Outcomes of GP Outreach Programme offering colonoscopic surveillance for IBD patients managed in primary care

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Introduction Colonoscopic surveillance in IBD patients can reduce the development of colorectal cancer (CRC) and the rate of CRC-associated death. We recently reported that 27% of IBD patients living in East Devon are managed exclusively in primary care of whom about 23% maybe eligible for colonoscopic surveillance. We devised an outreach programme, whereby we invited primary care physicians to enrol these patients in a colonoscopic surveillance programme.

Methods In December 2017 we contacted 37 general practices, where 161 patients with UC who were eligible for surveillance had been identified. Each practice was sent a letter explaining the goals of the project, a link to the National Institute for Healthcare and Clinical Excellence (NICE) guidance for CRC surveillance in IBD patients and patient information booklets. We informed the practices of their eligible patients and asked them to refer patients for secondary care IBD consults if appropriate. We included an outcome form that captured whether the patient was referred, was deemed inappropriate for surveillance, had surveillance elsewhere, had declined surveillance, or was no longer registered at the practice.

Results Sixty-five percent of practices (24/37) responded and we received responses for 57 of 161 (35%) potentially eligible patients. Thirty-five (61%) patients were referred to our IBD service; 7 (12%) patients declined surveillance; 7 (12%) patients were deemed by their GP to be unfit for surveillance and 5 (10%) were no longer registered at the identified GP practice; 2 (4%) had surveillance arranged elsewhere and 1 (2%) patient had died. Amongst the 35 patients referred to secondary care; 22 (63%) underwent surveillance colonoscopy, 12 (34%) declined surveillance after discussion or did not attend their booked appointments and one is awaiting colonoscopy. Half of patients who had a colonoscopy had active inflammation. We diagnosed one CRC. He was an elderly man with a locally invasive signet ring caecal tumour, without distant metastases, who went onto to have a curative right hemicolectomy without complication.

Conclusions Patients with longstanding IBD are frequently managed exclusively in primary care and maybe overlooked for colonoscopic CRC surveillance. There is a need to implement processes to facilitate identification and recall of patients eligible for surveillance across primary and secondary care.
and 52d) during food re-introduction (FR). Microbiome was assessed by 16s RNA gene sequencing of the V4 region performed on the MiSeq (Illumina). Community structure was resolved at 97% similarity operational taxonomic unit (OTU). Short chain fatty acids (SCFA) were quantified with gas chromatography and are expressed in μmol/g. Faecal calprotectin (FC) was measured using the CALPROLAB0170 (ALP) (Lysaker, Norway) ELISA kit. Continuous data are present as mean and standard deviation unless otherwise stated.

Results 66 CD patients were recruited (Female 25; age 13.4 yr). Clinical remission (wPCDAI<12.5) was achieved in 41 (62%). During EEN there was an increase in Shannon diversity (start: 0.3 [0.22] vs 30d EEN: 0.48 [0.2], p<0.001; vs 56d EEN: 0.43 [0.27], p=0.05). During FR these indices did not change.

Based on β-diversity dispersion analysis, estimated using Bray-Curtis distance, EEN induced clear alterations to the microbiome. Permutation ANOVA was used to identify significant changes to the microbiome during EEN. Most of the change that occurred was apparent within the first 4 weeks of treatment with R2: 4.7% (p=0.001) and by the end of EEN R2: 3.2%, (p=0.001).

In patients to enter remission using EEN, we observed a quick reversion in the microbiome composition to that of pre-treatment (p=0.23).

Assessing the metabolic activity of the microbiome we observed a significant decrease in the concentration of acetate (start: 423.6 [183.6], end: 224.9 [101.5]; p< 0.001), propionate (start: 93.8 [50.6], end: 55.7 [27.3]; p< 0.001) and butyrate (start: 95.0 [64.2], end: 41.0 [57.7]; p< 0.001). During FR, there was a rapid reversion in levels of acetate and propionate (acetate EEN end: 224.9 [101.5] vs 17d FR: 362.4 [179.7]; p=0.003; propionate EEN end: 55.7 [27.3] vs 17d FR: 93.0 [46.9]; p=0.002).

Faecal calprotectin significantly decreased during EEN (start: 1402.4 [586.3]; 4wk EEN: 877.5 [593.1], p<0.001; 8wk EEN: 720 [664], p<0.001) and was quickly reversed during food re-introduction (17d FR: 1025 [603], p=0.025; 52d FR: 1105 [651], p=0.003)

Conclusions EEN induces specific effects on faecal microbiome and markers of functional activity. This is characterised by a reduction in metabolic activity during EEN, with reversion to pre-EEN state during food re-introduction paralleling an elevation of faecal calprotectin.

### Abstracts

**WHOLE BLOOD PROFILING OF T-CELL DERIVED miRNA ALLOWS THE DEVELOPMENT OF PROGNOSTIC MODELS IN IBD**

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10.1136/gutjnl-2020-bsgcampus.14

Introduction There is an unmet need for blood-based biomarkers that help predict disease and its course at inception to allow tailoring of treatments, achieve early mucosal healing and improve clinical outcomes. In our study, we explore the clinical utility of miRNAs in Inflammatory bowel disease (IBD).

Methods A 2-stage prospective multi-centre case control study was performed. Small RNA sequencing was performed on a discovery cohort of immunomagnetically separated leucocytes (90 CD4+ & CD8+ T-lymphocytes and CD14+ monocytes) followed by validation in a larger case control cohort. 22 protemic miRNA were selected for qPCR analysis.

Results The selected miRNAs were significantly upregulated in IBD (CD) vs controls (C), particularly in CD14+ monocytes (3 were significantly higher in CD vs C and C14+ vs C). Results were validated in the larger cohort.

Conclusions We identified a significant signature of miRNAs differentially expressed in IBD, which can be used to discriminate IBD patients from healthy controls. We are currently validating these results in an expanded cohort of patients.