OC-048 ADENOMA DETECTION REDUCES DURING A COLONOSCOPY LIST

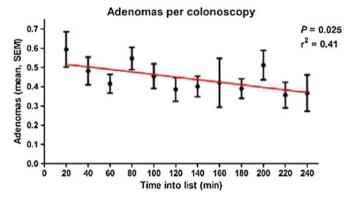
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R Gao,* G D Corbett, J Lee, F Sampaziotis, E Cameron. Department of Gastroenterology, Addenbrookes Hospital, Cambridge, UK

Introduction Recent studies have provided conflicting data on whether colonoscopy lesion detection varies with time of day.^{1–3} We aimed to assess whether the time of procedure within a list affected polyp and adenoma detection rate (PDR, ADR), surrogate markers for colonoscopy quality, at a busy tertiary centre endoscopy unit in the UK.

Methods All patients undergoing colonoscopy in 2009 were retrospectively identified and included in the study. Patient demographics and colonoscopic findings were obtained from endoscopy and histology reports. Polyp detection rate, adenoma detection rate, polyps per colonoscopy (PPC) and adenomas per colonoscopy (APC) were calculated. Morning and afternoon lists were combined as both are 4 h long and results were analysed in 20 min segments. Statistical significance was determined by linear regression.

Results During the 12-month study period, 3923 colonoscopy procedures (60.4 ± 15.3 years, 52.7% female) were performed by 42 endoscopists. 3718 procedures were available for analysis after excluding cases performed in the cross-over period between lists (1300-1400) and out of hours. The average PDR was 35.9% and the ADR 23.9%. Although PDR (p=0.27) and ADR (p=0.24) did not vary significantly during the course of a list, PPC (p=0.015, Abstract OC-048 figure 1) and APC (p=0.025) significantly reduced as lists went on. Results are shown in Abstract OC-048 table 1.



Abstract OC-048 Figure 1 Linear regression analysis for APC (p=0.025) during the course of a colonoscopy list.

Abstract OC-048 Table 1 PPC and APC at time periods during a list

Time into list	20 min	40 min	60 min	80 min	100 min	120 min
Cases	421	397	380	360	365	367
PPC	1.08	0.81	0.82	0.95	0.80	0.70
APC	0.59	0.48	0.42	0.55	0.45	0.39
Time into list	140 min	160 min	180 min	200 min	220 min	240 min
Cases	339	312	270	238	172	97
PPC	0.70	0.75	0.66	0.90	0.68	0.64
APC	0.40	0.42	0.39	0.51	0.35	0.37

Conclusion The mean number of polyps and adenomas detected falls significantly during the course of a colonoscopy list. Notably, our data shows no difference in the traditional "detection rate" measures (ADR and PDR) and thus highlights that, in isolation, these may be inadequate measures of colonoscopy quality, and some measure of

the number of adenomas detected is also required. The fall in number of adenomas per colonoscopy may relate to operator fatigue and implies that significant lesions could be missed at the end of a list. Further study is required to establish whether revising the length of endoscopy lists or providing a break during a list could prevent this drop off in performance.

Competing interests None declared.

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OC-049 DUODENAL BULB BIOPSIES—ARE THEY A NECESSITY IN COELIAC DISEASE?

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¹M Kurien,* ¹K E Evans, ¹I Aziz, ²S S Cross, ¹A D Hopper, ³M Hadjivassiliou, ¹D S Sanders. ¹Department of Gastroenterology, Royal Hallamshire Hospital, Sheffield, UK; ²Department of Pathology, Royal Hallamshire Hospital, Sheffield, UK; ³Department of Neurology, Royal Hallamshire Hospital, Sheffield, UK

Introduction Historically, Brunner's glands in the bulb were thought to cause histological interpretation difficulties, however recent studies have demonstrated that this area maybe the only site to demonstrate villous atrophy (VA) and thus detect Coeliac Disease (CD). This study evaluates the diagnostic yield of taking duodenal bulb biopsies in coeliac patients compared with controls.

Methods Patients undergoing clinically indicated oesophogastrodudoenoscopy (OGD) were prospectively recruited from a single tertiary referral centre between November 2008 and December 2011. Indications for OGD included positive coeliac serology, family history of coeliac disease, diarrhoea, iron deficiency anaemia, abdominal pain and weight loss. All biopsies were graded using the Marsh criteria, with patients being assigned to one of three groups: Group 1 (CD: New Diagnosis), Group 2 (CD: Remission) and Group 3 (Controls).

Results 550 patients (360 female) with median age 51 (range 15–89 years) were prospectively recruited. 153 had newly diagnosed celiac disease, 91 established celiac disease, and 306 controls. New diagnosis celiac disease (9%, p<0.0001) and established celiac disease (14%, p<0.0001) were more likely than controls to have VA in the bulb alone (Abstract OC-049 table 1). Overall, when comparing the histological lesion of the bulb against the distal duodenum, 36/91 (40%) with established celiac disease (p<0.0001) and 35/153 (23%) newly diagnosed (p<0.0001) had a discrepancy in the severity of the lesion between the two sites compared with 22/306 (7%) controls. In all, 28/36 with established celiac disease and 24/35 newly diagnosed had the more severe lesion in the bulb. One patient in the control group had VA. This patient was HIV positive with positive tTG and negative EMA, however the HLA status was incompatible with coeliac disease.

Abstract OC-049 Table 1 Histology, serology in coeliac disease and controls

	N	+ve Coeliac serology	VA in D1	VA in D2	VA in D1 only	VA in D2 only
Controls	306	55 (18%)	1 (0.0%)	1 (0.0%)	0	0
CD: new	153	142 (93%)	146 (95%)	139 (91%)	14 (9%)	7 (5%)
CD: remission	91	60 (66%)	40 (44%)	29 (32%)	13 (14%)	2 (2%)

Conclusion This is the largest prospective study evaluating the value of a duodenal bulb biopsy strategy. VA may only be present in the

duodenal bulb. In this study 14/153 (9%) of newly diagnosed coeliac disease patients and 13/91 (14%) of CD remission patients demonstrated VA in the bulb alone. We suggest that endoscopists should consider taking a duodenal bulb biopsy in patients suspected of having coeliac disease and in reassessment cases.

Competing interests None declared.

BSG transition symposium

OC-050 INPATIENT PAEDIATRIC UC CARE IN THE UK IN 2011 IS CHARACTERISED BY INCREASING RATES OF RESCUE THERAPY AND STOOL CULTURES BUT LOW USE OF PUCAI SCORES

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¹R K Russell,* ²A Protheroe, ²M Roughton, ³N Croft, ³M S Murphy, ³C Spray, ³A Rodrigues, ³D C Wilson, ³J Puntis, ³M Cosgrove, ³A Tarnock, ³P Rao, ²C Down, ²I Arnott, ⁴S Mitton. ¹Yorkhill Childrens Hospital, Glasgow, UK; ²Clinical Effectiveness and Evaluation unit; ³BSPGHAN IBD audit lead; ⁴St Georges Hospital, London, UK

Introduction Paediatric UC care is variable in the UK and appropriate clinical guidelines are very recent. Acute severe UC is rare with only a few cases presenting annually to each tertiary hospital.

Methods UC patients aged <17 years admitted to 23 UK paediatric hospitals had clinical details collected as part of the UK paediatric IBD audit (September 2010–2011). Each site was asked to enter up to 20 cases admitted electively or as an emergency, including patients who were having surgery. Day cases and patients who were admitted solely for diagnostic endoscopy were excluded. Comparative data for some items was available from the previous UK audit conducted in 2008.

Results 176 patients (98 males) of median age 13 years (IQR 10–13) were included in the audit; 22 were elective surgical admissions, 47 new diagnoses and 107 needed acute medical care for known UC. Median length of stay was 6 days (IQR 3-10); there were no deaths. 73% of patients with established disease had a pancolitis and 10% had co-existent liver disease. 88 (70%) of 126 patients with active disease had standard stool cultures performed (2% were positive) and 57 (45%) had C difficile toxin tested (none positive). Stool sample collection rates had improved significantly compared to the 2008 audit (70% vs 52%, p=0.001). 38% of emergency admissions had a plain abdominal XR taken on admission, but only 19% had a specific disease activity index (PUCAI score) recorded. There were three cases of toxic megacolon and 3 of thromboses. Rates of heparinisation were low but higher than in the 2008 audit (11% vs 2%, p=0.002). 71% of patients treated with steroids responded to treatment. 20 patients received 2nd line (rescue) therapy, of whom eight received infliximab, 11 Cyclosporin and one both, with an overall response rate of 90%; nine went to surgery without 2nd line medical therapy. Rescue therapy usage was significantly higher than in the 2008 audit (52% vs 26%, p=0.03). Overall, 71% of non-elective UC admissions were seen by an IBD nurse.

Conclusion There were signs of improving UC care from 2008 to 2011 with significantly increased rates of stool culture sampling and use of rescue therapy, but the majority of sites did not use PUCAI scores to assess patients on emergency admission.

Competing interests None declared.

OC-051 MICROBIAL, PHENOTYPIC AND GENETIC MARKERS OF RISK: ASPECTS OF CROHN'S DISEASE THAT ARE SHARED BY UNAFFECTED SIBLINGS

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^{1.2}C R Hedin,* ³K Taylor, ⁴P Louis, ⁴F Farquharson, ⁵S McCartney, ³N J Prescott, ²A J Stagg, ²J O Lindsay, ¹K Whelan. ¹Division of Diabetes and Nutritional Sciences, King's College London, London, UK; ²Blizard Institute, Queen Mary University of London, London, UK; ³Department of Medical and Molecular Genetics, King's College London, London, UK; ⁴Microbiology Group, Rowett Institute of Nutrition and Health, University of Aberdeen, Aberdeen, UK; ⁵Department of Gastroenterology, University College Hospitals NHS Foundation Trust, London, UK

Introduction Siblings of Crohn's disease (CD) patients have elevated risk of developing disease which may manifest as raised faecal calprotectin (FC) and increased intestinal permeability. Other features of CD that reflect the at-risk state and their interactions are not well described.

Aim Identify key facets of the at-risk state and establish their relationships.

Methods Faecal samples from 22 patients with quiescent CD, 21 siblings and 25 controls were analysed for FC (ELISA) and microbiota (quantitative PCR targeting bacterial 16S ribosomal RNA genes). Genotype RR at 72 known CD risk loci was determined by Illumina Immuno BeadChip, analysed with the REGENT R package. p Values from non-parametric analyses were Bonferroni corrected to adjust for multiple testing.

Results FC was elevated (>50 μ g/g) in 21 (96%) patients, 8 (38%) siblings and 2 (8%) controls. Compared with controls, patients had reduced Bacteroidetes, clostridial cluster IV, *Faecalibacterium prausnitzii*, *Ruminococci*, *Bifidobacterium adolescentis* and *Roseburia* spp. (Abstract OC-051 table 1). Similarly, in siblings vs controls there was reduced cluster IV clostridia, (specifically *F prausnitzii*) and *Roseburia* spp. (Abstract OC-051 table 1). This dysbiosis was evident in siblings with normal FC. Siblings with elevated/high genotype RR (n=4) had normal FC but reduced Bacteroidetes vs controls, (9.2 vs 10.4 log₁₀/g, p=0.007).

Conclusion In addition to enhanced genotypic risk and elevated FC, siblings share aspects of CD dysbiosis, in particular lower butyrate producing bacteria. Reduced Bacteroidetes comparable to that seen

Abstract OC-051 Table 1 Concentrations of bacteria between groups

Bacteria (log ₁₀ /g dry weight faeces (IQR))	Median concent	Median concentration			p Values			
	Patient	Sibling	Control	Patient vs sibling*	Patient vs control*	Sibling vs control*		
Universal	10.7 (0.73)	10.8 (0.48)	11.0 (0.54)	0.759	0.015	0.117		
Bacteroidetes	8.8 (1.70)	10.2 (1.20)	10.5 (0.69)	0.009	<0.005	0.639		
Clostridial cluster IV	7.8 (3.02)	9.3 (1.17)	9.7 (0.78)	0.018	<0.005	0.030		
F prausnitzii	6.9 (4.32)	9.3 (1.66)	9.6 (0.80)	0.006	<0.005	0.048		
Roseburia spp.	9.2 (2.50)	9.3 (2.49)	9.9 (0.77)	1.000	0.027	0.009		
Ruminococci	7.1 (2.34)	8.8 (1.29)	9.6 (1.65)	<0.005	<0.005	0.084		
Bifidobacteria adolescentis	5.8 (3.90)	9.0 (1.35)	9.2 (1.68)	0.042	0.027	1.000		

*Bonferroni corrected p values.