Correlation between colonic mural apparent diffusion coefficient and clinical/biochemical markers of inflammation in acute colitis

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Aim: to investigate the relationship of MRI derived colonic mural apparent diffusion coefficient (ADC) with clinical and biochemical markers of inflammation for acute colitis patients.

Introduction: Active inflammation in acute colitis may resolve with medical therapy, whilst failure to respond often necessitates surgery with removal of the large bowel. Currently, assessment of response to drug treatment is based on a combination of clinical (e.g. stool frequency, temperature) and biochemical markers (C-reactive protein [CRP]) of inflammation, imaging (abdominal radiograph, computed tomography) and endoscopy [1,2]. Despite these measures, assessment of response is often problematic; CRP maybe elevated in other pathologies and repeated conventional imaging imparts a subtstantial dose of ionising radiation to a predominantly young patient group. T2 weighted and contrast enhanced MR imaging has been used to assess inflammation within the small bowel, providing excellent depiction of both mural and extra-mural tissue [3]. High mural T2 signal intensity is related to inflammatory activity and likely caused by mural oedema [4], but to date no study has assessed mural MR diffusion changes. Diffusion weighted imaging is known to be extremely sensitive to changes in intracellular and extracellular water in neuroimaging, and increased Apparent Diffusion Coefficient (ADC) is seen with oedema [5]. The purpose of this study was to investigate whether a similar relationship exists between colonic mural ADC and inflammation.

Method: Local ethics committee permission was obtained for the study. Ten patients (4 male) were recruited. All patients had clinically diagnosed acute colitis requiring hospital admission. As part of routine clinical practice, CRP and stool frequency was measured on admission. In addition patients were invited to undergo MRI, which was performed within 24 hours of admission. For the MRI study, patients were fasted for four hours before scanning. Bowel motility was reduced by intravenous administration of 0.3mg/kg (maximum 20 mg) of spasmolytic (Buscopan, Boehringer Ingelheim, Germany) immediately prior to abdominal imaging. Images were acquired in the prone position using a 1.5T Siemens Avanto (Erlangen, Germany) magnet with the manufacturer's body and spine array coils. Coronal Half Fourier Acquisition Single Shot Turbo Spin Echo (HASTE) images of the abdomen and pelvis were produced during breath hold acquisitions to provide anatomical localisation (FOV variable to encompass patient anatomy, slices 19-27, stacks 1-2, TR 1200 ms, TE 52 ms, matrix 256 x 195, slice thickness 4 mm, interslice gap 5.2 mm, averages 4, echo train 256, iPAT 2). Coronal free gentle breathing echo planar diffusion weighted images (EPI-DWI) with incrementing b values (0, 50, 200 and 400) were acquired of the same imaging volume (FOV as for HASTE, slices 19-27, stacks 1, TR 7900 ms, TE 82 ms, matrix 192 x 154, slice thickness 4 mm, interslice gap 5.2 mm, averages 1, iPAT 2). Repeat CRP, stool frequency measurement and imaging was performed at a mean of 3.6 days (range 1 to 6 days) following initial assessment. Anonymized pre and post treatment MR images were evaluated by a radiologist (5 years of MRI experience) unaware of the clinical and biochemical results. The colon was anatomically divided into segments (rectum, sigmoid, descending, transverse, ascending and ceacum). Each segment was initially assessed on HASTE images for increased wall thickness, increased mural HASTE signal, loss of haustral pattern and peri-colonic mesenteric oedema. The most severely involved segment was selected and analysed on diffusion weighted images. Signal was measured from a single region of interest (ROI) placed within the selected segment bowel wall on diffusion weighted images. The ROI was manually repositioned to keep it in an anatomically constant location on subsequent increasing diffusion weighted images (thereby allowing calculation of mural ADC from the ROI). Pre and post treatment ROI placement was carefully matched allowing paired pre and post treatment ADC determination. Spearman statistics were used to test association of mural ADC with CRP and stool frequency; and change in mural ADC with change in CRP and change in stool frequency following treatment.

Results: Pre and post treatment mean mural ADC, CRP and stool frequency were 2.98 x 10^{-3}mm^{2}s^{-1} (range 1.77 x 10^{-3} to 5.04 x 10^{-3}), 31.6 mg/l (range 5 to 104) and 9.9 stools/day (range 8 to 12); and 3.03 x 10^{-3}mm^{2}s^{-1} (range 1.92 x 10^{-3} to 4.66 x 10^{-3}), 14.4 mg/l (range 5 to 41.4) and 6.2 stools/day (range 3 to 10) respectively. There was a strong positive correlation between CRP and ADC prior to treatment (Figure 1, p=0.003) but no such correlation was observed post treatment (p=0.303). Following therapy there was a positive association between ΔCRP and ΔADC (Figure 2, p=0.03). There was no significant correlation between ADC and stool frequency (pre: p=0.334, post: p=0.759) or ΔADC and change in stool frequency following treatment (p=0.166).

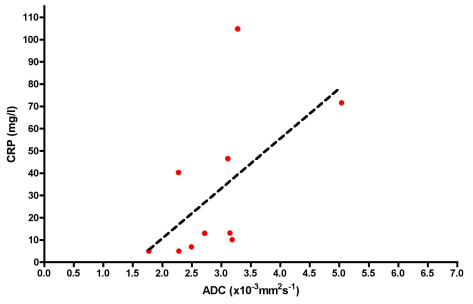

Figure 1: *Pre treatment mural ADC vs CRP*

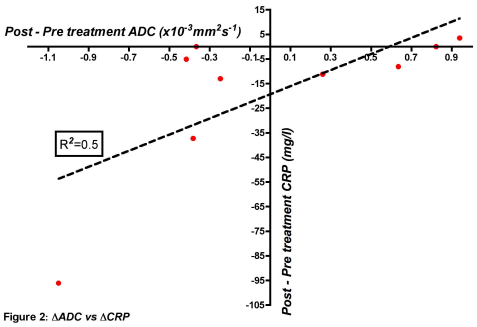

Figure 2: *ΔADC vs ΔCRP*

Conclusion: The pilot data presented suggest that colonic mural ADC is related to inflammation and that following medical therapy a reduction in inflammation is reflected by a reduction in mural ADC. This change in ADC may reflect decreasing mural oedema with resolving inflammation. Further studies are needed to assess whether ADC can provide a non-invasive quantitative site specific measure of colonic inflammation and to assess its potential utility as a predictor of treatment outcome.

References: [1] Ka Ho Lok, *Journal of Digestive Diseases, (2008)9; 219-224.* **[2]** da Luz Moreira A, *J Gastrointest Surg.,* (2008) Nov 1 Epub. **[3]** Maccioni F., *Radiology* (2006)238; 517-530. **[4]** Koh DM., *Americal Journal of Roentgenology,* (2001), 177; 1325-1332. **[5]** Guzman R., *Journal of Neuroradiology*, (2008), 35; 224-229.