On multivariate analysis, age, deprivation status, co-morbidity, and hospital length of stay were associated with increased 3-year mortality in both study periods.

Conclusion The overall mortality after hospitalisation for CD has not altered, although mortality associated with emergency medical admission has decreased, and now does not differ from rates after emergency surgical admission.

REFERENCE

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PWE-085 AUDIT OF OUTCOMES OF A MANAGED 5 ASA SWITCHING PROGRAM

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Introduction 5-aminosalicylic acid (5-ASA) preparations are used to induce and maintain remission in ulcerative colitis (UC). The cost of the recommended maintenance dose for oral 5ASA preparations varies from 86p and 208p per day (MIMMS 2011). Switching to cheaper 5ASAs has been suggested as a possible drug cost saving. The BNF states that preparations are not interchangeable but with little direct evidence to support this statement. The aim of this pilot study is to gain preliminary data on a primary care based 5ASA switching programme.

Methods Salofalk granules (Dr Falk Pharma) and Pentasa (Ferring Pharmaceuticals) were identified as the cheapest 5ASA items prescribed in 31 GP practices. An initial pilot study in one practice revealed that only 5/21 patients responded to a written invitation alone and successfully switched to Salofalk. We therefore developed a managed programme utilising a community based gastroenterology nurse specialist in 4 other practices.

A written invitation detailing the rationale for switching 5-ASA was sent to all appropriate patients who were then phoned a week later. If patients agreed to the switch, the GP surgery was contacted by the Nurse Specialist to change the repeat prescription. Patients were switched to Salofalk in 2 practices and Pentasa in the other 2. GP and hospital records were then examined 6 months post-switch to assess for evidence of patient acceptability and tolerability.

Results 120 patients (56 male, 64 female) with a mean age of 50 years were identified as being on a 5-ASA preparation (oral or topical). 56 (47%) were under either virtual or hospital gastroenterology follow-up. 64 patients with ulcerative colitis were taking oral 5ASAs. 21 (33%) were already taking Salofalk or Pentasa. Of the remaining 43 patients, 24 (56%) agreed to the switch, 10 (23%) declined, and 9 (21%) did

not respond to the invitation or telephone call. Of the 24 patients who agreed to switch, only 17 (71%) completed the process. 15 (88%) remained on the new 5-ASA for at least 6 months, with reasons for discontinuation cited as preference for the previous preparation or diarrhoeal symptoms.

Conclusion Conducting a managed 5ASA switching programme is feasible with 17/43 eligible patients successfully switched with 15/17 continuing on these preparations. Areas for development include following up patients who initially agreed but failed to switch, recording more robustly any flares, involving secondary physicians and assessing adherence and cost savings.

This study provides preliminary evidence to develop a large scale study in this important area.

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PWE-086 DETERMINING STEM CELL AND CRYPT DYNAMICS IN INFLAMMATORY BOWEL DISEASE

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Introduction Inflammatory bowel disease (IBD) confers a high risk of development of colitis-associated colorectal cancer (CACRC) in patients with extensive colitis. It is believed that a field effect, resulting from chronic inflammation and clonal outgrowth is present in ulcerative colitis (UC) patients, and this promotes the accumulation of protumourigenic clones via increased crypt fission rates. Increased rates of crypt fission may explain the mass expansion of protumourigenic mutations across the whole length of the bowel in a very short time period as observed in patients with CACRC (Leedham *et al.*, 2009; Galandiuk *et al.*, 2012).

Methods Fresh frozen normal colon (n = 15) and UC colon (n = 6) tissue samples were collected and sectioned in an *en face* orientation. Two-colour enzyme histochemistry for cytochrome c oxidase (CCO) activity was performed to identify clonal populations. Adjacent crypts completely deficient of CCO activity were recorded as a 'CCO deficient patch' and those with a fraction of the crypt that was CCO-deficient were designated as 'partial crypts'. The CCO-deficient clone fraction in partial crypts was estimated by recording the number of pixels within CCO-deficient (blue) and CCO-proficient (brown) areas. The number and size of clonal patches in colitis patients was compared to non-UC controls.

Results As in the healthy colon, CCO-deficient clonal patches accumulate in an age dependent fashion in the UC colon. The mean number of crypts within a CCO-deficient patch was statistically significantly larger (p < 0.05) in the UC colon than in the normal colon. In addition we observe a larger percentage of wholly and partially CCO-deficient crypts in the UC colon when compared to the normal controls.

Conclusion The proliferative drive induced by continuous inflammation and mucosal repair in UC appears to promote the expansion of CCO-deficient patches. The increase in the proportion of wholly and partially mutated crypts in UC could be explained by crypt hyperplasia with more stem cells present driving fission. This increased rate of clonal expansion may contribute to the