Conclusion In the setting of acute severe ulcerative colitis, serum calprotectin is comparable with serum CRP in predicting outcome. Further work is needed to establish if it may be a useful predictor of outcome in patients with ulcerative colitis who fail to mount a high CRP response despite endoscopic assessment confirming severe active inflammation. Work is also ongoing to establish its utility in the outpatient setting both in Crohn’s disease and ulcerative colitis.

Disclosure of Interest None Declared.

Abstract PTH-084 Table 1

<table>
<thead>
<tr>
<th>Variables</th>
<th>Flare up</th>
<th>No flare</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (mean ± SD)</td>
<td>51.2 ± 14.6</td>
<td>57.9 ± 12.5</td>
</tr>
<tr>
<td>Endoscopic score ≤1</td>
<td>22</td>
<td>182</td>
</tr>
<tr>
<td>Endoscopic score ≥2</td>
<td>35</td>
<td>78</td>
</tr>
<tr>
<td>Geboes score &lt; 2</td>
<td>33</td>
<td>224</td>
</tr>
<tr>
<td>Geboes score ≥2</td>
<td>24</td>
<td>36</td>
</tr>
</tbody>
</table>

Conclusion Histological activity and younger age are significant predictors of disease relapse in patients undergoing surveillance endoscopy. Endoscopic activity with standard white light endoscopy did not predict clinical relapse. Better non-invasive markers of disease relapse are required for patients with ulcerative colitis.

Disclosure of Interest None Declared.

REFERENCE


Disclosure of Interest None Declared.

REFERENCES

1. N Jawad, 1 T Graham, 3 M Novelli, 3 M Rodriguez-Justo, 1 N Wright, 1 S McDonald. Centre for Tumour Biology, Bart’s Cancer Institute, Bart’s and The London School of Medicine and Dentistry, Queen Mary University of London, London, UK; 2Centre for Evolution and Cancer, University of California at San Francisco, San Francisco, United States; 3Histo-pathology Department, University College Hospital, London, UK

Disclosure of Interest None Declared.

Disclosure of Interest

INTRODUCTION

Inflamatory bowel disease (IBD) confers a high risk of development of colitis-associated colorectal cancer (CACRC) in patients with extensive colitis. Crypt fission (a crypt bifurcating into two) has been shown to be a mechanism of clonal expansion in the intestinal epithelium. Although fission is rare in normal colon, many crypts in patients with colitis appear to be in the process of fission. A recent study from the host laboratory demonstrated that protumourigenic mutations can spread through the entire inflamed colon suggesting that this occurs at a considerable rate indicating stem cell dynamics are altered in IBD.

METHODS

Somatic mitochondrial DNA (mtDNA) mutations are a reliable marker of clonal expansion in human colon. Combining mtDNA mutations with additional markers of clonal expansion that change over time, such as methylation patterns of non-expressed genes, reveals whether populations of cells show a recent ancestry. This is measured by evaluating methylation pattern diversity between samples. Methylation patterns of CSX and MYOD1 genes were examined in clonally related and unrelated crypts from multiple areas in IBD patients by laser capture microdissection bisulphite sequencing. Clonality was demonstrated by cytochrome c oxidase deficient (CCO-) cells sharing an identical somatic mtDNA mutation.

RESULTS

In active inflammation, both adjacent clonally related CCO- crypts and adjacent unrelated crypts had similar methylation patterns, indicating recent crypt fission. In contrast, adjacent unrelated crypts in quiescent disease had dissimilar methylation patterns, indicating that crypt fission rates are slow and resemble that of normal colon. The number of unique methylation patterns in crypts from active IBD were significantly less than those obtained of normal colon. The number of unique methylation patterns, indicating recent crypt fission. In contrast, adjacent unrelated crypts in quiescent disease had dissimilar methylation patterns, indicating that crypt fission rates are slow and resemble that of normal colon. The number of unique methylation patterns in crypts from active IBD were significantly less than those obtained of normal colon. The number of unique methylation patterns, indicating recent crypt fission. In contrast, adjacent unrelated crypts in quiescent disease had dissimilar methylation patterns, indicating that crypt fission rates are slow and resemble that of normal colon. The number of unique methylation patterns in crypts from active IBD were significantly less than those obtained of normal colon. The number of unique methylation patterns, indicating recent crypt fission. In contrast, adjacent unrelated crypts in quiescent disease had dissimilar methylation patterns, indicating that crypt fission rates are slow and resemble that of normal colon. The number of unique methylation patterns in crypts from active IBD were significantly less than those obtained of normal colon. The number of unique methylation patterns, indicating recent crypt fission. In contrast, adjacent unrelated crypts in quiescent disease had dissimilar methylation patterns, indicating that crypt fission rates are slow and resemble that of normal colon. The number of unique methylation patterns in crypts from active IBD were significantly less than those obtained of normal colon.

CONCLUSION

Elevated crypt fission in active IBD may explain the extensive dispersion of protumourigenic clones previously observed in IBD. Subsequent cycles of crypt atrophy and mucosal healing by crypt fission, may provide a key growth stimulus in the inflamed colon. Furthermore, there appears to be an increased rate at which a single stem cell populates the niche within IBD crypts. Such expansion facilitates the establishment of protumourigenic mutations within crypts.

Disclosure of Interest None Declared.

REFERENCES


Disclosure of Interest None Declared.

INTRODUCTION

In active inflammation, both adjacent clonally related CCO- crypts and adjacent unrelated crypts had similar methylation patterns, indicating recent crypt fission. In contrast, adjacent unrelated crypts in quiescent disease had dissimilar methylation patterns, indicating that crypt fission rates are slow and resemble that of normal colon. The number of unique methylation patterns in crypts from active IBD were significantly less than those obtained of normal colon. The number of unique methylation patterns, indicating recent crypt fission. In contrast, adjacent unrelated crypts in quiescent disease had dissimilar methylation patterns, indicating that crypt fission rates are slow and resemble that of normal colon. The number of unique methylation patterns in crypts from active IBD were significantly less than those obtained of normal colon. The number of unique methylation patterns, indicating recent crypt fission. In contrast, adjacent unrelated crypts in quiescent disease had dissimilar methylation patterns, indicating that crypt fission rates are slow and resemble that of normal colon. The number of unique methylation patterns in crypts from active IBD were significantly less than those obtained of normal colon. The number of unique methylation patterns, indicating recent crypt fission. In contrast, adjacent unrelated crypts in quiescent disease had dissimilar methylation patterns, indicating that crypt fission rates are slow and resemble that of normal colon. The number of unique methylation patterns in crypts from active IBD were significantly less than those obtained of normal colon.