paradoxically harmful in IBD. However, disruption of the alternative pathway by deleting NFkB2 protected murine colon from developing inflammation and this was associated with reduced expression of TNF-α and IL-14. Pharmacological inhibition of the NFkB2 signalling pathway may therefore be a promising novel therapeutic strategy for IBD.

Competing interests None declared.

REFERENCES

None declared.

Competing interests None declared.

DIFFERING PHENOTYPE IN ELDERLY IBD; SHOULD MONTREAL INCLUDE AN A4 CATEGORY

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A A McNicol,* N Kennedy, J Satsangi, I D Arnott.

Introduction The phenotype of elderly-onset IBD is poorly described and knowledge lags behind that of other age-groups. While not the dominant age-group in the disease population, those diagnosed over 60 will compose a larger proportion it as the general population ages over the next decade. The Montreal classification for IBD stratifies related to age into three categories, 40(A3) the study aims to ascertain if disease phenotype varies between those aged 40–59 and those >60 with possibility of an A4 group becoming viable if variance is noted.

Methods 1957 patients with IBD were identified using the IBD database at the Western General Hospital. We selected all those UC (n=506) and CD (n=135) diagnosed at age 40 and over (A3) and subdivided the group in to those over and under 60. The diagnosis adhered to the criteria of Lennard-Jones and IBD was categorised according to the Montréal classification. Data collected included diagnosis, age at diagnosis, disease distribution, disease behaviour and smoking history. Follow-up were available for 5 years following diagnosis. Analysis of the groups was undertaken using χ² and Fishers exact test.

Results Gender of CD patients in the different age groups (40–59; M/F=55/96, >60; M/F=10/49; p=0.0115) illustrated a higher proportion of women in the >60 group. CD patients who were diagnosed over the age of 60 had more isolated colonic disease at diagnosis. (L2, 40–59 N=22/77, >60 N=57/55, p=0.0032 15; 40–59 N=15/77, >60 N=2/38 p=0.0073). By 5 years of follow-up these differences were no longer significant. There was no difference in disease behaviour or smoking history. UC patients had more left sided disease and less distal disease at diagnosis (E1; 40–59 N=70/204, >60 N=20/102, p=0.0079 E2; 40–59 N=83/204, >60 N=57/102 p=0.039). Smoking history showed a greater proportion of former smokers in the >60 group (40–59 N=108/216, >60 N=70/107 p=0.0058).

Conclusion Disease phenotype at Dx in both UC and Crohn’s differs in the over-60s at diagnosis but normalises to that of the A3 population at follow-up. This data suggests that the introduction of an additional Montreal age classification, A4, would be clinically meaningful. Further analysis will demonstrate whether response to treatment differs in this age group.

Competing interests None declared.

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Competing interests None declared.

COMPETING INTERESTS

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Competing interests None declared.

INFLIXIMAB INDUCTION THERAPY ALONE FOR ULCERATIVE COLITIS DOES NOT RESULT IN LONG TERM REMISSION

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A J Brooks,* K Robinson, A Wright, M E McAlindon, A Lobo.

Introduction Infliximab (IFX) has demonstrated efficacy in moderate to severe ulcerative colitis (UC) with a reduction in short-term colectomy rates. In the UK, the National Institute for Health and Clinical Excellence (NICE) guidance relates to an induction course of three-doses for severely active ulcerative colitis. The aim of this study was to determine outcomes following IFX induction, including colectomy rate, use of corticosteroids (CS) or repeat IFX induction.

Methods Patients with UC at a single large teaching centre received IFX induction for UC requiring hospitalisation or when urgent consideration of surgery was given for resistant or rapidly relapsing disease were retrospectively reviewed (2008–2011). All patients had a Simple Colitis Activity Index (SCAI) at 0, 2, 6 weeks.

Results Twenty-seven patients were studied, median age 38 (range 23–64), with 17 (63%) refractory to oral or intravenous CS (13 and 4 respectively). All received CS in the year preceding IFX; median 1course (range 1–4), 23 (85%) were on immunosuppression (IS) (16 thiopurines, 7 methotrexate), 3 intolerant or non-responsive and 1 naive to IS. Twenty (74%) received induction IFX alone. Median SCAI was 8 (range 4–13), 4 (range 0–9), 2 (range 0–10) at 0, 2 and
7 weeks respectively. Nine (45%) had a good clinical response, 6
(30%) had a partial response, and 5 (25%) had no response; median
SCAI at end of induction 0, 4 and 7 respectively. Colectomy rate at
1 year post IFX induction by response was 2/9 (22%) with a good
response, 3/6 (50%) with a partial response, 5/5 (100%) for no
response, with partial or no response significantly more likely to
result in colectomy compared a good response (p=0.02). Overall, the
coelectomy rate for induction IFX at 1 year was 0.50 (10/20; 0.30 in
1st 3 months, 0.20 in months 4–12). Seven (35%) required CS at a
median of 3 months (range 0–5) and 25% (5/20) a 2nd induction
course of IFX at a median of 4.5 months (range 2–25) post IFX
induction. Of these 5, 2 (53%) had a colectomy, 1 is receiving
maintenance IFX, 1 had 3rd induction with IFX, 1 had an infusion
reaction and commenced adalimumab. Following initial IFX induc-
ction, a further 26% (7/27) received maintenance IFX infusions,
median 3 (range 1–6). Of these, 1 receives maintenance IFX, 3
stopped due to lack of funding and are in remission, and 3 (45%) lost
response-requiring colectomy.

Conclusion Response to induction IFX determined by SCAI is useful
in predicting colectomy in challenging UC patients with resistant or
rapidly relapsing UC despite IS. Further induction or maintenance
IFX is unlikely to result in remission with partial response on SCAI
post induction IFX alone and in this group may be considered as a
bridge to surgery.

Competing interests None declared.

REFERENCES

PMO-228 VITAMIN D DEFICIENCY IN A COHORT OF IBD PATIENTS TREATED WITH ANTI-TNFα THERAPY
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Introduction There is a documented association between low
Vitamin D levels and IBD. In addition to the metabolic effects of
Vitamin D it also has an immunomodulatory role which includes
inhibition of the Th1 response (IL-2, IL-10 and TNF-α). Vitamin D
deficiency may be an effect of the inflammatory process resulting in
malabsorption of Vitamin D from the gastrointestinal tract or a
propagating factor in IBD through loss of Th1 suppression. Vitamin D
deficiency may affect the response of IBD patients to biologic
drugs that act through the same immunological pathway. The
primary aim of this study was to determine the prevalence of
Vitamin D deficiency in a cohort of IBD patients currently receiving
biologic therapy and investigate whether levels were associated with
disease activity as determined by the GI inflammatory marker, faecal
calprotectin. The secondary aim was to determine if Vitamin D level
was associated with the following parameters: treatment group,
corresponding serum CRP, history of small bowel Crohn’s, small
bowel resection.

Methods Patients receiving infliximab or adalimumab therapy for
IBD at Glasgow Royal Infirmary between 1 June and 31 July 2011
were included in this retrospective cohort study (n = 113). The
following patient information was extracted from the NHS Greater
Glasgow and Clyde Clinical Portal Database: treatment regime, start
date, underlying GI diagnosis, GI surgical history, previous biologic
therapy, Vitamin D level (serum 25OHD) and corresponding serum
CRP and faecal calprotectin.

Results 60 patients (53.1%) had a recorded Vitamin D level. Of
these, 63.4% (n = 38) were Vitamin D deficient (25 OHD <50 nmol/ l); 21.4% (n = 13) were severely Vitamin D deficient (25 OHD
<25 nmol/l). The median Vitamin D level of the active disease
group (faecal calprotectin >200 μg/g) was 41 nmol/l (range:
<14–122 nmol/l) vs 39 nmol/l (range: 17–108 nmol/l) in the
remission disease group (faecal calprotectin <200 μg/g), p = 0.63.
There were no significant associations between Vitamin D level and
biologic treatment group (p = 0.65), small bowel resection (p = 0.62),
history of small bowel Crohn’s (p = 0.42), and corresponding serum
CRP (p = 0.33).

Conclusion Significant Vitamin D deficiency is common in our
cohort of IBD patients receiving anti-TNFα therapy. Vitamin D level
appears to be independent of disease activity and other specified
parameters. There is evidence to consider the routine measurement
of Vitamin D levels in IBD patients receiving biologic therapy and
appropriate treatment of Vitamin D deficiency.

Competing interests None declared.

PMO-229 MICRORNA EXPRESSION PROFILING IN STRUCTURING
CROHN’S DISEASE IDENTIFIES MIR-34A AS A
FUNCTIONALLY RELEVANT INFLUENCE ON DISEASE
PHENOTYPE
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Introduction Intestinal fibrosis is a frequent complication in Crohn’s
disease (CD), with subsequent stricture development that may
require surgical intervention. MicroRNAs (miRNAs) are a novel
class of post-transcriptional gene regulators implicated in cardiac,
hepatic and pulmonary fibrosis. MiRNAs play a key role as modu-
lators of the potent pro-fibrotic cytokine transforming growth
factor (TGF)-β, which is up-regulated in CD intestinal strictures.
Here, we aimed to identify and investigate the functional charac-
teristics of miRNAs with differential expression between strictered
and non-strictered CD.

Methods Intestinal surgical specimens were collected from 17
patients with fibrosenosing CD, and total RNA was extracted from
uninflamed ileal mucosa. MiRNA expression profiling was performed using Illumina v2.0 microRNA array comparing matched
strictered to non-strictered areas from the same patient within each
experimental group. Subsequent validation of differentially
expressed miRNAs was performed using qRT-PCR. Primary mucosal
fibroblast cultures were derived from strictered CD and healthy
control tissue. Overexpression of miRNAs was induced by trans-
faction with Dharmafect agent under optimised conditions and
changes in mRNA expression were detected by RT-qPCR and
protein expression by IHC and Western Blotting.

Results We detected 11 miRNAs significantly up-regulated and 10
miRNAs significantly down-regulated (all p < 0.02) between the
stricted and non-stricted ileum. Validation experiments con-
fi~med the changes of mir-34a in an independent set of 3
matched strictered vs non-strictered tissues. MiR-34a has been
identified as a direct target of P53 which is involved in murine
kidney fibrosis. When overexpressed in primary fibroblast cell lines,
mir-34a increases expression of COL1A2 and COL3A1 mRNA
in strictered CD cell line. However, upregulation of P53 mRNA or
protein was not detected in 6 matched paired tissues, indicating a
P53-independent mechanism by which mir-34a exerts its
pro-fibrotic effects.