Intestinal inflammation in Crohn’s disease (CD) is associated with an increase in gut Polypeptide YY (PYY), Glucagon-like peptide 1 (GLP-1) and cholecystokinin (CCK). Patients with CD suffer from postprandial symptoms like fullness and nausea. These symptoms are believed to be linked to the increase expression of gut peptides and to the alteration in intestinal motility.

Our aim was to investigate the link between gut peptide, small bowel motility and patient symptom response after a standard test meal.

METHODS

Subjects underwent baseline and postprandial MRI scans, symptom questionnaires and blood sampling (GLP-1, PYY, CCK) at intervals for 270 min following a test meal: soup (400g) (chicken or mushroom) (Heinz, Wigan, UK); (kcal) 51, protein 1.5 g, carbohydrate 4.7 g, fat 2.9 g per 100g.

MRI scans were performed using a 1.5T Philips Achieva MRI scanner. Gastric volume, small bowel water content (SBWC) and small bowel motility were assessed using MRI. Patients also underwent a standard contrast enhanced clinical MR enterography (MRE) and the MaRIA score applied to quantify disease activity. All subjects gave informed written consent.

Trial registration number: NCT03052465. Data is presented as mean +/- SEM.

RESULTS

16 CD patients with active small bowel disease (table1) and 20 age-, BMI- and gender-matched healthy volunteers (HV) were recruited (table1). Results are summarised in table 3 and figure 1.

CD patients showed a significantly (P≤0.05) slower fasting small bowel motility (50±6a.u.) compared to HV (77±10a.u.). Postprandial SBWC was significantly greater in CD than HV (measured as area under the curve CD: 18452, HV: 13760, P≤0.05). Fasting PYY (CD: 236±16 pg/mL, HV: 118±11 pg/mL, P≤0.0001) and GLP-1 (CD: 50±8 µg/mL, HV: 13±3 µg/mL, P≤0.0001) were significantly higher in CD compared to HV with this difference persisting at each time point of the study (P≤0.0001). The meal induced a significant increase (P≤0.0001) in fullness, bloating and abdominal pain scores in patients (28±4mm, 22±3mm and 12±2mm respectively) compared to HV (12±4mm, 3±3mm and 1±2mm respectively). No differences were noted in gastric volumes, CCK concentration and postprandial motility.

CONCLUSIONS

The fasting hypomotility noted in CD may be ascribed to the increased fasting GI peptides. An increase postprandial SBWC and postprandial symptoms has been observed in CD. We plan to replicate these pilot data in a larger cohort with the aim of identifying key biomarkers for pharmacological modulation to improve patient symptoms.