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Project: Is surveillance for colorectal cancer in ulcerative colitis effective?

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Introduction:

Ulcerative Colitis (UC) is a chronic, relapsing condition characterised by inflammation of the mucosal layer of the colon and rectum. It has been shown that patients with UC are at an increased risk for colorectal cancer (also referred to as colitis associated cancer, CAC). An individual's specific risk of CAC is dependent on a number of factors such as: duration of disease, extent of disease, the presence of endoscopic findings such as post inflammatory polyposis (PIP) and stenoses, familial history of colorectal cancer and co-existing primary sclerosing cholangitis. The magnitude of this risk has been researched in a number of countries and different healthcare settings, which has resulted in a large degree of variation when quantifying the risk. Research has shown that the risk of CAC only becomes significant after 10 years of disease. With this knowledge national and international guidelines have been formed which advise frequent endoscopic surveillance for patients with UC. The aim of endoscopic surveillance is to detect dysplastic changes or lesions within the colonic mucosa. Currently, the detection of high grade dysplasia (HGD) is considered an indication for colectomy, as previous research has shown synchronous cancers at the time of colectomy for HGD in up to 40% of patients, while management of low grade dysplasia is more controversial with many favouring more frequent surveillance rather than colectomy. The pathway responsible for the development of CAC involves chronic inflammation of the colonic mucosa, which can lead to dysplastic changes with the possibility of further development to CAC. Recent research in this area has raised the question of whether published guidelines are still appropriate, and what the absolute risk of CAC really is. With appropriate surveillance and our increasing ability to detect and, conservatively, manage dysplasia, CAC is a potentially preventable condition. There is no local data available on the incidence of dysplasia or CAC, nor on compliance with surveillance guidelines.

Aim:

To use local data to determine:

1. The long term incidence of dysplasia and colorectal cancer and;
2. Compliance with surveillance guidelines in the UC population within Canterbury

Method:

The Canterbury Inflammatory Bowel Disease Project 2005 recruited all patients within the Canterbury region who had been diagnosed with Inflammatory Bowel Disease. For this project we only looked at patients diagnosed with UC, and excluded patients whose diagnosis of UC was later changed. We removed patients who were living outside of the Canterbury region at the start of the study (1/06/2005) and those who did not have any electronic notes available during the study period (1/06/2005-1/11/2015). All data was collected using electronic records; written records and notes were not sought. Each patient's medical records were reviewed for disease features and records of endoscopic surveillance. Endoscopy and histology reports were reviewed to determine disease extent, disease severity, the presence of endoscopic features such as PIP, and the presence of dysplasia or CAC. Clinical notes were used to review the presence of other known risk factors such as PSC and family history of CRC. Electronic records in private clinics within the Canterbury region were reviewed to ensure complete data retrieval occurred. Follow up was considered to end when there was note of a patient moving outside of the Canterbury region, at the time of death, at the time of colectomy,

or at the time dysplasia or CAC was detected. Surveillance intervals were noted for each patient and compared against the NZGG guidelines in place at the time to determine whether surveillance for each patient had been complaint. An allowance of +/- 6months was made when assessing compliance. Colitis associated neoplasia (CAN) was defined as dysplasia within the diseased region. Dysplasia outside of the diseased region was considered to be sporadic and not related to colitis.

Results:
Overall 521 patients were included in this study; 262 were considered eligible for surveillance and 259 ineligible due to age >75 years, previous colectomy or disease limited to the rectum only. Median follow up of the cohort was 16.8 years, 146 (28%) patients had extensive disease and 29 (5.6%) had PSC. Of the 521 patients, 34 developed CAN of any grade (6.53%); 6 (3.1%) patients had high-grade dysplasia or CAC detected histologically over the study period. Univariate analysis showed that the cohort who developed dysplasia had: a longer duration of disease (28.3yrs vs 21.0yrs), a greater proportion of patients had extensive disease (70.6% vs 23.8%), and a greater proportion had severe disease (20.6% vs 5.6%) when compared against the cohort who did not develop dysplasia. Of the 262 patients eligible for surveillance only 21.4% had surveillance that complied with the NZGG guidelines at the time. Of the cohort whose surveillance was non-complaint, 66.0% experienced a delay in receiving their surveillance, with a median delay time of 1.6years.

Conclusion:
Our study found that while colitis associated neoplasia and cancer are occurring locally, they are being detected at rates much lower than those reported in previous studies. We found that a majority of patients were receiving surveillance that did not comply with local NZGG guidelines, with most experiencing delays between colonoscopic procedures. With the findings of our study we think that a review of the current guidelines would be appropriate.