, researchers can generate three-dimensional liver organoids, miniature structures that replicate important features of human liver biology. These models offer a promising platform for studying drug toxicity in a more physiologically relevant environment.

OBJECTIVE

To examine how iPSC-derived liver organoids combined with artificial intelligence could improve current approaches to predicting drug-induced liver injury.

METHODOLOGY

- Review of recent literature on stem-cell derived liver organoids
- Analysis of studies evaluating organoid-based hepatotoxicity models
- Evaluation of artificial intelligence methods for toxicity prediction
- Integration of imaging and metabolic datasets from organoid experiments

RESULTS

Recent studies show that iPSC-derived liver organoids can model drug-induced hepatotoxicity, and AI-based analysis may enable earlier detection of toxicity signals.

DISCUSSION

Liver organoids derived from iPSCs reproduce key structural and metabolic characteristics of human liver tissue, making them valuable tools for studying hepatotoxicity in vitro. When exposed to candidate drugs, these organoids generate large datasets including high-resolution imaging, cellular morphology changes, and metabolic activity patterns.

Artificial intelligence can analyze these complex datasets and detect subtle patterns of cellular stress that may indicate early hepatotoxic effects. Importantly, iPSC technology allows organoids to be generated from individual patients, enabling researchers to study how different genetic backgrounds influence drug metabolism and toxicity. This patient-specific approach may be particularly relevant in genetically diverse populations such as those found in the United Arab Emirates, where variability in drug response can be significant.

CLINICAL IMPACT

- Earlier detection of hepatotoxic drugs
- Reduced drug-induced liver failure in patients
- Improved safety screening during drug development
- Reduced drug development costs and clinical trial failures
- Potential for personalized toxicity testing

CONCLUSION

The integration of iPSC-derived liver organoids with artificial intelligence represents a promising emerging strategy for improving the prediction of drug-induced liver injury. By providing human-relevant models and enabling patient-specific analysis of drug responses, these technologies may help transform how hepatotoxicity is studied and predicted, ultimately supporting the development of safer and more personalized therapies in hepatology.

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Complete Mucosal Healing In Ulcerative Colitis With Traditional Biologics In An Underprivileged Patient

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Introduction:

Ulcerative colitis is a chronic inflammatory condition of the colonic mucosa. The management has changed from simply controlling the symptoms to the goal of deep mucosal healing, which has been associated with better long-term outcomes.

Although aminosalicylates are the mainstay of treatment in mild and moderate ulcerative colitis, patients with persistent inflammatory activity or steroid-dependent disease require the addition of biologic agents. Anti-Tumor necrosis factor agents, traditional biologics, infliximab and adalimumab, have shown significant efficacy in the induction and maintenance of remission in moderate and severe ulcerative colitis.

This case demonstrates moderate ulcerative colitis with persistent inflammatory activity despite conventional management, which showed complete mucosal healing with the addition of a biologic agent.

Objectives:

To illustrate the role of biologic treatment in achieving clinical remission and mucosal healing in moderate disease by describing the clinical course of a patient with ulcerative colitis who had persistent inflammatory activity despite mesalazine therapy. The case also demonstrates how biologic agents can be successfully administered despite insurance limitations and how the patient support program (Axios-Madad) is helping patients with limited insurance access to receive treatment.

Clinical case:

A 46 year-old non-smoking male with a known history of Ulcerative Colitis since 2016, presented with active disease despite standard therapy. His past medical history included hypertension and IgA Nephropathy, with a family history of ulcerative colitis. He had flare up on mesalazine (despite full dosage) with fatigue, rectal bleeding, tenesmus and diarrhea.

During follow-up, surveillance colonoscopy demonstrated diffuse active colitis, with persistently elevated fecal calprotectin indicating ongoing inflammation. Repeat colonoscopy revealed two tubular adenomas, active right-sided colitis and diffuse active proctitis with histopathology suggestive of crypt abscesses without evidence of dysplasia.

Due to the ongoing disease activity, treatment was escalated to biologic therapy. Considering insurance coverage restrictions, treatment with adalimumab biosimilar (Hyrimoz) was initiated through a patient support program. Adalimumab was administered using the standard regimen of 160 mg at baseline, 80 mg at week 2, and 40 mg every other week starting from week 4.

Following induction and maintenance therapy, the patient showed clinical improvement. On recent follow-up, ileocolonoscopy demonstrated complete mucosal healing with a Mayo Ulcerative colitis endoscopic score of 0, and histopathology confirmed the absence of active inflammation.

The patient is covered by a basic health insurance plan that does not include biologic

therapy and comes from a very low socioeconomic background. Given these restrictions, he was enrolled in a patient support program (Axios-Madad), which is unique in UAE, that gave him access to biologic therapy. Through this program, he is currently receiving free treatment.

Conclusion:

This case highlights the importance of timely escalation to biologic therapy in patients with Ulcerative Colitis who have persistent inflammation despite standard therapy. Biologic treatment led to significant clinical and endoscopic improvement with complete mucosal healing. Access to treatment through a patient support program was fundamental in maintaining therapy continuity and achieving disease control.

References:

Effectiveness of adalimumab in severe ulcerative colitis: A systematic review and meta-analysis Summarizes clinical evidence supporting adalimumab efficacy in moderate-to-severe UC.

Adalimumab in ulcerative colitis: two cases of mucosal healing and clinical response

Case report demonstrating mucosal healing with adalimumab therapy.

Fecal Calprotectin Predicts Mucosal Healing in Patients With Ulcerative Colitis Treated With Biological Therapies

Evidence supporting the predictive value of biomarkers for response to biologic therapy (optional reference if you decide to include biomarker discussion).

Mayo Clinic – Ulcerative Colitis: Diagnosis and Treatment

Provides an overview of disease management, including indications for biologic therapy in moderate to severe UC.

Epidemiological and histopathological spectrum of gastrointestinal tract disorders: a decade of records from the UAE

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Abstract

Background

Gastrointestinal (GI) disorders represent a major public health concern, yet data from the UAE remain limited, particularly regarding histopathological trends. This study aimed to characterize the epidemiological spectrum and distribution of GI disorders reported to a tertiary care pathology center over a ten-year period. Histopathology-based surveillance offers unique insight into tissue-level patterns often missed by clinical registries.

Objectives

This study aimed to characterize the ten-year epidemiological and histopathological spectrum of GI disorders at a tertiary care center in the UAE, exploring demographic patterns, organ-specific involvement, and temporal disease trends.

Methods

A retrospective, record-based analysis of 12,688 GI-related histopathology reports was conducted from Thumbay Laboratories, UAE (2013–2023). Data was extracted using a structured checklist and analyzed in SPSS v29. Descriptive statistics and inferential tests were applied ($p < 0.05$).

Results

Of the 12,688 cases, 67.5% were male and 32.5% female ($p < 0.001$). Most patients were aged 21–40 years (60.3%). The stomach (29.6%), large intestine (21.2%), and appendix (20.4%) were the most affected organs. Gastritis (27.3%), intestinal conditions (20.6%), and appendicitis (20.0%) predominated. Males showed higher rates of gastritis and appendicitis, while cholecystitis was more common in females (21.4%). Appendicitis was dominant in those < 20 years (60.1%), while neoplasms and gastritis were more prevalent in older adults (≥ 61 years; $p < 0.001$).

Discussion

The findings reflect the UAE's unique demographic structure, particularly the high proportion of working-age male expatriates. The high burden of *H. pylori*-associated gastritis and the age-specific prevalence of acute versus chronic conditions align with regional trends but highlight the necessity for localized screening. The equal distribution of upper (\$51\%\$) and lower (\$49\%\$) GI disorders suggests a need for integrated surveillance strategies across the entire digestive tract.

Conclusion

This study provides a decade-long overview of GI pathology trends in the UAE. The findings highlight age- and sex-specific patterns and support future research integrating clinical and demographic data to inform targeted public health strategies.

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Late onset of De Novo Ulcerative Colitis in a Kidney Transplant Recipient

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Introduction Diarrhea is a common complication following kidney transplantation, typically caused by infection, rejection, or immunosuppressive toxicity. A much rarer cause is de novo inflammatory bowel disease (IBD), which may occur despite ongoing immunosuppression and is thought to result from intestinal dysbiosis and impaired mucosal immunity. Although reported across solid organ transplants, de novo IBD remains rare in kidney transplant recipients, and management approaches are not well standardized. Early recognition is essential in atypical or persistent post transplant diarrhea. We present a case of late onset de novo ulcerative colitis in a kidney transplant patient.

Case Description A 50 year old Bangladeshi man with ESRD of unknown etiology (long standing uncontrolled hypertension) underwent living related kidney transplantation in 2012, maintaining stable graft function (creatinine 99 $\mu\text{mol/L}$; eGFR 76 mL/min/1.73 m²). His baseline immunosuppression included Mycophenolate MMF 750 mg BID and tacrolimus 2 mg AM/1.5 mg PM.

In 2023, he presented with 2 weeks of fever, chills, diffuse abdominal pain, poor oral intake, and bloody diarrhea with tenesmus, along with a 2 month history of intermittent PR bleeding and abdominal discomfort. Initial work up for infectious colitis was unrevealing: stool culture, C. difficile testing, PCR, and ova/parasites were negative. ESR was 112 mm/h, and C ANCA was positive at 60 RU/mL. Symptoms did not improve with antimicrobial therapy. Colonoscopy showed moderate to severe pancolitis, and biopsies revealed active colitis with increased eosinophils in the terminal ileum. Due to atypical features for MMF related colitis (fever, leukocytosis, hematochezia) and negative infectious studies, MMF was discontinued, tacrolimus was up titrated, and methylprednisolone 60 mg/day was started. Repeat colonoscopy demonstrated pancolitis with backwash ileitis, supporting ulcerative colitis rather than MMF colitis. Azathioprine was initiated and optimized to 100 mg/day. Vedolizumab induction began with good clinical response. The patient developed steroid induced diabetes managed with insulin. Prior to the third vedolizumab dose, colonoscopy showed mucosal healing, and therapy was transitioned to infliximab.

Discussion: De novo IBD after kidney transplantation is rare but should be considered when post transplant diarrhea persists despite exclusion of infectious causes. Mycophenolate toxicity typically presents earlier, with characteristic endoscopic patterns different from ulcerative colitis. Calcineurin inhibitors, including tacrolimus, may contribute to IBD pathogenesis through impaired regulatory T cell function. Emerging data suggest that vedolizumab is a safe and effective therapy in transplant recipients due to its gut selective mechanism and minimal systemic immunosuppression, although evidence remains limited.

Conclusion: De novo ulcerative colitis is an uncommon but important diagnosis in kidney transplant recipients with persistent diarrhea. This case demonstrates successful management with vedolizumab induction followed by infliximab maintenance, while maintaining stable allograft function.

Keywords: ulcerative colitis, IBD, mycophenolate, kidney transplant

A Challenging Case of Cecal Mass Requiring Gastroenterological Expertise

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Introduction: Differentiating between benign and malignant lesions in the cecal region of the gastrointestinal tract is a significant diagnostic challenge. The differential diagnosis encompasses a wide array of conditions such as appendiceal abscesses, mucocèles, lipomas, tuberculous lesions, perityphlitic, and various neoplastic processes, including cecal carcinoma and lymphoma. Malignancies can also present as cecal masses, necessitating consideration of metastatic disease from distant sites. Additionally, inflammatory conditions such as Crohn's disease or diverticulitis may mimic malignancy. Given the broad spectrum of potential etiologies, surgical exploration is often requisite for definitive diagnosis and treatment. This report presents a complex case of a cecal mass associated with a rare disease, emphasizing the importance of thorough clinical evaluation.

Case Presentation: A 48-year-old male from Sudan, with a medical history of hypertension, presented with a 1-month history of fever, abdominal pain, weight loss, nausea, and poor oral intake. Laboratory investigations demonstrated normal liver and renal function, with a white blood cell count of $12.2 \times 10^9/L$, hemoglobin of 122 g/L, platelet count of $429 \times 10^9/L$, eosinophil count of $2.52 \times 10^9/L$, and C-reactive protein (CRP) at 98 mg/L. A CT scan of the chest and abdomen revealed a liver lesion (3 x 2.5 cm) in segment VI, alongside a substantial cecal and ascending colon mass causing significant luminal narrowing, mesenteric fat stranding, and multiple enlarged lymph nodes. The patient was initiated on broad-spectrum antibiotic therapy, with minimal improvement in his clinical status. Colonoscopic evaluation exhibited a large fungating cecal mass, and biopsy results indicated colonic ulceration with inflammation and was negative for malignancy. In light of persistent clinical deterioration, the patient underwent right hemicolectomy. His postoperative course was uneventful, but he was readmitted within 10 days due to worsening dyspnea and chest pain, accompanied by persistent fever. There was no recent exposure to tuberculosis. Tumor markers and autoimmune panels (ANCA, ANA) were negative, and follow-up imaging indicated an increase in the size of the hepatic lesion. Pathology of the cecal mass revealed extensive, geographic areas of necrosis associated with a granulomatous reaction, including multinucleated giant cells and a diffuse transmural infiltrate of eosinophils. These findings were consistent with the diagnosis of Churg-Strauss syndrome (eosinophilic granulomatosis with polyangiitis, EGPA). The patient was commenced on pulse intravenous steroids (500 mg) for 3 days, followed by a tapering regimen of 60 mg prednisolone. Subsequently, azathioprine (100 mg) was introduced. At 6 years of follow-up, the patient exhibited stable clinical status without immunosuppressive therapy.

Discussion: Eosinophilic Granulomatosis with Polyangiitis (EGPA) is an anti-neutrophil cytoplasmic antibody (ANCA)-associated vasculitis affecting small to medium-sized vessels, characterized by severe asthma, hypereosinophilia, extravascular granulomas, and necrotizing vasculitis. Patients commonly present with systemic symptoms such as fever, weight loss, and fatigue, with a subset experiencing multi-organ involvement, including cardiac, pulmonary, and upper respiratory manifestations. EGPA should be considered in patients with a relevant history of asthma, chronic rhinosinusitis with polyps, and chronic peripheral eosinophilia with end-organ involvement. Gastrointestinal manifestations, while observed in approximately 30% of cases, are comparatively rare at initial presentation. Diagnosing EGPA involving the gastrointestinal tract necessitates a high index of clinical suspicion, as symptoms can range from nonspecific to those indicating a surgical abdomen.

Conclusion: This case underscores the diagnostic complexity of a cecal mass and liver lesion as the initial manifestation of seronegative EGPA vasculitis. The patient's clinical condition markedly improved with steroid therapy and the addition of azathioprine.

Keywords: cecal mass, EGPA , eosinophilia

Pathogenic Aerobic and Anaerobic Bacterial Microbiomes in Colorectal Tumors versus Adjacent Normal Tissues

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Background

Colorectal cancer remains one of the leading causes of global cancer-related mortality. While microbial dysbiosis is linked to pathogenesis, the ecological role of the aerobe-anaerobe gut microbiome imbalance and its association with pathogenicity in colorectal cancer is poorly understood.

Objective

To evaluate whether the change in aerobe-anaerobe ratio (Δ AAR) defines unique, pathogen-enriched intratumoral microbiome ecological states specifically characterizing the pathogenic influence within colorectal cancer compared to adjacent normal tissues.

Methods

Using genus-level 16S rRNA metagenomics sequencing microbiome data from 492 paired tumor-normal colorectal samples, we addressed our objective by analysing aerobic and anaerobic microbial states and microbiome ecological risk in colorectal cancer versus normal tissues. Results Stratification by $\log_{10}$ (AAR) revealed a directionally concordant enrichment of anaerobe microbiome traits in High AAR tumors compared to normal tissues ($p < 0.001$), with reciprocal depletion of commensals. These High AAR tumors exhibited selective expansion of dominant pathogenic microbiomes, including *Fusobacterium*, *Escherichia-Shigella*, and *Campylobacter* relative to Low AAR tumors and normal tissues ($p < 0.001$). Both Pathogenic AAR and Pathogenic total relative abundance shifts were significantly higher in the High AAR group ($p < 0.001$). Furthermore, the shift in pathogenic total relative abundance was significantly greater in High AAR tumors compared to normal tissues ($p < 0.001$), indicating tumor-specific ecological remodelling driven by pathogenic pressure.

Discussion

These findings suggest that Δ AAR could be a biomarker for colorectal pathogenesis, moving beyond simple diversity metrics to functional ecological states. By linking the aerobe-anaerobe ratio to pathogenic trait enrichment, our results further suggest that the tumor microenvironment creates a unique niche that favours pathogenic anaerobic bacteria. This provides a molecular framework for developing microbiome-modulating therapeutic strategies tailored to a patient's specific anaerobic microbiomes ecological risk profile.

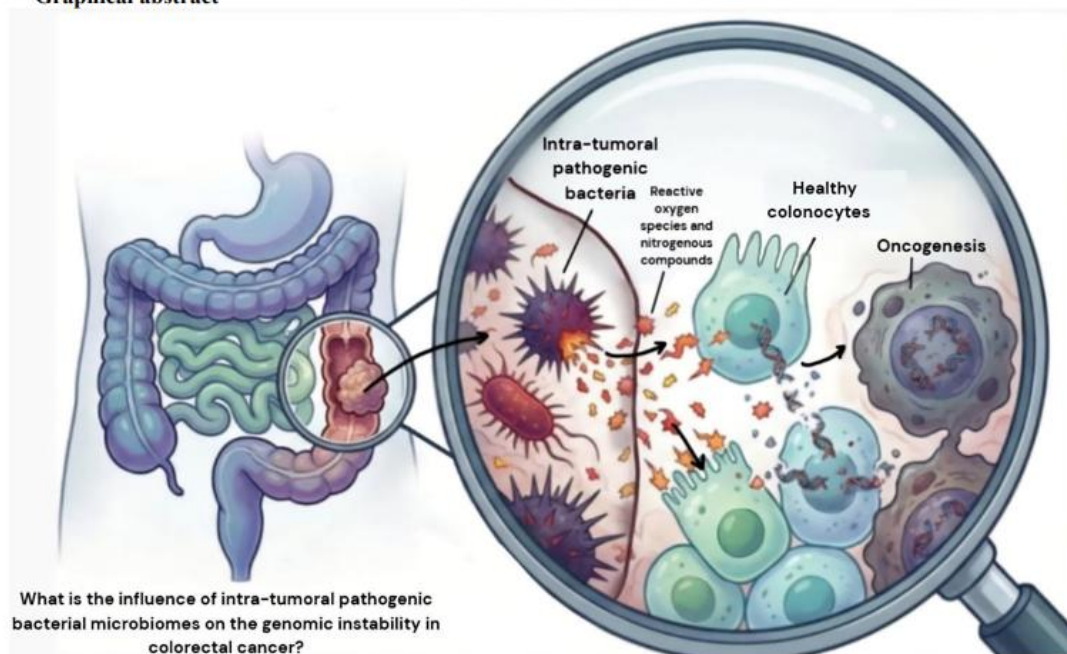
Conclusions

The aerobe-anaerobe gut pathogenic microbiol ratio defines a coherent, pathogen-enriched state in colorectal tumors, providing a robust, anaerobic microbiome-encoded biomarker for discriminating malignant tissue and guiding targeted clinical interventions. **Keywords:** Colorectal cancer, pathogenic microbiomes, aerobic and anaerobic microbial burden, intratumoral microbiome ecology.

Intratumoral Pathogenic Bacterial Microbiomes and Genomic Instability in Colorectal Cancer

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Graphical abstract



Introduction

Colorectal cancer involves complex interactions between the host genome and the intra-tumour microbiome. Pathogenic bacteria may influence tumorigenesis by promoting genomic instability. Understanding the association between pathogenic microbiomes enrichment and tumour genomic instability is crucial for identifying potential microbial risk factors and new prognostic biomarkers in colorectal cancer patients.

Objectives

This study investigated the association between pathogenic intratumoral bacterial microbiomes and the genomic instability in colorectal cancer.

Methods

We performed an integrated analysis of intratumoral gut bacterial microbiomes obtained via 16S rRNA sequencing and whole-genome metagenomics sequencing of 246 colorectal cancer samples using GATK-PathSeq and Kraken2 methodologies. Genomic instability markers included tumour mutational burden (TMB), microsatellite instability (MSI), and copy-number alteration (CNA) burden.

Results

As expected, MSI-high (MSI-H) tumours exhibited significantly higher tumour mutational burden (TMB) (median 104.70) than microsatellite stable (MSS) tumours (median 6.16, $p < 0.001$). However, specific bacterial pathogenic genera were enriched more in MSI-H tumours, notably *Selenomonas* (OR 3.07, $p < 0.001$) and *Hungatella* (OR 2.8, $p < 0.001$), while *Subdoligranulum* was depleted (OR 0.40). Genomic instability indicators, including the MANTIS score and CNA burden were significantly elevated in MSI-H tumours across right-sided colon, left-sided colon, and early-stage colorectal cancer strata (all $p < 0.001$). A high microbial

ecology measured by Microbial Burden Risk (MBR) was strongly associated with increased TMB and MANTIS scores ($p < 0.001$). Furthermore, the presence of *Selenomonas* specifically correlated with significantly higher TMB ($p < 0.001$) and a higher composite instability index ($p = 0.002$). Other pathogenic taxa, such as *Campylobacter*, also showed significant abundance differences between MSI-H and MSS groups ($p = 0.0167$).

Discussion

These findings suggest alignment between pathogenic microbial enrichment and high genomic instability. They further suggest that these bacterial microbiomes thrive in hypermutable environments. The strong association between MBR and instability markers across clinical strata implies that microbiome-based markers can effectively reflect the underlying tumour genomic state and pathological progression of colorectal tumours.

Conclusions

Pathogenic bacterial microbiomes are significantly associated with tumour genomic instability. Microbiomebased risk analyses using pathogenic bacterial microbiomes may serve as biomarkers for early genomic instability and enhance clinical risk stratification for colorectal cancer.

Keywords: Colorectal cancer, intratumoral bacterial microbiomes, genomic instability, biomarkers, tumour pathology.

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Table 1. Genomic and clinical characteristics of the study cohort

Characteristic	Overall	MSI-H	MSS	Odds Ratio	95% CI	P value
TMB - median (IQR)	6.97 (5.13-34.04)	104.70 (86.84-127.01)	6.16 (4.84-7.76)	n/a	25.78-182.15	<0.001
Sex						
FEMALE	115 (46.7%)	33 (66.0%)	82 (41.8%)	2.70	1.40-5.06	0.0026
MALE	131 (53.3%)	17 (34.0%)	114 (58.2%)	0.37	0.20-0.72	0.0026
Tumour side						
Right colon - no. (%)	36 (14.6%)	10 (20.0%)	26 (13.3%)	1.63	0.76-3.68	0.262
Left colon - no. (%)	95 (38.6%)	6 (12.0%)	89 (45.4%)	0.16	0.07-0.42	<0.001
AJCC pathological stage						
I	45 (18.3%)	13 (26.0%)	32 (16.3%)	1.80	0.88-3.77	0.15
II	87 (35.4%)	24 (48.0%)	63 (32.1%)	1.95	1.04-3.63	0.0464
III	77 (31.3%)	7 (14.0%)	70 (35.7%)	0.29	0.14-0.71	0.0033
IV	37 (15.0%)	6 (12.0%)	31 (15.8%)	0.73	0.31-1.90	0.658

Values are median (IQR) for continuous variables and no. (%) for categorical variables. Effect sizes are Hodges-Lehmann median shift for TMB and odds ratios (OR) for categorical contrasts. Two-sided 95% confidence intervals are reported in a separate column and P values are two-sided.

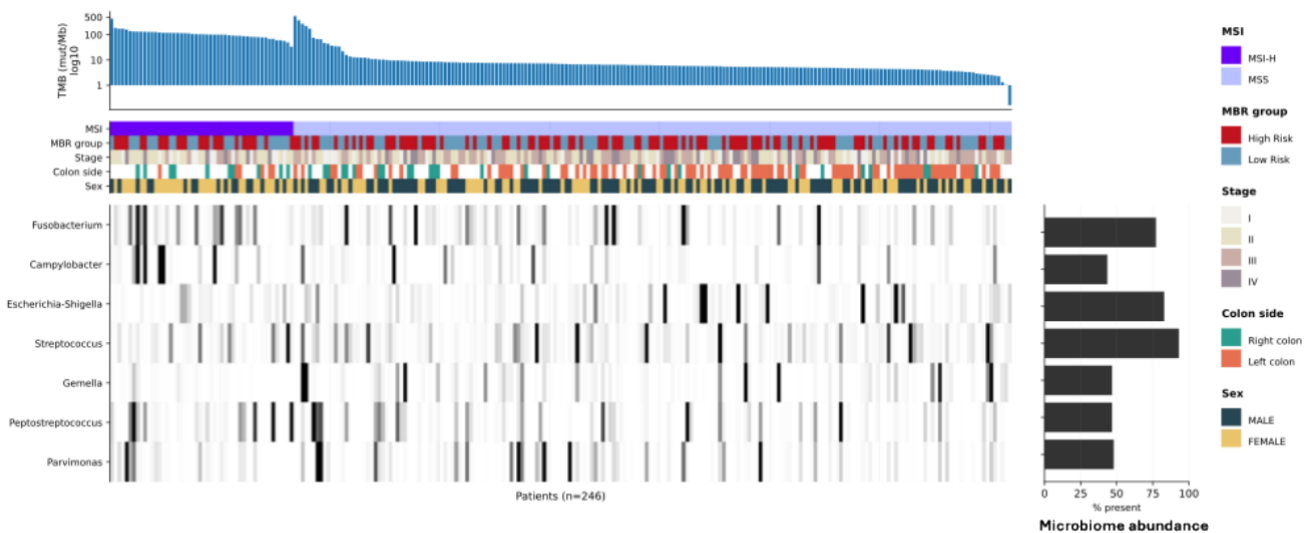


Fig 1. Integrated oncoprint linking intra-tumour pathogenic microbiomes with genomic instability and clinicopathological context in colorectal cancer.

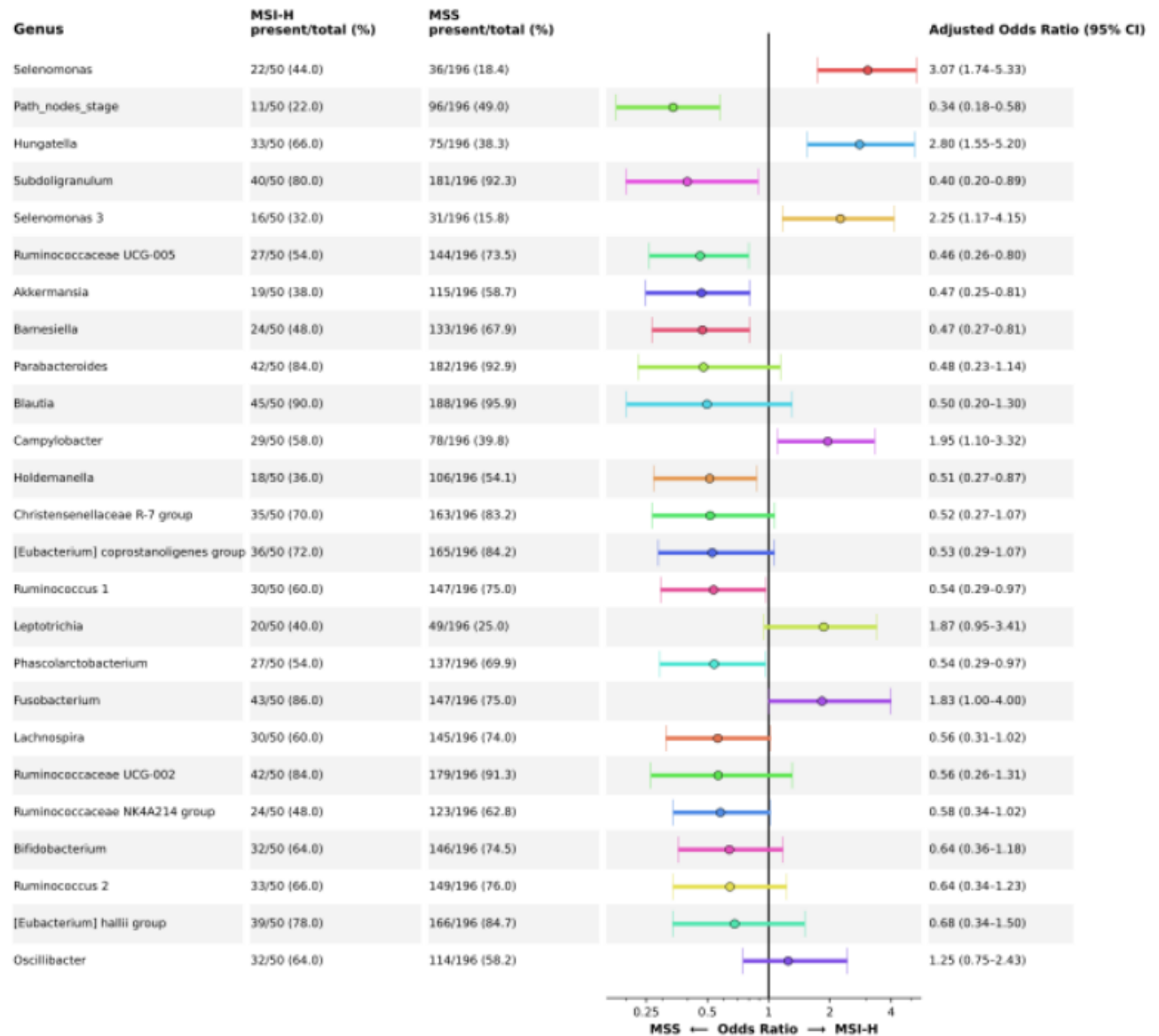


Fig 2. Pathogenic microbiomes and microsatellite instability clinicopathological associations in colorectal cancer.



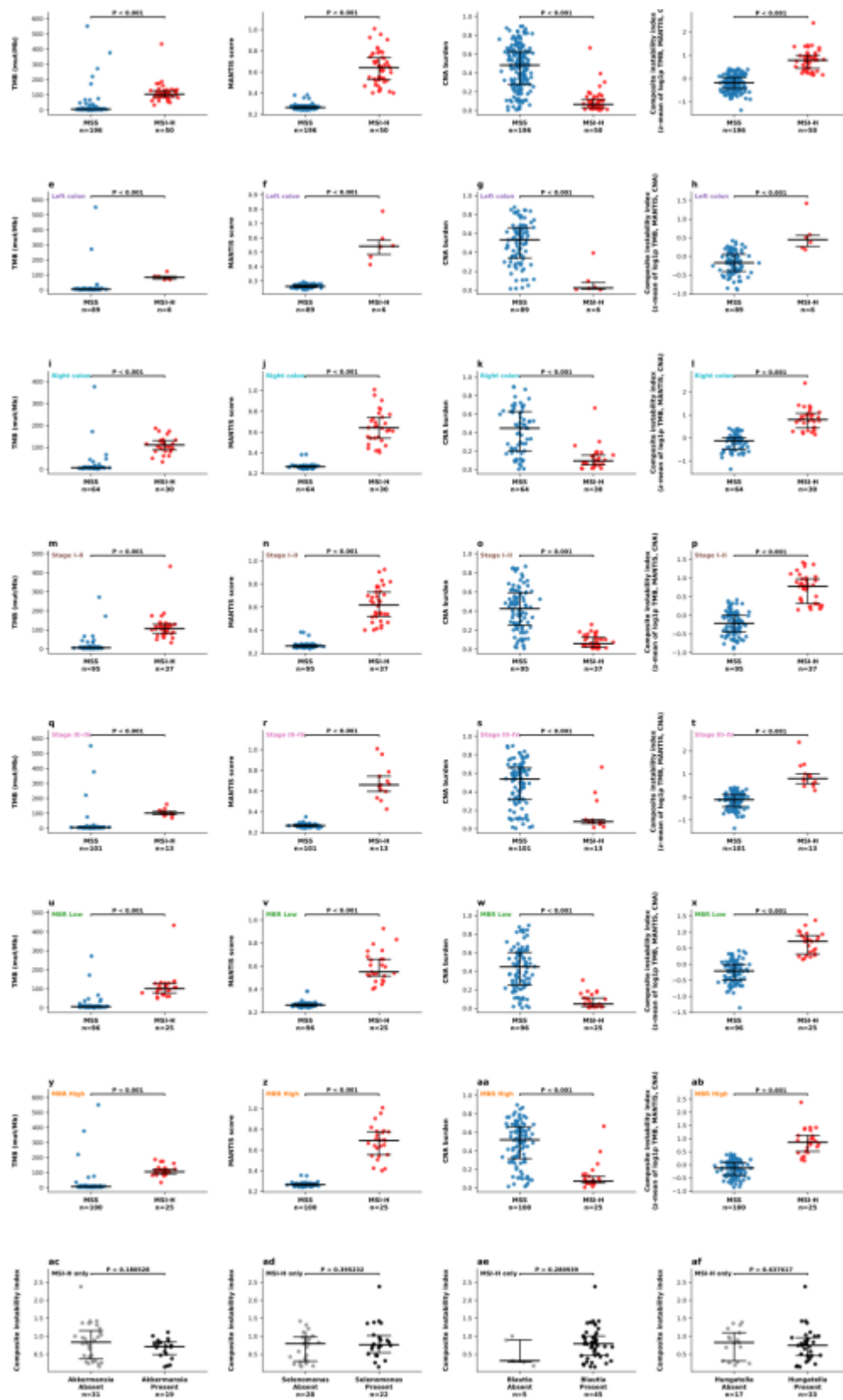


Fig 4. The relationships between specific pathogenic gut microbiomes, tumour mutational burden, microsatellite instability, and copy number alterations across clinical characteristics.

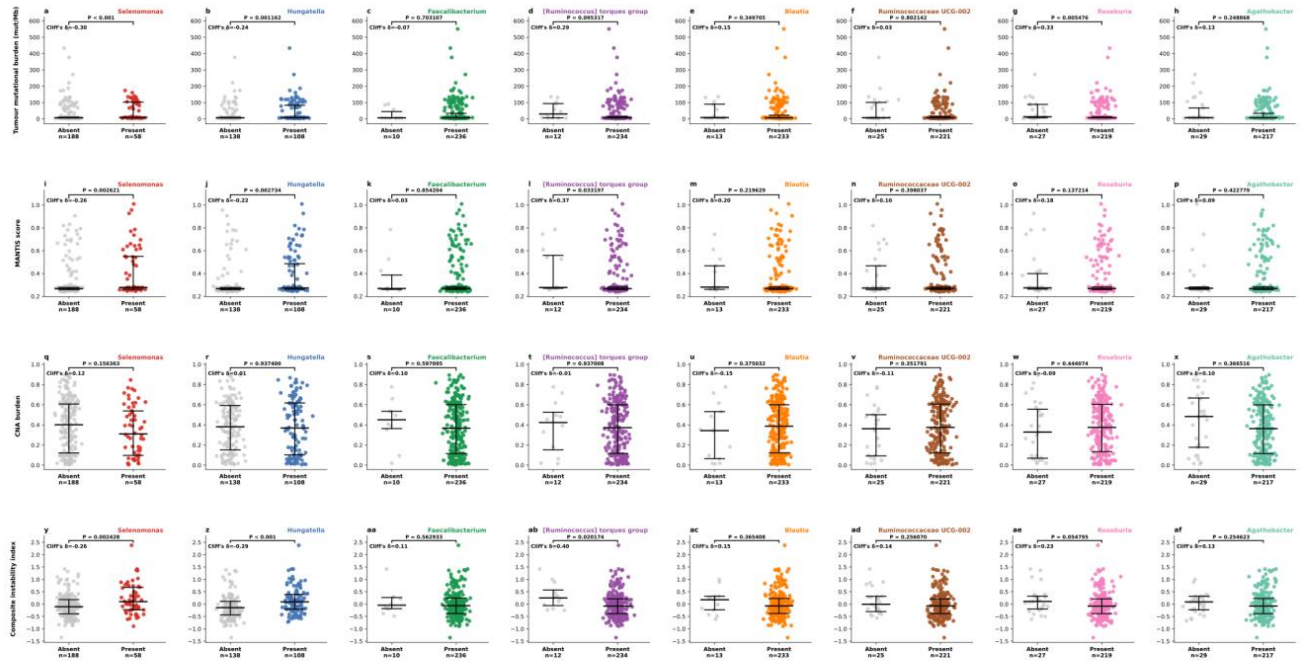


Fig 5. The relationships between different pathogenic gut microbiomes and the genetic instabilities across colorectal cancer samples, revealing potential associations between the microbiomes and tumour genomic profiles that may have clinical relevance.

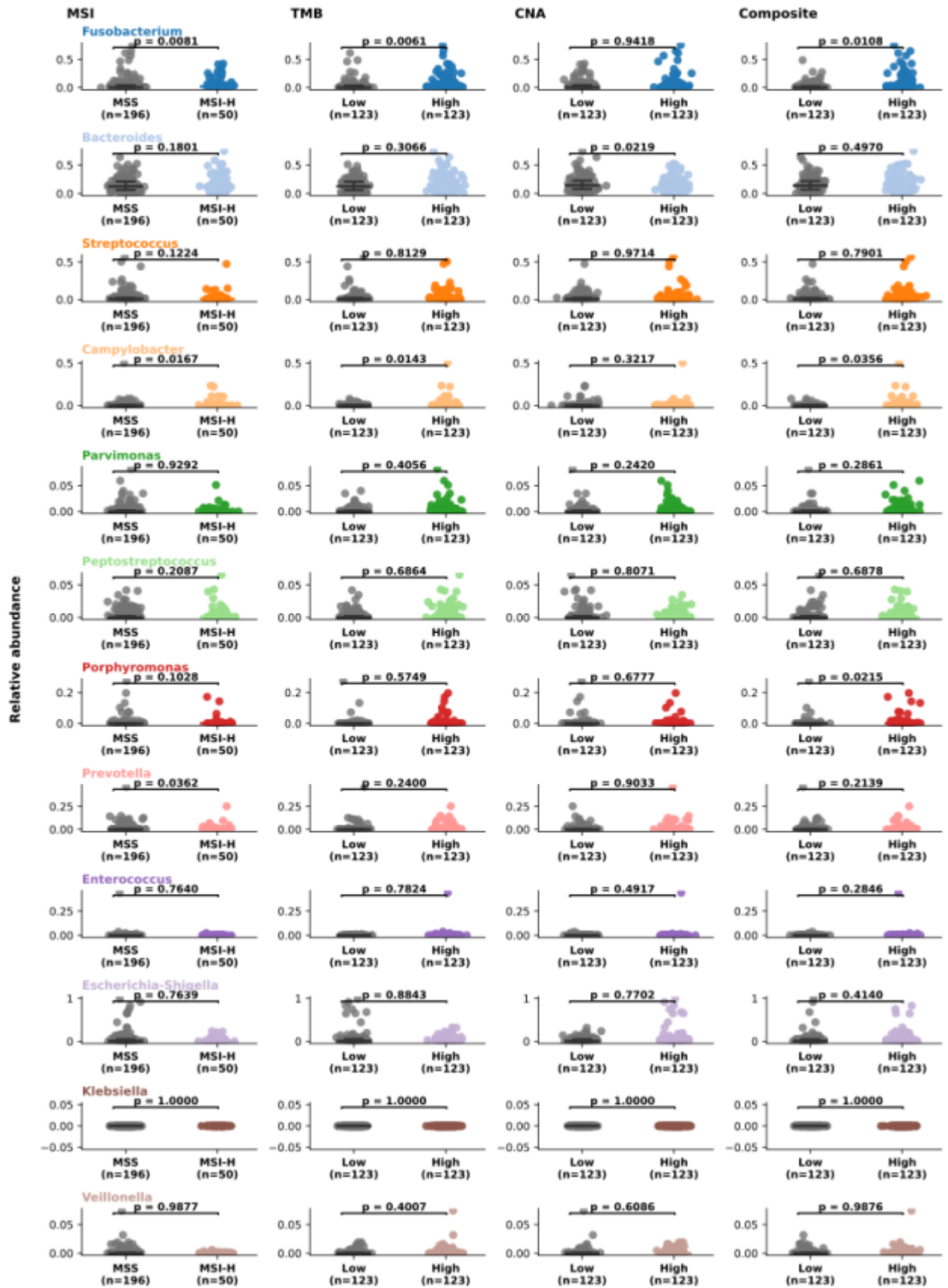


Fig 6. Pathogenic and oncogenic bacterial taxa associate with tumour genomic instability states at genus-level relative abundance for 12 canonical pathogenic/oncogenic taxa.

Rare Presentation of Lactulose Overdose Leading to Ischemic Colitis: A Case Report

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1. Introduction / Background

Lactulose is a synthetic, non-absorbable disaccharide widely prescribed both as an osmotic laxative for the management of constipation and as a first-line agent in the prevention and treatment of hepatic encephalopathy. Its therapeutic mechanism relies on colonic bacterial fermentation, which acidifies the luminal environment, trapping ammonia as ammonium ion and promoting its excretion. This fermentation process, however, concurrently generates significant volumes of hydrogen and carbon dioxide gas.

Ischemic colitis—the most common form of intestinal ischemia in adults—results from impaired colonic perfusion and manifests across a spectrum from transient mucosal injury to transmural gangrene. Established risk factors include advanced age, cardiovascular comorbidities, hypotension, vasopressor use, and hypercoagulable states. Drug-induced ischemic colitis is a well-recognized but less commonly considered aetiology, with oral bowel cleansing agents and certain laxatives implicated in case literature.

The association between lactulose and ischemic colitis is rare and incompletely understood. The proposed mechanism involves excessive gas production during fermentation leading to **colonic distension**, which increases intraluminal pressure, compresses submucosal vessels, and critically reduces mucosal perfusion. In patients with pre-existing vascular compromise—such as the elderly with multiple cardiovascular comorbidities—this mechanism may be sufficient to precipitate frank ischaemia or gangrene, particularly when exacerbated by an overdose of lactulose.

Only a small number of cases linking lactulose ingestion to ischemic colitis have been described in the literature, and fatal outcomes following overdose are exceptionally rare. This case illustrates the potential for lactulose toxicity to cause life-threatening colonic ischaemia in a vulnerable patient, and highlights the importance of dose awareness and clinical vigilance.

2. Objectives

The objectives of this case report are:

1. To present a rare and severe case of ischemic colitis precipitated by accidental lactulose overdose in a high-risk elderly patient.
2. To describe the clinical course, diagnostic evaluation, operative findings, and histopathological confirmation of lactulose-induced necrotizing ischemic colitis.
3. To explore the proposed pathophysiological mechanisms linking excessive lactulose ingestion to colonic ischaemia.
4. To raise clinician awareness of the potential for lactulose toxicity, particularly in elderly and multi-morbid patients, and to advocate for clear patient counselling regarding dosing.

3. Clinical Case

3.1 Presentation and History

A 77-year-old male with a significant burden of comorbidities—including hypertension, type 2 diabetes mellitus, chronic kidney disease (CKD) stage 2, chronic obstructive pulmonary disease (COPD), and a prior

cerebrovascular accident—presented to the emergency department with hypoglycaemia, hypoxia, and acute altered mental status. According to family members, the patient had ingested an entire bottle of lactulose solution for constipation, after which he developed profuse watery diarrhoea and progressive clinical deterioration, prompting emergency attendance.

3.2 Diagnostic Evaluation

On admission, investigations revealed a severe metabolic and lactic acidosis, hyperkalaemia, and acute-on-chronic kidney injury with a creatinine rise from baseline 86 $\mu\text{mol/L}$ to 365.9 $\mu\text{mol/L}$. Given respiratory compromise and haemodynamic instability, the patient required endotracheal intubation and immediate intensive care unit (ICU) admission.

Initial abdominal computed tomography (CT) demonstrated marked sigmoid colon dilatation without evidence of proximal mechanical obstruction, raising early concern for colonic ileus or pseudo-obstruction in the setting of toxic ingestion.

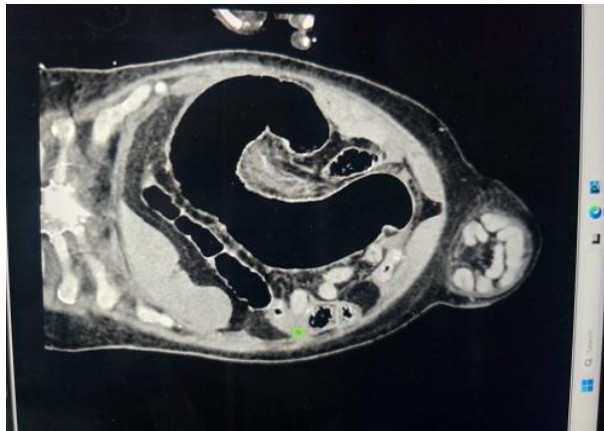


Figure 1. Abdominal CT demonstrating marked sigmoid colon dilatation without proximal obstruction.

Despite aggressive supportive management including fluid resuscitation and electrolyte correction, the patient's condition continued to deteriorate with escalating lactic acidosis (peak lactate 12,818 $\mu\text{mol/L}$), vasopressor requirement, and elevated intra-abdominal pressure of 23 mmHg, consistent with abdominal compartment syndrome.

Repeat CT angiography of the abdomen demonstrated further progression of sigmoid dilatation. Flexible sigmoidoscopy was subsequently performed and revealed circumferential black discolouration of the rectosigmoid mucosa, a finding pathognomonic of transmural ischaemia and impending gangrene. An extensive infectious and thrombotic workup—including stool cultures, *Clostridium difficile* toxin assay, vasculitis screen, and coagulation studies—was unremarkable, supporting a non-infectious, non-thrombotic aetiology.

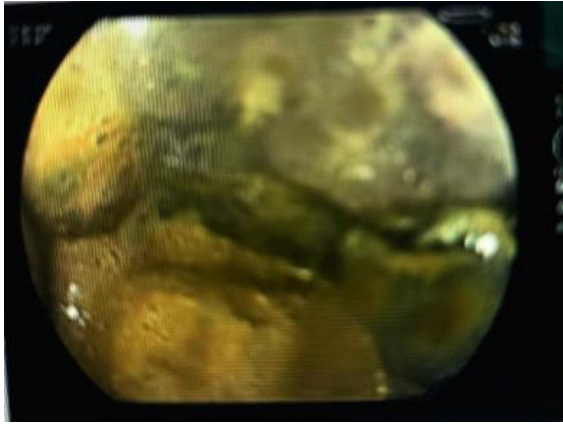


Figure 2. Flexible sigmoidoscopy demonstrating circumferential black discoloration of the rectosigmoid mucosa consistent with transmural ischaemia.



Figure 3. Gangrenous ischaemia of the sigmoid colon and upper rectum.

3.3 Treatment

Given the clinical and endoscopic findings consistent with gangrenous ischemic colitis, emergent surgical intervention was undertaken. Exploratory laparotomy confirmed extensive gangrenous ischaemia of the sigmoid colon and upper rectum with associated generalized peritonitis. The patient underwent low anterior resection with formation of an end colostomy (Hartmann's procedure). Resection of the ischaemic segment was performed without primary anastomosis given the degree of contamination and haemodynamic instability.

Histopathological examination of the resected specimen confirmed severe necrotizing ischemic colitis with full-thickness mucosal necrosis, consistent with the endoscopic and intraoperative appearances. Infectious causes (including *Clostridioides difficile* and enteric pathogens) and thrombotic causes were excluded on pathological and laboratory grounds.

3.4 Follow-Up and Outcomes

The postoperative course was prolonged and complex, reflecting the patient's multi-morbid baseline and severity of the acute insult. He remained ventilator-dependent and vasopressor-reliant in the ICU for several days following surgery. However, with ongoing critical care support, the patient demonstrated gradual clinical improvement with progressive weaning from vasopressors and eventual successful extubation. Renal function partially recovered without the need for renal replacement therapy. The patient was eventually transferred to the ward in a stable condition with an intact functioning colostomy.

4. Conclusions

This case represents a rare but instructive example of life-threatening ischemic colitis precipitated by accidental lactulose overdose in a high-risk elderly patient. In the absence of haemodynamic compromise prior to presentation, vascular thrombosis, or an identifiable infectious trigger, lactulose-induced colonic ischaemia was established as the most probable diagnosis—a diagnosis supported by histopathological confirmation.

The key lessons from this case are:

5. Lactulose-induced ischemic colitis, while rare, is a **potentially fatal complication** that clinicians should be aware of, particularly when encountering elderly or multi-morbid patients who may be more susceptible to colonic distension and vascular compromise.
6. The underlying mechanism—**excessive fermentative gas production causing colonic distension and mucosal hypoperfusion**—may be amplified substantially in the setting of overdose, underscoring the importance of clear patient and caregiver education around dosing.
7. Clinicians should maintain a **low threshold for abdominal imaging and endoscopic evaluation** in patients who develop abdominal distension, worsening lactic acidosis, or haemodynamic deterioration following high-dose lactulose ingestion.
8. Surgical intervention should not be delayed when endoscopic or imaging findings indicate transmural ischaemia, as prompt bowel resection is life-saving in the setting of gangrenous colitis.
9. This case adds to the limited body of literature on lactulose-associated ischemic colitis and reinforces the call for **robust dose labelling, patient counselling**, and cautious prescribing in vulnerable populations.

5. References

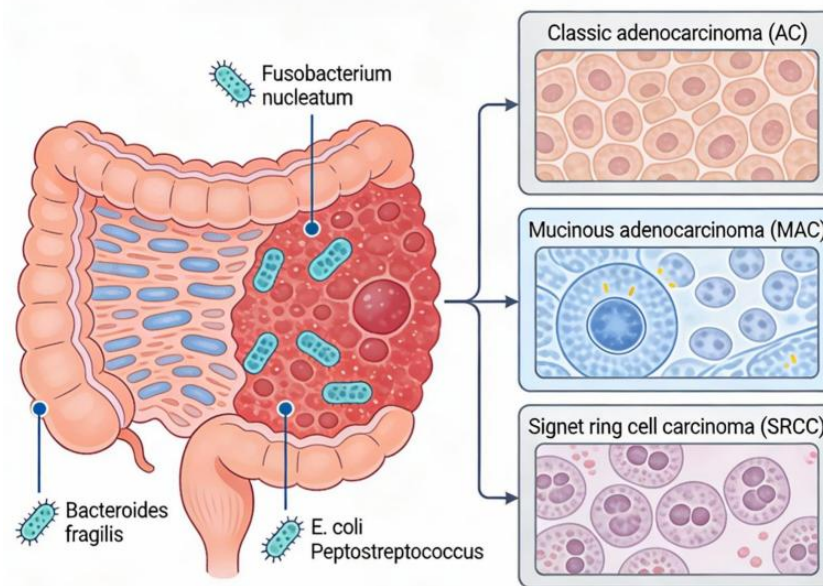
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Pathogenic Bacterial Microbiomes Profile of Histology-Specific Colorectal Adenocarcinomas Compared to Matched Adjacent Normal Tissues

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Graphical abstract



Background

The colorectal cancer microenvironment can host complex microbial ecosystems, yet the ecological consistency of these communities across divergent histological landscapes remains poorly understood. We investigated whether specific pathogenic microbiomes are conserved across histological subtypes or if distinct histological niches recruit unique pathobionts.

Methods

We performed a high-resolution intratumoral microbial characterization of 492 matched tumour and adjacent normal tissues from 246 colorectal cancer patients (Adenocarcinoma of no special type n=142, Intestinal-type n=55, Mucinous type n=44 and Other n=5). Using 16S rRNA metagenomics sequencing data, we analysed the microbial ecology by existing microbiome burden (MBR) score of key pathogenic genera abundance to assess the ecological differences across tumour histological subtypes compared to normal mucosa. We employed Kruskal-Wallis and chi-square tests with the Benjamini-Hochberg analysis for false discovery rate correction. Clinical outcomes including overall and progression-free survival rates were evaluated using adjusted Cox proportional hazards models and Log-rank tests.

Results

A distinct oncopathogenic microbiome core, including *Fusobacterium* (77.2%), *Parvimonas* (48.0%), *Peptostreptococcus* (46.7%) and *Porphyromonas* (33.3%), was significantly enriched in tumours across all histologies compared to matched normal tissue. Comparative analysis revealed that core pathogens did not vary significantly by histology, including *Fusobacterium* ($p=0.624$), *Parvimonas* ($p=0.69$), and *Peptostreptococcus* ($p=0.427$). However, histology-specific shifts were observed in other genera, such as *Lachnospiraceae* ($p=0.011$), *Anaerospirillum* ($p=0.014$), and *Dorea* ($p=0.032$), however none were significant when corrected for FDR q . Most critically, the microbiome emerged as a primary driver of clinical outcomes. Patients with high MBR score exhibited dramatically reduced overall survival (Log-rank $p < 0.001$). Adjusted Cox proportional hazards models identified MBR score as a highly significant independent predictor of mortality ($p < 0.001$). Furthermore, tumour *Porphyromonas* abundance specifically predicted poorer overall survival ($p=0.0228$) and progression-free survival (PFS, $p=0.0228$), with high-abundance groups showing significant PFS divergence (Log-rank $p=0.011$). These microbial associations remained robust despite significant baseline variations in sex ($p=0.014$) and anatomic site ($p=0.049$) across colorectal cancer histological subtypes.

Conclusions

Our findings suggest that while colorectal cancer histological subtypes share a common oncopathogenic microbial signature, the magnitude of this microbial presence and specific pathogens such as *Porphyromonas* are histology-independent determinants of treatment outcomes.

Keywords:

Colorectal cancer, histology subtypes, pathogenic microbiomes, microbial burden, survival analysis.

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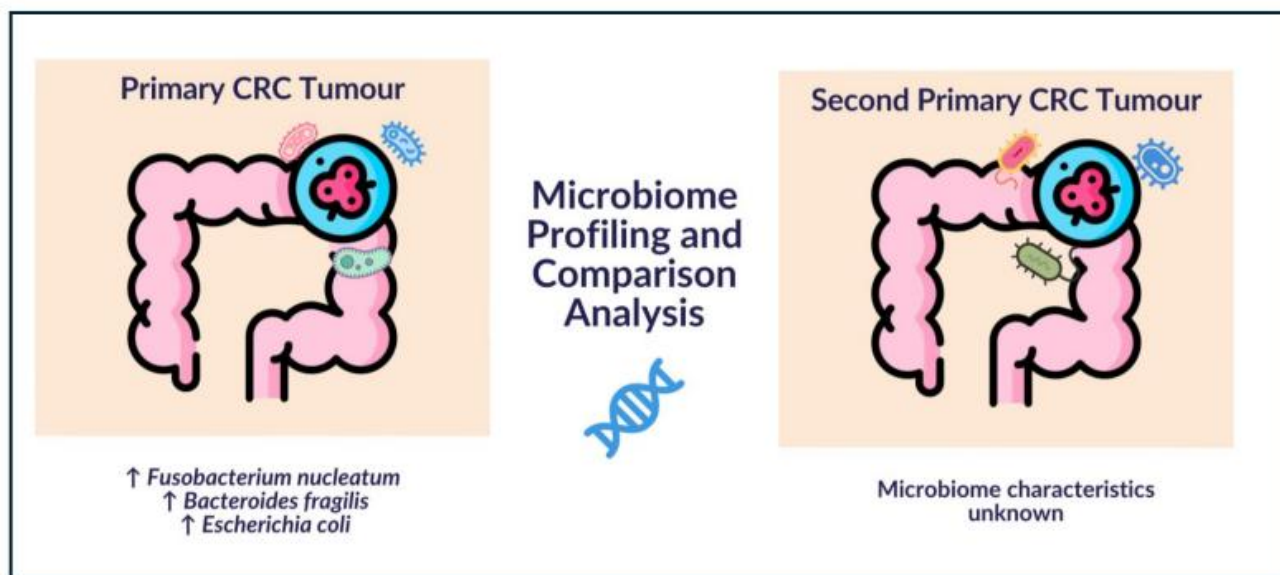
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ABSTRACT # 45

Bacterial Microbiomes Profile of the First Primary Colorectal Cancer and the Risk to Develop Second Primary Colorectal Cancer

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Graphical abstract



Introduction

Colorectal cancer remains a leading cause of global mortality and morbidity, and gut microbiomes contribute to this carcinogenesis. While colorectal cancer treatment is often successful, approximately 20% of treated colorectal cancer treated patients develop a metachronous second primary colorectal cancer. Identifying early predictive biomarkers for second primary colorectal cancer is critical, as current clinicopathological staging is limited in its ability to adequately predict these secondary events or identify high-risk individuals.

Objective

To investigate whether index intratumoral gut microbiome signatures can predict second primary colorectal cancer and enhance early clinical risk stratification.

Methods

We analysed 16S rRNA microbiome metagenomics sequencing data on 492 paired index cancers and adjacent-normal mucosa, this involved integrating microbial data with molecular features such as MSI and TMB using multivariable logistic regression and predictive analyses.

Results

Comparison of index tumours revealed that baseline *Streptococcus* relative abundance was significantly higher in patients who later developed second primary colorectal cancer ($p=0.027$). However, common colorectal

cancer pathobionts such as *Fusobacterium* and overall pathobiont burden showed no significant baseline differences between outcome groups. Furthermore, the magnitude of paired tumour-normal microbial shifts did not distinguish future cases of second primary colorectal cancer. In multivariable models, tumour *Streptococcus* and a history of prior cancer remained positive risk factors (aOR 1.8 and 2.2, respectively), whereas advanced index stage was associated with lower second-primary risk. Integrating *Streptococcus* ecological abundance improved model discrimination and precision-recall performance over clinical variables alone.

Discussion

These findings suggest that intratumoral *Streptococcus* enrichment marks a distinct biological state or field effect linked to metachronous recurrence. This signature provides independent prognostic value beyond standard markers such as MSI or TMB. This may shift the focus toward the microbial environment as a predictive biomarker of long-term colorectal cancer susceptibility, potentially refining personalized surveillance for patients otherwise deemed low-risk by traditional clinical staging.

Conclusions

Combining clinical features with baseline index tumour *Streptococcus* abundance independently predicts increased risk of metachronous second primary colorectal cancer, offering a potentially novel microbial biomarker for post-surgical colorectal cancer risk stratification and targeted surveillance.

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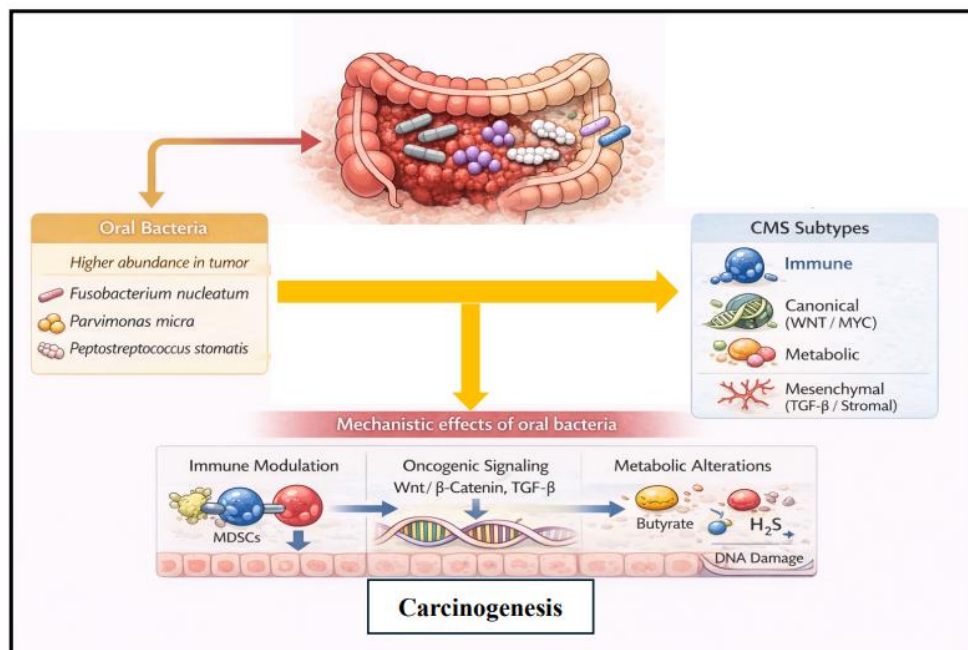
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ABSTRACT # 46

Presence of Oral Pathogenic Bacterial Microbiomes Across Different Tumor Molecular Subtypes in Colorectal Cancer

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Graphical abstract



Abstract

Introduction

Colorectal cancer remains a significant global health burden, with around 2 million annual diagnoses worldwide. While the gut microbiome influences oncogenesis, the role of oral-derived pathogenic microbiomes in specific molecular subtypes of colorectal cancer is unclear. Identifying these microbial-host interactions is critical for precision oncology.

Objectives

To determine whether oral pathogenic microbiomes preferentially align with specific colorectal cancer consensus molecular subtypes (CMS) and host traits.

Methods

We integrated whole-genome shotgun (WGS) and 16S rRNA metagenomics microbiome sequencing data from 347 colorectal tumours, and used to quantify oral-pathogen microbiomes richness and burden across transcriptomically defined CMS groups.

Results

Oral-pathogen bacterial enrichment was non-random, showing significant convergence toward

CMS1 (immune-activated/MSI-H). CMS1 tumours displayed the highest oral-pathogen microbiome burden (WGS genus panel, $p=0.03$) and species richness compared to other subtypes ($p=0.004$). Specifically, *Fusobacterium nucleatum* and *Gemella morbillorum* were significantly enriched in CMS1 ($p=0.03$ and $p=0.004$, respectively). High oral-pathogen burden correlated strongly with genomic instability, including elevated Tumour Mutational Burden (TMB) and MANTIS scores (both $p<0.001$). Furthermore, aggregate oral pathogenic bacterial microbial burden was positively associated with CMS1 probability (Spearman $R=0.25$, $p=0.0011$) and total tumour microbiome biomass (MBR). Oral pathogenic bacterial microbiomes also track with increased immune scores and neoantigen counts ($p<0.001$), suggesting a distinct ecological niche within the immune-activated tumour microenvironment.

Discussion

These findings suggest that oral-pathogen bacterial microbiome colonization is a subtype-specific hallmark of colorectal cancer. The association with CMS1 and genomic instability suggests these microbes thrive in an immunosurveillance-rich environment. Clinically, this highlights the potential for using oral-microbiome bacterial signatures as biomarkers for molecular stratification and suggests that targeting these specific oral pathogenic bacterial microbial-host axes could enhance precision therapeutic strategies by addressing the unique microbiome ecological context of CMS1 tumours.

Conclusions

Oral pathogenic bacterial microbiomes are significantly enriched in CMS1 colorectal tumours, where they correlate with genomic instability and distinct immune-activated host microenvironments.

Keywords: Colorectal cancer, oral microbiomes, pathogenic bacteria, immune microenvironments, carcinogenesis.

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Results Appendix

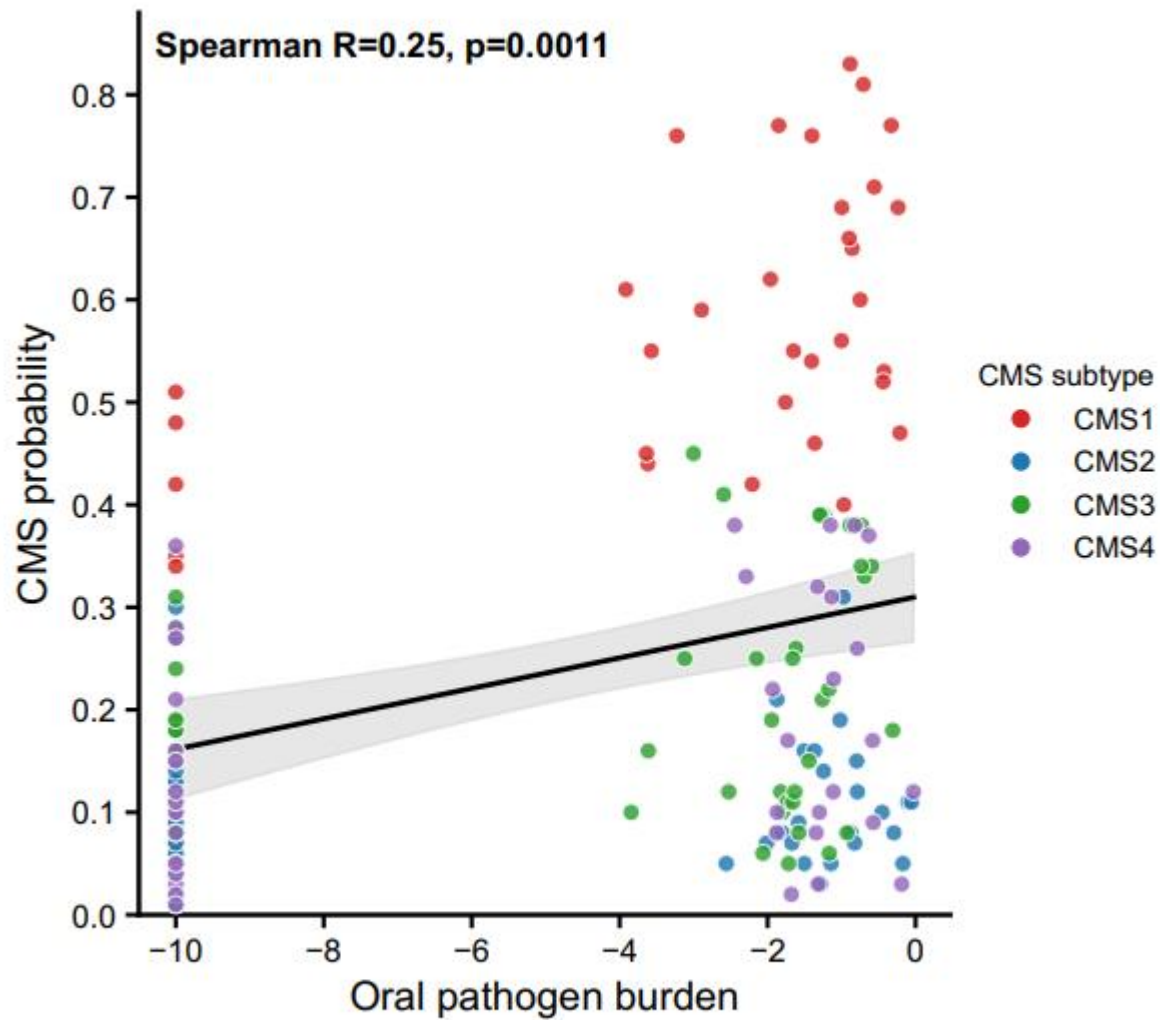


Fig 1. Scatterplot shows a significant positive association between pathogenic oral bacterial burden and CMS probability (Spearman $R=0.25$, $p=0.0011$).

Table 1. Baseline clinical, molecular, and oral-pathogen microbiome characteristics according to colorectal cancer consensus molecular subtype (CMS).

Characteristic	Overall (N=347)	CMS1 (n=63)	CMS2 (n=98)	CMS3 (n=84)	CMS4 (n=102)	P value
No. of patients	347	63	98	84	102	-
Clinical and pathologic characteristics						
Age at diagnosis -yr	68.2±11.5	68.0±14.0	68.8±10.8	68.2±12.3	67.6±9.6	0.90
Male sex -no./total (%)	181/347 (52.2)	25/63 (39.7)	53/98 (54.1)	37/84 (44.0)	66/102 (64.7)	0.005
Right-sided colon location -no./total (%)	183/347 (52.7)	52/63 (82.5)	30/98 (30.6)	52/84 (61.9)	49/102 (48.0)	<0.001
Left-sided/rectosigmoid location -no./total (%)	164/347 (47.3)	11/63 (17.5)	68/98 (69.4)	32/84 (38.1)	53/102 (52.0)	<0.001
Conventional adenocarcinoma -no./total (%)	273/347 (78.7)	40/63 (63.5)	94/98 (95.9)	55/84 (65.5)	84/102 (82.4)	<0.001
Mucinous adenocarcinoma -no./total (%)	64/347 (18.4)	22/63 (34.9)	3/98 (3.1)	25/84 (29.8)	14/102 (13.7)	<0.001
Pathologic T3-T4 stage -no./total (%)	278/347 (80.1)	54/63 (85.7)	76/98 (77.6)	53/84 (63.1)	95/102 (93.1)	<0.001
Pathologic node-positive disease (N1-N2) -no./total (%)	158/347 (45.5)	26/63 (41.3)	39/98 (39.8)	33/84 (39.3)	60/102 (58.8)	0.02
Distant metastasis (M1) -no./total (%)	39/324 (12.0)	5/61 (8.2)	8/92 (8.7)	10/78 (12.8)	16/93 (17.2)	0.24
AJCC stage III-IV -no./total (%)	171/347 (49.3)	28/63 (44.4)	43/98 (43.9)	39/84 (46.4)	61/102 (59.8)	0.09
Molecular and immune context						
MSI-H -no./total (%)	57/280 (20.4)	37/53 (69.8)	1/78 (1.3)	11/70 (15.7)	8/79 (10.1)	<0.001
Hypermutated tumours -no./total (%)	69/280 (24.6)	40/53 (75.5)	2/78 (2.6)	17/70 (24.3)	10/79 (12.7)	<0.001
ImmuneScore -median [IQR]	280.6 [-145.1-701.1]	681.2 [206.1-1,009.5]	-12.3 [-358.7-414.4]	16.8 [-259.6-381.2]	446.2 [175.4-1,005.0]	<0.001
StromalScore -median [IQR]	-199.5 [-618.3-288.6]	-1.2 [-275.1-236.6]	-414.905 [-674.815--210.807]	-732.805 [-1100.052--474.639]	558.1 [263.6-917.9]	<0.001
ESTIMATEScore -median [IQR]	113.0 [-722.0-892.9]	703.0 [-134.4-1,325.8]	-470.9 [-995.8-184.1]	-772.961 [-1238.179--187.939]	1,152.9 [524.8-1,774.2]	<0.001
MANTIS score -median [IQR]	0.270 [0.261-0.283]	0.545 [0.303-0.693]	0.267 [0.259-0.274]	0.266 [0.257-0.278]	0.267 [0.259-0.277]	<0.001
Oral-pathogen microbiome features						
Tumour mutational burden per Mb -median [IQR]	6.8 [5.0-34.4]	95.5 [47.3-119.3]	5.787 [4.581-7.462]	7.4 [5.3-31.8]	5.800 [4.562-7.500]	<0.001
Predicted neoantigen count (500 nM) -median [IQR]	10.0 [6.0-33.8]	104.0 [55.0-136.0]	7.0 [5.0-10.0]	10.0 [6.0-28.5]	8.0 [6.0-13.5]	<0.001
Oral-pathogen microbiome features						
Any oral pathogen by 16S genus panel -no./total (%)	236/245 (96.3)	46/46 (100.0)	68/69 (98.6)	60/64 (93.8)	62/66 (93.9)	0.17
Total oral-pathogen burden by 16S genus panel -median [IQR]	0.010 [0.003-0.034]	0.020 [0.008-0.045]	0.007 [0.003-0.048]	0.008 [0.001-0.028]	0.012 [0.003-0.028]	0.05
Fusobacterium detected by 16S -no./total (%)	148/245 (60.4)	35/46 (76.1)	40/69 (58.0)	37/64 (57.8)	36/66 (54.5)	0.11
Streptococcus detected by 16S -no./total (%)	220/245 (89.8)	44/46 (95.7)	63/69 (91.3)	56/64 (87.5)	57/66 (86.4)	0.38
Any oral pathogen by WGS genus panel -no./total (%)	132/166 (79.5)	31/33 (93.9)	33/47 (70.2)	38/44 (86.4)	30/42 (71.4)	0.02
Total oral-pathogen burden by WGS genus panel -median [IQR]	0.051 [0.003-0.204]	0.197 [0.039-0.363]	0.031 [0.000-0.172]	0.036 [0.003-0.104]	0.055 [0.000-0.181]	0.03
Fusobacterium detected by WGS -no./total (%)	98/166 (59.0)	24/33 (72.7)	22/47 (46.8)	31/44 (70.5)	21/42 (50.0)	0.03
Streptococcus detected by WGS -no./total (%)	83/166 (50.0)	20/33 (60.6)	21/47 (44.7)	28/44 (63.6)	14/42 (33.3)	0.02
Any oral pathogen by WGS species panel -no./total (%)	106/166 (63.9)	28/33 (84.8)	24/47 (51.1)	32/44 (72.7)	22/42 (52.4)	0.004
Total oral-pathogen burden by WGS species panel -median [IQR]	0.013 [0.000-0.073]	0.039 [0.000-0.139]	0.003 [0.000-0.065]	0.015 [0.000-0.054]	0.004 [0.000-0.066]	0.09
Fusobacterium nucleatum detected by WGS -no./total (%)	85/166 (51.2)	22/33 (66.7)	19/47 (40.4)	27/44 (61.4)	17/42 (40.5)	0.03

a Data are shown as mean±SD, median [IQR], or no./total (%), with percentages calculated among participants with available data for that variable.

b P values were calculated with one-way analysis of variance for age, Kruskal-Wallis tests for non-normally distributed continuous variables, and chi-square tests for categorical variables.

c Oral-pathogen features were restricted to available assay subsets: 16S genus panel, n=245; WGS genus/species panel, n=166.

d Relative-abundance burden variables represent the summed relative abundance across the predefined oral-pathogen panel in each platform.

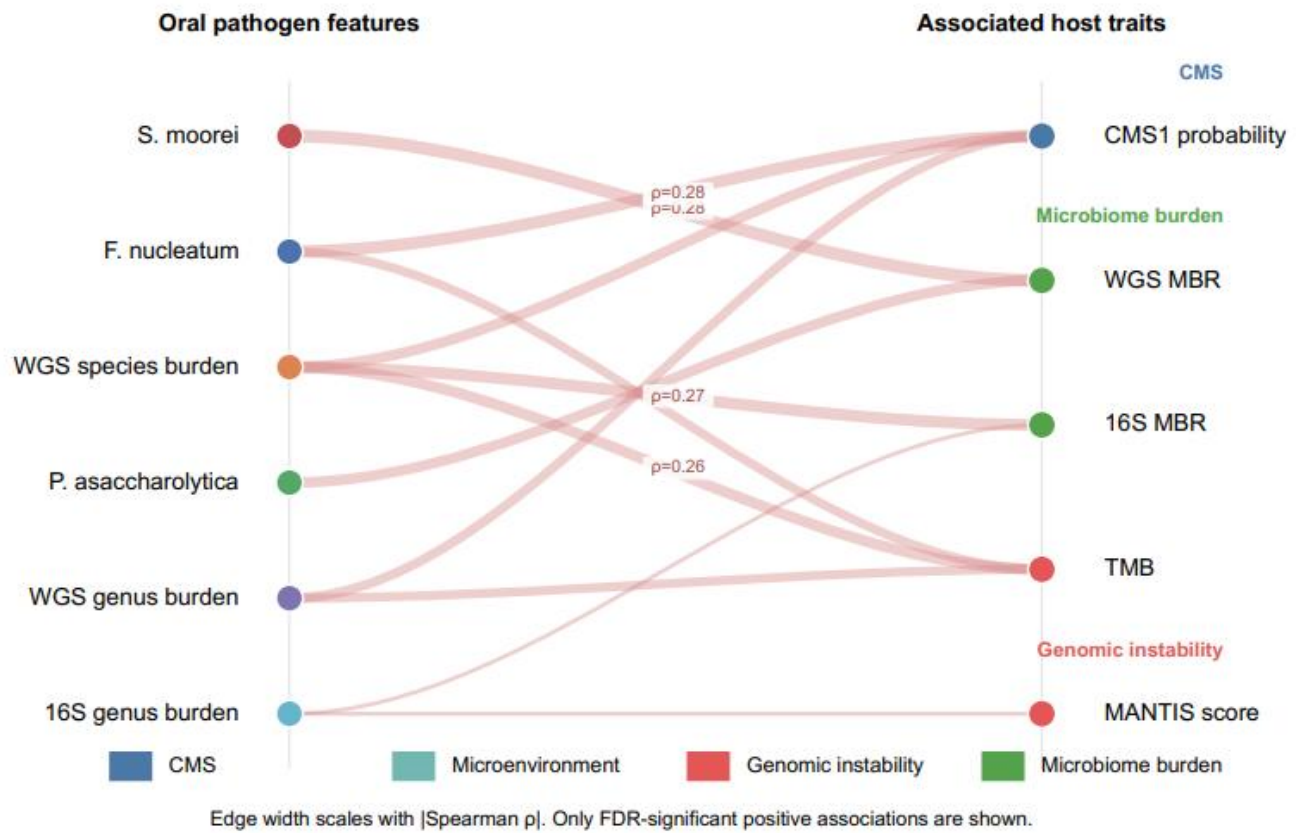


Fig 2. Trait map shows that oral pathogen features align positively with host molecular states rather than acting as isolated microbial markers.

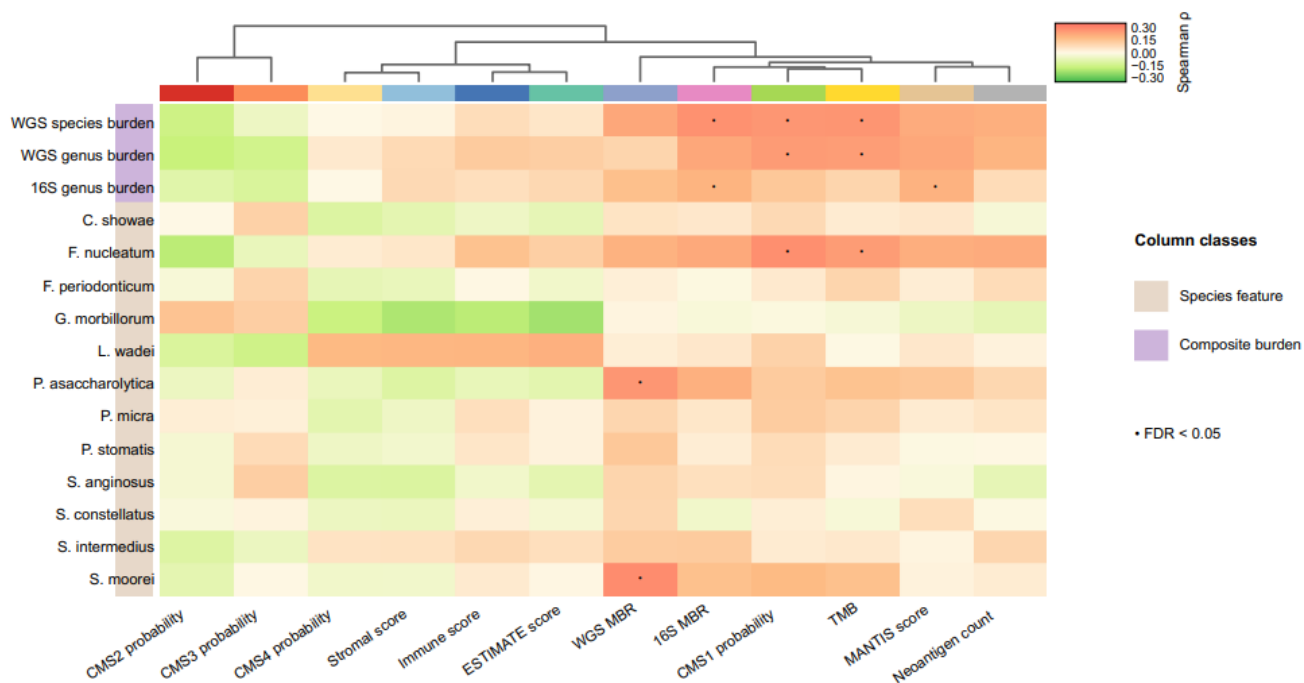
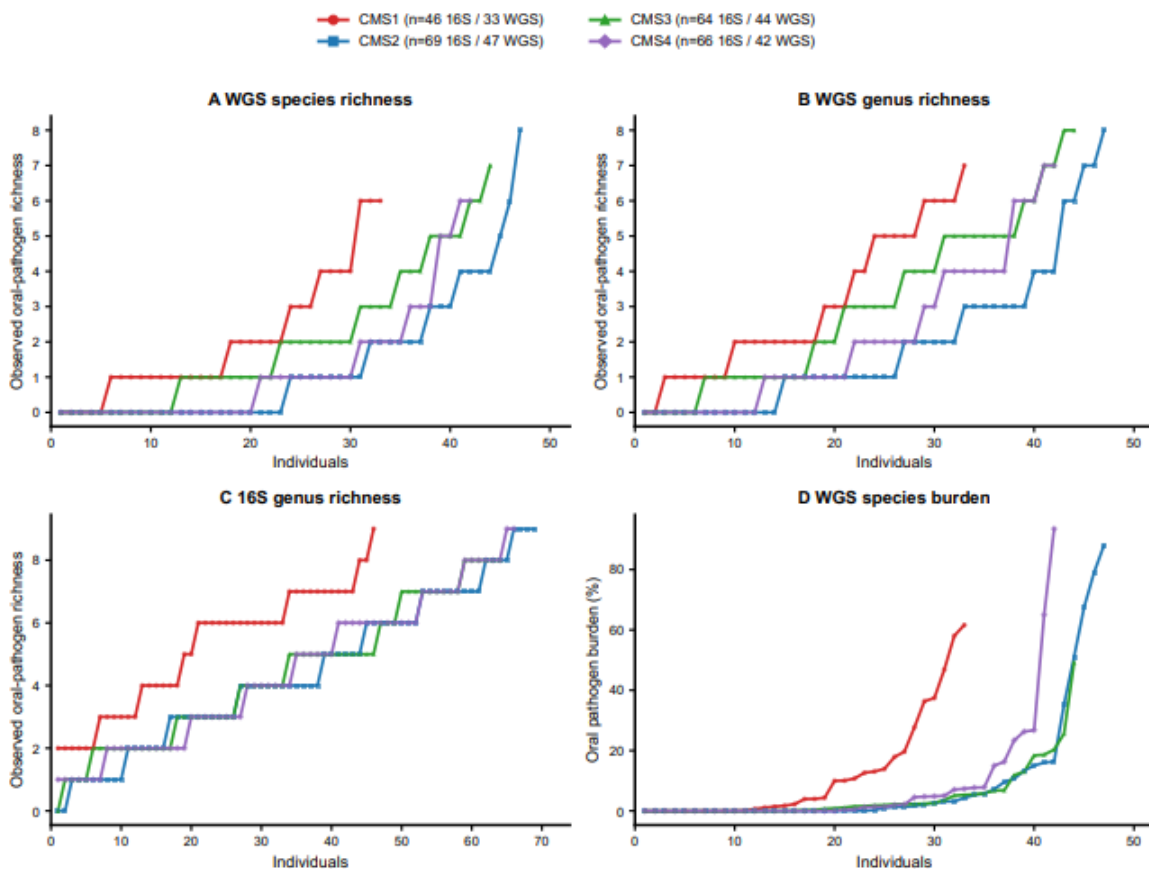


Fig 3. Clustered heatmap shows that oral pathogen burden features, especially WGS species burden, WGS genus burden, and 16S genus burden, correlated positively with CMS1 probability.



Curves show ranked individual tumors within each CMS group. Richness was defined as the number of oral-pathogen taxa detected above 1×10^{-4} relative abundance.

Fig 4. Ranked curves show that oral-pathogen ecology differs by CMS subtype, but burden and richness are not identical phenomena.

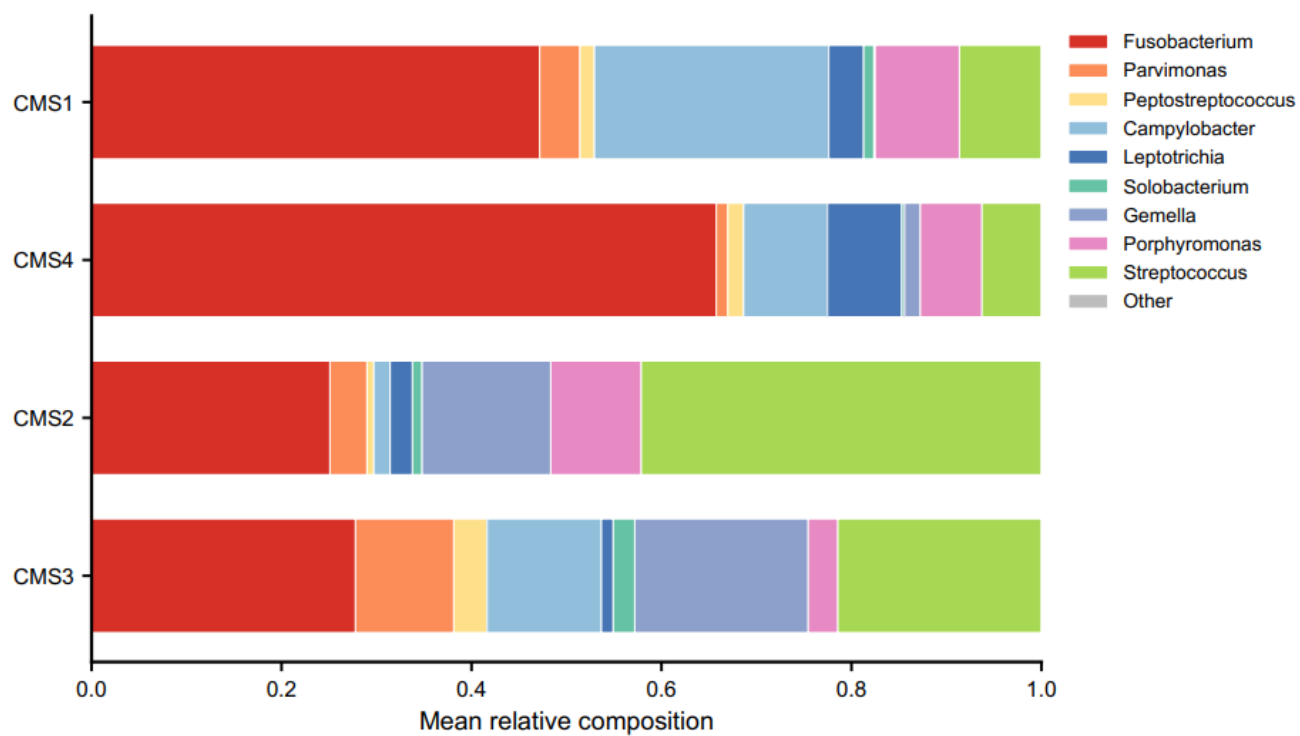


Fig 5. Stacked composition plot shows clear CMS-specific oral-pathogen architectures, CMS1 and CMS4 are dominated by Fusobacterium.

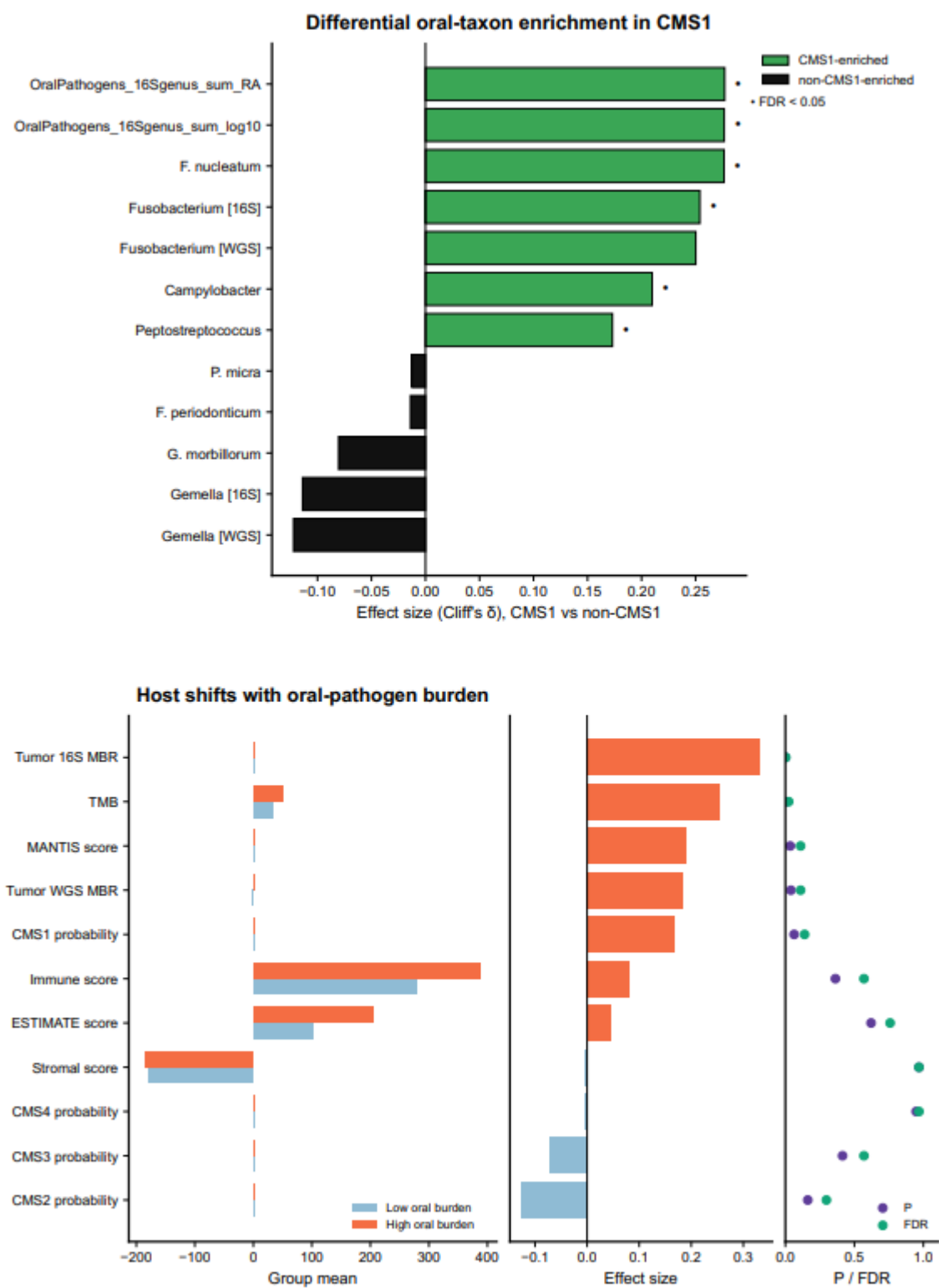


Fig 6. CMS1 is preferentially enriched for oral-pathogen burden and classic oral taxa, especially *F. nucleatum*, *Fusobacterium*, *Campylobacter*, and *Peptostreptococcus*.

Distribution of Aerobic and Anaerobic Bacterial Microbiomes in Tumour versus Normal Tissue and the Prognostic Outcomes in Colorectal Cancer

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Abstract

Introduction

The composition of gut microbiome has been linked to colorectal cancer prognosis and has necessitated deeper insights into the microbial contributions to tumorigenesis. The spatial oxygen gradient within the gut microbiome is often disrupted, yet the specific shift between aerobic and anaerobic populations remains insufficiently characterised as a prognostic biomarker for colorectal cancer.

Objectives

This study aimed to quantify intratumoral aerobic versus anaerobic gut microbiome ecological shifts and evaluate their prognostic potential.

Methods

We analysed 492 paired colorectal tumour-normal samples after 16S rRNA metagenomics sequencing. Microbiome taxa were classified by oxygen-tolerance phenotypes as aerobic or anaerobic. Statistical significance was assessed via Wilcoxon signed-rank tests and rank-biserial correlations to quantify community-level aerobic-anaerobic shifts.

Results

The total anaerobic ecological microbiome composition was higher in tumours compared to normal tissues ($p=0.002$), while the aerobic (oxygen-tolerant) fraction significantly decreased in tumours compared to normal tissue ($p<0.001$). Consequently, the anaerobe-aerobe log₁₀ ratio (AAR) increased significantly in tumour tissue compared to normal tissue ($p<0.001$), with 71.5% of pairs showing an increased anaerobic share. Notably, specific anaerobes like *Fusobacterium* ($p<0.001$) and *Parvimonas* ($p<0.001$) were enriched in tumours, alongside aerobes including *Streptococcus* ($p<0.001$) and *Haemophilus* ($p=0.004$). The receiver operation characteristic (ROC) curves analyses for the AAR yielded an area under the curve (AUC) of 0.63 ($p<0.001$), while a combined model reached an AUC of 0.66 ($p<0.001$). Furthermore, a high Δ Anaerobic fraction correlated with significantly different survival outcomes ($p=0.0117$), suggesting these dynamics influence clinical prognosis.

Discussion

These findings suggest that tumorigenesis may drive a microbial oxygenation shift, where aerobic expansion disrupts the typically anaerobic-dominant gut environment. This metabolic transition may reflect altered vascularisation or oxidative stress within the tumour niche. Clinically, the AAR may serve as a novel prognostic biomarker in colorectal cancer. Understanding these molecular shifts offers the potential for targeted therapies that manipulate the tumour aerobic-anaerobic microbiomes ecology.

Conclusions

Colorectal tumours exhibit a significant increase in the anaerobe-to-aerobe ratio. This shift may serve as a prognostic biomarker and correlates with patient survival outcomes.

Keywords: Colorectal cancer, aerobic-anaerobic microbiomes, bacterial composition, treatment outcomes, survival analysis.

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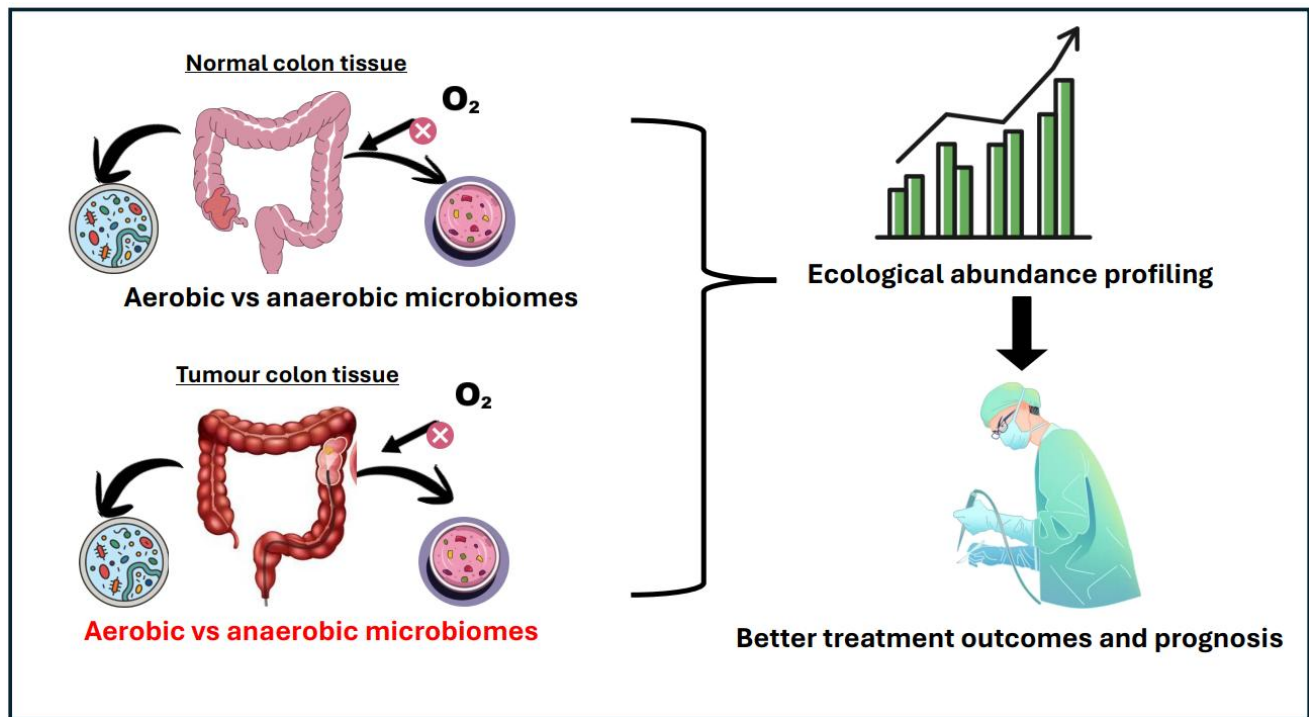


Table 1. Paired tumour versus matched normal summary of aerobic-anaerobic microbiome composition and community ecology

Characteristic	Normal (n=246)	Tumour (n=246)	Paired Δ (Tumour-Normal)	95% CI for	Effect size	% pairs with Tumour > Normal	P value	FDR q value
Anaerobic fraction (assigned taxa)	0.594 [0.520, 0.662]	0.578 [0.482, 0.654]	-0.025 [-0.080, 0.042]	-0.034 to -0.007	-0.23	39.8%	0.002	0.004
Aerobic (oxygen-tolerant) fraction (assigned taxa)	0.020 [0.004, 0.075]	0.044 [0.016, 0.117]	0.013 [-0.002, 0.050]	0.007 to 0.019	0.44	69.5%	<0.001	<0.001
Anaerobe: Aerobe log10 ratio (AAR)	1.485 [0.850, 2.200]	1.123 [0.654, 1.561]	-0.351 [-0.944, 0.047]	-0.476 to -0.221	-0.56	28.5%	<0.001	<0.001
Assigned phenotype coverage	0.648 [0.583, 0.710]	0.654 [0.592, 0.727]	0.011 [-0.038, 0.066]	-0.001 to 0.021	0.15	55.3%	0.038	0.060
Unassigned fraction (no phenotype evidence)	0.352 [0.290, 0.417]	0.346 [0.273, 0.408]	-0.011 [-0.066, 0.038]	-0.021 to 0.001	-0.15	44.7%	0.038	0.060
Anaerobe share among assigned taxa	0.968 [0.876, 0.994]	0.930 [0.818, 0.973]	-0.018 [-0.074, 0.002]	-0.026 to -0.013	-0.46	28.5%	<0.001	<0.001
Aerobe share among assigned taxa	0.032 [0.006, 0.124]	0.070 [0.027, 0.182]	0.018 [-0.002, 0.074]	0.013 to 0.026	0.46	71.5%	<0.001	<0.001
Shannon diversity	2.822 [2.543, 3.024]	2.811 [2.485, 3.059]	0.016 [-0.210, 0.192]	-0.021 to 0.054	-0.01	51.6%	0.865	0.865
Simpson diversity	0.894 [0.838, 0.917]	0.892 [0.832, 0.920]	-0.000 [-0.029, 0.025]	-0.005 to 0.005	-0.04	49.2%	0.546	0.601
Observed genera richness	60 [53, 67]	60 [54, 68]	2 [-5, 8]	0 to 3	0.14	55.3%	0.052	0.072

Values are summarized as median [IQR]. Paired differences were computed within patient (Tumour - Normal). Two-sided Wilcoxon signed-rank tests were used for paired hypothesis testing; effect sizes are rank-biserial correlations (r).

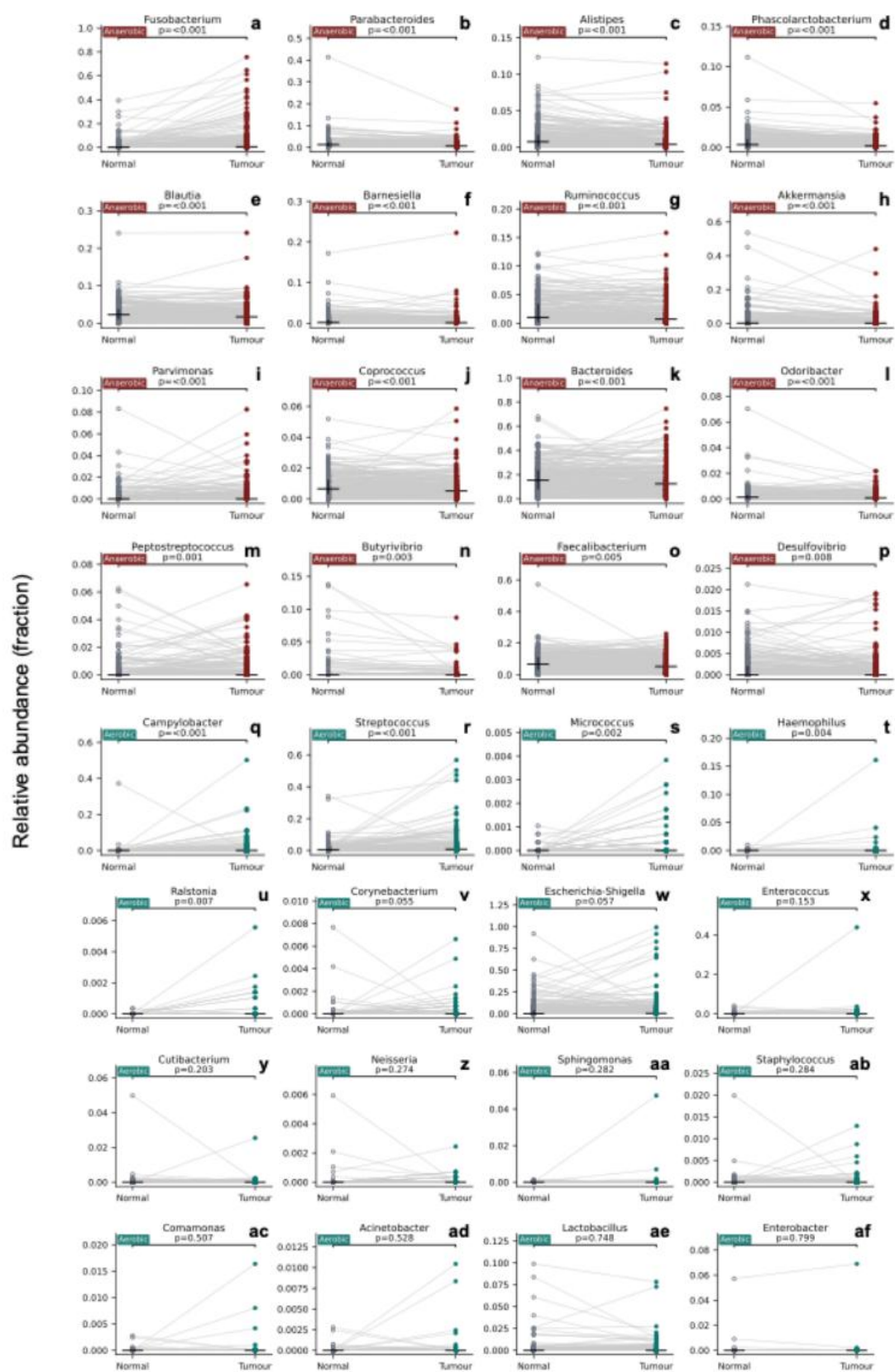


Fig 1. Multi-panel paired plot compares matched normal versus tumour relative abundance across bacterial genera. Tumours show consistent enrichment of several anaerobes

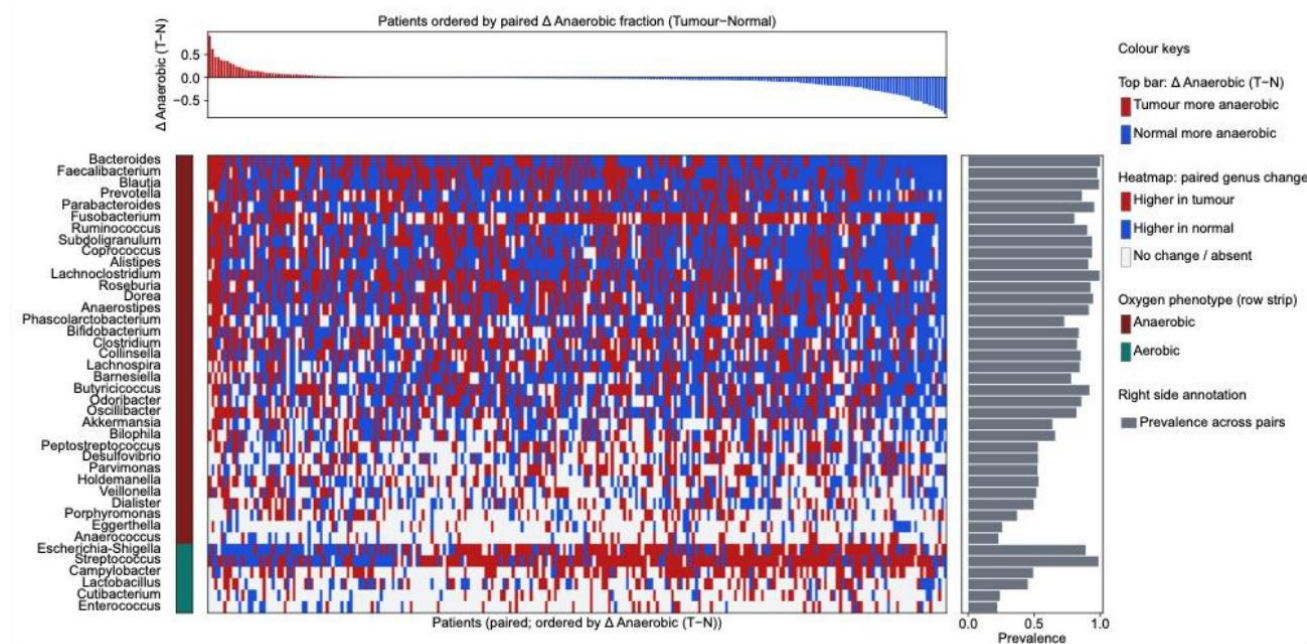


Fig 2. Ordered paired heatmap shows patient-specific genus shifts between tumour and matched normal tissue, ranked by the change in total anaerobic fraction.

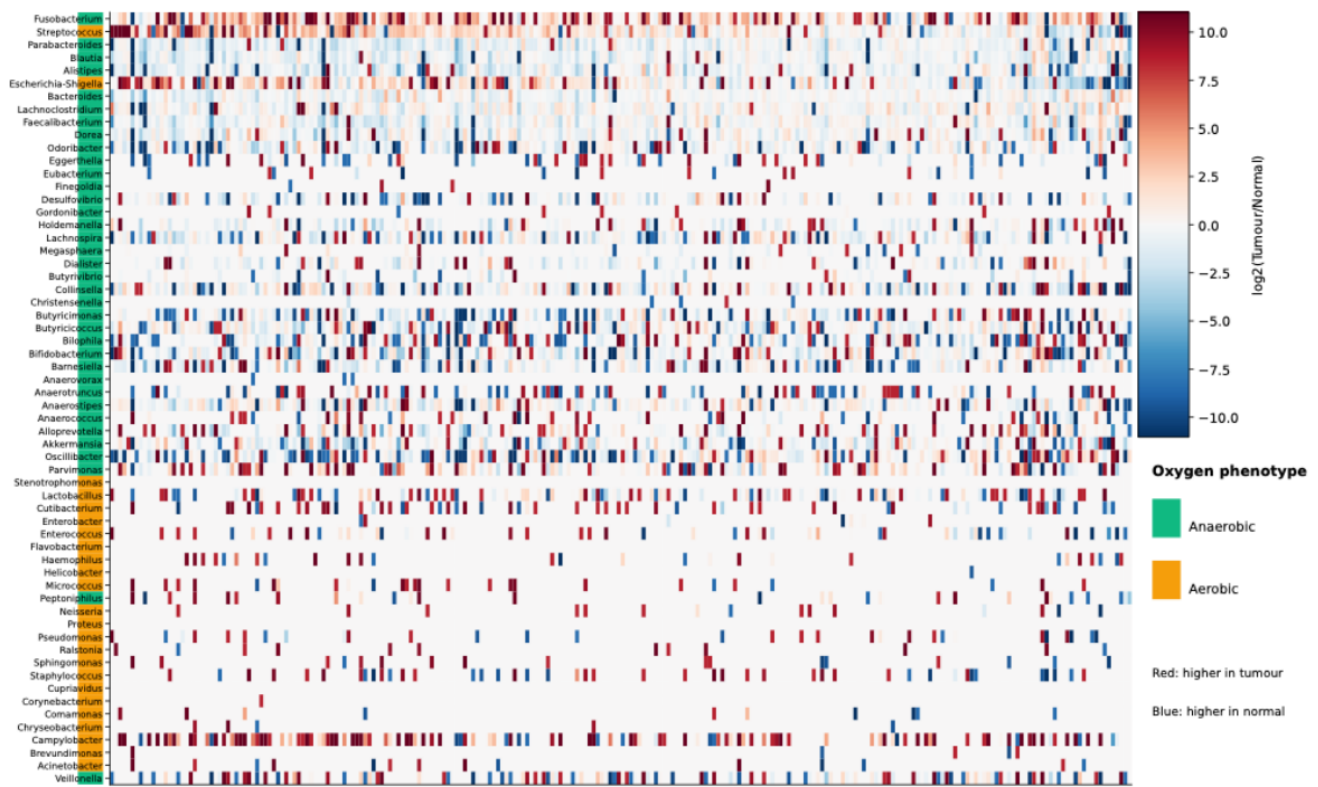


Fig 3. $\log_2(\text{tumour/normal})$ heatmap shows paired, patient-level microbial shifts across genera, with red indicating tumour enrichment and blue normal enrichment.

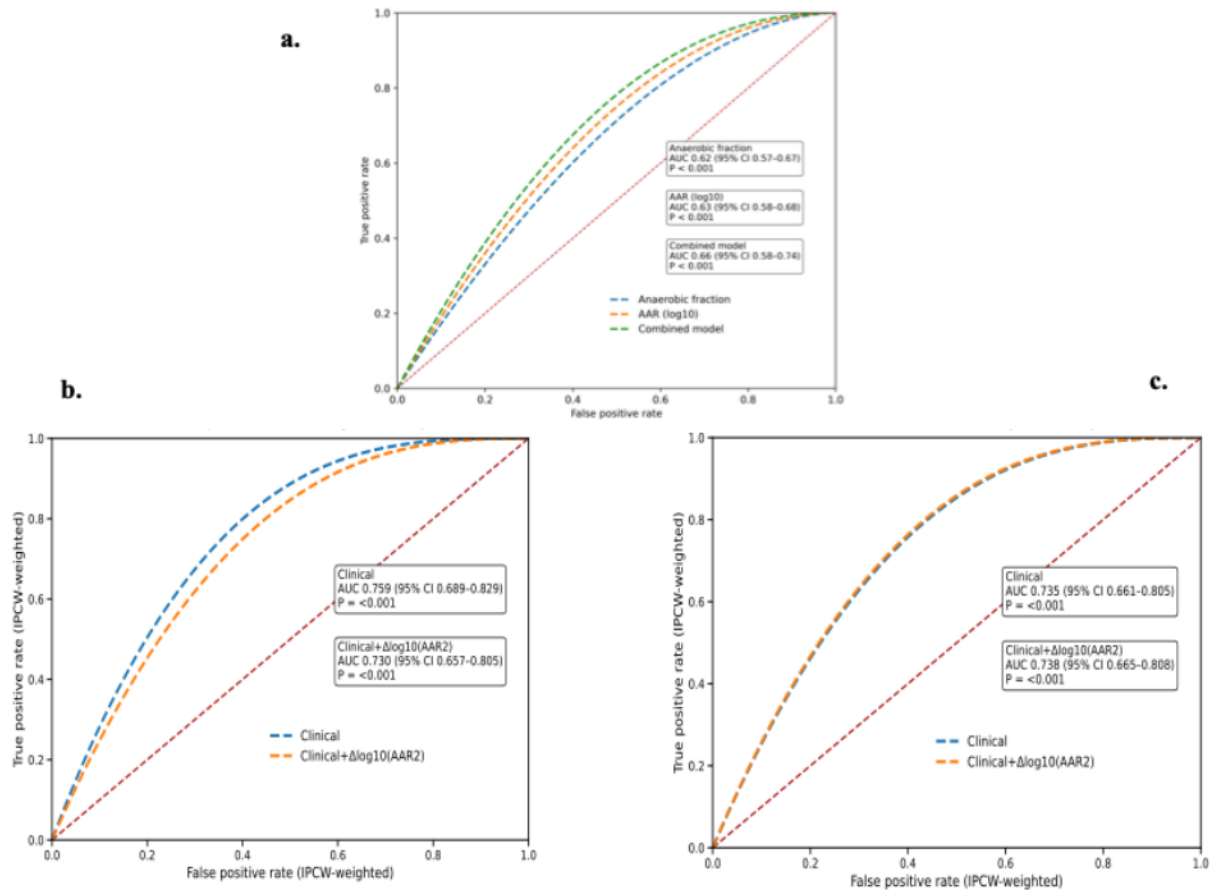


Fig 4. ROC analyses show modest but significant discriminative value for tumour-normal microbiome-derived oxygen metrics.

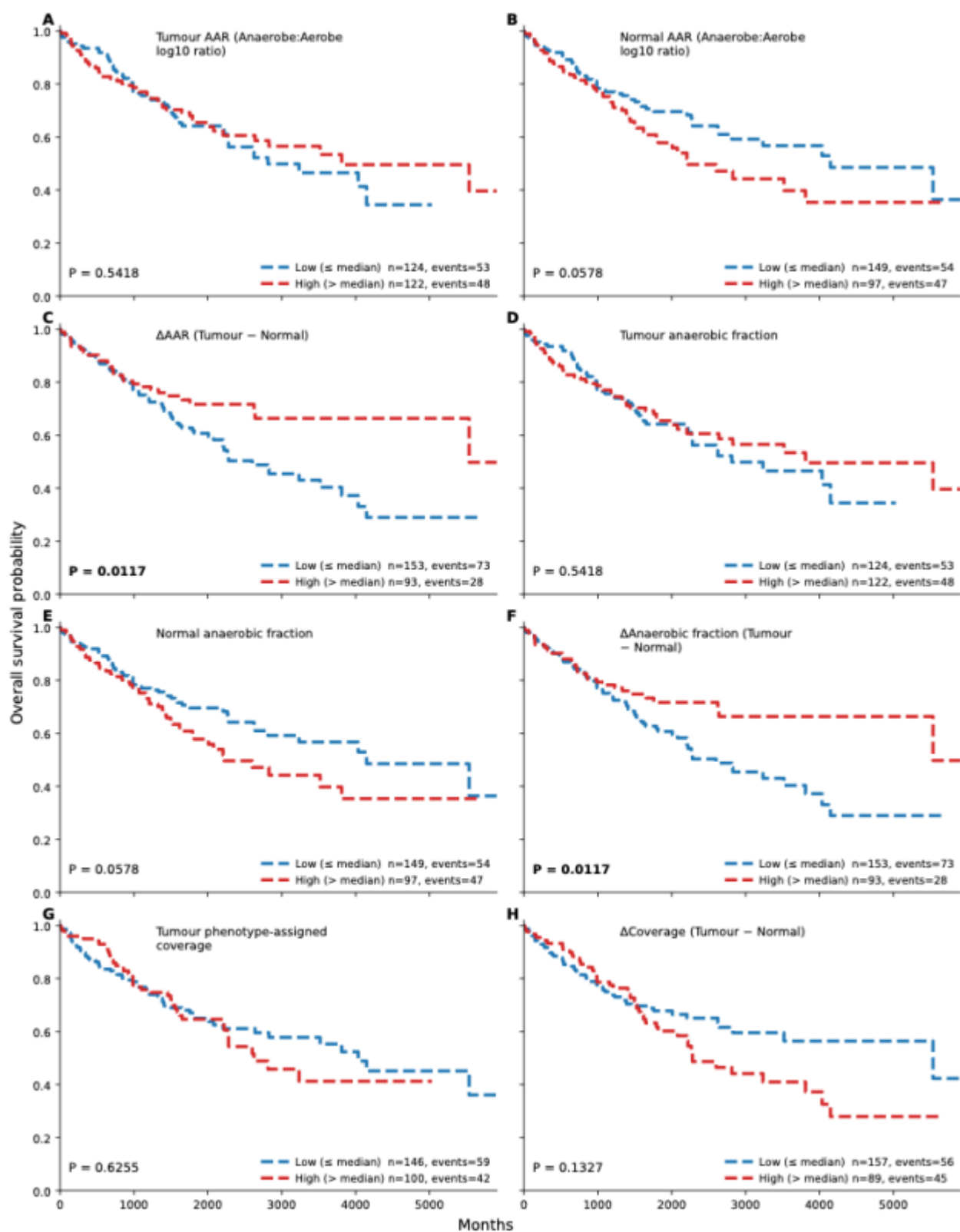


Fig 5. Kaplan-Meier analyses of paired tumour-normal shift showing both Δ AAR and Δ anaerobic fraction significantly stratify survival ($p=0.0117$).

Acute Hepatitis A Complicated by Secondary Hemophagocytic Lymphohistiocytosis: A Rare Hyperinflammatory Presentation

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1. Introduction / Background

Hepatitis A virus (HAV) infection typically follows a self-limited course, presenting as acute hepatocellular injury that resolves without intervention in the majority of immunocompetent individuals. However, in rare instances, HAV infection can precipitate severe systemic complications that carry substantial morbidity and mortality. Hemophagocytic lymphohistiocytosis (HLH) is one such complication—a life-threatening hyperinflammatory syndrome driven by uncontrolled immune activation and dysregulated cytokine release, culminating in multi-organ dysfunction if left unrecognized and untreated.

HLH is broadly classified into primary (familial/genetic) and secondary forms. Secondary HLH occurs in the context of triggering conditions such as infections, malignancies, and autoimmune diseases. Among infectious triggers, viral etiologies—including Epstein-Barr virus (EBV), cytomegalovirus (CMV), and influenza—are most frequently implicated. The association between HAV and HLH, however, remains exceptionally rare, with only isolated case reports documented in the global literature.

The clinical overlap between severe acute hepatitis and early HLH presents a diagnostic challenge, as both conditions share features such as fever, hepatosplenomegaly, and elevated liver enzymes. Extreme hyperferritinemia ($>10,000$ ng/mL), cytopenias, and hypertriglyceridemia serve as key discriminating features that should prompt early consideration of HLH. Given the high mortality associated with delayed diagnosis, heightened clinical awareness is paramount.

2. Objectives

The primary objectives of this case report are:

1. To describe a rare case of secondary HLH triggered by acute HAV infection in a young, immunocompetent adult.
2. To outline the diagnostic approach, including application of the HLH-2004 criteria in the context of viral hepatitis.
3. To emphasize the importance of early multidisciplinary management in preventing clinical deterioration and improving patient outcomes.
4. To contribute to the limited body of literature on HAV-associated HLH and raise awareness among clinicians managing severe acute hepatitis.

3. Clinical Case

3.1 Presentation and History

A 29-year-old previously healthy male presented to the emergency department with an eight-day history of persistent high-grade fever, right upper quadrant abdominal pain, nausea, and progressive jaundice accompanied by dark urine. He reported recent travel to Pakistan approximately one month prior to symptom onset. He denied alcohol consumption, use of hepatotoxic medications, herbal supplements, or intravenous drug use. There was no family history of liver disease or immunodeficiency. Physical examination was notable for marked icteric sclerae, jaundice, and hepatosplenomegaly, with no peripheral lymphadenopathy.

3.2 Diagnostic Evaluation

Initial laboratory investigations revealed severe hepatocellular injury: alanine aminotransferase (ALT) 4,868 IU/L, aspartate aminotransferase (AST) >4,000 IU/L, with significant hyperbilirubinemia (total bilirubin 164 μ mol/L; direct bilirubin 121 μ mol/L) and mildly elevated alkaline phosphatase (ALP) at 185 IU/L. These findings were consistent with acute hepatocellular hepatitis.

Markers of systemic inflammation were markedly deranged: serum ferritin was profoundly elevated at 19,751 ng/mL (>10,000 ng/mL threshold), lactate dehydrogenase (LDH) was 503 IU/L, and the lipid panel demonstrated severe hypertriglyceridemia at 10.6 mmol/L. Hematologic parameters revealed an initial white blood cell count of 5.7×10^9 /L with subsequent development of thrombocytopenia, indicative of evolving cytopenia.

Serological workup confirmed acute HAV infection with positive anti-HAV IgM antibodies. An extensive infectious screen—including EBV, CMV, HIV, hepatitis B and C—was negative. Autoimmune hepatitis panel and metabolic liver disease workup were unremarkable. Bone marrow biopsy was not performed at initial presentation given rapid clinical improvement; however, the constellation of findings met five of eight HLH-2004 diagnostic criteria:

5. Fever (persistent, high-grade)
6. Splenomegaly
7. Cytopenias (thrombocytopenia)
8. Extreme hyperferritinemia (>10,000 ng/mL)
9. Hypertriglyceridemia (10.6 mmol/L)

A diagnosis of secondary HLH triggered by acute HAV infection was established following urgent multidisciplinary consultation with hematology and hepatology.

3.3 Treatment

Following diagnosis, the patient was initiated on HLH-directed therapy. High-dose intravenous dexamethasone was commenced as the cornerstone of immunosuppressive management, targeting the underlying cytokine storm. Concurrently, supportive care was provided including correction of coagulopathy with fresh frozen plasma (FFP) and cryoprecipitate. Etoposide-based therapy (as per the HLH-94/2004 protocol) was deferred pending the initial response to steroids, given the secondary and potentially reversible nature of HLH in the context of a self-limited viral infection.

3.4 Follow-Up and Outcomes

The patient demonstrated a favorable clinical response to therapy. Fever resolved within the first week of treatment, and serial laboratory monitoring confirmed a progressive decline in ferritin levels and improvement of transaminases, triglycerides, and bilirubin. Platelet count recovered without the need for transfusion. The patient was discharged in stable condition with outpatient follow-up arranged for repeat liver

function tests and ferritin surveillance. No escalation of HLH-directed therapy was required. At follow-up, the patient remained well with no evidence of recurrence or residual hepatic dysfunction.

4. Conclusions

This case illustrates that hemophagocytic lymphohistiocytosis, while rare, represents a potentially fatal complication of acute hepatitis A infection that demands early clinical recognition and prompt multidisciplinary intervention. The overlap of severe hepatitis and systemic hyperinflammation may obscure the diagnosis of HLH if clinicians are not vigilant for discriminating features.

Key clinical take-aways from this case include:

10. Extreme hyperferritinemia (>10,000 ng/mL) in the context of acute hepatitis should prompt immediate evaluation for HLH, irrespective of the underlying aetiology.
11. The HLH-2004 diagnostic criteria provide a validated framework for early diagnosis and should be applied systematically in patients presenting with fever, organomegaly, and cytopenias.
12. Timely multidisciplinary collaboration between gastroenterology, hematology, and critical care is essential to guide appropriate therapy and prevent disease escalation.
13. HLH secondary to a self-limited viral infection may respond to corticosteroid monotherapy alone, potentially avoiding the toxicity of cytotoxic regimens, as demonstrated in this case.

This report adds to the limited literature on HAV-associated HLH and underscores the need for clinicians to consider this diagnosis in patients with atypical or severe presentations of acute viral hepatitis.

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ABSTRACT # 50

From fatty liver to fatal cancer - Metabolic associated steatohepatitis

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Keywords: Metabolic Associated Steatohepatitis, Nonalcoholic Steatohepatitis, Fatty Liver Disease, Metabolic Syndrome, Liver Fibrosis

Background:

Metabolic Associated Steatohepatitis (MASH), previously referred to as Non-Alcoholic Steatohepatitis (NASH), has emerged as a leading cause of chronic liver disease worldwide. It affects approximately 25% of the global population and is strongly associated with metabolic disorders such as obesity, insulin resistance, and type 2 diabetes mellitus. The condition is characterized by hepatic steatosis accompanied by inflammation and progressive fibrosis, which may eventually lead to cirrhosis and hepatocellular carcinoma. Due to the increasing prevalence of metabolic syndrome globally, MASH represents a growing public health concern with significant morbidity and mortality.

Objective:

This narrative review aims to provide a comprehensive overview of Metabolic Associated Steatohepatitis, focusing on its pathophysiology, risk factors, diagnostic approaches, and emerging therapeutic strategies, while highlighting its systemic complications and public health impact.

Methods:

A narrative review was conducted using literature from PubMed, Cochrane Library, and Google Scholar databases. Keywords including "Metabolic Associated Steatohepatitis," "Non-alcoholic steatohepatitis," "fatty liver," "metabolic liver disease," "liver fibrosis," "obesity-associated liver disease," and "insulin resistance" were used. Relevant studies published between 2015 and 2024 were included to summarize current evidence regarding epidemiology, diagnosis, and management.

Results:

MASH is highly prevalent among individuals with metabolic disorders, particularly type 2 diabetes and obesity. The prevalence among patients with type 2 diabetes has been reported to reach 43.6%, while obesity significantly increases the risk across both adults and children. Diagnosis typically involves a combination of liver function tests, imaging modalities, and liver biopsy, with biopsy remaining the gold standard. Non-invasive techniques such as transient elastography and magnetic resonance elastography have improved early detection and fibrosis assessment. Management primarily focuses on lifestyle modification, where a weight reduction of 7–10% can significantly improve hepatic inflammation. Pharmacologic therapies including GLP-1 receptor agonists such as liraglutide and semaglutide, as well as pioglitazone, have shown promising results. Resmetirom, a thyroid hormone receptor- β agonist, has recently become the first FDA-approved pharmacological treatment for MASH.

Conclusion:

MASH represents a significant and rapidly growing global health challenge due to its strong association with metabolic diseases and its potential progression to severe liver and systemic complications. Early detection, lifestyle modification, and emerging pharmacologic therapies are crucial to reducing disease progression and associated morbidity and mortality. Increasing awareness and continued research are essential for improving prevention and management strategies.

BRAF Mutated High-Risk Multisystem Langerhans Cell Histiocytosis in a 2-years old child

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Introduction:

Langerhans cell histiocytosis (LCH) is an inflammatory myeloid neoplasm that most commonly affects bones and skin, but it can also involve the bone marrow, liver, spleen, lungs, pituitary gland/central nervous system, and other organs. (1) LCH is a rare disease in both adults and children under 15, occurring respectively in up to 0.07 and 8.9 per million cases per year. (2) LCH most commonly presents as a painful bone lesion, with skin being the second most commonly involved organ. Systemic symptoms of fever, weight loss, diarrhea, edema, dyspnea, polydipsia, and polyuria relate to specific organ involvement. Diabetes insipidus is the most frequent endocrine manifestation in LCH. (3) Specific organs are considered high risk including the liver, spleen, and hematopoietic system, when involved at disease presentation. Involvement of every organ except kidney and gonads has been described. The most frequent site of LCH in children is a lytic lesion of the skull vault.(4)

Case report:

A 2-year-old girl initially presented in December 2025 while on vacation in Thailand with seborrheic dermatitis-like scalp lesions, a maculopapular rash involving the hands and lower extremities, central diabetes insipidus (DI), and perioral candidiasis. Skin biopsy findings were consistent with multisystem Langerhans Cell Histiocytosis (LCH). Further evaluation demonstrated multiorgan involvement including the liver, bone, spleen, lymph nodes, and hypothalamic-pituitary axis.

A liver biopsy confirmed hepatic involvement by LCH, and repeated skin biopsy from the hand demonstrated BRAF mutation positivity. The patient later was referred to our centre for further multidisciplinary evaluation and management.

At presentation, the patient appeared alert and well perfused. She was not clinically jaundiced but appeared mildly pale. Nutritional status was poor, with reduced subcutaneous fat and muscle bulk. Marked scratch marks were noted over the skin, consistent with pruritus. There were no spider nevi or bruises. Oral examination revealed good oral hygiene without oral ulceration. Chest examination demonstrated good bilateral air entry without respiratory distress. Cardiovascular examination revealed normal heart sounds without murmurs. Abdominal examination showed mild distension with hepatomegaly approximately 4 cm below the right costal margin.

Laboratory investigations demonstrated persistent liver dysfunction. Erythrocyte sedimentation rate was 17 mm/hr. Coagulation studies showed mildly prolonged partial thromboplastin time (45.9 seconds). Liver enzymes were significantly elevated, with AST up to 382 U/L, ALT up to 254 U/L, and GGT up to 372 U/L. Direct hyperbilirubinemia and hypoalbuminemia were present. Hemoglobin was 10.9 g/dL with low MCV and MCH, consistent with iron deficiency anemia. Dyslipidemia with elevated cholesterol and triglycerides was also noted. Antithrombin III was elevated, likely secondary to liver dysfunction.

Imaging studies demonstrated extensive multisystem disease involvement. Maxillofacial CT revealed patchy bony destruction involving the bilateral mandible and maxilla with alveolar involvement. MRI of the brain demonstrated abnormal signals in the cranial diploic space and clivus suggestive of skull involvement as well as pituitary involvement causing central diabetes insipidus.

Abdominal ultrasound demonstrated hepatomegaly, splenomegaly, and features of diffuse liver disease. Liver elastography revealed a markedly elevated stiffness measurement of 16.9 kPa (normal <5.5 kPa in children), indicating severe fibrosis or early cirrhosis. Chest CT demonstrated bilateral axillary lymphadenopathy and interval worsening of bronchovascular bundle thickening. Multiple enlarged mediastinal, hepatic portal, mesenteric, and cervical lymph nodes were identified on imaging. Temporal bone CT identified external auditory canal lesions, and audiometry demonstrated left-sided middle ear dysfunction. Vascular imaging revealed a chronic catheter-related thrombus in the right internal jugular vein. Echocardiography identified an incidental atrial septal defect with mild tricuspid regurgitation.

Initial BRAF testing in Thailand was negative, but subsequent next-generation sequencing performed in China confirmed a BRAF mutation, supporting the diagnosis of MAPK pathway-driven multisystem disease. Histopathological examination of the scalp biopsy confirmed LCH with immunohistochemistry positive for CD1a and Langerin, Ki-67 proliferation index of 10%, focal S100 positivity, and BRAF staining in the scalp lesion specimen.

The patient was diagnosed with high-risk multisystem BRAF-mutated LCH with severe hepatic disease and evolving cirrhosis, central diabetes insipidus, osseous lesions, lymph node involvement, splenomegaly, hearing impairment, iron deficiency anemia, dyslipidemia, and catheter-related thrombosis.

Initial therapy with vinblastine and prednisolone was initiated but discontinued after two doses due to worsening liver enzyme levels. Escalation to high-dose cytarabine was subsequently attempted; however, it similarly resulted in further deterioration of hepatic function. Tremetinib, which is a MEK inhibitor, was initiated. Despite partial improvement in some lesions following trametinib therapy, progressive hepatic dysfunction remained the predominant clinical concern. Supportive management included optimization of ursodeoxycholic acid dosing (20–30 mg/kg/day divided doses), supplementation with fat-soluble vitamins (DEKAs), and nutritional support using medium-chain triglyceride-based formula. Referral to a dietitian and repeat FibroScan monitoring were recommended. Given the presence of hepatomegaly, hypoalbuminemia, cholestasis, and significant fibrosis suggestive of evolving cirrhosis, referral to a liver transplant center for further assessment and possible future transplant evaluation was advised.

Current management considerations include reassessment and optimization of targeted therapy, including dose escalation of trametinib or addition of a BRAF inhibitor therapy such as vemurafenib or dabrafenib, with consideration of combination BRAF/MEK inhibition in view of persistent high-risk disease activity and progressive liver involvement.

Discussion

Workup for Langerhans cell histiocytosis involves complete comprehensive history and physical examination. Our case initially presented with seborrheic dermatitis-like scalp lesions, maculopapular rash over the hands and lower extremities, central diabetes insipidus, and perioral candidiasis. A history of excessive thirst and urination points towards diabetes insipidus, caused by damage to the antidiuretic hormone-secreting cells of the posterior pituitary. (5) Blood tests to be done include complete blood count with leukocyte differential and platelet count, liver function tests (e.g., bilirubin, albumin, aspartate aminotransferase, alanine aminotransferase, gamma glutamyl transferase, and prothrombin time or international normalized ratio (INR)/partial thromboplastin time in patients with hepatomegaly, jaundice, elevations of liver enzymes, or low albumin), and serum electrolytes.(6) In our patient, laboratory evaluation demonstrated persistent liver dysfunction with markedly elevated AST, ALT, and GGT levels, hyperbilirubinemia, hypoalbuminemia, prolonged partial thromboplastin time, and dyslipidemia. Hemoglobin was reduced with low MCV and MCH, consistent with iron deficiency anemia.

For children with LCH in the lung, pulmonary function testing and chest CT scans are sensitive methods for detecting disease progression. (7) Imaging modalities helpful includes CT, MRI and PET scans. PET scans often

demonstrate lesions not found by other modalities and are becoming more widely used because of superior diagnostic index.(8) MRI findings in patients with diabetes insipidus include thickening and nodularity of the pituitary stalk with loss of the posterior pituitary bright spot, reflecting absence of antidiuretic hormone. (9) In our case, imaging demonstrated extensive multisystem disease including bilateral axillary, mediastinal, mesenteric, cervical, and hepatic portal lymphadenopathy; hepatomegaly and splenomegaly with coarse hepatic echotexture. There was severe liver fibrosis on elastography with liver stiffness measurement of 16.9 kPa, suggestive of advanced fibrosis or evolving cirrhosis. Craniofacial and skull involvement with patchy bony destruction involving the bilateral mandible and maxilla, abnormal cranial diploic and clival signals was also noted on MRI. Pituitary MRI demonstrated hypothalamic-pituitary involvement causing central diabetes insipidus. The patient additionally developed complications including severe liver dysfunction with cirrhosis, iron deficiency anemia, dyslipidemia, catheter-related internal jugular vein thrombosis, and mild hearing impairment secondary to temporal bone involvement.

The diagnosis of LCH is based on histological and immunohistochemical examination of affected tissue. The main feature is the identification of the characteristic LCH cells. Additionally, positive staining of the lesional cells with CD1a and/or Langerin (CD207) is mandatory for definitive diagnosis. (10, 11) Lytic bone lesions, skin, and lymph nodes are the sites most frequently biopsied for diagnosis of LCH. Histopathological examination of the scalp biopsy in our case confirmed LCH with immunohistochemistry positive for CD1a and Langerin, Ki-67 proliferation index of 10%, focal S100 positivity, and BRAF staining in the scalp lesion specimen.

A bone marrow aspirate and biopsy is indicated for patients with multisystem disease who have unexplained anemia or thrombocytopenia, which was advised for our patient. The biopsy specimens should be stained with anti-CD1a and/or anti-CD207 (langerin) and anti-CD163 immunostains to facilitate the detection of LCH cells.(11)

Genetic profiling has demonstrated several genomic mitogen-activated protein kinase (MAPK) alterations in LCH, all of which uniformly result in extracellular-signal regulated kinase (ERK) activation. Mitogen-activated protein kinase (MAPK) pathway activation is a key driver of this neoplastic disorder. (12) Modern classification of the histiocytic diseases subdivides them into dendritic cell related, monocyte/macrophage-related, or true malignancies. (13, 14) The BRAFV600E mutation, which is present in more than half of LCH cases in children, but less common in adult LCH, is associated with more severe disease phenotype and an increased risk for relapsing course in patients with multisystem LCH. (15) Although assessment of the RAS-RAF-MEK pathway is not a mandatory requirement of the workup for patients with LCH, the BRAF variant can be assessed for. While initial BRAF testing of our case in Thailand was negative, subsequent next-generation sequencing performed in China confirmed a BRAF mutation, supporting the diagnosis of MAPK pathway-driven multisystem disease.

Treatment of LCH depends on whether the disease is considered low risk or high risk, as well as the location of the LCH cells in the body. Based on the number of body systems involved, LCH is classified as either single-system disease or multisystem disease. In single-system LCH, the disease affects one organ or body system. Multisystem LCH is less common than single-system LCH, where two or more organs or body systems are affected, and in some cases the disease may spread throughout the body. (16)

Our patient had confirmed BRAF-mutated multisystem LCH with risk-organ involvement, particularly severe hepatic disease. The standard front-line therapy for patients with MS-LCH should consist of a 6-12 weeks of oral steroids and weekly vinblastine injections, followed by pulses of prednisolone/vinblastine every 3 weeks, for a total treatment duration of 12 months. This is to be followed by a continuation therapy of prednisolone (day 1–5)/ vinblastine (day 1) pulses given every 3 weeks for a total treatment duration of 12 months.(17-19) In our case, initial therapy with vinblastine and prednisolone was discontinued because of worsening liver enzymes after two doses, and escalation to high-dose cytarabine similarly resulted in further hepatic

deterioration. A standard of care for patients who fail front-line therapy with suboptimal response, disease progression or relapse, but the disease is not life-threatening, remains unestablished.

But in our case, following confirmation of BRAF mutation positivity, therapy with trametinib (MEK inhibitor) at a dose of 0.25 mg daily was initiated. Although partial improvement was observed in some lesions, including temporal bone and external auditory canal involvement, progressive hepatic dysfunction persisted despite treatment. This highlights the therapeutic challenges associated with refractory high-risk multisystem LCH and severe liver involvement. Given the progressive fibrosis and suboptimal hepatic response, reassessment of the therapy strategy with possible dose optimization or addition of BRAF inhibitor therapy such as vemurafenib or dabrafenib may be required. There are reported cases with a successful outcome with this approach. (20-22) The overall response to therapy is influenced by the duration and intensity of treatment. Response to therapy at 6 to 12 weeks has been shown to be a more important prognostic factor than age.(19)

Survival is closely linked to the extent of disease at presentation when high-risk organs are involved, For many years, lungs were thought to be high-risk organs, but isolated lung involvement in pediatric LCH is no longer considered to pose a significant risk of death (23) In this case, severe liver involvement represented the predominant and most urgent clinical issue. The patient demonstrated biochemical and elastographic evidence of evolving fibrosis/cirrhosis, with hepatomegaly, hypoalbuminemia and cholestasis. Management therefore required close multidisciplinary collaboration involving oncology, hepatology, endocrinology, ENT, cardiology, nutrition, and transplant services. Supportive management included optimization of ursodeoxycholic acid dosing, supplementation with fat-soluble vitamins, and nutritional support using medium-chain triglyceride-based formula. Referral to a liver transplant center was also recommended given the severity of hepatic disease and ongoing progression despite targeted therapy.

A significant proportion of patients who survive LCH experience disease relapses and/or develop permanent conditions. Central diabetes insipidus is the most common condition, and CNS neurodegenerative LCH is the most severe condition.(24)

Conclusion:

This case highlights the aggressive and complex nature of high-risk multisystem Langerhans Cell Histiocytosis in a young child with BRAF-mutated disease and severe hepatic involvement. The patient demonstrated extensive multisystem manifestations including liver, bone, lymph node, spleen, and hypothalamic-pituitary axis involvement, complicated by central diabetes insipidus, fibrosis/cirrhosis, thrombosis, dyslipidemia, hearing impairment, and malnutrition. Despite initiation of therapy with trametinib and partial response in selected lesions, progressive liver dysfunction remained the major clinical challenge and prognostic concern. This case emphasizes the importance of early recognition of risk-organ involvement, comprehensive molecular evaluation including BRAF mutation analysis, and prompt multidisciplinary management. It also underscores the limitations of conventional chemotherapy in selected patients with significant hepatic dysfunction and the evolving role of targeted therapy in refractory multisystem LCH. Close monitoring of treatment response, optimization of targeted therapy strategies, and timely hepatology and transplant referral are essential in improving outcomes for patients with advanced hepatic disease. Further studies are needed to establish optimal targeted treatment approaches and long-term management strategies for children with high-risk BRAF-mutated multisystem LCH.

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Adult Annular Pancreas Presenting as Gastric Outlet Obstruction Following Semaglutide Initiation: A Case Report

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Introduction / Background

Adult annular pancreas is a rare congenital anomaly in which pancreatic tissue encircles the duodenum, often remaining clinically silent until adulthood. With the widespread and growing use of glucagon-like peptide-1 receptor agonists (GLP-1 RAs) such as semaglutide, which delay gastric emptying, upper gastrointestinal symptoms in these patients are frequently misattributed to drug related effects. This diagnostic pitfall risks masking an underlying fixed mechanical obstruction and delaying appropriate surgical intervention.

Objective

To report a novel presentation of adult annular pancreas unmasked by semaglutide initiation, and to highlight the importance of systematic multimodal evaluation including cross sectional imaging, endoscopic assessment, and intraoperative frozen section in distinguishing structural from pharmacological causes of gastric outlet obstruction, thereby avoiding unnecessary radical surgery.

Clinical Case

A 55 year old man with type 2 diabetes mellitus presented with a three-month history of progressive postprandial vomiting and 9 kg of unintentional weight loss following semaglutide initiation. Symptoms persisted after discontinuation, arguing against drug induced gastroparesis as the primary aetiology. Tumour markers (CA 19-9, CEA) were normal. CT and MRI demonstrated a partial annular pancreas causing extrinsic stenosis of the second part of the duodenum, with a pancreas divisum variant and mild common bile duct dilatation, without a discrete mass. MRCP excluded obstructing biliary or ductal pathology, and endoscopic ultrasound with biopsy revealed chronic duodenitis only.

Exploratory laparotomy confirmed a fibrotic duodenal stricture encircled by pancreatic tissue with marked proximal dilatation. Intraoperative frozen section excluded malignancy, avoiding unnecessary pancreaticoduodenectomy. The patient underwent Roux en Y gastrojejunostomy and concurrent cholecystectomy for symptomatic cholelithiasis. Histopathology confirmed benign pancreatic acinar tissue with chronic fibrosis, without atypia or malignancy. Recovery was uncomplicated, vomiting resolved completely, and progressive nutritional recovery was documented at follow up.

Conclusion

When a patient on a GLP-1 receptor agonist develops severe gastrointestinal symptoms, the instinct is to blame the drug. This case challenges that instinct. Symptom persistence after semaglutide discontinuation was the critical red flag that uncovered a congenital anomaly silent for five decades. Structural pathology must be excluded before symptoms are attributed to pharmacological effects alone. As GLP-1 receptor agonist use grows globally, so does the risk of missing the structural diagnosis hiding behind the drug.

ABSTRACT # 56

Evaluation of the appropriateness and safety of Guselkumab use in SSMC (MUE)

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1. Introduction / Background

Isolated hepatic tuberculosis is a rare manifestation of extrapulmonary tuberculosis and may closely mimic primary hepatic malignancy because of its nonspecific clinical presentation and radiological findings. In patients with chronic hepatitis B infection, a focal liver lesion often raises strong suspicion for hepatocellular carcinoma (HCC), making accurate diagnosis particularly challenging.

2. Objectives

To highlight the importance of considering isolated hepatic tuberculosis in the differential diagnosis of hepatic masses and to demonstrate the role of diagnostic laparoscopy with histopathological and microbiological confirmation in establishing the correct diagnosis and guiding appropriate management.

3. Clinical Case – Including Diagnostic Evaluation, Treatment and Follow-up

A 52-year-old man with chronic hepatitis B infection and type 2 diabetes mellitus presented with fatigue, headache, and an unintentional 5–6 kg weight loss. Liver function tests were elevated, while serum alpha-fetoprotein remained within the normal range. Contrast-enhanced abdominal computed tomography demonstrated an ill-defined 6 × 3 × 4 cm lesion in the left hepatic lobe with internal calcifications and biliary compression, raising suspicion for hepatocellular carcinoma. Diagnostic laparoscopy was performed for tissue diagnosis. Intraoperatively, a localized hepatic lesion corresponding to the imaging findings was identified and biopsied. Histopathological examination demonstrated necrotizing granulomatous inflammation with epithelioid histiocytes and Langhans-type giant cells. Ziehl–Neelsen staining was positive for acid-fast bacilli, confirming isolated hepatic tuberculosis. The patient completed six months of standard anti-tubercular therapy. At follow-up, he was asymptomatic, had gained approximately 5 kg in body weight, and repeat abdominal ultrasonography and chest radiography demonstrated complete radiological resolution.

4. Conclusion(s)

This case highlights the importance of considering isolated hepatic tuberculosis in the differential diagnosis of hepatic masses, even in patients with established risk factors for hepatocellular carcinoma. Diagnostic laparoscopy with histopathological and microbiological evaluation enabled an accurate diagnosis, facilitated appropriate medical treatment, and avoided mismanagement of a lesion initially suspected to be malignant.

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Hepatocellular Carcinoma Cell Reprogramming: Exploring the Role of Lineage-Specific Transcription Regulators

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Abstract— Hepatocellular carcinoma (HCC) is the most common primary liver malignancy associated with chronic liver disease and cirrhosis. Despite advances in treatment, the prognosis for HCC remains poor. Emerging evidence emphasizes how disturbed transcriptional networks contribute to tumorigenesis, especially when lineage-specific transcription factors (LSTFs) of cell identity and differentiation are dysregulated. The study proposes that fetal liver-specific master TFs are upregulated during liver regeneration and tumorigenesis, while mature liver-specific master TFs are downregulated. This antagonistic relationship disrupts cell identity, promoting proliferation and tumorigenesis. The study aims to investigate the interplay between LSTFs of cell identity by reintroducing downregulated LSTFs in HCC cell lines, with a focus on their potential as therapeutic targets to reprogram cancer cells into a differentiated, nonproliferative state. The findings of this research could provide critical mechanical insights into the role of LSTFs in hepatocellular carcinoma, offering a potential avenue for the treatment of this cancer. **Keywords-** Hepatocellular carcinoma; transcription factors; overexpression; differentiation

I. INTRODUCTION Hepatocellular carcinoma (HCC) is the most common primary liver malignancy associated with chronic liver disease and cirrhosis. Despite advances in treatment, the prognosis for HCC remains poor due to late-stage diagnosis and limited therapeutic options. Emerging evidence emphasizes how disturbed transcriptional networks contribute to tumorigenesis, especially when master transcription factors (TFs) of cell identity and differentiation are dysregulated. The study proposes that fetal liver specific master TFs are upregulated during liver regeneration and tumorigenesis, while mature liver specific master TFs are downregulated. This antagonistic relationship disrupts cell identity, promoting proliferation and tumorigenesis. We aim to investigate the interplay between master regulators of cell identity with a focus on their potential as therapeutic targets to reprogram cancer cells into a differentiated, non-proliferative state. This study aims to reintroduce downregulated LSTFs and evaluate their potential in reprogramming HCC cells. Furthermore, the study aims to elucidate the molecular mechanisms underlying HCC reprogramming mediated by identified master regulators of hepatic lineage, with a focus on their therapeutic implications.

II. MATERIALS AND METHODS

A. Cell line and culture conditions: Hepatocellular carcinoma (HCC) cell lines HepG2, Huh7, and Hep3B were cultured based on ATCC guidelines for each respective cell line under standard conditions (37°C, 5% CO₂). Cell lines were authenticated and tested for mycoplasma contamination before use. Maintaining the Integrity of the Specifications.

B. Lentiviral-mediated transduction: Lentiviral vectors were designed to overexpress the transcription factors (TFs). Lentiviral particles were produced in HEK293T cells using a third-generation packaging system with the packaging plasmid psPAX2 and pMD2.G. Supernatants containing lentiviral particles were harvested, filtered (0.45 µm), and used for transduction. HCC cells were transduced with lentiviral particles in the presence of 8 µg/mL polybrene. After 48 hours, transduced cells were selected using puromycin to establish stable cell lines.

C. Phenotypic and morphological analysis: Phenotypic changes in transduced HCC cells were observed. Changes in cell morphology, including alterations in size, shape, and clustering behaviour, were documented and compared to untransduced controls.

D. Immunocytochemistry and microscopy: Cells were fixed in 4% paraformaldehyde, permeabilized with 0.1% Triton X-100, and blocked in 5% bovine serum albumin (BSA). Primary antibodies against hepatic differentiation markers (e.g., albumin, Ecadherin) and proliferation markers (BrdU) were applied. Stained cells were imaged using a fluorescence microscope.

E. RNA expression profiling: Total RNA was extracted from transduced and control cells, and cDNA was synthesized to evaluate the expression of hepatic differentiation markers and proliferation related genes. Gene expression was normalised to RPL13 and 18srRNA.

F. Functional Assays: To assess whether molecular changes in LSTFs re-expressing cells translate into behaviour and function, functional assays will be employed. These will include the LDH assay (quantitative index of membrane damage or necrosis) to detect cytotoxicity and stress in transduced cells. The CYP3A4 activity (drug detoxification activity of liver cells, often lost in cancer owing to their drug resistance) assay will be used to check for hepatic function. TUNEL quantification of DNA fragmentation will be used to detect apoptosis to determine if reprogrammingbased differentiation is also able to trigger apoptosis in these cells. Furthermore, to assess the effect of LSTF expression on cancer cell motility, metastatic potential, and tumour invasiveness, Migration and invasion assays will be done.

III. RESULTS Results suggest that reintroducing downregulated TFs as single TFs and combinations significantly impacts HCC cell morphology and proliferation patterns. Immunostaining and qPCR analyses revealed synergistic effects in reducing proliferation while restoring markers of hepatic differentiation. LDH and CYP3A4 assay also shows increased cell death and CYP3A4 functional activity in transduced cells.

IV. CONCLUSION In hepatocellular carcinoma, the disrupted network of TFs drives tumorigenesis and loss of cell identity. Reintroducing mature liver-specific TFs can partially reverse the malignant phenotype of HCC cells. Identification of master TFs and their overexpression highlights the importance of TF hubs in reestablishing cellular identity. These findings lay the groundwork for therapeutic strategies aimed at reprogramming HCC cells into their normal differentiated state, offering potential advancements in regenerative medicine and cancer therapy. The ultimate aim of the study is to reprogram cancer cells into normal differentiated hepatocytes.

V. ACKNOWLEDGMENT UAEU, CMHS, Department of Biochemistry and Molecular Biology.

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Impact of Gestational Diabetes Mellitus on Maternal and Neonatal Outcomes in the United Arab Emirates and Global Population: A Multicenter Systematic Review and Meta-Analysis Faiqa Quasim

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Review and Meta-Analysis

Gestational diabetes mellitus (GDM) is an increasingly prevalent metabolic disorder of pregnancy, particularly in the Gulf region, and is associated with adverse maternal and neonatal outcomes. It has been linked to increased risks of cesarean section, preterm birth, fetal overgrowth, neonatal hypoglycemia, neonatal jaundice, and preeclampsia. However, the overall impact of GDM on pregnancy outcomes in the United Arab Emirates (UAE) and comparable populations has not been systematically quantified.

To evaluate maternal and neonatal outcomes associated with GDM in the UAE, Gulf region, and other populations, with particular focus on cesarean section, preterm birth, fetal overgrowth/macrosomia, neonatal hypoglycemia, neonatal jaundice, and preeclampsia.

A systematic search of PubMed, Scopus, Web of Science, and Cochrane Library was conducted for studies published between 2000 and 2024. Studies comparing maternal and neonatal outcomes in GDM versus non-GDM pregnancies were included. Two independent reviewers screened studies and extracted data. Risk of bias was assessed using ROBINS-I for cohort studies and the Cochrane Risk of Bias Tool for randomized controlled trials. Pooled odds ratios (ORs) with 95% confidence intervals (CIs) were calculated using a random-effects model in accordance with PRISMA 2020.

Heterogeneity was assessed using I^2 , with sensitivity and subgroup analyses performed where appropriate. Ten studies were included in the quantitative synthesis. GDM was associated with increased odds of cesarean section (OR 1.80, 95% CI 1.28–2.53; $I^2=96.2\%$), preterm birth (OR 1.58, 95% CI 1.09–2.30; $I^2=88.2\%$), and fetal overgrowth/macrosomia (OR 2.45, 95% CI 2.00–3.01; $I^2=58.7\%$). Neonatal hypoglycemia was significantly increased

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abdomen to be done one year after surgery. If normal, yearly follow-up is advised by the oncologist for five years.

4. Conclusion- ICP N, being a preinvasive gallbladder neoplasm, is less invasive and has a better prognosis than gallbladder carcinoma. The presence of an invasive component at the time of preoperative detection cannot be determined. Invasive carcinomas may be associated with lymph nodal metastasis. Standardized guidelines are required for preoperative evaluation, investigations, and surgical planning in patients diagnosed with ICPN

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Intrabiliary Rupture of a Hepatic Hydatid Cyst Causing Mechanical Biliary Obstruction: Endoscopic Management of Cystobiliary Communication

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I. INTRODUCTION

Hepatocellular carcinoma (HCC) is the most common primary liver malignancy associated with chronic liver disease and cirrhosis. Despite advances in treatment, the prognosis for HCC remains poor due to late-stage diagnosis and limited therapeutic options.

Emerging evidence emphasizes how disturbed transcriptional networks contribute to tumorigenesis, especially when master transcription factors (TFs) of cell identity and differentiation are dysregulated. The study proposes that fetal liver specific master TFs are upregulated during liver regeneration and tumorigenesis, while mature liver specific master TFs are downregulated. This antagonistic relationship disrupts cell identity, promoting proliferation and tumorigenesis. We aim to investigate the interplay between master regulators of cell identity with a focus on their potential as therapeutic targets to reprogram cancer cells into a differentiated, non-proliferative state.

This study aims to reintroduce downregulated LSTFs and evaluate their potential in reprogramming HCC cells. Furthermore, the study aims to elucidate the molecular mechanisms underlying HCC reprogramming mediated by identified master regulators of hepatic lineage, with a focus on their therapeutic implications.

II. MATERIALS AND METHODS

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F. Functional Assays: To assess whether molecular changes in LSTFs re-expressing cells translate into behaviour and function, functional assays will be employed. These will include the LDH assay (quantitative index of membrane damage or necrosis) to detect cytotoxicity and stress in transduced cells. The CYP3A4 activity (drug detoxification activity of liver cells, often lost in cancer owing to their drug resistance) assay will be used to check for hepatic function. TUNEL quantification of DNA fragmentation will be used to detect apoptosis to determine if reprogramming-based differentiation is also able to trigger apoptosis in these cells. Furthermore, to assess the effect of LSTF expression on cancer cell motility, metastatic potential, and tumour invasiveness, Migration and invasion assays will be done.

III. RESULTS

Results suggest that reintroducing downregulated TFs as single TFs and combinations significantly impacts HCC cell morphology and proliferation patterns. Immunostaining and qPCR analyses revealed synergistic effects in reducing proliferation while restoring markers of hepatic differentiation. LDH and CYP3A4 assay also shows increased cell death and CYP3A4 functional activity in transduced cells.

IV. CONCLUSION

In hepatocellular carcinoma, the disrupted network of TFs drives tumorigenesis and loss of cell identity. Reintroducing mature liver-specific TFs can partially reverse the malignant phenotype of HCC cells.

Identification of master TFs and their overexpression highlights the importance of TF hubs in reestablishing cellular identity. These findings lay the groundwork for therapeutic strategies aimed at reprogramming HCC cells into their normal differentiated state, offering potential advancements in regenerative medicine and cancer therapy. The ultimate aim of the study is to reprogram cancer cells into normal differentiated hepatocytes.

V. ACKNOWLEDGMENT

UAEU, CMHS, Department of Biochemistry and
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Terminal Ileal Endometriosis Mimicking Crohn's Disease: A Rare Cause of Mechanical Obstruction and Ileo-Ileal Intussusception

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1. Introduction / Background

Intestinal endometriosis is an uncommon manifestation of endometriosis and a rare cause of small-bowel obstruction. Although the rectosigmoid colon is most frequently involved, the terminal ileum may also be affected.(1,2) Unlike inflammatory bowel disease, intestinal endometriosis predominantly involves the serosa and muscularis propria, with relative preservation of the mucosa.(2-4) This distribution can result in progressive fibrosis, muscular hypertrophy, and fixed luminal stenosis despite limited endoscopic abnormalities.(3,4)

Terminal ileal endometriosis may therefore closely mimic Crohn's disease on cross-sectional imaging, with bowel-wall thickening, luminal narrowing, mesenteric inflammatory change, and proximal dilatation.(3-5) We present a case of terminal ileal endometriosis causing recurrent mechanical obstruction and ileo-ileal intussusception, initially managed as presumed Crohn's disease.

2. Objectives

- To highlight intestinal endometriosis as an important differential diagnosis for terminal ileal stricturing disease.
- To demonstrate the diagnostic limitations of radiological and mucosal assessment in predominantly mural and serosal endometriosis.
- To emphasize the importance of integrating clinical history, imaging, endoscopy, and histopathology when evaluating an unexplained ileal stricture.
- To describe an unusual presentation of terminal ileal endometriosis complicated by ileo-ileal intussusception.

3. Clinical Case – Diagnostic Evaluation, Treatment and Follow-up

A 39-year-old woman with previously surgically confirmed endometriosis, including prior sigmoid resection, presented with recurrent abdominal pain and vomiting progressing to complete obstipation. She had no established history of Crohn's disease, chronic diarrhoea, or perianal manifestations.

Serial cross-sectional imaging demonstrated persistent terminal ileal wall thickening and luminal narrowing, mesenteric inflammatory change, regional lymphadenopathy, and proximal small-bowel dilatation. (Figure 1) These findings were repeatedly interpreted as inflammatory ileal stricturing, with probable Crohn's disease. Such imaging features overlap substantially with those described in intestinal endometriosis.(3-5) Corticosteroid therapy was therefore initiated. Although symptoms temporarily improved with bowel rest,

obstruction recurred following dietary advancement.

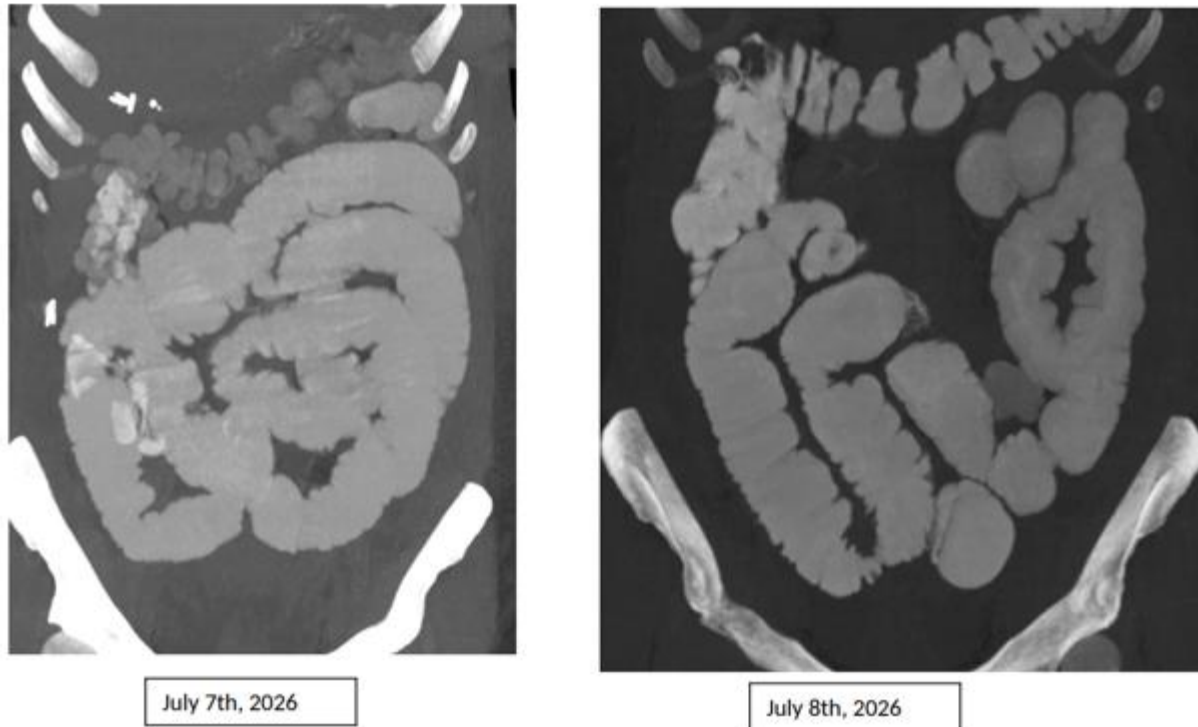


Figure 1. Preoperative CT.

Sequential images demonstrating terminal ileal wall thickening, luminal narrowing, and proximal bowel dilatation

Colonoscopy demonstrated a fixed, non-traversable narrowing approximately 7–10 cm proximal to the ileocecal valve. (Figure 2) Despite the severity of the obstruction, the visualized mucosa was relatively preserved, without significant ulceration or convincing features of chronic inflammatory bowel disease. This finding is consistent with the predominantly serosal and muscular distribution of intestinal endometriosis, which substantially limits the diagnostic yield of mucosal biopsies.(4,6)



Figure 2. Colonoscopy findings.

Colonoscopic evaluation demonstrating a fixed, non-traversable distal ileal narrowing

Persistent mechanical obstruction prompted diagnostic laparoscopy. An ileo-ileal intussusception was identified approximately 3 cm proximal to the ileocecal junction, associated with severe distal ileal stenosis. A second stenotic segment was identified approximately 30 cm proximally. A limited right hemicolectomy with ileocolic anastomosis was performed and the diseased segment was removed. (Figure 3)

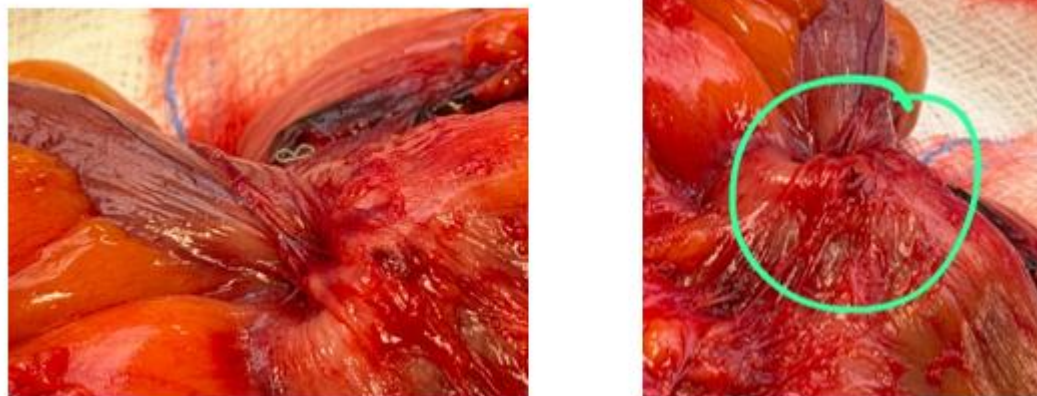


Figure 3. Gross specimen

Gross intraoperative specimen demonstrating telescoping of the distal ileum (ileo-ileal intussusception) with marked mural thickening and focal luminal stenosis.

Histopathological examination demonstrated endometrial-type glands and stroma infiltrating the muscularis propria and attached serosal fat, with marked mural thickening and luminal stenosis. The overlying mucosa showed only mild nonspecific inflammation and focal erosion. No granulomas, crypt architectural distortion, chronic active ileitis, dysplasia, or malignancy were identified, confirming intestinal endometriosis involving the muscularis propria and serosal fat of the distal ileum.

The postoperative course was uncomplicated apart from transient electrolyte abnormalities and symptomatic anaemia, which were appropriately corrected. The patient was discharged on postoperative day 5 and remained clinically well at follow-up without recurrent obstructive symptoms. Gynaecological follow-up was recommended.

4. Conclusion

Terminal ileal endometriosis is an important but frequently overlooked differential diagnosis for an ileal stricture, particularly in patients with a previous history of endometriosis. Its predominantly muscular and serosal distribution may produce substantial mechanical obstruction despite relatively preserved mucosa, limiting the diagnostic yield of colonoscopy and potentially mimicking stricturing Crohn's disease on cross-sectional imaging.(3-6)

In this case, the combination of a persistent fixed ileal stricture, recurrent mechanical obstruction, minimal mucosal abnormality, absence of histological features of inflammatory bowel disease, and established endometriosis ultimately supported the diagnosis. The associated ileo-ileal intussusception represents an unusual mechanical complication in this setting. Adult intussusception is uncommon and is frequently associated with an underlying structural lesion.(7,8)

This case emphasizes that discordance between the severity of obstructive physiology and the extent of mucosal disease should prompt consideration of predominantly mural or serosal pathology, including intestinal endometriosis. Definitive diagnosis may require full-thickness surgical resection and histopathological examination.

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Real-world effectiveness of guselkumab for induction of remission in Crohn's disease: a prospective multicentre cohort study with propensity score-matched comparison to Risankizumab

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Background & Aims:

Selective interleukin-23 (IL-23) inhibitors have entered the Crohn's disease (CD) treatment algorithm rapidly, but no head-to-head randomised trial compares them. Clinicians therefore choose between guselkumab and risankizumab without direct comparative evidence, and real-world data from Middle Eastern populations are almost entirely absent. We evaluated the real-world induction effectiveness of guselkumab in moderately to severely active CD and compared it against risankizumab using propensity score matching.

Methods:

Prospective multicentre cohort study of adults with moderately to severely active CD initiating guselkumab in the United Arab Emirates. The primary outcome was clinical remission at week 12 (Harvey-Bradshaw Index, HBI <5). Secondary outcomes were clinical response, C-reactive protein (CRP) and faecal calprotectin. Full propensity score matching against a contemporaneous risankizumab cohort controlled for 12 covariates, including disease phenotype, baseline endoscopic activity (SES-CD), baseline biochemical activity, surgical history and current smoking status.

Results:

Fifty-one guselkumab and 60 risankizumab patients were included. In the guselkumab cohort, 71% were advanced therapy-exposed, with a median of 2 prior advanced therapies. At week 12, clinical remission was achieved in 51% (26/51) and clinical response in 86% (44/51). Median CRP fell from 8.5 to 3.0 mg/L ($P = .002$) and median faecal calprotectin from 216 to 80 $\mu\text{g/g}$ ($P < .001$). Effectiveness was consistent regardless of prior treatment history. On univariable analysis, higher baseline HBI (OR 0.77, $P = .011$), SES-CD (OR 0.87, $P = .049$) and CRP (OR 0.94, $P = .019$) were associated with lower likelihood of remission. Maintenance dosing was 100 mg every 8 weeks in 73% and 200 mg every 4 weeks in 27%. After propensity score matching, clinical remission (51% vs 50%, $P > .9$) and clinical response (86% vs 87%, $P = .87$) were comparable between guselkumab and risankizumab, with no significant between-group biomarker differences.

Conclusions:

Guselkumab demonstrated robust real-world induction effectiveness in Crohn's disease, including in heavily pre-treated patients, and performed comparably to risankizumab across clinical and biochemical outcomes. These are the first regional comparative effectiveness data for selective IL-23 inhibitors and warrant confirmation in larger, longer-term studies with endoscopic endpoints.

Real-world outcomes of subcutaneous infliximab in a Middle Eastern inflammatory bowel disease cohort: a prospective study of switch and de novo treatment strategies

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Background & Aims:

Subcutaneous (SC) infliximab offers pharmacokinetic and convenience advantages over intravenous (IV) administration, with higher and more stable trough concentrations. Registrational data have established efficacy against placebo in maintenance, but real-world evidence from the Middle East is lacking, and the relative performance of the two available strategies, switching stable IV-treated patients versus de novo SC initiation after IV induction, remains unsettled. We evaluated clinical, biochemical and pharmacokinetic outcomes and drug persistence with SC infliximab in a prospective Middle Eastern inflammatory bowel disease (IBD) cohort across both strategies.

Methods:

Prospective observational cohort study conducted in the United Arab Emirates. Adult patients with IBD were enrolled either as switchers, transitioning from stable IV infliximab to SC infliximab 120 mg or 240 mg every 2 weeks, or as new starters receiving SC infliximab 120 mg every 2 weeks following IV induction. Clinical outcomes, C-reactive protein (CRP), serum albumin, faecal calprotectin and serum trough concentrations were assessed, and drug persistence was compared between groups by log-rank test.

Results:

Fifty-eight patients were included: 33 switchers (10 receiving 120 mg every 2 weeks and 23 receiving 240 mg every 2 weeks) and 25 new starters, with a median follow-up of 10.1 months (IQR 7.3-12.9). In the switcher cohort, 90.9% maintained clinical remission after the switch, with stable CRP, serum albumin and faecal calprotectin, and significant increases in median trough concentration: 9.5 to 20.0 µg/mL in the 120 mg group ($P = .044$) and 8.0 to 42.0 µg/mL in the 240 mg group ($P = .001$). Among new starters, 88% achieved clinical remission after induction, with a median post-induction trough of 16.0 µg/mL and significant improvements in CRP, serum albumin and faecal calprotectin; dose intensification was required in 28%. Drug persistence was high and comparable across all groups (log-rank $P = .61$), with 12-month rates exceeding 92% in every cohort.

Conclusions:

SC infliximab is effective for both switch and de novo management of IBD in a Middle Eastern population, delivering high clinical remission rates, robust drug exposure and durable treatment persistence exceeding 92% at 12 months. These are the first prospective regional data on SC infliximab and support its use as a default maintenance route in appropriately selected patients.

Advanced combination treatment in inflammatory bowel disease: first real-world experience from the United Arab Emirates

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Background & Aims:

Advanced combination treatment (ACT), the concurrent use of two advanced therapies, is increasingly used in refractory inflammatory bowel disease (IBD). No data have been reported from the United Arab Emirates (UAE).

Methods:

Retrospective analysis of all ACT courses at a tertiary IBD centre in Abu Dhabi between June 2021 and May 2026. A course was defined as concurrent use of two advanced therapies with documented intent, or co-induction. Baseline activity and all outcomes were adjudicated against the clinic record, so proportions cover the full cohort; the unit of analysis was the treatment course. The primary outcome was composite remission, clinical and biochemical remission together; endoscopic outcomes were reported separately.

Results:

Sixteen patients (11 Crohn's disease, 5 ulcerative colitis) received 21 courses of 12 distinct regimens; 13 (81%) were Emirati. Patients had failed a median of 4 prior advanced therapies. Twenty of 21 courses (95%) began with objective evidence of active disease (median calprotectin 450 µg/g, IQR 223-856). Median course duration was 218 days (IQR 134-359). The commonest combination was guselkumab with upadacitinib (8 courses, 7 patients). A selective anti-IL-23 agent (guselkumab or risankizumab) was paired with a JAK inhibitor in 10 courses (48%) and with an anti-TNF in 5 (24%); the remaining 6 spanned five further pairings. Composite remission was achieved in 8 of 21 courses (38%, 95% CI 18-62), clinical remission in 14 of 21 (67%) and biochemical remission in 8 of 21 (38%); 14 of 21 (67%) were corticosteroid-free. Endoscopic assessment was performed in 14 courses, 4 reaching remission. Eleven courses (52%) were discontinued, 5 for treatment failure. Four courses added a thiopurine to two advanced therapies; one produced the only serious adverse event, an EBV-related mucocutaneous ulcer with B-cell lymphoproliferative disorder, which regressed after withdrawal.

Conclusions:

In the first ACT cohort reported from the United Arab Emirates, composite remission was achieved in just over a third of courses in multiply refractory disease. Short-term safety signals are encouraging, although careful monitoring remains essential.

Extraintestinal manifestations and immune-mediated inflammatory diseases in inflammatory bowel disease: a population-based study from the United Arab Emirates

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Background & Aims:

Extraintestinal manifestations (EIMs) and immune-mediated inflammatory diseases (IMIDs) in inflammatory bowel disease (IBD) are described almost entirely in tertiary cohorts, where the apparent excess cannot be separated from coding opportunity, and none from the United Arab Emirates has been measured against a general population.

Methods:

We analysed routinely recorded diagnostic data from the complete public hospital network of Abu Dhabi, United Arab Emirates, to July 2026, ascertaining both arms by diagnostic coding alone. Burden within IBD was described for the whole cohort and, among Emirati adults, compared with the general population over a reconstructed denominator, with sex adjustment and, by design, no age standardisation.

Results:

Of 3,552 people with IBD, 483 (13.6%) had at least one EIM or IMID. A classic EIM was present in 260 (7.3%) and a shared IMID in 282 (7.9%), with 59 carrying both. Enteropathic arthropathy was present in 90 patients and was the sole classic manifestation in 76; excluding it, the classic group was 184 (5.2%). The commonest conditions were sacroiliitis (3.7%), psoriasis (3.3%), ankylosing spondylitis (2.7%), enteropathic arthropathy (2.5%), autoimmune hepatitis (2.2%) and primary sclerosing cholangitis (1.7%). Composition differed in children, classic 5.1% against shared 2.4%. Among Emirati adults prevalence was 15.4% against 2.74% in the general population, a crude ratio of 5.62 (5.02 to 6.28) and 5.65 (5.05 to 6.31) after sex adjustment.

Conclusions:

In the first analysis from the United Arab Emirates to use a general-population denominator, an extraintestinal manifestation or associated immune-mediated disease was present in 13.6% of patients with IBD and was 5.6-

fold more common than in the general population, an upper figure because the reference population is unselected. The burden was dominated by sacroiliitis, psoriasis and ankylosing spondylitis, conditions largely diagnosed and coded outside gastroenterology.

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Conflicts of interest: MNQ has received speaker fees from Johnson and Johnson, Takeda, AbbVie, Hikma, Tillotts and Lilly, and consultancy fees from Johnson and Johnson and Lilly. IA, KA, SAM, AAT and LLB declare no conflicts of interest. TJ: to be confirmed.

Predictors of Chronic Liver Disease in Adults: The Role of Metabolic and Lifestyle Factors in Healthy Aging

Authors

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Keywords

Chronic liver disease; Healthy aging; Body mass index; Alcohol consumption

Background

Chronic liver disease (CLD) is an increasing public health burden driven by metabolic and lifestyle factors. This study identified demographic, clinical, and behavioral predictors of CLD to support targeted prevention and healthy aging. **Materials and Methods** A hospital-based case-control study included 149 adults with confirmed CLD and 300 controls. Demographic, clinical, and lifestyle variables were analyzed using chi-square tests and multivariable logistic regression to identify independent predictors. **Results** Male sex, abnormal body mass index (BMI), and alcohol consumption were independently associated with CLD. Male participants had threefold higher odds of CLD (adjusted OR 3.03; 95% CI 1.88–4.90). Abnormal BMI increased the odds more than twofold (adjusted OR 2.33; 95% CI 1.30–4.20). Alcohol consumption was the strongest predictor, with current users demonstrating markedly increased odds (adjusted OR 53.35; 95% CI 6.87–414.15).

Although adults aged 40–60 years were more frequently affected, age was not an independent predictor after adjustment. **Conclusions and Recommendations** Male sex, abnormal BMI, and alcohol consumption are key predictors of CLD. Targeted screening and lifestyle interventions focusing on metabolic health and alcohol reduction may reduce disease burden and promote healthy aging. **References** (APA 7th Edition) Paik, J. M., Kabbara, K., Eberly, K. E., et al. (2022). Global burden of NAFLD and chronic liver disease among adolescents and young adults. *Hepatology*, 75, 1204–1217. Cheemerla, S., & Balakrishnan, M. (2021). Global epidemiology of chronic liver disease. *Clinical Liver Disease*, 17, 365–370. Younossi, Z. M., Tacke, F., Arrese, M., et al. (2019). Global perspectives on nonalcoholic fatty liver disease and nonalcoholic steatohepatitis. *Hepatology*, 69, 2672–2682. Drake, I., Giontella, A., Miari, M., et al. (2024). Lifestyle and genetic risk of chronic liver disease in metabolically healthy and unhealthy individuals. *JHEP Reports*, 6, 101105. Alkaabi, J., Afandi, B., Alhaj, O., et al. (2024). Identifying metabolic dysfunction-associated steatotic liver disease in patients with type 2 diabetes mellitus using clinic-based prediction tools. *Frontiers in Medicine*, 11.

Mapping of Colonic Lesions in Patients Aged 65 Years and Older: Experience from a Tertiary University Center

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INTRODUCTION: Population aging is associated with an increasing number of indications for colonoscopy in older adults. Although more demanding in this population, colonoscopy retains a high diagnostic yield and enables immediate therapeutic intervention, warranting a rigorous assessment of its value.

OBJECTIVE: To describe the clinical characteristics, indications, and lesion profile of colonoscopies performed in patients aged 65 years and older.

METHODS: We conducted a retrospective descriptive study in the digestive endoscopy unit of the Gastroenterology Department at Ibn Rochd University Hospital, Casablanca. All patients aged 65 years and older who underwent colonoscopy were included, based on data from medical records and endoscopy reports.

RESULTS: One hundred patients were included (mean age: 73 years; balanced sex ratio). Patients aged 65–74 years accounted for 65% of the cohort, followed by those aged 75–84 years (29%) and ≥85 years (6%). Hemorrhagic indications predominated (44%), followed by functional indications (31%) and endoscopic surveillance (24%). Colonoscopy was complete in 47% of cases, with satisfactory bowel preparation reported in the same proportion. The lesion profile was dominated by angiodysplasias and polyps (19% each), followed by colorectal cancers (17%) and active bleeding (10%). A clinically significant lesion was identified in more than half of the patients (51%).

CONCLUSION: In older adults, colonoscopy has a high diagnostic yield, with clinically significant lesions identified in more than half of patients. Angiodysplasias, polyps, and colorectal cancers were the predominant findings, supporting the diagnostic value of colonoscopy in this population

Upper Gastrointestinal Bleeding: Comparison of Etiological Profile and In-Hospital Prognosis According to Antithrombotic Treatment

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INTRODUCTION: Upper gastrointestinal bleeding (UGIB) is a life-threatening emergency. Antithrombotic agents complicate its management.

OBJECTIVE: To assess the impact of antithrombotic treatment on the in-hospital prognosis of patients admitted for UGIB.

METHODS: A retrospective descriptive and analytical study conducted at Ibn Rochd University Hospital, Casablanca, based on medical records, comparing patients receiving antithrombotic therapy with those not receiving such therapy.

RESULTS: Among 327 patients, 61.5% were male and 38.5% were female (M/F ratio: 1.6); mean age was 55 ± 17.8 years. Antithrombotic therapy was being received by 12.5% of patients, versus 87.5% who were not: antiplatelet agents in 58.5% of cases, direct oral anticoagulants in 22%, and vitamin K antagonists in 19.5%. Upper gastrointestinal endoscopy was performed in 322 patients (98.5%). Portal esophagogastric varices were the leading etiology (38.2%), followed by gastroduodenal ulcers (29.7%), tumoral lesions (8%), and angiodysplasia (3.4%). Endoscopy findings were normal in 6.7% of cases, and 1.5% of patients died before endoscopy could be performed. Outcome was favorable in 74.6% of patients. Rebleeding occurred in 9.2%, transfer to intensive care, surgery, or oncology in 8.9%, and in-hospital mortality reached 5.8%. Patients on antithrombotic therapy were older (70.3 ± 13.0 vs 52.8 ± 17.4 years; $p < 0.0001$), had fewer esophagogastric varices (9.8% vs 42.3%; $p < 0.001$), and required transfusion more frequently (78.0% vs 59.8%; $p = 0.037$). Peptic ulcers were not significantly more frequent in this group (36.6% vs 28.7%; $p = 0.29$), whereas angiodysplasia was (9.8% vs 2.4%; $p = 0.037$).

CONCLUSION: Antithrombotic therapy influenced the etiological profile and transfusion requirements but did not constitute an independent prognostic factor. These findings highlight the need for digestive risk assessment, and for endoscopic evaluation in at-risk patients, before initiating anticoagulant therapy.

AI-Assisted Non-Invasive Liver Fibrosis Staging vs. Conventional Elastography/Serum Scores: A Systematic Review of Diagnostic Accuracy

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1. Introduction/Background

Liver biopsy remains the diagnostic reference standard for fibrosis staging but is invasive, subject to sampling error, and impractical for serial monitoring. Non-invasive alternatives like transient/magnetic resonance elastography and serum-based scores (FIB-4, NAFLD Fibrosis Score) are widely used but show variable accuracy, particularly in early-stage disease and non-Caucasian populations. Machine learning (ML) models integrating clinical, biochemical, and imaging data have recently been proposed to improve on these tools, but head-to-head diagnostic accuracy synthesis across AI models and conventional methods remains limited.

2. Objectives

To systematically review and compare diagnostic accuracy (sensitivity, specificity, AUROC) of AI/ML-based fibrosis staging models versus conventional elastography and serum-based scores across liver disease etiologies.

3. Methods

A systematic search of PubMed, Embase, Cochrane Library, and Web of Science (2015–2026) will be conducted per PRISMA-DTA guidelines, using terms combining "machine learning," "artificial intelligence," "liver fibrosis," "elastography," "FIB-4," and "diagnostic accuracy." A preliminary evidence-mapping search identified 21 multimodal-AI studies in a recent scoping review of 2,849 records, with additional potentially eligible studies published subsequently. However, many studies lacked direct comparison with elastography or established serum scores. A new systematic search and study-level screening are therefore underway to identify the subset providing eligible head-to-head diagnostic-accuracy data. Eligible studies report AUROC, sensitivity, or specificity for both an AI/ML model and a conventional comparator against histologic or established non-invasive reference standards. QUADAS-2 will assess risk of bias.

4. Results

Established benchmarks for conventional methods are well-characterized: magnetic resonance elastography (MRE) outperforms transient elastography (Fibroscan) for significant fibrosis detection (AUROC 0.981 vs. 0.796), while serum-based FIB-4 shows more modest accuracy for advanced fibrosis in NAFLD (sensitivity 69%, specificity 64%). Emerging ML approaches show promise: a 2026 metabolite-augmented FIB-4 machine learning panel (Met-FIB) demonstrated superior rule-in specificity for advanced fibrosis/cirrhosis compared to Fibroscan, and a dedicated systematic review of gut-microbiome-based ML models (10 studies, 838 participants) reported diagnostic potential but insufficient head-to-head comparator data. No identified systematic review has pooled AI/ML diagnostic accuracy directly against conventional elastography and serum scores across a broad, etiology-spanning evidence base most existing syntheses compare conventional methods to each other, or evaluate a single ML approach (e.g., microbiome-based) in isolation.

5. Discussion

Available evidence suggests ML-augmented panels may offer complementary particularly rule-in diagnostic value alongside, rather than as outright replacement for, elastography. However, the absence of pooled head-to-head diagnostic accuracy data limits confident clinical positioning of AI tools relative to established non-invasive standards.

6. Conclusion

Non-invasive fibrosis staging tools each carry distinct accuracy trade-offs; AI/ML models show emerging but not yet systematically synthesized comparative advantage. This review aims to consolidate head-to-head evidence to guide clinical tool selection and prioritize future validation study design.

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Multisystem Complications in Cephalosporin-Resistant Typhoid Fever: Glomerulonephritis and Pleural Effusion in a Pediatric Traveler

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1. Introduction / Background

Typhoid fever, caused by *Salmonella enterica* serovar Typhi (S. Typhi), remains a substantial health burden in resource-limited settings and among returning travelers, disproportionately affecting children. While gastrointestinal and constitutional symptoms predominate, renal and respiratory involvement is rare, especially in pediatric patients. The emergence of cephalosporin-resistant S. Typhi has further complicated management, increasing the risk of severe and atypical presentations.

2. Objectives

To report a rare case of cephalosporin-resistant typhoid fever complicated by glomerulonephritis and pleural effusion in a previously healthy adolescent, highlighting the importance of early recognition of treatment failure, multisystem monitoring, and timely antimicrobial escalation.

3. Clinical Case

A previously healthy 13-year-old South Asian female presented with high-grade fever, vomiting, and gross hematuria two days after returning from travel to an endemic region, where she had received incomplete treatment with an oral cephalosporin. Examination showed a tender, distended abdomen, fever, dry mucous membranes, and tachycardia.

Initial workup showed a normal CBC, elevated CRP (106.5 mg/L), brown urine with 3+ hematuria and 2+ proteinuria, negative H. pylori and dengue NS1 tests, and a positive Widal test. She was admitted for further management.

Treatment includes IV fluids, antipyretics, and IV ceftriaxone were started. CRP rose to 133.2 mg/L, Hb and platelets dropped, and blood cultures grew Gram-negative bacilli. Urinalysis revealed >100 RBC casts/HPF, confirming glomerulonephritis. IV azithromycin was added. Despite this, ALT continued rising, RBC casts persisted, and the patient developed cough, chills, and shortness of breath with absent breath sounds. Chest X-ray on day 4 confirmed a mild right-sided pleural effusion. Treatment was escalated to IV meropenem (ceftriaxone and azithromycin discontinued) due to poor response and suspected antimicrobial resistance. Follow-up: Over the next several days, CRP, Hb, and platelet counts progressively normalized; repeat imaging on day 6 showed resolution of the pleural effusion. By day 7, CRP had fallen to 8.9 mg/L and urinary findings had markedly improved. The patient was discharged on IV ertapenem (5 days) with oral rehydration and dietary advice. At one-week follow-up, hematuria and RBC casts had further decreased, confirming continued clinical recovery.

4. Conclusion(s)

Travel history is a critical component of clinical assessment, and typhoid fever should be suspected in recent travelers presenting with fever. As it can involve multiple organ systems simultaneously, thorough systemic evaluation is essential. Early recognition of treatment failure and prompt antimicrobial escalation—particularly in the context of rising cephalosporin resistance—are key to preventing morbidity. Adherence to hygiene,

sanitation, and immunization remains crucial given the increasing prevalence of multidrug-resistant *Salmonella* Typhi strains.

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ABSTRACT # 73

Bowel Preparation as Residual Gastric Content Protection: Perioperative Management of GLP-1/GIP Receptor Agonists in Adults Undergoing Combined Gastroscopy and Colonoscopy. Systematic Review and Meta-Analysis

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1. Introduction / Background: GLP-1 receptor agonists and dual GLP-1/GIP receptor agonists delay gastric emptying and impair colonic motility, raising concerns about clinically significant residual gastric content (RGC) during upper endoscopy. Combined same-session gastroscopy and colonoscopy is mechanistically distinct because bowel preparation may mitigate upper-GI retention while potentially impairing colonoscopy preparation quality.

2. Objectives: To determine RGC rates in GLP-1/GIP agonist users undergoing combined gastroscopy and colonoscopy, evaluate the effect of temporary drug interruption versus continuation, and assess colonoscopy preparation adequacy.

3. Methods: A systematic review and meta-analysis was conducted according to PRISMA 2020 (PROSPERO CRD420261392931). Adults receiving GLP-1 or dual GLP-1/GIP receptor agonists undergoing elective endoscopy were included. Non-randomized comparative studies were assessed using ROBINS-I. The primary outcome was clinically significant RGC, with secondary outcomes including aborted procedures and aspiration events.

4. Results: Seven studies reported combined gastroscopy and colonoscopy data. RGC rates were consistently 0–2%. The pooled odds ratio for concurrent colonoscopy versus EGD alone was 0.24 (95% CI 0.16–0.36; $I^2=0\%$), representing a 76% reduction in RGC odds. In two EGD-only hold-versus-continue studies ($N=1,444$), temporary interruption reduced RGC (pooled OR 0.34, 95% CI 0.21–0.55; $I^2=0\%$). No procedures were reported as aborted due to RGC in the combined-procedure subgroups. GLP-1 therapy was associated with modestly impaired colonoscopy preparation quality.

5. Discussion: Standard colonoscopy preparation appears to substantially mitigate GLP-1-mediated upper-GI retention during combined procedures. However, no included study directly compared drug interruption versus continuation specifically in the combined-procedure population.

6. Conclusion(s): In adults undergoing combined same-session gastroscopy and colonoscopy, standard bowel preparation is associated with very low RGC rates (0–2%) and a 76% reduction in RGC odds compared with EGD alone. Routine GLP-1 interruption may therefore not be necessary in adherent patients undergoing combined procedures, although individualized decisions remain appropriate for higher-risk patients. 7.

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Gastritis Reporting with Olmesartan Versus Losartan: A FAERS Disproportionality Analysis

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BACKGROUND: Olmesartan and losartan are widely prescribed angiotensin II receptor blockers for hypertension in the United States. Gastritis is a potentially serious but underrecognized adverse event reported with both agents and may be attributed to more common gastrointestinal disorders. Although olmesartan is associated with sprue-like enteropathy, the clinical characteristics and pharmacovigilance signals of gastritis with these drugs remain poorly defined.

OBJECTIVE: To characterize FAERS gastritis reports involving olmesartan and losartan, quantify drug-specific disproportionality signals, and compare gastritis reporting odds between the two agents.

METHODS: The Food and Drug Administration Adverse Event Reporting System (FAERS) was queried from 2004 through 2026 for reports involving olmesartan or the active comparator losartan using generic, salt-form, and brand-name variants. Gastritis was identified using the MedDRA Preferred Term “Gastritis.” Disproportionality was assessed using reporting odds ratios (RORs) with 95% confidence intervals (CIs) versus all other FAERS reports and directly between olmesartan and losartan. Signals required an ROR >1.0 with a lower 95% CI >1.0. Case listings were deduplicated by FAERS Case ID and descriptively characterized by age, sex, hospitalization, death, suspect-product status, and the most frequently co-reported reactions.

RESULTS: Significant disproportionality signals for gastritis were identified for olmesartan versus all other FAERS reports (ROR, 18.84; 95% CI, 16.77–21.17) and losartan versus all other reports (ROR, 4.07; 95% CI, 3.33–4.97). In the active-comparator analysis, gastritis reporting odds were significantly higher with olmesartan than losartan (ROR, 4.60; 95% CI, 3.65–5.79). The listings contained 291 olmesartan and 97 losartan reports. Among reports with known age, median age was 66 years (IQR, 61–74; n=102) for olmesartan and 67 years (IQR, 56–78; n=48) for losartan. Among reports with known sex, 71/105 (67.6%) olmesartan and 66/89 (74.2%) losartan reports involved females. Non-mutually exclusive outcomes included hospitalization in 162 olmesartan reports (55.7%) and 51 losartan reports (52.6%), life-threatening events in 0 (0.0%) and 9 (9.3%), and death in 2 (0.7%) and 11 (11.3%), respectively. The index drug was the sole suspect ingredient in 167 (57.4%) olmesartan and 25 (25.8%) losartan reports. Selected non-mutually exclusive co-reported events included weight loss in 122 olmesartan reports (41.9%) and 15 losartan reports (15.5%), diarrhea in 89 (30.6%) and 20 (20.6%), respectively, and sprue-like enteropathy in 38 olmesartan reports (13.1%) and none with losartan.

DISCUSSION: Olmesartan showed a stronger gastritis signal than losartan, consistent with prior reports of reversible gastric epithelial injury. Gastric CD8-positive infiltration, supported by olmesartan-associated IL-15 upregulation and tight-junction disruption observed in intestinal models, provides a plausible but unconfirmed mechanism; comparable evidence for losartan is limited.

CONCLUSION: Olmesartan had substantially higher reporting odds of gastritis than losartan in FAERS (ROR, 4.60; 95% CI, 3.65–5.79). Because FAERS lacks exposure denominators and sufficient clinical information for causal adjudication, these signal-generating findings cannot establish causality or incidence and require confirmation in controlled epidemiological and mechanistic studies.

KEYWORDS: Olmesartan; Losartan; Gastritis; Pharmacovigilance; Disproportionality analysis.

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ABSTRACT # 76

Hexosamine Biosynthesis Pathway: A Metabolic Rheostat In Hepatic Lineage Commitment and Hepatocellular Carcinoma

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ABSTRACT

Hepatocellular carcinoma (HCC) is the most common form of primary liver cancer and is a leading cause of cancer-related mortality. In the United Arab Emirates (UAE), the incidence rate has doubled over the past three decades, rising rapidly with increasing rates of obesity, diabetes and metabolic dysfunction-associated fatty liver disease. Emerging research evidence that dysregulated metabolism is a prominent driver of this progression, and flux through the hexosamine biosynthesis pathway (HBP) culminating in protein O-GlcNAcylation by O-GlcNAc transferase (OGT) functions as a nutrient sensing biochemical pathway that sustains oncogenic transcription in HCC. This study proposes that that reducing HBP flux would impair HCC progression by destabilising oncogenic signalling and restoring tumour suppressor function and on the other hand, tightly regulated HBP activity is essential for pluripotency, definitive endoderm specification and hepatic lineage commitment during embryonic development. To elucidate the role of the HBP, OGT knockout and knockdown models in HCC cell lines and GFPT1 and GFPT2 knockdown in H9 human embryonic stem cells were generated. OGT knockout in Huh-7 HCC cell line resulted in impaired cell division confirming that O-GlcNAcylation is essential for hepatocellular carcinoma proliferation whereas OGT knockout in HepG2 hepatoblastoma cell line resulted in a markedly different, distinct morphological phenotype, indicating cell-line specific dependency on O-GlcNAcylation. Immunostaining of OGT-knockout HepG2 cells showed a marked loss of the oncofetal marker alpha-fetoprotein (AFP), increased hepatocyte-specific albumin, reduced proliferation (Ki-67), unchanged E-cadherin and increased vimentin, indicating a shift towards a more differentiated yet partially mesenchymal phenotype. Because this AFP-to-albumin switch is thought to be gated epigenetically, at the level of chromatin-associated transcriptional elongation, the checkpoint that controls RNA polymerase II (Pol II) processivity - total Pol II, Pol II Ser2P, Pol II Ser5P, CDK9, HEXIM1, HEXIM2 and BRD4 - was profiled alongside PI3K-AKT signalling (pan-AKT, phospho-AKT), to test whether reduced O-GlcNAc flux facilitates the differentiation-associated transcriptional switch observed. In parallel, knockdown of the HBP-committing enzymes GFPT1 and GFPT2 in H9 human embryonic stem cells is being used to define how HBP flux at an earlier pathway node governs pluripotency maintenance and hepatic lineage commitment. By integrating molecular and functional studies, this work seeks to identify HBP enzymes as potential targets to therapeutically reprogram hepatocellular carcinoma towards a more differentiated and less proliferative state.

Chyme reinfusion therapy: First report from UAE

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Introduction

Chyme reinfusion (CR) restores intestinal continuity by reintroducing proximal effluent into the distal limb, aiming to reduce parenteral nutrition (PN) requirements, and improve intestinal function. Manual CR (MCR) is often impractical, particularly in patients with separate stomas. The Insides Channel[®] was created to facilitate automated CR across the abdomen.

Objective

CR with the Insides Channel[®] was performed on a 55-year-old gentleman who was referred to our Integrated Intestinal Rehabilitation Centre, for management of Type II Intestinal Failure (IF). The patient had undergone an umbilical hernia repair, complicated by multiple jejunal perforations. He had a left-sided jejunostomy and a right-sided ileostomy and was being treated with a combination of liquid diet, PN, distal feeding, and MCR. We aim to describe the first UAE experience of the Insides Channel[®] in the management of Type II IF.

Materials and methods

The patient's nutrition was optimised by stopping PN, distal feeding, and MCR. The Insides Channel system, comprising the Insides tube, pumps, driver, and channel tubing, was introduced to enable automated CR between ostomies. The patient was trained to self-administer reinfusion. Daily fluid balance, reinfusion volumes, weight, and serum electrolytes were monitored.

Results

CR was well tolerated, with minimal symptoms reported. Oral diet commenced, and within 1 week parenteral support ceased. The patient was discharged home after 2 weeks and was reinfusing up to 90% of his stomal output. He gained weight and was able to return to work using the system. He has subsequently undergone successful delayed continuity surgery.

Conclusion

CR via the Insides Channel was feasible and showed good results in maintaining nutritional status and rehabilitating the distal intestine, even with separated stomas.

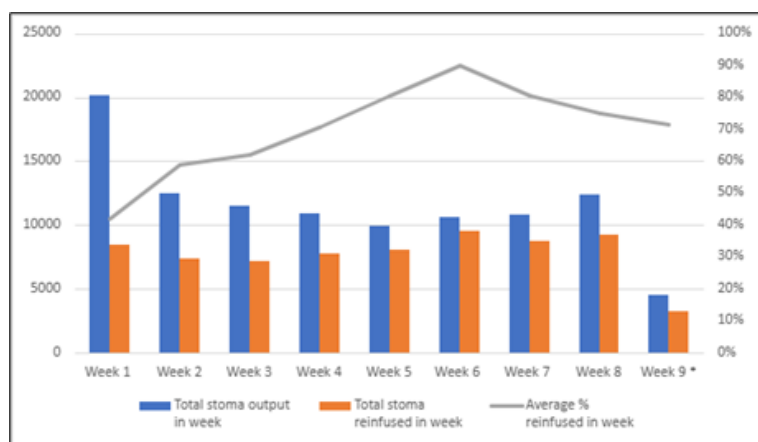


Figure 1: Weekly stoma output and reinfusion volumes