**PTH-113**  CLINICAL RESPONSE TO INDUCTION ANTI-TNF THERAPY HAS NO EFFECT ON HAEMOGLOBIN LEVELS IN PATIENTS WITH IBD

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**Introduction** Iron deficiency anaemia (IDA), as a consequence of intestinal bleeding and inflammation, is arguably the most common complication of inflammatory bowel disease (IBD). In controlled trials, oral iron is reportedly as effective as IV iron; however, few studies have included patients with active disease where inflammation through the release of hepcidin may limit the efficacy of oral or dietary iron.

**Methods** To further study the relationship between inflammation and anaemia, we hypothesised that haemoglobin (Hb) levels would rise more in anaemic patients that responded, than in patients who did not respond, to induction anti-TNF therapy. Using electronic case note review, we assessed the prevalence, severity, type and mean change in Hb (ΔHb) of 174 [97 Male] consecutive patients undergoing induction therapy with an anti-TNF agent [Infliximab 139; Adalimumab 35]. Anaemia was defined according to age-sex adjusted WHO criteria. Primary response was assessed at 4 weeks and defined as, at least two of; the absence of symptoms, having withdrawn from steroids, and/or a normal serum C-reactive protein. Non-response was defined as one or none of the above.

**Results** 89% [156/174] patients had Crohn’s disease: the mean [SD] age, age at diagnosis, and disease duration were 34 [17], 24 [13], 10 [10] years respectively. Overall 49% [85/174] patients were anaemic at initiation of anti-TNF treatment with a mean [SD] haemoglobin of 10.9 [1.3] g/dl. Only 48% [41/85] had haematinsics checked within 3 months of commencing an anti-TNF. 65% [26/41], 31% [13/41] had anaemia of chronic disease and 5% [2/41] were folate deficient. Overall, 37% [31/85] of the anaemic patients were prescribed iron therapy. Considering all anaemic patients, there were no differences in the baseline Hb in patients who responded (11.1 [1.2] g/dl) compared with those who did not respond (10.6 [1.6] g/dl, p = 0.11) to anti-TNF therapy. Regardless of concurrent iron therapy, there was no difference in the mean [SD] change in Hb (ΔHb) between patients that responded to anti-TNF therapy (0.51 [1.2] g/dl) and those who did not (0.47 [1.4] g/dl, p = 0.85).

**Conclusion** Iron deficiency anaemia is common but frequently undertreated in IBD patients receiving anti-TNF therapy. Induction anti-TNF therapy, with or without oral iron therapy, has no effect on haemoglobin levels. In patients with IDA receiving anti-TNF therapies, gastroenterologists should consider IV iron therapy.

**Disclosure of Interest** None Declared.

**REFERENCES**


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**PTH-114**  SKIN PATHOLOGY ASSOCIATED WITH ANTI-TUMOUR NECROSIS FACTOR (ANTI-TNF) THERAPY- A SINGLE UK IBD CENTRE EXPERIENCE

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**Introduction** With the increasing use of biologic therapy in the treatment of inflammatory bowel disease (IBD) there has been a reported increase in dermatological conditions associated with therapy in patients with IBD. We carried out a prospective audit to identify the proportion of IBD patients at The Royal Free Hospital on anti-TNF therapy developing therapy related inflammatory skin pathology.

**Methods** 141 IBD patients on anti-TNF therapy (either infliximab or adalimumab) were sent a postal questionnaire to identify patients who had experienced identifiable and associated skin conditions. The questionnaire included information regarding the body site affected, dermatology opinion and whether therapy had to be stopped. Data for infliximab and adalimumab were analysed.

**Results** Of 141 patients, 105 replied (71 (74%) infliximab and 34 (74%) adalimumab). In both groups 32% of patients described new skin complaints attributable to anti-TNF therapy (n = 23 in infliximab group, n = 11 in adalimumab group). Sites of skin inflammation were common to both groups; face (29%), trunk (21%), legs (14%) and arms (14%). Combined data showed only 44% of patients were reviewed by a dermatologist and received a formal diagnosis. No patients on adalimumab stopped treatment, while 3 stopped therapy in the infliximab group (9% overall).

**Conclusion** Although IBD is itself associated with skin pathology, recent studies have demonstrated that patients on anti-TNF therapy develop inflammation of the skin (1), and our data support the concept that paradoxical skin inflammation related to anti-TNF therapy is a class effect. [c1] In our cohort few patients had to stop therapy which is similar to some (2), but not all reported studies (3). Less than half of affected patients received consultant dermatological review.

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**REFERENCES**


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**PTH-115**  OUTCOME OF ULCERATIVE COLITIS PATIENTS THAT HAVE FAILED CONVENTIONAL THIOPURINE THERAPY; ALLOPURINOL CO-THERAPY AND BEYOND

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**Introduction** Treatments available for Ulcerative Colitis (UC) are limited in those who fail thiopurines. British society of gastroenterology guidelines suggest the use of methotrexate (MTX) or colectomy in this group as biologics are not funded in UC in England or Wales. Clinical treatments used in our unit include low dose thiopurine and allopurinol co-therapy (LDTA) and topical arsenic. We report our experience of patients who have failed standard dose thiopurines and their subsequent treatment and clinical outcomes.

**Methods** UC patients who failed conventional thiopurine therapy were identified from our prospective inflammatory bowel disease database. Reasons for failure, disease extent, length of follow-up and subsequent treatment pathway were identified. Data from patients with <3 months follow-up was disregarded.

**Results** 20 patients were identified, 12 initially started LDTA and 10 started MTX (2 patients received LDTA and then MTX). Mean age was 47 (range: 22–60yrs), 15 were male. 8 had pancolitis, 9 left sided UC and 4 proctitis. Reasons for failure of conventional dose thiopurine were; Hepatits (n = 5), therapeutic 6-thioguanine levels without clinical remission (n = 11), intolerable side effects (n = 3), dyserthropoiesis (n = 1) and gout (n = 1).
Of those patients commenced on LDTA; 10 (83%) entered clinical remission. 2 (17%) failed due to lack of clinical response and commenced MTX. 10 remain in a sustained clinical remission at a mean length of follow up of 16.2 months (range: 4–23).

Of the 10 patients commenced on MTX; 7 (70%) failed due to lack of clinical response and 1 (10%) due to side-effects (Pneumonitis). Of those patients that failed; 4 (40%) underwent colectomy, 2 (20%) received arsenic suppositories and entered a sustained clinical remission. 2 (20%) were reclassified to Crohn’s disease, were treated with biologic therapy and entered a sustained clinical remission. Mean length of follow up in this group was 17.6 months (range: 2–30).

**Conclusion**

Thiopurines remain the mainstay of treatment for patients with UC. A significant number of patients fail this conventional treatment and represent a clinical challenge. Novel treatments such as LDTA can be effective in a significant proportion of this group. Data for the efficacy of MTX remains less effective and topical arsenic is useful and can be helpful.

**Disclosure of Interest**

T. Hollingworth: None Declared, H. JOHN-SON Conflict with: SPONSORSHIP FROM FALK, ABBOTT & WARNER CHILCOTT TO ATTEND MEETINGS, R. BASUROY: None Declared, S. MCLAUGHLIN Conflict with: SPONSORSHIP FROM FALK TO ATTEND MEETINGS, S. WEAVER Consultant for: MSD ADVISORY BOARD, Conflict with: SPONSORSHIP FROM FALK, ABBOT , MSD & FERRING TO ATTEND MEETINGS

**REFERENCE**

1. Microscopic Colitis in Tayside: Clinical features, associations and behaviour. Mowat C, Heron T, Walsh S. Gastroenterology 2005: 128 (4); A331

**Microscopic Colitis in Tayside – Further Observations on Clinical Features and Outcome**

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**Introduction**

The aetiology of microscopic colitis remains unknown. We have previously reported our experience of microscopic colitis diagnosed in our region between 1999 – 2004(1). Although data continue to emerge, the natural history of microscopic colitis remains unclear. Furthermore, there are reports of macroscopic changes in the mucosa at endoscopy.

**Methods**

Cases from 2004–2011 were identified from pathology records. Case notes were retrospectively reviewed and data extracted including subtype of microscopic colitis and clinical details where possible.

**Results**

82 case notes were obtained and reviewed. 8 cases did not have a clear diagnosis and were excluded. Of the remaining 74 cases, 56 were collagenous colitis, 18 lymphocytic colitis. 17 patients had macroscopic abnormalities (excluding diverticulosis) at endoscopy, 16 of these in the collagenous colitis group, representing 28% of this subgroup. The mean age was 46.1 (range 33–87), female: male ratio of 4.3:1. 18 reported an autoimmune condition including 2 coeliac disease and 7 hypothyroidism. 30(40%) were on a proton pump inhibitor at the time of diagnosis and 15(20%) were on non-steroidal anti-inflammatory drugs. Follow up data was available for 66 patients. Of these 37(71%) reported complete resolution of symptoms and 15(22%) partial resolution. 5(7%) did not respond in the follow up period. Therapeutic strategies included either alone or in combination of stopping/switching PPI, loperamide, mesalazine and steroids. 24/47(65%) of complete responders required simple intervention (PPI withdrawal, switch in brand of PPI, loperamide or even spontaneous resolution) whereas 10/47(21%) required steroids. 7/15(47%) partial responders received steroids.

**Conclusion**

Since microscopic colitis was last studied in our region, the female predominance has increased, the mean age has dropped by almost 20yrs, and the ratio of collagenous : lymphocytic colitis has increased from 2:1 to 3:1. This could represent a change in the number of younger people investigated or missing data in our cohort. A significant number of patients with a diagnosis of collagenous colitis had endoscopic abnormalities in comparison to the lymphocytic colitis group, which does raise the question of the nomenclature. The majority of patients have complete resolution of symptoms with simple intervention.

**Disclosure of Interest**

None Declared.

**REFERENCE**

1. Microscopic Colitis in Tayside: Clinical features, associations and behaviour. Mowat C, Heron T, Walsh S. Gastroenterology 2005: 128 (4); A331

**Predicting the Need for Dose Escalation in Patients with Crohn’s Disease Treated with Adalimumab**

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**Abstract PTH-116 Figure 1**

**PTH-117**

**Predicting the Need for Dose Escalation in Patients with Crohn’s Disease Treated with Adalimumab**

**REFERENCE**


**Disclosure of Interest**

None Declared.