Methods Mucosal biopsy samples were taken from ulcerative colitis patients with pouchitis (chronic pouchitis $n=9$, acute pouchitis $n=4$) and those without pouchitis ($n=11$). Epithelial cells were isolated from biopsy tissue after incubation with DTT and EDTA. Epithelial cell expression of ZO-1, claudin 1 and claudin 2 were measured by multicolour flow cytometry. Cytokines were assessed by multiplex ELISA of biopsy supernatants. The t-test was used for statistical analysis.

Results In acute pouchitis ZO-1 was elevated compared with both chronic pouchitis and non-pouchitis (p = 0.008), whilst in chronic pouchitis ZO-1 expression was reduced compared with non pouchitis (p = 0.006). Claudin 1 expression was reduced in chronic pouchitis (p = 0.04), but was not significantly reduced in acute pouchitis. In acute pouchitis, claudin 2 expression was elevated (p<0.001), but was not increased in chronic pouchitis. IL6 levels were elevated in chronic pouchitis compared with non pouchitis patients (p = 0.01).

Conclusion Epithelial tight junction expression was altered in pouchitis in association with increased IL6 levels. Increased claudin 2 expression in acute, but not chronic pouchitis may represent early pathological changes in the development of pouch inflammation. In chronic inflammation the tight junction complex was deranged with reduced expression of both claudin 1 and ZO-1. Increased epithelial barrier permeability due to altered tight junction expression may be a critical mechanism in the development and perpetuation of pouch inflammation.

Disclosure of Interest None Declared.

A PROSPECTIVE CONTROLLED PILOT STUDY OF FECAL MICROBIOTA TRANSPANTATION FOR CHRONIC REFRACTORY POUCHITIS

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Introduction Faecal microbiota transplantation (FMT) is an effective therapy for *Clostridium difficile* and possibly inflammatory bowel diseases (IBD). Published data of FMT for inflammatory bowel diseases are reported in case series and case reports. To our knowledge, there are no controlled studies of FMT for IBD. We aimed to conduct a prospective study of FMT for chronic refractory pouchitis.

Methods Patients with clinically, endoscopically and histologically confirmed chronic refractory pouchitis; with a pouch disease activity index (PDAI) > 7 were included. Donors were screened by clinical history and serology for HAV, HBV, HCV, HEV, Treponema, HIV, HTLV 1/II and stool for M, C+S, C. difficile toxin and parasites. Fresh donor stool was collected within six hours of nasogastric tube feeding and stored at -80°C.

Prior to FMT, patients were screened for: (a) immunosuppression; (b) CD vaccinated patients; (c) drugs that alter the gut microbiota; (d) open infection; (e) abdominal surgery; (f) use of antibiotics prior to FMT; and (g) previous FMT.

Methods Participants undergoing RPC from the ileostomy afferent loop, the pouch pre-ileoectomy closure (P) and the pouch 6 and 12 months post-ileoectomy closure (n = 5). Epithelial cell expression of zona occludens (ZO-1), claudin 1 and claudin 2 and DC expression of TLR 2 and 4, CCR9, β7 and CD40 were measured by multicolour flow cytometry. Cytokines were assessed by multiplex ELISA of biopsy supernatants. The paired t-test was used for statistical analysis.

Results Expression of claudin 2 was increased (p = 0.04) at 6 months and remained elevated at 12 months. No changes were seen in ZO-1 or claudin 1 expression. There was a significant increase in β7 expression on lamina propria DC (p = 0.02), but no differences in DC TLR and CD40 expression were seen at 6 months. DC expression of β7 was further elevated (p = 0.005) as well as significantly increased TLR 4 and CD40 expression (p = 0.04). No cytokines were found to be elevated at 6 months, but at 12 months there was a trend towards increased IL6 (p = 0.05).

Conclusion In patients with UC, altered tight junction expression with increased epithelial expression of the “pore-forming” tight junction claudin 2 was an early event after ileoectomy closure that preceded the onset of mucosal inflammation. In parallel, more lamina propria DC expressed gut homing markers possibly in response to increased exposure to the changing microbial signals and a more permeable epithelial barrier. These changes in parallel may lead to increased microbial stimulation of DC with increased TLR and costimulatory molecule expression that could predispose to the development of inflammation.

Disclosure of Interest None Declared.

PREVALENCE OF FAECAL INCONTINENCE IN ADULTS WITH INFLAMMATORY BOWEL DISEASE

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