

that were recommended to them. Although 77% were aware of a form of recommended vaccine, 39% were not receiving them. Wider education of our IBD patients as well as their primary care doctors should be implemented to increase awareness and uptake of vaccines to provide adequate protection to this vulnerable group.

Disclosure of Interest None Declared.

PWE-102 THE USE OF TPMT ENZYME TESTING IN MANAGING THIOPURINE THERAPY

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¹M Widlak, ¹M Smith, ¹J Slater, ¹L Wood, ¹S De Silva. ¹Gastroenterology, Russells Hall Hospital, The Dudley Group NHS Foundation Trust, Dudley, UK

Introduction Thiopurine methyl transferase (TPMT) enzyme deficiency is associated with azathioprine-induced myelosuppression and hepatotoxicity. We aimed to assess thiopurine methyl transferase levels (TPMT) in patients with inflammatory bowel disease (IBD) and to determine how these levels impact on thiopurine dosing and further treatment.

Methods Retrospective analysis of all adult IBD patients treated with azathioprine (AZA) who had TPMT levels checked in our department 2008 – 2012. Patient demographics, AZA dose, blood parameters, adverse effects and reason for discontinuation were recorded. Patients were treated to try and achieve a target dose of 2.0 – 2.5 mg/kg of AZA. Our laboratory enzyme assay defined normal TPMT level as 68–150 mU/L.

Results A cohort of 135 patients receiving azathioprine were identified. Mean age was 43 years (SD 17), M:F ratio 1:1. 72 patients (53%) had Crohn's disease, 60 (44%) ulcerative colitis and 3 (3%) indeterminate colitis. Mean starting dose of AZA 1.7 mg/kg (SD 0.6), maintenance dose 1.8 mg/kg (SD 0.5). 63 (47%) were initially dosed with ≥ 2.0 mg/kg. The mean TPMT level was 100 mU/L (SD 23); 14 patients (10%) had low levels. Patients with low TPMT levels were generally started at lower doses of AZA 1.3 mg/kg (SD 0.7) vs 1.8 mg/kg (SD 0.6).

Mean length of follow up on AZA was 10.6 months (range 0.1 – 44). 50 out of 135 patients (37%) exhibited adverse effects during treatment (representing 44 of the 121 (36%) with normal TPMT levels, 6 of 14 (43%) with low TPMT). Median time to adverse effects was 3 months (range 0.1–22). Leucopenia developed in 11 (22%), deranged liver biochemistry 24 (48%), adverse symptoms 24 (48%). There was one case of pancreatitis and lymphoma respectively. Adverse effects were managed by discontinuation in 30 (60%), dose reduction 14 (28%), monitoring only 6 (12%).

In the 6 patients with low TPMT levels, 4 developed deranged liver biochemistry and 2 had leucopenia; in 50% AZA was discontinued. The median time to adverse effects was 11 months (range 2–22), with 5 out of 6 patients developing adverse effects after more than 6 months of treatment.

Conclusion In our cohort low TPMT enzyme activity was not associated with an early onset of adverse effects or an excess of adverse effects when compared to the normal TPMT group. We would suggest that the value of TPMT level testing prior to thiopurine therapy may be less of a priority than vigilant monitoring of blood parameters during AZA therapy in identifying serious adverse effects.

Disclosure of Interest None Declared.

PWE-103 VITAMIN D DEFICIENCY IN CROHNS DISEASE AND ULCERATIVE COLITIS

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¹M S Mohammad, ¹J Mayberry. ¹Gastroenterology, University Hospitals of Leicester, Leicester, UK

Introduction The role of vitamin D in inflammation and its possible cancer protective effects are now coming to the fore. Inflammatory bowel disease (IBD) provides a model where both roles can be assessed. Early studies have indicated that vitamin D deficiency is common amongst patients with this condition. Leicester in the UK has a significant population of patients from both English and South Asian backgrounds and so allows comparisons to be made between people of different ethnicities and different experiences of vitamin D deficiency.

Methods Vitamin D (25 OH D) status was measured in 148 consecutive patients with IBD, who attended the gastroenterology clinics during a period of one year (Jan to Dec 2011). Vitamin D levels were graded as severe deficiency (< 15 nmol/l), moderate deficiency (16–30 nmol/l), insufficiency (31–50 nmol/l), adequate (51–75 nmol/l), and ideal (> 76 nmol/l). All case notes were reviewed and data analysed using χ^2 statistic.

Results 148 patients had IBD, 64% were women and 36% men. 93 patients (61%) with ulcerative colitis (UC) and 55 (39%) Crohns disease (CD). Of the 93 patients with UC, 50 were English and 43 South Asian. Of the 55 patients with CD, 43 were English and 12 of South Asian background. The proportions amongst both communities and across the disease groups were not significantly different when analysed with a χ^2 statistics.

Abstract PWE-103 Table Vitamin D Levels in patients with inflammatory bowel disease in Leicester

Ulcerative colitis	< 15nm/lt	15–30nm/lt	30–50nm/lt	> 50nm/lt
English (50 pts)	3(6%)	17(34%)	12(24%)	18(36%)
Asian (43 pts)	16(37%)	13(31%)	12(28%)	12(28%)
Crohns disease				
English (42 pts)	7(6%)	14(34%)	8(19%)	13(31%)
Asian (12 pts)	2(37%)	4(31%)	3(25%)	3(25%)

Conclusion Vitamin D deficiency is common in all patients with inflammatory bowel disease. It plays a vital role in bone health, immune regulation and cancer prevention in IBD. The optimal target level of 25(OH) vitamin D in IBD patients is uncertain as is the best therapeutic modality. The precise threshold for 25(OH) vitamin D level is poorly defined in the literature and so is the dosing and duration of treatment. Its frequency is almost similar in South Asian and English patients with Crohn's disease which suggests that mal-absorption of the vitamin may play a part in the vitamin D deficiency. In contrast it is commoner in South Asian patients with ulcerative colitis than amongst English patients and this warrants further investigation. It may suggest that in the future South Asian patients will be at greater risk of colonic carcinoma as a complication of ulcerative colitis.

Disclosure of Interest None Declared.

PWE-104 VITAMIN D IN INFLAMMATORY BOWEL DISEASE. A SEMI-QUALITATIVE ASSESSMENT OF THE PATIENT EXPERIENCE

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¹M S Mohammad, ¹J Mayberry. ¹Gastroenterology, University Hospitals of Leicester, Leicester, UK

Introduction Vitamin D plays a vital role in bone health, immune regulation and cancer prevention in inflammatory bowel disease (IBD). Our understanding has increased remarkably in the past decade, although the mechanism of its influence in IBD

remains unclear as does its role in treatment. Although the efficacy of vitamin D as an immunomodulator remains to be established, given current evidence it appears reasonable to screen and treat vitamin D deficiency in patients with IBD.

Methods Two hundred consecutive patients with vitamin D deficiency were identified from IBD clinics at Leicester General Hospital who had been seen during a 12 month period. A postal questionnaire was sent to these patients. It was anonymous and requested whether: Respondents believed they had been told of their vitamin D deficiency. General practitioners had prescribed vitamin D supplements. Vitamin D supplements had been bought at local pharmacies. Brand of vitamin D supplements taken. Compliance with treatment. Symptoms had improved with treatment. They had enough sunlight exposure

Results Ninety eight of the 200 patients responded to the questionnaire, a response rate of 49%. Sixty five were English and 33 Asian. The response rate was 48% in Asians and 50% in English which is not significantly different. Seventy (71%) of the 98 patients, recalled being told they were vitamin D deficient. Thirty nine patients (40%) believed they had sufficient exposure to sunlight. 54 (71%) had been prescribed this medication by general practitioners and 22 (29%) had bought vitamin D supplements over the counter at local pharmacies or herbal stores. General practitioners predominantly prescribed Adcal D3 tablets. Sixty four (84%) patients said they were adherent to vitamin D treatment. Symptoms improved significantly in 29 (38%) patients. However, this figure rose to 45% when only those 64 patients who were compliant with therapy were considered.

Conclusion Vitamin D plays a significant role in intracellular functions which extends beyond its effects on bone metabolism. It is an important regulator of the immune system which may have implications for the development, severity and management of immune related disorders such as IBD. In summary the relationship between the vitamin D axis and IBD is multifaceted. It should comprise maintenance of musculoskeletal health and control of disease through immunomodulation and modification of associated malignancy.

Disclosure of Interest None Declared.

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PWE-105 DO GASTROENTEROLOGISTS MONITOR THEIR PATIENTS TAKING 5-AMINOSALICYLATES FOLLOWING INITIATION OF TREATMENT ?

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¹N Siddique, ²C Farmer, ³J Irving, ⁴A Muller. ¹Dept of Gastroenterology; ²Dept of Nephrology, The Kent & Canterbury Hospital, Canterbury, UK

Introduction 5-Amino salicylate (5-ASA) medications may rarely be associated with a significant decline in renal function and interstitial nephritis. The British Society of Gastroenterology guidelines¹ advise regular renal function monitoring for patients (pts) taking these drugs. This study assessed whether clinicians were following best practise guidelines.

Methods Using longitudinal community and regional pathology databases for the East Kent population (720 k), our renal unit regularly screens a total population of 300,000 for evidence of renal disease. The data extracted is analysed using the SEIK (System for Early Identification of Kidney disease), an automated computerised

system to identify pts requiring intervention for kidney disease. As part of this process, all patients taking 5-ASA medication were identified. The pathology database was studied to identify the pts on treatment who had had renal function tests and could differentiate from results initiated in primary and secondary care. Data analysis can be performed over many years of treatment.

Results 800 adult pts (M : 341, F 459) identified taking 5-ASA therapy (median duration 1.5 years (range 1–24 yr); mean (+/- SD) age 52.7 +/- 16.2 yr (range 18.2–94.4). The mean estimated Glomerular Filtration Rate (eGFR) on commencing 5-ASA therapy was 82 ml/min (range 28 - > 90). Pts with an eGFR < 60 were regarded as having chronic kidney disease (stage 3–5). 612 pts received 5-ASA's for 3 months or more (median 3.2; range 1 – 24 yr) and these were included in the final analysis.

293 (48%) pts had no renal function cheques whilst on treatment. 79 (12%) pts had renal function tests less than once every 4 years and 36 pts once every 2 – 4 yrs. 204 pts had renal function measurements in 50% or more of years of treatment, of whom 116 were checked every year. 72 pts with a baseline eGFR < 60 ml/min were treated with 5-ASA's for 3 or more months. Of these, 8 had no renal function cheques. The eGFR fell in 24 pts and in 8 by > 2ml/min/yr.

Conclusion The majority of pts receiving 5-ASA compounds do not have regular renal function monitoring. Some are started on treatment with abnormal results at baseline and some with identified kidney disease continued on their 5-ASA's.

Gastroenterologists are failing to follow best practise guidelines.

Disclosure of Interest None Declared.

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PWE-106 ASSOCIATION STUDY OF IL23R AND ATG16L1 VARIANTS IN IBD MOROCCAN PATIENTS

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¹N Serbati, ¹N Senhaji, ¹B Diakite, ²W Badre, ¹S Nadifi. ¹Laboratory of Medical Genetics, Medical school of Casablanca - University Ain Chock Hassan II, Center Of Doctoral Sciences "In Health Sciences"; ²Department of Gastro-enterology, CHU Ibn Rochd - Casablanca - Medical school of Casablanca, Casablanca, Morocco

Introduction IBD (Crohn's disease and Ulcerative Colitis) is chronic and multifactorial disease of the gastrointestinal tract. Although several studies have tried to explore these diseases, their pathogenesis is still unclear. Recently, CD has been associated with the variants in interleukin 23 receptor (*IL23R*) and autophagy-related 16-like 1 (*ATG16L1*) genes. The aim of our study was to assess the frequency of *ATG16L1* (T300A) and *IL23R* (L310P) variants in Moroccan IBD patients and to determine a possible effect of these variants on Disease's phenotype and clinical course.

Methods we genotyped a group of 96 Moroccan IBD patients and 114 unrelated volunteers for *ATG16L1*(T300A) and *IL23R* (L310P) variants.

Results Our results showed no significantly increased risk of Crohn's disease among individuals carrying the GG genotype or the G allele for the (Thr300Ala) polymorphism, in contrast to the (L310P) polymorphism which confers a protective effect. We also noticed the presence of a positive correlation between Crohn's disease Type and *ATG16L1* polymorphism. For UC, the carriage of the mutated allele in the *ATG16L1* gene confers a protective effect.

Conclusion Our results showed a limited role of *ATG16L1* and *IL23R* variants in the Moroccan population

Disclosure of Interest None Declared.