Original Paper

The Effect of Endogenous Hypergastrinemia on Lower Esophageal Sphincter Pressure and Esophageal Motility

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ABSTRACT

Background/Aims: The effect of exogenous hypergastrinemia on esophageal motor function has been well documented. However, it is not known whether chronic endogenous hypergastrinemia influences esophageal motility and lower esophageal sphincter pressure. The purpose of this study was to investigate the effect of chronic hypergastrinemia on lower esophageal sphincter pressure and esophageal motility in patients with significantly elevated serum gastrin levels. **Methodology:** 37 patients (28 women; mean age, 53.7 years) with autoimmune gastritis and 35 functional dyspepsia patients participated in this study. Esophageal motility testing was performed by using an eight-lumen water-perfused catheter. Ten wet swallows were given and each contraction was analysed for lower esophageal sphincter pressure, lower esophageal sphincter relaxation, contraction amplitude and peak velocity. **Results:** Mean serum fasting gastrin level was 1382.8±731.9pg/mL in patients with autoimmune gastritis and 107±83.9pg/mL in the control group (p=0.000). Mean lower esophageal sphincter pressure (31.6±14.42mmHg *vs.* 20.5±8.05mmHg, p=0.000) and mean contraction amplitude (82.48±35.0mmHg *vs.* 58.11±21.75mmHg, p=0.001), in hypergastrinemic patients were significantly higher than in the control group. **Conclusions:** These results suggest that in patients with autoimmune gastritis, prolonged and significant elevation of serum gastrin levels, increases lower esophageal sphincter pressure and esophageal body contraction amplitude. However, this increase in lower esophageal sphincter pressure does not cause upper gastrointestinal symptoms in patients with autoimmune gastritis.

Key Words: Hypergastrinemia; Autoimmune gastritis; Lower esophageal sphincter pressure; Esophageal motility.

Abbreviations: Autoimmune Gastritis (AIG); Lower Esophageal Sphincter (LES); Lower Esophageal Sphincter Pressure (LESP); Gastrointestinal (GI).

INTRODUCTION

Autoimmune gastritis (AIG) is an asymptomatic disease and has an indolent course in the early stages of the disease and is demonstrable only by serological detection to gastric parietal cells. It is characterized by elevated serum gastrin levels due to loss of gastric parietal cells (1). Gastrin is the main stimulant of gastric acid secretion during meal ingestion. Injection or continious infusion of gastrin results in an increase in lower esophageal pressure (2,3), however when gastrin is given in doses close to those reached after a meal, lower esophageal sphincter pressure (LESP) remains the same, or even decreases (4-6).

There are no specific clinical findings or symptoms that indicate AIG. It is only when stores of vitamin B12 are depleted that pernicious anemia manifests itself clinically with hematological and/or neurological findings (1). However, some of these patients may have upper gastrointestinal symptoms such as bloating, nausea, belching and abdominal pain without significant endoscopic findings (7). Esophageal motility has not been systematically investigated in patients with AIG that is characterized by chronic endogenous hypergastrinemia. Therefore, the purpose of this study was to investigate the effect of chronic hypergastrinemia on lower esophageal sphincter pressure and esophageal motility in a group of patients with autoimmune gastritis and elevated serum gastrin levels. Our hypothesis was that chronic endogenous hypergastrinemia increases LESP.

METHODOLOGY

The study population consisted of 37 patients with atrophic gastritis and hypergastrinemia, who were referred to the gastroenterology outpatient clinic due to dyspeptic symptoms and/or vitamin B12 and/or iron deficiency anemia. The diagnosis of chronic autoimmune gastritis was based on the pathological findings in the gastric biopsy tissue (8). Serum gastrin level, presence of antiparietal cell antibody and gastric juice pH were also investigated in each patient. Gastrointestinal symptoms if present, were also noted. Thirty-five patients diagnosed as having functional dyspepsia served as a control group in this study. None of the patients was on drugs that might alter esophageal motor function during motility testing.

Esophageal motility

Esophageal motility studies were performed with a pull-through technique that runs microperfusion system by using a single catheter containing 8 pressure transducers spaced at 5cm intervals and attached to an online computer (Medical Measurement Systems (MMS), The Netherlands). Patients came to the laboratory after at

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least 8h of fasting. The 8-channel catheter was lubricated and passed nasally and advanced into the stomach. A slow station pull-through was performed at 1cm increments. Once the lower esophageal sphincter (LES) was profiled, the distal pressure transducer which included four lumens was placed in the high-pressure zone of the LES, so that the proximal pressure transducers were located at 5, 10, 15 and 20cm above the LES. A series of 10 wet swallows (with 5ml water bolus) were given at 20-30s intervals. Lower esophageal sphincter pressure (reference 6-25mmHg), percentage of peristaltic waves (reference: 80%) and esophageal body amplitude (reference 30-160mmHg) were determined. Each contraction was recorded and then analyzed by a computerized software system (MMS) for amplitude, contraction and velocity. The present study was approved by the Institutional Review Board of Ankara University Medical School and all patients gave their informed consent before entering the study.

Statistical analysis

Data were analyzed with the Statistical Package for Social Sciences (SPSS; version 11.0; SPSS Inc. Chicago, IL, USA) for Windows software. The Mann Whitney U test and Kruskal Wallis test were used for group comparisons. For categorical variables the chi-square test was used. Mean±SD was given for continuous measurements. A p value <0.05 was considered as statistically significant.

RESULTS

The mean age of the patients enrolled in the study (n=37, 28 women) was 55.7 years and mean age of the control group (n=35, 20 women) was 47.6 years. The clinical manifestations of atrophic gastritis and GI symptoms in our cases are summarized in **Table 1**. All patients in this study complained of upper GI symptoms, predominantly of abdominal pain and abdominal distantion. Seventy-five percent of patients had at least one gastrointestinal symptom. Anemia was found in seven patients (vitamin B12 deficiency in 4 and iron deficiency amenia in 3 patients). The mean serum fasting gastrin level was 1382.8±731.9pg/mL in patients with autoimmune gastritis and 107±83.9pg/mL in the control group (p=0.000).

As for manometric findings, median LES pressure was significantly higher in patients with endogenous hypergastrinemia compared to the control group (31.59±14.42mmHg *vs.* 20.51±8.05mmHg, respectively, p=0.0001). There were significant differences between mean LES relaxation (88.27±14.12% *vs.* 95.08±6.02%, p=0.01), mean LES relaxation duration (7.12±1.71s *vs.* 8.54±1.81s, p=0.001), mean contraction amplitude (82.48±35.10mmHg *vs.* 58.11±21.75mmHg, p=0.001) and median peak velocity (2.98±0.85s *vs.* 3.67±1.76s, p=0.041) in patients with endogen hypergastrinemia compared to the control group (**Table 2**). Esophageal motor function was also investigated in patients (n=24) with serum gastrin >1000pc/mL and compared to patients (n=13) with serum gastrin <1000pg/mL by using the same parameters: LESP (31.37±13.14mmHg *vs.* 32.0±17.09mmHg, p=0.902), mean contraction amplitude (81.20±40mmHg *vs.* 86.91±22.01mmHg, p=0.649), LES relaxation (90.91±11.28% *vs.* 83.61±17.94%, p=0.137) and peak velocity (2.98±0.81s *vs.* 3.03±0.94s, p=0.877). There were no statistical significant between the two groups (**Table 3**). In order to delineate the relationship between the change of LESP/ esophageal motor function and gastrointestinal symptoms, we also investigated LESP and esophageal motor function in patients with gastrointestinal symptoms (n=26) and compared them to patients (n=11) without gastrointestinal symptoms (9=anemia patients, 2=check-up patients). There were no statistical significance between the two groups except in peak velocity. Patients with gastrointestinal symptoms *vs.*without gastrointestinal symptoms: LESP: 30.8±15.0mmHg *vs.* 33.2±13.3mmHg p=0.648; mean contraction amplitude: 76.54±27.7mmHg *vs.* 89.2±49.4mmHg p=0.439; LES relaxation: 87.3±13.7% *vs.* 94.2±7.9%, p=0.701), peak velocity: 3.1±0.7s *vs.* 2.5±0.8s, p=0.049).

TABLE 1. Symptoms of patients with endogenous hypergastrinemia

Symptoms	n	%
Abdominal pain	8	21.6
Epigastric pain	3	8.1
Abdominal distantion	18	48.6
Check-up	2	5.4
Belching	2	5.4
Burning sensation in the epigastrium	3	8.1
Nausea	4	10.8
Intermittan diarrhea	1	2.7
Anemia: Iron deficiency and/ or vitamin B12 deficiency	9	24.3

(Patients could have more than one symptom)

TABLE 2. Manometric findings in patients with hypergastrinemia compared to control group

	Hypergastrinemia patients (n=37)	Control group (n=35)	p
LES pressure (mmHg)	31.59±14.42	20.51±8.05	0.000
LES relaxation (%)	88.27±14.12	95.08±6.02	0.01
Duration of LES relaxation	7.12±1.71	8.54±1.81	0.001
Esophageal body contraction amplitude (mmHg)	82.48±35.10	58.11±21.75	0.001
Peak velocity (seconds)	2.98±0.85	3.67±1.76	0.041

LES: Lower esophageal sphincter

DISCUSSION

In this study, we investigated the effect of chronic hypergastrinemia on esophageal motility and lower esophageal sphincter pressure in patients with significantly elevated serum gastrin levels and in patients with autoimmune gastritis. When manometric findings of endogenous hypergastrinemia patients were compared with the control group, there were significant differences between lower esophageal sphincter pressure and contraction amplitude in the body of the esophagus. LESP and esophageal body motility are effected indirectly through cholinergic neural or hormonal stimulation. LESP is known to be regulated by intrinsic smooth muscle tone, neuorgenic components and gastrointestinal hormones. Of neural components, excitatory vagal cholinergic fibers, in particular, regulate basal LESP tone (9,10). Several GI hormones when infused intravenously have been shown to either increase LESP, such as gastrin, pancreatic polypeptide and motilin, or decrease LESP, such as cholecystokinin, glucagon and vasoactive intestinal polypeptide (11-14). Gastrin is thought to be a stimulator of the LES by increasing LESP. Intravenous injection of the synthetic analog pentagastrin in pharmacological doses increases LESP (2) but in physio-

logical postprandial doses gastrin does not affect LESP or even decreases LESP (4). Heil *et al.* evaluated the effect of gastrin and somatostatin on the lower esophageal sphincter in a total of ten metabolically healthy volunteers and one patient with Zollinger-Ellison syndrome. Only unphysiologically high concentrations of gastrin produce a rise in pressure in the LES, while somatostatin has neither an effect on the LESP, nor was it able to inhibit the pharmacological effect of exogenic gastrin administration (15). Strathof *et al.* studied the effect of gastrin-17 on LES and transient lower esophageal sphincter relaxations in nine healthy volunteers. The study group participated in two experiments performed in random order during continuous infusion of saline (control) or gastrin-17 (15pmol/kg/hr). During continuous gastrin infusion, LESP decreased significantly ($p<0.05$) compared to the controls but the rate and duration of transient lower esophageal sphincter relaxations were not influenced by gastrin-17 (16).

In this study, endogenous hypergastrinemia patients were separated into two groups according to their serum gastrin level. Abnormal esophageal motility pattern was observed in 29.1% (7/24) of patients with a serum gastrin level >1000pg/mL. Patients with serum gastrin level >1000pg/mL did not show different patterns of esophageal motor function. The reason why serum gastrin level was chosen as 1000pg/mL was because PA is related with chronic achlorhydria and serum gastrin levels are elevated to levels ≥1000pg/mL, which is the range observed in many patients with Zollinger Ellison syndrome (17,18). Strader *et al.* (19) evaluated the esophageal function in 92 consecutive patients with Zollinger Ellison syndrome (66 with active disease and 26 disease-free after curative resection). Esophageal manometry revealed normal motility in 85% of the patients. Eleven percent had low LES pressures and only 1% of patients had an elevated LES pressure. No correlation was noted between the LES pressures or manometric abnormalities and the fasting serum gastrin level. Farrell *et al.* studied lower esophageal sphincter pressure in nine patients with pernicious anaemia and compared their results with 14 healthy male subjects (20). The resting sphincter pressure was found to be significantly lower in the pernicious anaemia patients. In our study we also evaluated patients with gastrointestinal symptoms by means of esophageal manometry and compared them to patients without gastrointestinal symptoms who were referred for the investigation of anaemia etiology. However, we did not find any correlation between LESP and gastrointestinal symptoms. In previous studies on the effect of gastrin on LESP the hormone has been given by intravenous injection as a bolus or continuous infusion. Watanabe *et al.* evaluated the effect of endogenous gastrin on LESP in fifteen dogs (21). Animals were divided into three groups by the type of operation: 5 dogs with antral excision and Billroth-II gastrojejunostomy; 5 dogs with a denervated antral pouch and Billroth-II; and 5 dogs with an innervated antral pouch and Billroth-II. Fasting serum gastrin levels and LESP were determined preoperatively (basal) and at 2 and 4 weeks postoperatively. The fasting serum gastrin rose significantly at 2 weeks and 4 weeks in only innervated antral pouch and Billroth-II dogs. The mean LESP at 2 and 4 weeks did not significantly change from the basal LESP in the three groups. Dennis *et al.* investigated the effect of endogenous hypergastrinemia on LESP in canine esophagus (22). They transplanted the isolated vagally innervated antrum as a diverticulum into the transverse colon which produced endogenous hypergastrinemia. Although antral transplantation resulted in a marked increase in serum gastrin levels, LESP remained unchanged. They concluded that endogenous gastrin plays no significant role in the regulation of LESP.

There are a number of limitations of our study. The patients recruited into this study did not demonstrate symptoms suggestive of esophageal dysmotility or reflux disease, therefore the clinical relevance of these results may be redundant. Additionally, the increased LESP and esophageal body contraction amplitude may be related the autoimmune nature of the disease.

In conclusion, the results of our study suggest that in patients with autoimmune gastritis, prolonged and significant elevation of serum gastrin levels increase LESP and esophageal body contraction amplitude. However, the increase in LESP does not cause upper gastrointestinal symptoms in patients with autoimmune gastritis and the clinical significance of these findings warrants further investigations.

TABLE 3. Manometric findings in patients with serum gastrin >1000pg/mL compared to patients with serum gastrin <1000pg/mL.

	Gastrin >1000pg/mL 24 (64.9%)	Gastrin <1000pg/mL 13 (35.1%)	*p*
LES pressure (mmHg)	31.37±13.14	32.00±17.09	0.902
LES relaxation (%)	90.91±11.28	83.61±17.94	0.137
Duration of LES relaxation	7.19±1.81	6.98±1.56	0.725
Esophageal body contraction amplitude (mmHg)	81.20±40.00	86.91±22.01	0.649
Peak velocity (seconds)	2.98±0.81	3.03±0.94	0.877

LES: Lower esophageal sphincter.

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ASNEMGE	Mark Hull – United Kingdom
ISDS	Roger Leicester – United Kingdom
ESCP	Anna Martling – Sweden
EPC	Julia Mayerle – Germany
EHSG	Francis Megraud – France
Independent	Tania Roskams – Belgium
Independent	Wolff Schmiegel – Germany
ESPGHAN	Raanan Shamir – Israel
ESGAR	Jaap Stoker – The Netherlands
Independent	Severine Vermeire – Belgium

class II genes. In particular, *HLA-DRB1*0103*, a rare allele with a frequency of less than 2% in European and white North American populations has been associated with both extensive and severe UC in Caucasians. *HLA-DRB1*1502* has also been associated with UC. This allele is much more prominent in Japanese and Korean populations than Caucasians. However, despite variable background frequencies, the odds ratios for *HLA-DRB1*1502* in UC remains similar suggesting it is a disease causing variant. There have been no previous reports on the frequency of *HLA* class II alleles in Indian IBD populations.
AIMS & METHODS: To investigate the association between HLA class II alleles and UC in an Indian Asian population. The study included 129 unrelated Indian Asian UC patients and 199 healthy Indian Asian controls. All participants had four grandparents originating from the Indian subcontinent. *HLA-DRB1* allele frequency was determined using sequence-specific primers and PCR. The clinical phenotype of the patients were analysed with regard to maximal disease extent and statistical analyses were performed using the Chi-squared test.
RESULTS: The *HLA-DRB1*0103* allele was absent in all Indian Asian UC patients and controls. However, *HLA-DRB1*1502* was significantly more frequent in the UC cohort (28.9%) than controls (17.6%) ($p = 0.016$). There was no significant difference in the frequency of *HLA-DRB1*1502* when patients with extensive colitis were compared with left-sided/proctitis (29.5% vs 28%; $p = 0.856$). Patients requiring colectomy did not have a significantly increased incidence of *HLA-DRB1*1502* (2/6; 33% $p = 0.315$).
CONCLUSION: The HLA-*DRB1*0103* allele is rare or absent in the Indian Asian population but *HLA-DRB1*1502* is positively associated with ulcerative colitis. However, *HLA-DRB1*1502* was not associated with disease extent in UC and does not therefore account for the higher incidence of pancolitis previously reported in South Asians compared to Caucasians. Further genetic studies are warranted to determine whether other genes may account for the differences in UC phenotype in this ethnic group.
Disclosure of Interest: None Declared *Gut 2010; 59 (Suppl III)* A405

P1487 REAUDIT ON APPLICATION OF BSG DEXA SCAN GUIDELINES IN INFLAMMATORY BOWEL DISEASE SUBJECTS

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INTRODUCTION/OBJECTIVES: There is a high prevalence of osteopenia and osteoporosis in patients with inflammatory bowel disease (IBD). This increased risk of metabolic bone disease translates into a greater risk of fracture among IBD patients compared to the general population. The British Society of Gastroenterology (BSG) guidelines for prevention and treatment of osteoporosis in patients with IBD were last published in a revised version in 2007 [1]. We have a long-established tradition of re-auditing these recommendations, which is used to improve our practice and for in-house training.
AIMS & METHODS: *Aim*: To monitor and improve the use of DEXA scanning for the diagnosis of osteopenia and osteoporosis in patients with IBD.
Methods: 3 audit cycles have been completed. Unselected subjects attending the IBD clinic for follow-up were audited (107 in 2002, 138 in 2006, 106 in 2009). Information was collected from questionnaires completed on day of attendance and was focused on age, sex, pre/postmenopausal state, recent corticosteroid therapy, previous fragility fractures, current diseases and treatment that could affect bone metabolism, as well as smoking status and alcohol intake.
RESULTS: 2009 Data was summarised from 59 patients with ulcerative colitis and 47 with Crohn's disease. There were 59 males (age range 21–71) and 47 females (age range 20–80). 28 out of 48 IBD patients treated with systemic steroids over last 12 months had bone protecting agents co-prescribed. 14 men were over 55 and 12 women were postmenopausal. 7 patients with Crohn's disease and 1 with ulcerative colitis had a previous fragility fracture. The numbers requiring DEXA scans and the numbers who have actually had scans are shown in the table.

Table 1. DEXA requirement, previous and actual use

Year	Number Requiring DEXA as per BSG guidelines (%)	DEXA performed
Ulcerative colitis		
2002	30/58 (51.7%)	12/30 (40%)
2006	39/67 (48%)	20/39 (51%)
2009	30/59 (50.8%)	11/30 (36.6%)
Crohn's disease		
2002	26/51 (51%)	12/26 (46.2%)
2006	26/71 (45%)	15/26 (58%)
2009	31/47 (65.9%)	19/31 (61.3%)

CONCLUSION: Our results show that we continue to make progress in the application of BSG guidelines in surveillance for osteopenia and osteoporosis with DEXA scanning in patients with Crohn's disease. The findings in patients with ulcerative colitis are disappointing. The use of DEXA scans was higher in the Crohn's patients possibly reflecting better awareness of their increased risk by their attending physician. Further training and subsequent re-audit is still required, with a particular focus on patients with ulcerative colitis.
REFERENCE(S): [1] Lewis NR, Scott BB. Guidelines for Osteoporosis in Celiac Disease and Inflammatory Bowel Disease. British Society of Gastroenterology, June 2007.
Disclosure of Interest: None Declared *Gut 2010; 59 (Suppl III)* A406

P1488 DEVELOPMENT AND VALIDATION OF A NEW ASSAY FOR MONITORING FUNCTIONAL SERUM ANTI-TUMOUR NECROSIS FACTOR ANTIBODY LEVEL IN CROHN'S DISEASE PATIENTS WHO MAINTAINED AND THOSE WHO LOST RESPONSE TO ANTI-TNF

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INTRODUCTION/OBJECTIVES: Infliximab (IFX) is an anti-tumour necrosis factor (TNF)-α antibody used to treat patients with Crohn's disease (CD). However, antibodies to IFX (ATI) emerge, which potentially can impair its efficacy as well as invoking hypersensitivity reactions, having a negative impact on the safety of IFX therapy. We were interested to develop a new assay for measuring IFX as well as ATI.
AIMS & METHODS: In this study, we developed a fluid-phase enzyme immunoassay (FP-EIA) by immunizing rabbits with IFX Fab fragments to produce ATI, which we used to measure serum functional IFX (f-IFX) in patients with CD on maintenance IFX therapy. Thirty-one patients, 16 had maintained response to IFX (group I) and 15 had lost response in spite of good initial response to IFX (group II) were included. Serum f-IFX was measured just before and immediately after IFX infusion, and the values together with CD activity index (CDAI) and C-reactive protein (CRP) were compared between the two groups.
RESULTS: The immunized rabbits produced very high levels of anti-idiotypic antibody titer as measured at a very finite dilution of 1:4000. The new FP-EIA produced a smooth dose-response curve for increasing amounts of IFX vs TNF-α. The binding capacity of IFX for TNF-α was almost abolished by the addition of 1% serum from a patient who had lost response to IFX (had the lowest f-IFX level even immediately after IFX-infusion) or by the addition of 1% serum from an immunized rabbit. The assay could also detect antibodies that had emerged against IFX. Further, the duration of IFX therapy in groups I and II were 1.8±1.2 and 2.7±1.5 years, respectively, while the median dose frequency was 56 days in group I and 29 days in group II. On the infusion day, CRP and CDAI in group II were significantly higher than in group I, while the median trough f-IFX for groups I and II were 4.7 μg/mL and 6.3 μg/mL, respectively. The median f-IFX immediately after IFX infusion for groups I and II were 149.5 μg/mL and 126.3 μg/mL respectively ($P = 0.0488$), showing negative correlation with CRP ($r = -0.516$, $P = 0.0035$) and CDAI ($r = -0.468$, $P = 0.0092$).
CONCLUSION: This new FP-EIA could accurately measure f-IFX. High serum ATI strongly impacted f-IFX levels even immediately after IFX infusion. The f-IFX level immediately after an infusion was associated with clinical response. f-IFX level should be valuable in decision making to optimize treatment efficacy with this biologic.
Disclosure of Interest: None Declared *Gut 2010; 59 (Suppl III)* A406

P1489 FICOLIN-2 AS A POSSIBLE NEW SERUM MARKER FOR DISEASE ACTIVITY IN CD PATIENTS

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INTRODUCTION/OBJECTIVES: Besides mannan binding lectin (MBL), the ficolins also represent microbial pattern recognition receptors that can activate the lectin pathway of complement. By analyzing the role of the lectin pathway of complement regarding the development of anti-Saccharomyces cerevisiae mannan antibodies (ASCA), we found that in the serum of IBD patients, especially in Crohn's disease (CD), ficolin-2 (L-ficolin), had an acute-phase like expression pattern. With the present study we further investigated the acute-phase like expression of ficolin-2 in CD patients including a possible correlation with disease activity.
AIMS & METHODS: Ficolin-2 concentrations were measured in the sera of IBD patients by ELISA. For the same patients also CRP and fecal calprotectin concentrations were determined. Harvey-Bradshaw index, Mayo score and medication were assessed at the time point of sample collection. Sera and stool samples were collected in a prospective manner from 48 patients of our local IBD cohort.
RESULTS: In our IBD cohort serum concentrations of ficolin-2 were significantly increased in CD patients compared to healthy controls. It was also increased in UC patients compared to HC but this was not statistically significant. The increase of ficolin-2 was higher than 40% compared to healthy controls and a moderate but significant correlation with CRP concentration was measured. Furthermore, CD patients with a Harvey-Bradshaw index (HBI) >3 had significantly higher ficolin-2 concentrations than patients with a HBI ≤3.
CONCLUSION: Ficolin-2 in CD showed an expression pattern like an acute-phase protein. Ficolin-2 was the only marker in this study which was significantly correlated with disease activity represented by the HBI. There fore ficolin-2 may represent a new, easy measurable serum marker, to objectively assess disease activity of CD patients.
Disclosure of Interest: None Declared *Gut 2010; 59 (Suppl III)* A406

P1490 BOWEL DISEASE IN PATIENTS WITH SPONDYLOARTHROPATHY-EVIDENCE FOR SUBCLINICAL CROHN'S DISEASE?

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INTRODUCTION/OBJECTIVES: Increasingly, Ankylosing spondylitis (AS) and Inflammatory Bowel Diseases (IBD) are seen as part of a spectrum of inter-related diseases which share a similar genetic predisposition, with phenotypical overlap with respect to intestinal inflammation and peripheral and axial arthritis. Clinically significant bowel symptoms have not previously been thought to be a feature of AS.
AIMS & METHODS: To determine the severity of bowel symptoms in AS in comparison with 3 other groups: patients with Crohn's Disease (CD), healthy controls and controls with osteoarthritis (OA) who used long-term NSAIDs and by doing so to assess the validity of the Dudley Inflammatory Bowel Symptom Questionnaire (DISQ) in AS.
Thirty-one patients with CD were recruited. A further 26 patients with active AS (according to the Bath AS Disease Activity Index: BASDAI) and 10 patients with inactive AS were recruited, and compared with 35 healthy controls, and 21 patients with OA on NSAIDs. The DISQ was administered to all groups on a single occasion. The IBDQ was administered to all patients with CD.
RESULTS: AS patients had more severe bowel symptoms – mean 9.81 (±5.45) – than healthy controls – mean 2.61 (±2.57) $p < 0.0001$, and controls taking NSAIDs – mean 5.80 (±5.14) $p = 0.02$. There was no significant difference in severity of symptoms between AS and CD patients in remission – mean 13.56 (±8.41) $p = 0.06$.
The DISQ correlated strongly with the IBDQ in patients with CD – Spearman's rank correlation (rho) between DISQ and IBDQ of = 0.773 ($p < 0.001$).
CONCLUSION: Bowel symptoms are significantly associated with active AS in comparison to healthy controls. Active AS is associated with more severe bowel symptoms than inactive AS and comparable to Crohn's disease in remission. The incidence of bowel symptoms cannot be attributed to the use of NSAIDs alone. The DISQ correlates well with the IBDQ.
Disclosure of Interest: None Declared *Gut 2010; 59 (Suppl III)* A406

P1491 SERUM VITAMIN B12 AND FOLATE STATUS IN PATIENTS WITH INFLAMMATORY BOWEL DISEASES

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INTRODUCTION/OBJECTIVES: Vitamin B_{12} (cobalamin) deficiency is a common cause of macrocytic anemia and has been implicated in a spectrum of disorders. Crohn's disease (CD) is a chronic inflammatory disease involving small and/or large bowel. CD frequently involves terminal ileum, which is the site of B_{12} absorption. Ulcerative colitis (UC) is also an inflammatory disease limited to the colon, with little or no ileal

involvement, and the prevalence of B_{12} deficiency in UC patients is similar to the general population.
AIMS & METHODS: The aims of this study were to define the prevalence of serum vitamin B_{12} and folate abnormalities in patients with inflammatory bowel disease and to identify risk factors associated with B_{12} and folic acid abnormalities in CD. 138 patients with inflammatory bowel disease (45 Crohn's disease, 93 Ulcerative colitis) and 53 healthy subjects were enrolled into the study. Fasting serum B_{12} and folic acid levels were measured and clinical data regarding inflammatory bowel diseases were gathered.
RESULTS: While the mean serum B_{12} concentration in CD patients was 281±166 pg/ml, the mean serum vitamin B_{12} concentration in UC patients was 348±218 pg/ml (p = 0.224). The number of patients with vitamin B_{12} deficiency in CD group were greater than number of patients with UC [n = 10 (22%) vs. n = 4 (7.5%), p = 0.014]. The number of patients (n = 10, 22%) with B_{12} deficiency in CD group was also greater than controls (n = 4, 7.5%) (p = 0.039). With regard to folate levels, the median serum folate level in CD patients was 7.7±5.3 ng/ml, 8.6±8.3 ng/ml in UC patients and 9.9±3.3 ng/ml in the control group (p = n.s). Patients with a prior ileocolonic resection had an abnormal B_{12} concentration compared to patients without surgery (p = 0.008). In CD patients, ileal involvement was the only independent risk factor for having a low folate level.
CONCLUSION: Serum vitamin B_{12} and folate deficiencies are common in patients with CD compared to UC patients and controls. In CD patients, prior small intestinal surgery is an independent risk factor for having a low serum vitamin B_{12} level. It would be recommended that patients with CD shoul be screened routinely for B_{12} and folate deficiency and patients who have B_{12} and folate deficiency, adequate vitamin supplementation should be provided.
REFERENCE(S): [1] Headstrom PD, Rulyak SJ, Lee SD. Prevalence and risk factors for vitamin B_{12} deficiency in patients with Crohn's disease. Inflamm Bowel Dis. 2008;14:217–223. [2] Duerksen DR, Fallows G, Bernstein CN. Vitamin B_{12} malabsorption in patients with limited ileal resection. Nutrition. 2006;22:1210–1213.
Disclosure of Interest: None Declared *Gut 2010; 59 (Suppl III)* A406

P1492 CORRELATION OF CT ENTEROGRAPHY FINDINGS TO HSCRP, PLATELETS, HEMATOCRIT AND BMI

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INTRODUCTION/OBJECTIVES: We hypothesized that specific Computed Tomography Enterography (CTE) findings would correlate with low body mass index (BMI), elevated high sensitivity C-reactive protein (hsCRP), anemia, thrombocytosis and elevated white blood cell count (WBC) in patients with Crohn's disease.
AIMS & METHODS: A retrospective study was performed on 37 outpatients with established CD who underwent CTE between August 2008 and August 2009 and whose laboratory findings and BMI were assessed within 1 month of their CTE. CTE scans were reread by an abdominal radiologist blinded to the clinical history and original radiographic impression. The presence of findings associated with inflammatory activity (mural enhancement, wall thickness, increased mesenteric fat density, and comb sign) and the presence of skip lesions, fistula, inflammatory mass, abscess, stricture, and small bowel dilation were noted separately. Patient's hsCRP, hematocrit, WBC, platelets, hematocrit and BMI was also noted. Spearman correlation coefficients were calculated between CTE findings and BMI, hsCRP, hematocrit, platelet count, and WBC.
RESULTS: Markers of mural disease (both inflammatory and fibrotic) are statistically more likely to be associated with lower BMI as compared to extraintestinal markers (mesenteric fat, comb sign, fistula, inflammatory mass, abscess) suggesting a role of malnutrition. Patients who have inflammatory mass are more likely to be anemic. CTE findings associated with inflammatory activity are associated with thrombocytosis. There was no correlation between WBC and any CTE finding, including abscess. Elevated hsCRP correlated with presence of fistula, inflammatory mass and abscess.

Statistically significant correlations between CTE findings versus BMI, hematocrit, platelets, and hsCRP

	r	p
Mural enhancement and BMI	−0.57	<0.001
Wall thickening and BMI	−0.49	0.002
Fibrosis and BMI	−0.44	0.007
Inflammatory mass and hematocrit	−0.36	0.042
Mural enhancement and platelets	0.38	0.036
Comb sign and platelets	0.43	0.015
Skip lesions and platelets	0.47	0.008
Stricture and platelets	0.37	0.001
Fistula and hsCRP	0.52	0.004

CONCLUSION: Presence of inflammatory mass, fistula and abscess but not mural enhancement, wall thickening, comb sign or mesenteric fat enhancement correlate strongly with elevated hsCRP. Patients who have mural disease tend to have lower BMI suggesting a role of malnutrition.
Disclosure of Interest: None Declared *Gut 2010; 59 (Suppl III)* A407

P1493 CT ENTEROGRAPHY FINDINGS CORRELATE WITH COMPONENTS OF HARVEY BRADSHAW INDEX

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INTRODUCTION/OBJECTIVES: We hypothesized the Harvey-Bradshaw Index (HBI) and HBI's clinical components (well-being, abdominal pain, diarrhea, and abdominal mass) would correlate with specific Computed Tomography Enterography (CTE) findings.
AIMS & METHODS: 37 patients with established Crohn's disease (CD) who underwent CTE between August 2008 and August 2009 and whose HBI scores were assessed within 1 month of their CTE were included in this study. CTE scans were reread by an abdominal radiologist blinded to the clinical history and original radiographic impression. The presence of findings associated with inflammatory activity (mural enhancement, wall thickness, increased mesenteric fat density, and comb sign) and the presence of skip lesions, fistula, inflammatory mass, abscess, stricture, and small bowel dilation were noted separately. The individual component of each CTE finding was split into two groups, absent or present. The mean total HBI score of each group was determined and compared using analysis of variance. Spearman correlation coefficients were calculated between CTE findings and total HBI score as well as components of HBI.
RESULTS: Patients with the presence of wall thickness and comb sign had significantly higher total HBI scores (10.3 vs6.1, p = 0.036 and 11.5 vs7.5, p = 0.028, respectively). The presence of the other CTE findings were not associated with significantly higher total HBI scores, although patients with mural enhancement also trended towards having a higher HBI (9.9 vs7.3, p = 0.18). Notably diarrhea did not correlate with any CTE finding. The presence of a higher number of CTE findings associated with inflammatory activity correlated with general well being (r = 0.39, p = 0.020) and abdominal mass (r = 0.40, p = 0.016) and trended toward a correlation with total HBI score (r = 0.33, p = 0.054) and with abdominal pain (r = 0.33, p = 0.053), but correlated less well with total number of liquid stools daily (r = 0.18, p = 0.28).

Correlation of individual CTE parameters with clinical measures of HBI

Clinical features	Mural enhancement	Wall thickness	Comb sign
General well being	r = 0.33, p = 0.05	r = 0.43, p = 0.009	r = 0.43, p = 0.008
Number of liquid stools per day	r = 0.15, p = 0.37	r = 0.20, p = 0.22	r = 0.21, p = 0.19
Abdominal pain	r = 0.30, p = 0.007	r = 0.22, p = 0.19	r = 0.39, p = 0.01
Abdominal mass	r = 0.24, p = 0.16	r = 0.22, p = 0.19	r = 0.43, p = 0.008

CONCLUSION: The findings of wall thickness and comb sign most correlate with clinical disease activity as measured by the HBI. Diarrhea did not correlate with CTE findings and may be overrepresented in CD activity indices. Further study is needed to incorporate CTE data into a newer CD activity index.
Disclosure of Interest: None Declared *Gut 2010; 59 (Suppl III)* A407

P1494 PROPOSAL OF AN MRI ACTIVITY INDEX FOR CROHN'S DISEASE

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INTRODUCTION/OBJECTIVES: Assessment of Crohn's disease activity is crucial for the clinical management of the disease. So far no clinical (Crohn's Disease Activity Index [CDAI]) tests have proven to be fully satisfactory to assess the activity. The endoscopic score (Crohn's Disease Endoscopic Index of Severity [CDEIS]) obtained by the optical evaluation and associated biopsies is widely considered as a valuable, although invasive, score for the assessment of activity. To propose an MRI index of Crohn's activity.
AIMS & METHODS: 50 patients with clinically active (n = 30) moderately active (11) or inactive (n = 9) Crohn's disease underwent lower gastrointestinal endoscopy (reference standard) and MRI. T2-weighted and contrast-enhanced T1-weighted sequences were acquired, after oral administration of a superparamagnetic contrast agent. The following parameters were considered as predictors of activity: degree of bowel wall oedema (scored 0–2), degree of wall thickening (between 0–3 mm: score 0; 4–7 mm: score 1; >8 mm: score 2) perivisceral mesenteric oedema or fluid (scored 0–2) on T2w images; pattern of Gd-enhancement (stratified: scored 2), homogenous (scored 1) or absent (0) and its degree (0–2). The presence and number (0, 1–3, <3) of inflamed lymphnodes on T1-T2 w images, scored 0–2. This parameters produce a basic activity score ranging between 0–12. This score was multiplied for a factor 1 or 2 or 3 according to the length of the affected segment, scored as 1 (1–7 cm) 2 (7–25 cm) 3 (more than >25 cm <50 cm) 4 (>50 cm). The final score (12–48) was further multiplied for a factor 1.5, 2 or 3 in presence of perianal disease (1.5) complex severe perianal disease (2), intestinal abscesses or phegmon with entero-enteric fistulas (3), in relation to their extent and severity. The final MRI activity score ranged 0-maximum of 144. A 0–5 score suggests inactivity (0); 6–20 inactive or minimally active disease (1); 20–60 moderate activity (2), 60–100 active disease (3), >100 severe active disease (4). Gold standard for activity was considered an association of biological activity (BA) scored 0–3 and endoscopic activity evaluated by CDEIS (Crohn's Disease Endoscopic Index of Severity); endoscopic activity was classified as absent, mild, active or severe (0–3).
RESULTS: A significant correlation (p < 0.001) between gold standard and MRI activity index was obtained.
CONCLUSION: MRI may become a reference non invasive diagnostic tool for assessment of disease activity, due to the high sensitivity for inflammation and to the capability to evaluate both the transmural and longitudinal spread of the disease in the small and large bowel.
Disclosure of Interest: None Declared *Gut 2010; 59 (Suppl III)* A407

P1495 VACCINATION STATUS AND OPPORTUNISTIC INFECTIONS IN INFLAMMATORY BOWEL DISEASE PATIENTS

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INTRODUCTION/OBJECTIVES: It has been estimated by the European Crohn's and Colitis Organisation (ECCO) in 2009 that 80% of IBD patients will be treated with corticosteroids, 40% antimetabolites (such as 6-mercaptopurine, methotrexate or azathioprine) and 20% biologic therapies (such as infliximab and adalimumab). Whilst the use of immunomodulator treatment has led to improved disease outcomes, patients are at risk of opportunistic infections. Some of these infections can be prevented by appropriate vaccinations.
AIMS & METHODS: We assessed whether our IBD patients were appropriately vaccinated. The IBD patients who attended the gastroenterology outpatient clinic for review were asked to complete a specifically designed structured questionnaire. The questions included diagnosis, age, sex, medications and immunisation history.
RESULTS: We reviewed 82 patients, median age 48 (range 21–89). 42 of the patients were females and 40 were males. 52 (63%) patients reported current or previous use of immunosuppressive treatment. 34 (41%) were aware of the increased infection risk. 19 (23%) had suffered of either recurrent fungal, bacterial or viral infections. Only 31 (38%) patients had influenza immunisation on a yearly basis.