difference in disease severity. The number of patients referred directly to gastroenterology rose from 3% (2013) to 17% (2016), while 10% of diagnoses were made during emergency admissions (unchanged, table 1). Referrals via the 2-week wait pathway remained high (38% 2013, 28% in 2016), whilst many had initial investigations at independent centres contracted to provide NHS care (16% in 2013, 24% in 2016).

Time from referral to diagnosis of IBD increased non-significantly from 2013 (0.79 months) to 2016 (1.27, $p=0.2$). 48 patients had FC checked prior to referral and 37.5% of these were referred direct to gastroenterology. Time to diagnosis was marginally longer in patients with FC compared to those without (1.47 vs 0.86 months, $p=0.06$). Time from diagnosis to treatment reduced from 1.37 (2013) to 0.72 months (2016, $p=0.01$).

Abstract PTH-121 Table 1 Routes of initial referral

	2013 cohort (n=104)			2016 cohort (n=144)		
	Initial referral	Time to diagnosis (mean months)	Time to treatment (mean months)	Initial referral	Time to diagnosis (mean months)	Time to treatment (mean months)
Independent NHS providers	17	N/A	1.0	34	N/A	0.8
GP to Gastro	3	2.0	0.67	25	1.16	0.44
GP to Surgeons	18	2.0	1.0	14	3.8	1.62
Colorectal 2 week wait pathway	39	0.64	1.62	40	0.6	0.89
Hospital Admission	13	0.31	0.8	14	0.07	0.31
Hepatology	3	N/A	1.33	2	3.0	0
Private care	1	0	0	7	1.14	0.8
Bowel Cancer screening	3	N/A	5.0	5	N/A	1.67
Others	7	N/A	1.6	2	N/A	0.5

Conclusion FC has contributed to an increased proportion of referrals direct to gastroenterology, but time to diagnosis in patients with FC was marginally longer. There are considerable differences in time to diagnosis and treatment depending on referral routes. Importantly, 30% present via the 2-week wait pathway and 10% as emergency admissions. Further work is required to ensure patients with suspected IBD get referred to the most appropriate service in a timely manner.

PTH-122 FAECAL CALPROTECTIN SUGGESTS PRESENCE OF GUT INFLAMMATION IN AXIAL SPONDYLOARTHRITIS WITHOUT IBD

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Studies since early 1980s have shown the presence of subclinical gut inflammation in up to 60% of the patients with axial spondyloarthritis, 6% of which proceeded to become inflammatory bowel disease (IBD) in 10 years. Here we aim to further explore the presence of gut inflammation using acute inflammatory markers in stool and serum and correlate the results with disease characteristics.

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 with or without IBD (axSpA-IBD and axSpA not-IBD, respectively) and psoriatic arthritis as disease controls. Eligible patients were called prior to their clinic appointments and were sent stool containers. On the day of their clinic appointments, patients were consulted for 15–30 minutes where their disease was carefully phenotyped, drug history obtained and they were consented in to the study. Stool and blood samples were collected and analysed for faecal calprotectin (FC) and ESR and CRP respectively.

Results 116 patients with axial spondyloarthritis (79 axSpA-not IBD, 22 axSpA-IBD and 15 psoriatic axSpA [axSpA-PsA] and 22 patients with psoriatic peripheral spondyloarthritis (pPsA) were recruited.

Total of 81 stool samples were analysed. Faecal calprotectin (FC) was elevated to above 50ug/g in 36 stool samples; 21 out of 44 (48%) patients with axSpA-not IBD (range 51–587), 9 out of 18 (50%) patients with axSpA-IBD (range 51–>2000), 2 out of 7 (29%) patients with axSpA-PsA (range 51–87) and 6 out of 14 (43%) patients with pPsA (range 57–122). Of the elevated FC samples, only one patient with known Crohn's disease had significant GI symptoms.

10 out of 36 (28%) patients with elevated FC were taking daily NSAIDs compared to 4 out of 48 (8%) patients with normal FC. Daily NSAID users did not have higher mean FC.

9 out of 11 (82%) patients who had disease duration of more than 10 years had elevated FC.

18 out of 45 (40%) patients who were younger than 30 years old when they developed axSpA had elevated FC (median 51ug/g) compared to 19 out of 35 (54%) of patients who were older or equal to 30 years of age (median 36ug/g). 13 out of 28 (46%) patients with BASDAI index (axSpA disease activity score) of > 4 had elevated FC and 13 out of 33 (39%) with BASDAI < 4. BASDAI of above 4 reflects active disease.

Patients on biological therapy were equally distributed across all cohorts.

Discussion Our study shows that significant proportion of patients with axSpA-not IBD have evidence of gut inflammation relatively independent of their NSAID use. There is some correlation between gut inflammation and age of symptoms onset, disease duration and serum inflammatory markers. As we had expected, mean faecal calprotectin in patients with axSpA-IBD is higher than patients with axSpA-not IBD. Based on this study, a discussion needs to be had for lowering the threshold of performing colonoscopies in axSpA-not IBD cohort.