Up to 40% of the patients with inflammatory bowel disease (IBD) have MRI evidence of axial spondyloarthritis compared to 1.5% in the non IBD population. Here we aim to distinguish axial spondyloarthritis associated with IBD (axSpA-IBD) from axSpA without IBD (axSpA-not IBD) and psoriatic axSpA (axSpA-PsA)

Method A collaborative group comprising of gastroenterologists and rheumatologists was formed between 2 healthcare trusts. Clinic lists and electronic operating systems were interrogated with appropriate ethical approval to identify patients with axSpA-IBD, axSpA-not IBD and axSpA-PsA. Patients were contacted prior to their clinic appointments and were sent stool containers. On the day of their clinic appointments, they were consulted for 15–30 minutes where their diseases were carefully phenotyped, drug history obtained, and informed consent taken. Stool and blood samples were collected and analysed for faecal calprotectin and acute inflammatory markers.

Results 62 patients were analysed for this study; 22 patients (10 Crohn’s/12 UC) with axSpA-IBD, 24 with axSpA-not IBD and 16 patients with axSpA-PsA. Of the patients with IBD, 13 out of 22 (59%) were diagnosed with IBD first. Median duration of IBD before the diagnosis of axSpA was 3.5 years. Remaining 9 patients who were diagnosed with axSpA first, the median duration of disease before the diagnosis IBD was 3.5 years as well.

Median age of onset for joint disease in axSpA-IBD is 30 years compared to 27 and 22 in axSpA-not IBD and axSpA-PsA cohorts.

All three cohorts had gender predominance towards the male sex with 82% in axSpA-IBD, 86% in axSpA-not IBD and 94% in axSpA-PsA.

Association with HLA B*27 is stronger in the axSpA-not IBD group with 76% patients possessing the polymorphism compared to 50% in axSpA-IBD and 40% in axSpA-PsA. Median BASDAI (ankylosing spondylitis disease activity score) for axSpA-IBD, axSpA-not IBD and axSpA-PsA are 4.3, 3.95 and 3.2 respectively. Scores of above 4 indicate active disease.

Mean faecal calprotectin in axSpA-IBD is 233.7 ug/g compared to 99.1 and 28.2 in axSpA-not IBD and axSpA-PsA.

Pearson correlation coefficient between faecal calprotectin and BASDAI scores was 0.0451.

Discussion In our cohort, we have shown that IBD related axial spondyloarthritis is different to axSpA-not IBD in terms of its weaker association with HLA B*27 antigen, higher degree of gut inflammation and increased age of onset for joint disease. Mean faecal calprotectin in axSpA-not IBD was still significantly higher than axSpA-PsA.

Weaker association with HLA B* 27 antigen in axSpA-IBD points to the possibility of a different genetic loci or mode of aetio-pathogenesis.

Contrary to what we had expected, male gender predominance, joint disease activity and functional disability are similar across both axSpA-IBD and axSpA-not IBD group. We have also shown that there is no correlation between gut inflammation and joint disease activity (R= 0.0451) which again is consistent with previous findings that inflammatory axial disease is independent of gut inflammation.