

**PTH-102 FERRIC MALTOL, UNLIKE FERROUS SULPHATE, DOES NOT ADVERSELY AFFECT THE INTESTINAL MICROBIOME**

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**Introduction** We have previously shown that altering dietary ferrous iron consumption exacerbates murine models of inflammatory bowel disease (Mahalhal *et al.*, 2018) and is associated with dysbiosis. We have now investigated the effect of oral ferric maltol and ferrous sulphate on the microbiome of patients with iron deficiency anaemia (IDA) and on mice treated with dextran sodium sulphate (DSS) to induce colitis. We report results of changes at the phylum level.

**Methods** Studies were performed on three groups of wild-type mice: acute colitis was induced with 2% DSS for 5 days, followed by 5 further days on water while mice were fed one of three diets (from day-1 of DSS treatment): normal diet (200ppm iron ferrous sulphate) [ND (n=8)], 400ppm iron ferrous sulphate [FS (n=16)] or 400ppm iron ferric maltol [FM (n=16)]. Clinical and pathological data were compared at day-1 and day-10, when faecal samples were collected. 16 patients with IDA were also recruited: 10 were treated with FS and 6 with FM supplements for 4 weeks: paired faecal samples (pre and post-treatment) were collected. Faecal bacterial gDNA was extracted from murine and human samples and the microbiota composition was determined from the sequence of V4 region of 16S rDNA. Statistical inferences were made using Welch's t-test with post-hoc analysis: Shannon Diversity Index (SDI) and Principal Component Analysis (PCA) were used to compare population and phylum-level changes.

**Results** DSS-induced colitis was worse in ND and FS mice than FM mice (determined by weight loss [3, 7 & 0%, at day-8 respectively] and histology [median score 2, 2 & 1 at day-10]). FS supplementation was associated with an increase in *Bacteroidetes* (15% in mice, 4% in humans) whereas FM led to a reduction in *Bacteroidetes* (3% in mice and 15% in humans). There was a 4% increase in *Firmicutes* with FM supplementation in mice and 20% in humans whereas FS led to a 15% reduction in *Firmicutes* in mice and 5% in humans.

**Conclusion** This study has demonstrated differential and unique influences of ferric maltol and ferrous sulphate supplements on the faecal microbiome of mice with DSS induced colitis and patients with iron deficiency. These differences might contribute to the difference in side effects that are associated with these preparations.

**PTH-103 THE GASTROENTEROLOGIST'S BIGGEST FEAR – WHEN THE CURE IS WORSE THAN THE DISEASE**

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**Introduction** One of the most sinister, whilst rare, complications of combination treatment with biological and thiopurines therapy is lymphoproliferative disorders. We present a case of a 35 year old gentleman with a background of Crohn's

disease. He presented with symptoms of chest pain, dyspnoea and an atypical rash, to the gastroenterology ward. Following extensive investigations he was diagnosed with hepatosplenic T-cell lymphoma.

The patient had a longstanding history of penetrating small bowel Crohn's disease with two previous small bowel resections and multiple stricturoplasties. After failing monotherapy with Azathioprine, he was started on combination treatment with Adalimumab and Azathioprine, which brought his disease into symptomatic remission for 5 years.

On presentation to A&E, in addition to the symptoms detailed above, he had a 3 month history of B symptoms (fevers, night sweats and weight loss). Therefore, on consideration of diagnosis our top differential was of possible haematological malignancy whilst his cutaneous manifestation led us to consider inflammatory or vasculitic processes.

**Results** Radiologic investigations including computed tomography scan [CT] and positron emission tomography scan [PET] showed long standing hepatosplenomegaly with portal hypertension, but with no evidence of lymphadenopathy, malignancy or large vessel vasculitis. He continued to spike fevers and showed evidence of haemolysis and severe anaemia requiring daily transfusions. His lactate dehydrogenase [LDH] was over 9600U/L initially and then continued to rise above 15000U/L, and his ferritin was significantly elevated at 12025ng/ml with otherwise minimally raised inflammatory markers.

The main differentials remained haematological including lymphoma or Hemophagocytic Lymphohistiocytosis. He subsequently had a bone marrow biopsy which confirmed the diagnosis of hepatosplenic T-Cell lymphoma.

He was transferred to a tertiary centre where he was treated with chemotherapy and was deemed to be in remission, awaiting sibling allograft.

**Conclusion** Literature suggests the risk of Hepatosplenic T-cell lymphomas [HSTCLs] occur almost exclusively in males under the age of 35 who are exposed to thiopurines and the risk increases for those treated with combination of thiopurines and anti-TNF agents. In the latter group risk increases when the duration of combination treatment exceeds two years. Therefore, question should be posed whether we should avoid combination therapy in this cohort of patients for more than two years or whether alternative concomitant immunomodulation or biologics should be considered in high risk cases.

**PTH-104 REMISSION AND RECURRENCE IN IBD AFTER BIOLOGICAL THERAPY**

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**Introduction** Biological agents have revolutionised the treatment of inflammatory bowel disease (IBD) with usage continually rising. Decisions regarding de-escalation of therapy require balancing the financial implications, risks of long-term immunosuppression against the risk of relapse, and the potential of unsuccessful re-challenge. Approximately one third of IBD patients relapse within 12 months of discontinuing biologics.<sup>1</sup> We aimed to identify the factors associated with relapse and remission.

**Methods** The IBD register at a single district general hospital was analysed for all patient who had received biologic therapy