

The relationship between inflammatory bowel disease and type 1 diabetes mellitus: a study of relative prevalence in comparison with population controls

To the Editor,

Genome wide association studies have identified that an overlap exists in the genetic architecture underpinning inflammatory bowel disease (IBD) and other immune-mediated inflammatory diseases [1]. Epidemiological studies have established that IBD patients have a higher prevalence of asthma, psoriasis, rheumatoid arthritis and multiple sclerosis, than persons without IBD [2, 3]. However, data remains unclear regarding the association between IBD and type-1 diabetes mellitus (T1DM). We have examined the prevalence of IBD in T1DM and T1DM in IBD and assessed the effect of concurrent IBD in T1DM patients on glycaemic control and quality of life (QoL).

Type 1 diabetes mellitus (n= 662) and IBD (n= 622) patients were recruited during attendance at outpatient clinics. Non-diabetic controls (n= 602) were recruited from general practices within the South Yorkshire region. Demographic information was recorded from patient case notes, alongside stated diagnoses of T1DM and/or histology confirmed IBD. Diabetic controls were selected from the diabetes cohort matched for age and sex in a 2:1 ratio for comparison of QoL and glycaemic control. Glycaemic control was assessed using HbA1c values and QoL using the Short Form-36 Version 2 (SF-36) questionnaire.

We found that the prevalence of IBD was 12/662 (1.5%) in those with T1DM and 2/602 (0.3%) in controls (OR 5.5, 1.2-24.9; $p=0.03$). The prevalence of T1DM in IBD patients was 4/662 (0.6%), which is comparable with the UK adult population prevalence of T1DM (0.4% [4]; OR 1.5, 0.38-6.07; $p=0.56$). In T1DM-IBD patients, QoL scores were significantly lower in the general health and vitality domains compared to T1DM-only patients ($p=0.004$ and $p=0.041$, respectively; Fig. 1). Adverse QoL was not explained by changes in the glycaemic control (Fig. 2).

In conclusion, the prevalence of IBD in T1DM was increased six-fold compared with that in the control population. However,

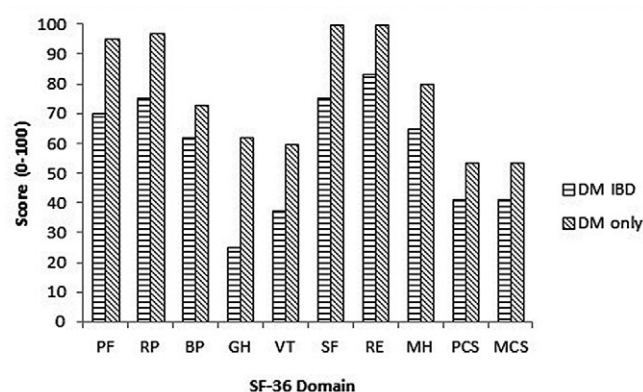


Fig. 1. Bar chart showing SF-36 scores for T1DM-IBD and T1DM-only patients. (PF, physical functioning; RP, role-physical; BP, bodily pain; GH, general health; VT, vitality; SF, social functioning; RE, role-emotional; MH, mental health; PCS, physical component score [combines PF, RP, BP, GH]; MCS, mental component score [combines VT, SF, RE, MH]). QoL scores were significantly different for GH and VT domains ($p=0.004$ and 0.041 , respectively).

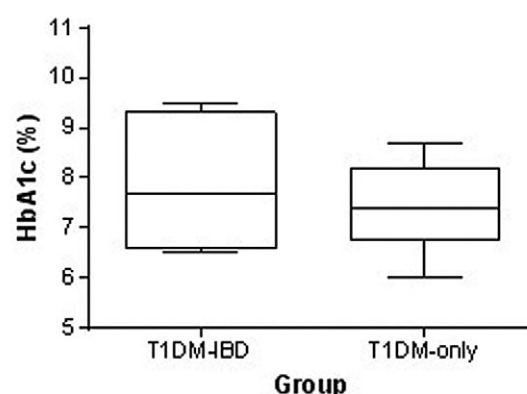


Fig. 2. Box and whisker plot of HbA1c results for T1DM-IBD and T1DM-only. HbA1c was 7.7% in T1DM-IBD and 7.5% in T1DM-only ($p=0.43$).

our data suggest that there is no increase in the prevalence of T1DM in IBD patients. Similar to our findings, a recent Swedish

study found an increase in the incidence of ulcerative colitis in the offspring of parents with T1DM [5]. Moreover, two large North American studies also reported no difference in the prevalence of T1DM in IBD patients compared with healthy controls [2, 3]. Multiple shared susceptibility loci between IBD and T1DM have been described [6]. However, the clinical significance of these genes has yet to be established given the absence of a clear epidemiological link between these two diseases. Further studies are required to characterise the association between these two conditions.

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Does anatomical distribution of colorectal polyps show a rightward shift? Analysis of 2,372 colorectal polyps in 1,558 patients from Turkey

To the Editor,

We read the article of Visovan et al. [1] with great interest. In the last two decades, the literature has reported a change in the topographic distribution of colorectal cancer (CRC), comprising a shift towards the proximal colon [2, 3]. But as stated by the authors, data from the East are scarce. Since the majority of CRC arise from polyps, we aimed to evaluate the topographic distribution of colorectal polyps in our population over a six year period in order to assess any proximalization.

Colonoscopy procedures performed in Sisli Hamidiye Etfal Education and Research Hospital Gastroenterology Department between 2009 and 2014 were evaluated retrospectively. The gender, age and polyp localization in patients who were reported to have polyp(s) in colonoscopy were recorded from the hospital database.

A total of 1,558 patients who had 1,780 total colonoscopies accompanied with polypectomy(ies) were enrolled in the study. The mean age of the patients was 61.1±18.3 years, similar to the study mentioned above [1], as was the male predominance: 933 (60%) males, and 625 (40%) females. Polyp locations were evaluated according to a total of 2,372 polypectomies performed in 1,780 procedures. One thousand and sixty one (48.9%) of the polyps were located in the rectosigmoid region. The other sites of the polyps are shown in Table I. The frequency of the right-sided polyps (from cecum up to the splenic flexure) was 26.6 % in 2009, 25 % in 2010, 23.3 % in 2011, 27.9 % in 2012, 26.2 % in 2013 and 28.5% in 2014.

We did not detect a shift in the localization of colorectal polyps from the left to the right side of the colon, at least 25% of the polyps were found in the right colon in our group. We could not confirm colonic polyp proximalization. However, we agree that rectosigmoidoscopy should not be considered sufficient and patients should be encouraged to undergo a total colonoscopy.

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Table I. The number and topographic sites of colorectal polyps distributed by years

	No. of patients	No. of colonoscopies	No. of polypectomies	Rectosigmoid region	Ascending colon	Transverse colon	Descending colon	Cecum
2009	124	143	184	95	40	35	9	5
2010	232	271	343	165	92	70	7	9
2011	198	225	297	145	83	53	11	5
2012	279	318	445	213	108	91	18	15
2013	444	490	637	318	152	102	39	26
2014 (8 months)	281	333	466	225	108	85	27	21
Total (n)	1558	1780	2372	1161	583	436	111	81
Total (%)			100	48.9	24.6	18.4	4.7	3.4

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Neutrophil-lymphocyte ratio in Crohn's disease patients predicts sustained response to infliximab 52-week therapy

To the Editor,

The neutrophil-lymphocyte ratio (NLR) is a simple, inexpensive and effective marker of subclinical inflammation, linked with inflammatory and neoplastic diseases [1-3]. The NLR also provides information on neutrophils, infiltrating leukocytes that significantly contribute to the tissue injury and intestinal wall inflammation, and lymphocytes [4-6]. Therefore, we hypothesized that NLR may predict maintenance or loss of response to infliximab (IFX) maintaining treatment during a 52-week therapy in Crohn's disease (CD) patients.

To test our hypothesis, we performed a retrospective analysis of 30 CD patients who underwent full 52-week IFX therapy and of 15 healthy subjects. Total white blood cell, neutrophil, and lymphocyte counts were obtained. In CD patients similar counts were performed before the initiation of

a 52-week IFX therapy and after induction treatment at week 14. The NLR was calculated as the ratio of the neutrophil to the lymphocyte count, both obtained from the same sample.

The study showed that CD patients had a higher NLR before the first dose of the 52-week IFX therapy compared to controls, which confirms its association with the inflammatory process in CD (4.62 ± 2.43 vs. 1.49 ± 0.76 ; $p < 0.0001$). In those CD patients who responded to induction treatment at week 14 and maintained the response, a significantly lower NLR at baseline (3.39 ± 1.28 vs. 5.85 ± 2.71 ; $p = 0.0036$) and at week 14 (2.58 ± 1.23 vs. 4.79 ± 2.61 ; $p = 0.0062$) was found, compared with those who lost response to maintenance IFX treatment (Fig. 1). Receiver operating characteristic (ROC) curves were constructed to assign optimal cut-off values associated with the prediction of a sustained response to IFX maintenance treatment. Estimated areas under ROC curves were 0.85 for baseline NLR and 0.76 for NLR at week 14. The analysis of cut-off values showed that a baseline NLR lower than 4.068 predicts sustained response to IFX treatment with 80% sensitivity and 87% specificity. The negative predictive value (NPV) was 81% and the positive predictive value (PPV) was 86%. The NLR at week 14 higher than 3.667 predicts loss of response to IFX with 67% sensitivity and 80% specificity; NPV was 71% and PPV was 77%.

Before the first dose of a 52-week IFX therapy, 17 (17/30) CD patients had $\text{NLR} < 4.068$ and 4 (23.5%) of them lost response to maintenance IFX treatment. Thirteen (13/30) CD patients had baseline $\text{NLR} \geq 4.068$ and 11 (84.6%) of them lost response to 52-week therapy ($p < 0.001$). Eighteen (18/30) CD patients had $\text{NLR} < 3.667$ in week 14 and 6 (33.3%) of them lost response to maintenance IFX treatment. Twelve (12/30) CD patients had $\text{NLR} \geq 3.667$ in week 14 and 9 (75.0%) of them lost response to IFX ($p = 0.023$).

In conclusion, NLR is a subclinical systemic inflammation biomarker and a good predictor of the sustained response to maintaining treatment in the 52-week therapy with IFX in CD patients. The NLR, as an easily accessible and cheap tool, may allow the application of the most appropriate therapy, based on an individualized approach. In case of a possible relapse after successful induction treatment, physicians may be able to

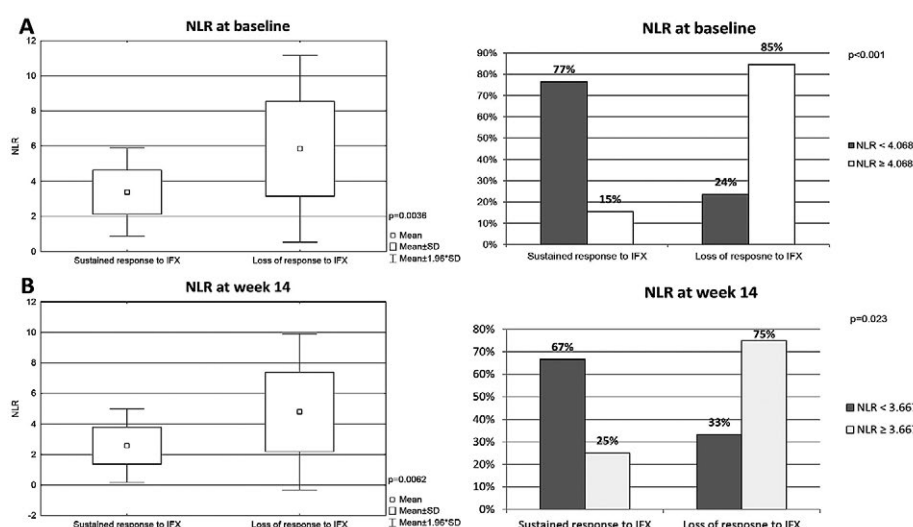


Fig. 1. Relationship between neutrophil lymphocyte ratio (NLR) at baseline (A) and after induction treatment in week 14 (B) with a sustained response to maintaining infliximab treatment. Optimal predicting cut off points were assigned by ROC curve analysis.

escalate the dose of IFX, change drugs within the same group or search for other treatments (e.g. combo therapy, antibodies against cell adhesion molecules).

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Conflicts of interest: No conflict to declare.

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Appendiceal mucocele in ulcerative colitis: a rare association and a crucial preoperative diagnosis

To the Editor,

After a long period of clinical remission, a 71-year-old male with longstanding history of ulcerative colitis (UC) complained of rectal bleeding, urgency, increased stool frequency (8/day), abdominal pain, fever and weight loss. Abnormal laboratory findings included leukocytosis and increased C-reactive protein (80 mg/L). Colonoscopy with biopsies revealed moderately active ulcerative pancolitis with predominant rectosigmoid involvement consistent with acute UC relapse, which was treated with oral and topical steroids with clinical benefit and endoscopic improvement. Meanwhile, multidetector CT (Fig.1) revealed a fluid-filled tubular structure located between the cecum and the right iliac fossa, with thin enhancing walls and a distal cyst-like configuration suggesting appendiceal mucocele (AM), without inflammatory changes of the surrounding fat. Laparoscopic appendectomy was performed, and pathology confirmed distal spherical

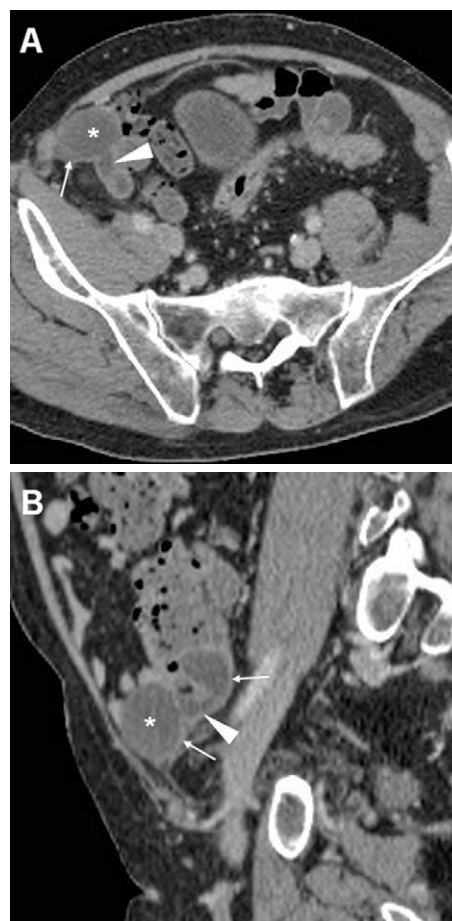


Fig. 1. Axial (A) and sagittal (B) contrast-enhanced multidetector CT images show a roundish structure (*) in the right iliac fossa (3.5-cm transverse diameter, homogeneous near-water (10-12 Hounsfield Units) attenuation and absent internal enhancement, corresponding to a cyst-like dilatation of the distal end of the vermiform appendix (arrowheads). Note the uniformly thin enhancing walls (thin arrows) without calcifications and nodularities, and the lack of inflammatory changes of the surrounding fat planes.

dilatation of the vermiform appendix containing mucoid material, consistent with low-grade mucinous neoplasia.

A rare entity with an incidence of 2-3/1000 appendectomies, AM may occur as an incidental surgical or imaging finding, or manifest with unspecific symptoms such as right lower quadrant pain, palpable mass and vomiting often mimicking acute appendicitis. Occasional complications include bowel obstruction, intussusception, bleeding and pseudomyxoma peritonei from peritoneal rupture. The descriptive term AM refers to appendiceal dilatation from intraluminal accumulation of mucoid substance, regardless of the underlying pathology which is currently categorized as non-neoplastic (retention cyst or mucosal hyperplasia), cystadenoma, or cystadenocarcinoma [1].

Sporadic (less than ten) literature reports described the occurrence of mucinous appendiceal cystadenoma in patients with UC. Although the relation between AM and UC remains debated, some authors have suggested that chronic

inflammation of the appendiceal orifice may cause luminal obstruction. Endoscopically, AM is suggested by cecum indentation, elevation or yellowish mucous discharge of the appendiceal orifice [2, 3].

Since CT imaging is increasingly performed to investigate elective cases and acute exacerbations of UC, awareness of this rare disorder is required to avoid misinterpretation of AM as acute appendicitis, abscess, salpingitis or adnexal tumour [4]. Typical CT features of AM include a well-demarcated round or tubular structure with homogeneous near-water attenuation and enhancing wall in the expected site of the vermiform appendix. Cystic appendiceal dilatation with maximal diameter ≥ 15 mm has been reported to confidently diagnose AM with 71-83% sensitivity and 88-92% specificity. Furthermore, although cystadenocarcinoma is uncommon (approximately 10% of AM cases), enhancing mural nodules should be reported as suggestive of malignancy [1-3, 5].

Appendectomy is the treatment of choice for AM. Correct preoperative diagnosis is crucial to avoid complications such as unintended intraperitoneal dissemination. Careful appendix resection and intraoperative handling are warranted to prevent the development of pseudomyxoma peritonei, and currently most surgeons prefer open over laparoscopic resection [1].

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Comment to the article Gastric Cat Scratch - an Upper Endoscopy Finding?

To the Editor,

We read with great interest the article by Carvalho et al. in the last issue of your journal [1]. The authors claimed that

they saw the multiple bright red linear lesions in the antrum which they called a “gastric cat scratch”, and this finding has never been reported. We appreciate the efforts of the Authors for making this topic available to the journal, and we would like to comment on this image.

First, we do not agree with description of the gastric lesion as a “gastric cat scratch”. To our knowledge from the literature, these lesions are described as a “gastric reddish streak”, and are well known to many expert endoscopists with this name [2]. Also this kind of endoscopic finding is not rare. Although not having enough literature data, Chen et al. demonstrated that those lesions were “gastric reddish streak” in 63 patients with functional dyspepsia, and commented on those lesions based on the histopathological features as “transient reactive gastropathy” due to the factors of their association with *Helicobacter pylori*, non-steroidal anti-inflammatory drug (NSAID) use and bile retention. Consistent with this diagnosis, we observed a 5-mm round erosion (at 1 o'clock) in the image (available on <http://www.jgld.ro/2014/4/2.html>), probably NSAID-related. Chen et al. also claimed that the fundamental histological features of gastric reddish streaks are transient unified epithelial and vascular changes owing to the mentioned factors and not to barotrauma. Secondly, those lesions do not resemble colonic cat scratch lesions in terms of the endoscopic view. In all case series, colonic cat scratch lesions are commonly observed endoscopically as linear mucosal breaks that sometimes are very thin (1-2 mm) and a little irregularly shaped like a lightning streak, sometimes thicker and more irregular, but those gastric lesions commonly are thicker (>5 mm), very regular and parallel corkscrew shaped as glove fingers or watermelon [3, 4]. Moreover, gastric lesions are always located in thick-walled regions such as the antrum, not in thin-walled regions such as the fundus, but colon cat scratch lesions are commonly located in thin-walled places such as the cecum, ascending colon. Colonic cat scratch lesions are considered to be the result of barotrauma due to a long and difficult process, but the barotrauma is not possible for gastric lesions due to the short and easy process. On the other hand, the two cases of “esophageal cat scratches” may not be exactly considered as real cat scratch lesions, because those lesions have prominent esophageal peeling and tearing features and the biopsy-proven [5]. Moreover, these cases have been published before as esophageal parakeratosis [6].

We prepared and sent a mini-questionnaire form to 21 experts, having a minimum of 10 years endoscopic experience, with the image and clinical information. Questions were listed as follows: What is your diagnosis of this image? Does this image look like a cat scratch lesion in the colon? Have you ever seen such a lesion? Answers were as follows: 85.7% diagnosed this lesion as a gastric reddish streak (18/21), 14.2% diagnosed this as watermelon-like stomach (3/21), 100% did not accept this lesion as cat scratch (21/21), and 95.2% already had observed such lesions in the antrum.

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Reply,

We would hereby like take this opportunity to express our highest gratitude for your interest in our case, along with the forwarded comments relating to our Gastric scratch article. We considered the expression “gastric cat scratch” as the most

appropriate to our observations. First, contrary to the lesions studied by Chen et al. [1], the gastric reddish streaks, our patient did not have any risk factor correlated with such lesions, denying recent or past consumption of NSAIDs. Furthermore, the biopsies performed did not show atrophy related lesions, or *H. pylori* presence.

In order to categorize our observations, we discussed our findings profoundly. We realized that the image presented had a different endoscopic appearance when compared with the described lesions, a fact that enhanced its interest, due to the impressive precise definition of the linear lesions. Such lesions appeared when the endoscope was removed, were not shown at insertion, thus not consistent with the typical watermelon-like stomach or the typical gastric reddish streaks.

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