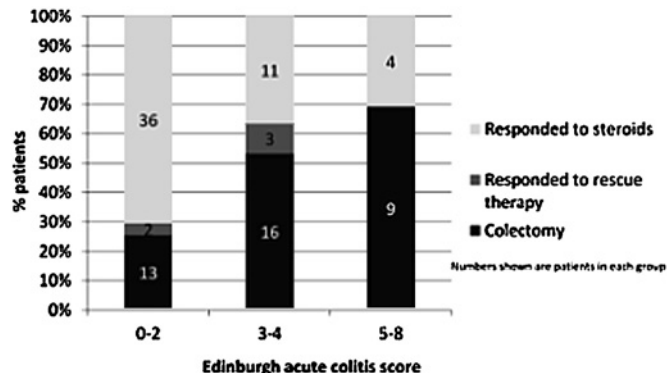


of 10%, 31%, 61% and 75%. For day 3 parameters, both the Edinburgh acute colitis (Ho) score (Abstract PTU-123 figure 1) and Travis criteria performed well.



Abstract PTU-123 Figure 1

Conclusion ASUC remains an important cause of colectomy. This study confirms the prognostic value of the Ho score and Travis criteria at day 3, but also indicates that day 0 CRP and albumin are strong predictors of outcome.

Competing interests None declared.

REFERENCES

1. Dinesen LC, et al. *J Crohns Colitis* 2010;**4**:431–7.
2. Ho GT, et al. *Aliment Pharmacol Ther* 2004;**19**:1079–87.
3. Turner D, et al. *Clin Gastroenterol Hepatol* 2007;**5**:103–10.

PTU-124 ARE PSEUDOPOLYPS THE SOURCE OF TUMORIGENIC MUTATIONS IN ULCERATIVE COLITIS?

doi:10.1136/gutjnl-2012-302514c.124

¹N Jawad,* ²T Graham, ³M Novelli, ³M Rodriguez-Justo, ⁴R Feakins, ¹A Silver, ¹N Wright, ¹S McDonald. ¹Digestive Diseases Department, Blizard Institute, Barts and The London School of Medicine and Dentistry, Queen Mary, University of London, London, UK; ²Histopathology Department, Cancer Research UK, Lincoln's Inn Fields, London, UK; ³Histopathology Department, University College Hospital, London, UK; ⁴Histopathology Department, Bart's and The London, London, UK

Introduction Pseudopolyps develop as a result of mucosal ulceration and epithelial regeneration.¹ They appear as islands of relatively normal epithelium in otherwise denuded mucosa and are the likely source of epithelium from which the mucosa repairs. They can be associated with areas of dysplasia but are thought to be benign. We have previously shown that protumourigenic mutations can spread through the entire colon in patients with UC-associated cancer.² We hypothesise that pseudopolyps are clonal expansions of crypts that have acquired a protumourigenic survival advantage over surrounding normal epithelium that frequently perishes in the inflammatory milieu.

Methods To determine the genetic status of pseudopolyps and frequency of mutated pseudopolyps. DNA extracted from macro-dissected UC-associated pseudopolyp tissue sections underwent nested PCR sequencing of common tumour suppressor and oncogenes known to be mutated in colitis associated cancers, using well established published protocols.

Results Of 30 pseudopolyp samples analysed from 30 different patients, four patients had identifiable mutations; in *CDKN2A* (c.C387T p.129Y), *TP53*-exon7 (c.C735T p.245G), and *K-RAS* (c.G37A p.G13S, and c.G35C p.Gly12Ala).

Conclusion We have shown that pseudopolyps are a potential source of protumourigenic mutations in UC. Pseudopolyps may possibly be the site within the inflamed epithelium where mutations are

harboured, and may be the source for restituted bowel. More numbers are needed to be analysed and this is planned for future work and comparison with normal matched tissue is required. These lesions have been traditionally thought to be benign, genetically inert, incidental findings, characteristic of chronic inflammation. Although this data are preliminary, these findings propose an exciting paradigm shift in the way we consider pseudopolyps and may alter endoscopic management of these lesions in the future.

Competing interests None declared.

REFERENCES

1. Jagan KN, Sircus W, Walker RJ, et al. Pseudopolypositis in ulcerative colitis. *Lancet* 1969;**2**:255.
2. Leedham SJ, Graham TA, Oukrif D, et al. Clonality, founder mutations, and field cancerization in human ulcerative colitis-associated neoplasia. *Gastroenterology* 2009;**136**:542–50.

PTU-125 POSITIVE CALPROTECTIN BUT NEGATIVE INVESTIGATIONS-WHAT NEXT?

doi:10.1136/gutjnl-2012-302514c.125

¹N Mohammed,* ²S Smale. ¹Gastroenterology, Leeds Teaching Hospitals NHS Trust and Leeds Institute of Molecular Medicine, St James University Hospital, Leeds, UK; ²Gastroenterology, York teaching Hospital NHS Foundation Trust, York, UK

Introduction Faecal markers are an increasingly established markers of gastrointestinal inflammation. Faecal calprotectin has an important role in differentiating functional from organic disease.^{1 2} However a small proportion of patients have an initial abnormal calprotectin and go on to complete normal series of gastrointestinal investigations. We submit this project where in we studied the outcome of those patients who had abnormal faecal calprotectin followed by normal endoscopic/radiological investigations for gastrointestinal diseases, over a period of 3 years.

Methods We identified all 600 patients in our unit who had faecal calprotectins performed between January and June 2007. We then selected all those with abnormal calprotectins (>50 µg/g) whose initial investigations were normal (n=67) and assessed the long term follow-up of these patients.

Results All patients whose initial calprotectin was <225 µg/g had not been found to have any inflammatory or neoplastic gastro-intestinal disease in the following 3 years follow-up. In those whose calprotectin was initially >225 µg/g (n=25) nine were found to have inflammatory bowel disease, 5 were found to have other organic pathology (Clostridium difficile associated Diarrhoea, Coeliac disease, Diverticular disease, Ischaemic colitis) over the subsequent 3 years. In those who had organic disease repeat calprotectin was elevated (3/14). All other patients with an initial calprotectin of >225 µg/g were concluded to have functional disease of whom 8/11 had subsequent faecal calprotectins all of which were normal. Summary: 1. At a calprotectin of <225 µg/g all patients were subsequently concluded to have functional disease. 2. Among those in whom initial calprotectin was >225 µg/g with normal investigation only those with sequentially abnormal calprotectins (mean interval to second calprotectin 6 months, range 60–550 days) had organic disease on follow-up.

Conclusion Negative faecal calprotectin can be a very useful tool to exclude organic pathology. Borderline positive results can be misleading. Serial Calprotectin may hold promise for defining those with post-inflammatory/infective IBS or organic disease. It also reflect response to treatment and aid in disease monitoring, but further longer term study is required.

Competing interests None declared.

REFERENCES

1. Tibble, et al. *Gut* 2000;**47**:506–13.
2. Limburg, et al. *Am J Gastroenterol* 2000;**95**:2831–7.