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Original Research Article

Inflammatory Bowel Disease in Patients with Chronic Abdominal Pain: A Colonoscopy-Based Cross-Sectional Study

Shrusti Joshi[1], N.L. Vyas[2], Randhir Singh Rao[3], Shaffi Sharma[4]

[1]Final year PG resident, Department of General Surgery, Jaipur National University Institute for Medical Sciences and Research Centre, Jaipur, Rajasthan, India

[2]Professor and Head of Department, Department of General Surgery, Jaipur National University Institute for Medical Sciences and Research Centre, Jaipur, Rajasthan, India

[3]Associate Professor, Department of General Surgery, Jaipur National University Institute for Medical Sciences and Research Centre, Jaipur, Rajasthan, India

[4]Final year PG resident, Department of General Surgery, Jaipur National University Institute for Medical Sciences and Research Centre, Jaipur, Rajasthan, India

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Corresponding author: Dr. Shrusti Joshi

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Abstract

Background. Chronic abdominal pain is a common and non-specific complaint that may be the first sign of inflammatory bowel disease (IBD), yet it overlaps heavily with functional and infectious disorders. We examined how often IBD underlies chronic abdominal pain in patients referred for colonoscopy, and characterised the features that separate ulcerative colitis (UC) from Crohn's disease (CD).

Methods. In this cross-sectional observational study, 50 adults with chronic abdominal pain (> 3 months) undergoing colonoscopy at a tertiary teaching hospital in Jaipur were evaluated. Diagnosis rested on colonoscopy with targeted biopsy, supported by serology (pANCA, ASCA) and faecal calprotectin. Disease was classified by the Montreal system and graded by Truelove–Witts/Mayo and CDAI scores. Groups were compared with the chi-square or Fisher exact test ($p < 0.05$ significant).

Results. Ulcerative colitis accounted for 42 patients (84.0%) and Crohn's disease for 8 (16.0%). The cohort was young (mean age 36.6 ± 11.5 years) and predominantly female (70.0%), with no demographic difference between subtypes. Rectal bleeding, mucus discharge and tenesmus were significantly commoner in UC, while weight loss and perianal disease characterised CD. CD presented later (15.6 vs 8.2 months; $p = 0.006$) and carried higher ESR, CRP and calprotectin and lower albumin. Colonoscopy and histology separated the subtypes cleanly, and pANCA (UC) and ASCA (CD) reached predictive values approaching 97% in combination. Complications were commoner in CD (75.0% vs 38.1%; $p = 0.046$).

Conclusion. IBD was a frequent diagnosis in patients with chronic abdominal pain, with UC predominating but CD behaving more aggressively and presenting later. Colonoscopy with biopsy was the cornerstone of diagnosis, with serology and calprotectin as useful non-invasive adjuncts.

Keywords: inflammatory bowel disease; ulcerative colitis; Crohn's disease; colonoscopy; chronic abdominal pain; faecal calprotectin.

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Introduction

Inflammatory bowel disease (IBD) is a chronic, immune-mediated disorder of the gut that runs a relapsing and remitting course and steadily erodes quality of life, work capacity and long-term health [1]. It is conventionally divided into two overlapping entities — ulcerative colitis (UC) and Crohn's disease (CD) — which share immunological roots but diverge in where and how they damage the bowel [2,3]. Ulcerative colitis is confined to the colon and rectum, producing continuous mucosal inflammation that begins at the rectum and spreads proximally [3]. Crohn's disease, by contrast, can strike anywhere from mouth to anus, most often the terminal ileum and colon, and its transmural, patchy inflammation tends to throw up strictures, fistulae and abscesses [4,5]. Why the disease develops at all is still not fully settled. The prevailing view is that IBD emerges when an environmental trigger meets a susceptible genome and a disturbed gut microbiome, tipping mucosal immunity into chronic inflammation [6]. Genome-wide studies have catalogued many risk loci, yet

genetics alone cannot explain how quickly incidence has shifted within a single generation [7], which points firmly at environment and lifestyle. For most of the twentieth century IBD was a disease of the industrialised West, with incidence rates of roughly 2 to 24 per 100,000 person-years [8]. That geography is now changing. Over the past two decades incidence has climbed sharply across Asia, India, Latin America and the Middle East, regions where the disease was once considered a rarity [9,10] — a transition widely attributed to urbanisation, dietary change, antibiotic exposure and the other accompaniments of modern living [11]. India, in particular, now carries a substantial and growing burden.

Chronic abdominal pain is among the commonest reasons patients seek gastroenterological care, and it is frequently the opening symptom of IBD. The difficulty is that pain overlaps heavily with irritable bowel syndrome, infectious colitis and other benign causes, so the diagnosis is easily missed or delayed — a delay that, especially in Crohn's disease, translates into more complications and more surgery [12]. Colonoscopy with targeted biopsy is the reference standard for sorting these patients out: it allows the mucosa to be inspected directly and tissue to be sampled for histology, distinguishing UC from CD and excluding mimics [13,14].

In a setting such as India, where chronic pain is often attributed reflexively to functional or infectious causes [15], a low threshold for colonoscopy is especially valuable. Against this background, we set out to determine how often IBD underlies chronic abdominal pain in patients referred for colonoscopy, and to characterise the clinical, endoscopic, histological and serological features that separate the two major subtypes.

Materials and Methods

This cross-sectional observational study was conducted in the Department of General Surgery of a tertiary teaching hospital in Jaipur over approximately eighteen months, following institutional ethics committee approval. We enrolled adults aged 18 years or older who presented with chronic abdominal pain of more than three months' duration — with or without altered bowel habit, bleeding per rectum or mucus discharge — and who were scheduled for colonoscopy. Patients with a prior diagnosis of IBD, those positive for HIV, hepatitis B or hepatitis C, pregnant women, those with intestinal obstruction, and anyone unfit for or unwilling to undergo the procedure were excluded. Written informed consent was obtained from every participant. The required sample size, calculated for comparison of two means at 5% significance and 90% power using variance estimates from an earlier study, was 50 patients.

Every eligible patient underwent colonoscopy, during which biopsies were taken from visible lesions or representative mucosa and sent for histopathological examination. To sharpen the distinction between subtypes, two serological markers were measured in all patients: perinuclear anti-neutrophil cytoplasmic antibody (pANCA), which tends to accompany ulcerative colitis, and anti-Saccharomyces cerevisiae antibody (ASCA), which is more often seen in Crohn's disease; the two were interpreted together where histology alone was equivocal. Faecal calprotectin was also measured in every patient as a marker of intestinal inflammation and to help separate inflammatory from non-inflammatory causes of pain. Disease extent and behaviour were classified by the Montreal system, and activity was graded by the Truelove–Witts criteria and Mayo score for UC and the Crohn's Disease Activity Index (CDAI) for CD.

Data were analysed in SPSS. Categorical variables were expressed as frequencies and compared with the chi-square or Fisher exact test; continuous variables were summarised as mean ± standard deviation or median with interquartile range according to distribution (Shapiro–Wilk test) and compared with the t-test or Mann–Whitney U test. A two-sided p value below 0.05 was considered significant.

Results

Of the 50 patients, 42 (84.0%) had ulcerative colitis and 8 (16.0%) had Crohn's disease — a roughly five-to-one preponderance of UC. The cohort was young, with a mean age of 36.6 ± 11.5 years and most patients under 46, and women outnumbered men more than two to one (70.0%). None of the demographic variables — age, sex, urban versus rural residence or socioeconomic class — differed significantly between the two subtypes (Table 1).

Table 1: Demographic characteristics by IBD subtype (N = 50)

Variable	UC (n = 42)	CD (n = 8)	Total (N = 50)	p
Mean age, years	37.2 ± 11.8	33.5 ± 9.4	36.6 ± 11.5	0.398
Female, n (%)	29 (69.0)	6 (75.0)	35 (70.0)	0.714
Urban residence, n (%)	28 (66.7)	5 (62.5)	33 (66.0)	0.682
Middle socioeconomic class, n (%)	23 (54.8)	5 (62.5)	28 (56.0)	0.756

Chronic abdominal pain was almost universal (96.0%), as expected given the entry criterion, and chronic diarrhoea was reported by 86.0%. Several symptoms, however, separated the subtypes cleanly (Table 2).

Bleeding per rectum (95.2% vs 50.0%; p = 0.001), mucus discharge (p = 0.024) and tenesmus (p = 0.038) were all far more frequent in ulcerative colitis, mirroring its rectal, mucosal pattern of inflammation. Crohn's disease, in turn, was marked by unintentional weight loss (75.0% vs 33.3%; p = 0.022) and perianal symptoms (37.5% vs 7.1%; p = 0.014), in keeping with its transmural, often small-bowel disease.

Table 2: Presenting symptoms by IBD subtype (N = 50)

Symptom	UC (n = 42)	CD (n = 8)	Total	p
Chronic abdominal pain	40 (95.2)	8 (100)	48 (96.0)	0.512
Chronic diarrhoea	37 (88.1)	6 (75.0)	43 (86.0)	0.298
Bleeding per rectum	40 (95.2)	4 (50.0)	44 (88.0)	0.001
Mucus discharge per rectum	28 (66.7)	2 (25.0)	30 (60.0)	0.024
Tenesmus	22 (52.4)	1 (12.5)	23 (46.0)	0.038
Unintentional weight loss	14 (33.3)	6 (75.0)	20 (40.0)	0.022
Perianal symptoms	3 (7.1)	3 (37.5)	6 (12.0)	0.014

The two groups also differed in how long they had been unwell before reaching care. Crohn's patients had endured symptoms for far longer than those with UC (mean 15.6 vs 8.2 months; p = 0.006), and a quarter of them presented only after more than two years — a delay that fits the disease's insidious, intermittent and bleeding-poor course.

The laboratory picture told the same story of a heavier inflammatory and nutritional toll in Crohn's disease: ESR (p = 0.048), CRP (p = 0.012) and faecal calprotectin (p = 0.042) were all higher, while serum albumin (p = 0.008) and total protein (p = 0.048) were lower than in UC, consistent with transmural inflammation and malabsorption.

Colonoscopy and histology drew the sharpest line between the subtypes (Table 3). Continuous involvement, rectal disease, loss of vascular pattern and friability were the hallmarks of ulcerative colitis, present in nearly all such patients, whereas skip lesions, cobblestoning, terminal-ileal involvement and strictures clustered almost exclusively in Crohn's disease.

Histopathology reinforced the distinction: crypt architectural distortion, basal plasmacytosis and goblet-cell depletion favoured UC, while transmural inflammation, non-caseating granulomas and skip lesions on biopsy were essentially confined to CD.

Table 3: Key colonoscopic and histopathological features differentiating UC from CD

Feature	UC (n = 42)	CD (n = 8)	p
Continuous involvement (endoscopy)	42 (100)	1 (12.5)	<0.001
Rectal involvement (endoscopy)	40 (95.2)	3 (37.5)	<0.001
Loss of vascular pattern (endoscopy)	40 (95.2)	3 (37.5)	<0.001
Skip lesions (endoscopy)	0 (0)	6 (75.0)	<0.001
Cobblestone appearance (endoscopy)	0 (0)	5 (62.5)	<0.001
Terminal ileum involvement (endoscopy)	0 (0)	6 (75.0)	<0.001
Transmural inflammation (histology)	0 (0)	7 (87.5)	<0.001
Non-caseating granuloma (histology)	0 (0)	5 (62.5)	<0.001

Serology performed well as an adjunct (Table 4). pANCA was positive in 81.0% of UC patients but only 12.5% of those with CD, while ASCA showed the reverse pattern (87.5% in CD vs 14.3% in UC; both p < 0.001). The reciprocal pANCA-positive/ASCA-negative combination identified UC with a positive predictive value of 97.1%, and the pANCA-negative/ASCA-positive pattern identified CD with a negative predictive value of 97.4%. Faecal calprotectin tracked disease activity closely, correlating with the Mayo score in UC (r = 0.66) and the CDAI in CD (r = 0.74; both p < 0.001), and a level above 250 µg/g flagged active disease with 82.6% sensitivity.

Table 4: Diagnostic performance of serological markers and faecal calprotectin

Marker / combination	Sens (%)	Spec (%)	PPV (%)	NPV (%)
pANCA-positive (for UC)	81.0	87.5	97.1	43.8
ASCA-negative (for UC)	85.7	87.5	97.3	50.0
ASCA-positive (for CD)	87.5	85.7	53.8	97.3
pANCA-negative / ASCA-positive (for CD)	87.5	90.5	63.6	97.4
Faecal calprotectin > 250 µg/g (active disease)	82.6	70.4	86.4	63.3

Complications were more than twice as common in Crohn's disease (75.0% vs 38.1%; p = 0.046), driven by strictures, fistulae, abscesses and perforation, whereas the few cases of toxic

megacolon and massive haemorrhage occurred in UC. Most patients in both groups had mild-to-moderate disease at presentation, with a mean Mayo score of 6.6 in UC and a mean CDAI of 256.8 in CD.

Discussion

The headline finding — that ulcerative colitis outnumbered Crohn's disease by more than five to one — fits the traditional South Asian pattern, in which UC has long been the commoner form. It runs counter, though, to the large community-based colonoscopy study of Banerjee and colleagues, who screened more than 30,000 symptomatic Indians and found Crohn's disease to be the more frequent diagnosis among the 5.4% with IBD [16]. The discrepancy probably reflects differences in setting and referral: a tertiary surgical clinic seeing patients with bleeding and chronic pain will inevitably enrich for UC, whereas a community screen captures a broader spectrum. Our distribution sits closer to the colonoscopic-biopsy series of Karve and colleagues, in which ulcerative colitis clearly outnumbered Crohn's disease [17].

The patients were young — most in their thirties and forties — and predominantly female, a pattern that diverges from the male or equal sex distribution reported in much of the Western literature and deserves further study. Clinically, the symptom profile did much of the diagnostic work before the scope was even passed. Rectal bleeding, mucus and tenesmus pointed to UC, while weight loss and perianal disease pointed to CD — exactly the discriminators that Passos and Hong each highlighted when arguing that careful endoscopic and clinical assessment reliably separates the two conditions [18,19].

One of the more clinically important observations was the long diagnostic lead-time in Crohn's disease, with a mean symptom duration nearly twice that of UC and a quarter of patients presenting beyond two years. Crohn's announces itself quietly — intermittent pain and weight loss rather than the overt bleeding that drives UC patients to seek help — and that quietness carries a cost in strictures and surgery. Raghuveer and colleagues made much the same point in their tertiary-care series, noting the high diagnostic yield of colonoscopy in precisely this group of patients with chronic abdominal symptoms [20].

The laboratory and endoscopic findings were internally consistent and biologically sensible. The higher ESR, CRP and calprotectin and the lower albumin in Crohn's disease reflect its transmural, systemically active inflammation, and the endoscopic and histological features split almost perfectly along textbook lines. This is the territory in which colonoscopy is indispensable: Kim, Spiceland and Kilcoyne have all underlined that direct visualisation with targeted biopsy, complemented where needed by cross-sectional imaging, is what allows UC and CD to be distinguished and their complications detected [21,22,23].

Our serological results echo the wider literature in showing that pANCA and ASCA, while neither perfectly sensitive nor specific on their own, become powerful when combined — the reciprocal pANCA-positive/ASCA-negative and pANCA-negative/ASCA-positive patterns carried predictive values approaching 97% for UC and CD respectively. Faecal calprotectin earned its place too, correlating strongly with both the Mayo score and the CDAI; as a cheap, non-invasive readout of mucosal inflammation it offers a practical way to monitor activity between colonoscopies. These markers do not replace histology — Tanaka's classic work showed how accurately defined microscopic criteria can categorise IBD [24] — but they complement it usefully.

Several limitations temper these conclusions. The sample was small (50 patients) and drawn from a single tertiary centre, so the marked UC predominance may partly reflect referral bias rather than true community prevalence, and the rural population was under-represented. Cross-sectional enterography was not applied uniformly, which may have under-detected small-bowel Crohn's disease, and there was no irritable-bowel or infectious-colitis comparison group and no long-term follow-up. Larger, multicentre studies will be needed to pin down the true prevalence of IBD among patients with chronic abdominal pain in this population.

Conclusion

In patients presenting with chronic abdominal pain, inflammatory bowel disease was a frequent and important diagnosis, and within it ulcerative colitis clearly predominated. Crohn's disease, though less common, behaved more aggressively — presenting later, carrying a heavier inflammatory and nutritional burden, and causing more complications. Colonoscopy with targeted biopsy was the linchpin of diagnosis, reliably separating the two subtypes on the basis of characteristic endoscopic and histological patterns, while serology and faecal calprotectin added useful, non-invasive corroboration and a means of gauging activity. The practical message is simple: in anyone with chronic abdominal pain, particularly when accompanied by diarrhoea or rectal bleeding, early colonoscopy shortens the road to diagnosis and offers the best chance of heading off the long-term complications of IBD.

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