ORIGINAL ARTICLE

Prevalence of Celiac Disease in Inflammatory Bowel Disease: A Prospective Cohort Study from Northern India

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Abstract

Background: Celiac disease (CeD) and inflammatory bowel disease (IBD) share immune and genetic pathophysiological pathways, suggesting a possible increased coexistence. Indian data on biopsy-confirmed CeD in IBD are limited, and false-positive anti–tissue transglutaminase (tTG) antibody results complicate interpretation.

Aim: To determine the prevalence of biopsy-confirmed CeD in an Indian IBD cohort and compare clinical features between CeD-positive and CeD-negative IBD patients.

Materials and methods: In this prospective observational study, 477 IBD patients were assessed. All patients underwent IgA anti-tTG screening. Seropositive patients underwent upper gastrointestinal endoscopy with duodenal biopsies. The standardized prevalence ratio (SPR) for CeD was calculated, and clinical and laboratory parameters were compared between patients with and without CeD.

Results: Among 477 IBD patients (UC 421; CD 56), 14 (2.94%) were anti-tTG positive, and 11 had biopsy-confirmed CeD, giving a prevalence of 2.31% (95% confidence interval: 1.29–4.08%). Prevalence was 2.14% in UC and 3.57% in CD (p = 0.59). The SPR, compared with the Indian population prevalence of 1.04%, was 2.22. The anti-tTG false-positive rate was 21.4%. No significant differences were observed between CeD-positive and CeD-negative patients, although UC patients with CeD showed a non-significant trend toward pancolitis.

Conclusion: In conclusion, CeD prevalence in Indian IBD patients was numerically higher than the Northern Indian general population but did not reach statistical significance. Larger multicenter studies are needed to definitively establish the CeD–IBD association in Indian populations. Selective screening is preferable, and histological confirmation remains essential due to high false-positive serology.

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Introduction

Celiac disease (CeD) is a chronic immune-mediated enteropathy triggered by dietary gluten exposure in genetically predisposed individuals.[1] The global prevalence is estimated at 1.0–1.4%,[2] while studies from India report a prevalence of about 1.04% in Northern regions and between 0.3% and 1.23% across different parts of the country.[3–5] Diagnosis requires a combination of serological testing, upper gastrointestinal endoscopy (UGIE), and characteristic histopathological findings.[6]

Ulcerative colitis (UC) and Crohn's disease (CD) are the major forms of inflammatory bowel disease (IBD), characterized by chronic intestinal inflammation in genetically susceptible individuals. CeD and IBD share several clinical and biological features, including genetic susceptibility through human leukocyte antigen (HLA) DQ2/DQ8 haplotypes and compromised mucosal barrier integrity.[7]

Coexisting CeD in patients with IBD has been associated with extensive colitis, refractory symptoms, and increased risk of disease flares,[8] making early detection clinically important. However, evidence regarding the prevalence of CeD among IBD patients remains inconsistent. Early work by Salem and Truelove[9] suggested an association between UC and villous atrophy, while subsequent studies have reported both higher and comparable CeD rates relative to the general population.[10–12] These variations likely reflect differences in population characteristics, diagnostic criteria, and serological assay performance. Furthermore, false-positive anti–tissue transglutaminase (tTG) antibody results in IBD further complicate interpretation.[13] Determining the true prevalence of biopsy-confirmed CeD in Indian IBD patients is therefore essential for appropriate screening and clinical decision-making.

Indian data remain limited, with few prospective studies using standardized diagnostic methods to evaluate biopsy-confirmed CeD in IBD. This study aimed to determine the prevalence of biopsy-confirmed CeD in a well-characterized IBD cohort from Northern India and identify clinical factors associated with CeD–IBD overlap.

Materials and Methods

This prospective observational study was conducted in the Department of Gastroenterology, Sawai Man Singh Medical College and Group of Hospitals, Jaipur, between April 2021 and June 2024. Institutional Ethics Committee approval was obtained, and written informed consent was taken from all participants. The study adhered to the Declaration of Helsinki (1975; revised 2013).

All consecutive adult patients (≥18 years) with a confirmed diagnosis of IBD-UC or CD, attending outpatient or inpatient services during the study period, were enrolled. Patients below 18 years of age, those unwilling to participate, and patients already following a gluten-free diet (as this could lead to false-negative anti-tTG results) were excluded from the study. The diagnosis of IBD was based on European Crohn's and Colitis Organisation–European Society of Gastrointestinal and Abdominal Radiology (ECCO–ESGAR) guidelines[14,15] using combined clinical, endoscopic, radiologic, and histopathologic criteria. UC was classified as proctitis (E1), left-sided colitis (E2), or pancolitis (E3), and CD was classified by disease location as ileal (L1), colonic (L2), ileocolonic (L3), or upper gastrointestinal (L4).[16] Extraintestinal manifestations (EIMs) were systematically documented.

All enrolled patients underwent screening for IgA anti-tTG antibodies using a commercial enzyme-linked immunosorbent assay (ELISA) kit (CHORUS tTGA, Siena, Italy). Serum IgA concentrations were measured in all subjects; patients with IgA deficiency

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(IgA <7 mg/dL) underwent additional IgG-based tTG testing. Seropositive patients were advised to undergo UGIE with at least six duodenal biopsies (two from the bulb, four from the second part), which were evaluated using the modified Marsh–Oberhuber grading system.[17] Patients with discordant serology and histology underwent human leukocyte antigen (HLA) DQ2/DQ8 genotyping using polymerase chain reaction (PCR)-based typing. The diagnosis of CeD followed European Society for Pediatric Gastroenterology, Hepatology and Nutrition (ESPGHAN) recommendations,[18] requiring compatible histology (Marsh ≥2) and/or HLA positivity with symptomatic response to a gluten-free diet.

Statistical Analysis

Data were analyzed using IBM SPSS Statistics version 23.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD) or median (interquartile range, IQR), and categorical variables as frequencies and percentages. Group comparisons used chi-square or Fisher's exact test for categorical variables and Student's *t*-test or Mann–Whitney *U* test for continuous variables. A p-value < 0.05 was considered significant. The standardized prevalence ratio (SPR) was calculated using the Northern Indian general population prevalence of 1.04% reported by Makharia et al.,[3] a community-based study from the same geographic region as our cohort, ensuring appropriate demographic and genetic comparability for prevalence estimation. Diagnostic accuracy parameters of IgA anti-tTG testing were calculated using duodenal histology as the reference standard.

Results

Between April 2021 and June 2024, 477 consecutive patients with IBD were screened for CeD. The final cohort of 477 patients represents one of the largest prospective Indian cohorts systematically screened for CeD in IBD. This cohort comprised 421 patients with UC (88.3%) and 56 with CD (11.7%). The mean age was 37.8 ± 13.2 years, with a male-to-female ratio of 1.26:1. Baseline characteristics are summarized in Table 1. The mean hemoglobin level was 10.24 ± 2.64 g/dL, with no significant difference between UC and CD (10.26 ± 2.71 vs 10.08 ± 2.23 g/dL; $p = 0.65$). The mean platelet count was 3.08 ± 1.33 × 10^5/mm^3. The average disease duration was 4.9 ± 0.9 years.

Among UC patients, left-sided colitis (E2) was the most common extent (54.4%), followed by pancolitis (E3, 31.1%) and proctitis (E1, 10.0%); extent was unclassified in 4.5%. Among CD patients, ileocolonic disease (L3) predominated (68.8%), followed by colonic disease (L2, 18.8%), ileal disease (L1, 6.3%), and upper gastrointestinal involvement (L4, 6.3%). EIMs were documented in 72 of 477 patients (15.1%), occurring more frequently in CD (37.5%) than UC (12.1%) ($p < 0.001$) (Table 2).

Figure 1 illustrates the screening and diagnostic pathway. 14 patients (2.94%) were seropositive for IgA tTG antibodies. All underwent upper gastrointestinal endoscopy with duodenal biopsies. 11 patients were confirmed to have CeD based on Marsh criteria, supported by HLA-DQ2/DQ8 genotyping when indicated. The overall prevalence of biopsy-confirmed CeD in the IBD cohort was 2.31% (95% confidence interval (CI): 1.29–4.08%). Prevalence was 2.14% among UC patients (9/421) and 3.57% among CD patients (2/56), with no significant difference ($p = 0.59$). The SPR of CeD in IBD patients, compared with the reported prevalence in the Indian general population (1.04%), was 2.22, although this difference was not statistically significant ($p = 0.655$) (Table 3). The false-positive rate of IgA anti-tTG testing was 21.4% (3/14). Test sensitivity was 100%, specificity 99.4%, positive predictive value 78.6%, and negative predictive value 100%.

Among patients with UC–CeD overlap ($n = 9$), pancolitis was the predominant phenotype (55.6%), followed by left-sided colitis (44.4%). None had isolated proctitis. Both CD-CeD patients had ileocolonic disease (L3). Although extensive disease (E3) was numerically more frequent in CeD-positive UC patients compared with CeD-negative UC patients (55.6% vs 30.6%), the difference was not statistically significant ($p = 0.42$). Compared with CeD-negative patients,

Table 1: Baseline characteristics of study patients

Parameter	*IBD (n = 477)*	*UC (n = 421)*	*CD (n = 56)*
Age (years, mean ± SD)	37.8 ± 13.2	37.8 ± 13.0	37.9 ± 14.8
Male: female (%)	266:211 (55.8: 44.2)	239:182 (56.8: 43.2)	28:28 (50: 50)
Hemoglobin (g/dL)	10.24 ± 2.64	10.26 ± 2.71	10.08 ± 2.23
Platelet count (×10^5/mm^3)	3.08 ± 1.33	3.06 ± 1.28	3.21 ± 1.59
Disease duration (years)	4.9 ± 0.9	5.0 ± 0.9	4.3 ± 1.0
UC extent			
E1–Proctitis	–	42 (10.0%)	
E2–Left-sided	–	229 (54.4%)	
E3–Pancolitis	–	131 (31.1%)	
Extent not recorded	–	19 (4.5%)	
CD location			
L1–Ileal	–	–	4 (6.3%)
L2–Colonic	–	–	10 (18.8%)
L3–Ileocolonic	–	–	38 (68.8%)
L4–Upper gastrointestinal	–	–	4 (6.3%)

Table 2: Extraintestinal manifestations in ulcerative colitis and Crohn's disease

EIM	*UC [n = 421 (%)]*	*CD [n = 56 (%)]*	*Total [n = 477 (%)]*
Musculoskeletal			
Arthralgia/arthritis	9 (2.2)	0 (0)	9 (1.9)
Ankylosing spondylitis	5 (1.1)	2 (3.6)	7 (1.5)
Endocrine			
Hypothyroidism	14 (3.3)	0 (0)	14 (2.9)
Diabetes mellitus	1 (0.2)	1 (1.8)	2 (0.4)
Infectious/mucocutaneous			
Candidiasis (oral or esophageal)	19 (4.5)	3 (5.4)	22 (4.6)
Other*	≤1	≤1	7(1.5%)
Gastrointestinal complications			
Perianal fistula	0 (0)	7 (12.5)	7 (1.5)
Stricture	0 (0)	4 (7.1)	4 (0.8)
Any EIM present	51 (12.1)	21 (37.5)	72 (15.1)

*Includes primary sclerosing cholangitis, cholangiocarcinoma, neuropathy, rectovaginal fistula, etc.

CeD-positive patients showed no significant differences in age, sex, disease duration, hemoglobin, platelets, or EIM frequency except hypothyroidism [27.3% (3/11) vs 2.4% (11/466), $p = 0.005$] (Table 4).

Characteristics of the 11 IBD–CeD overlap cases are described in Table 5. The median age was 36 years (range: 22–58), with near-equal sex distribution (6 males, 5 females). Nine patients had UC, and two had CD. Median disease duration was 5 years (range: 2–8). Marsh 3a, 3b, and 3c lesions were seen in 4 (36.4%), 5 (45.5%), and 2 (18.2%) patients, respectively. Chronic diarrhea was universal (100%). Weight loss (72.7%), anemia (81.8%), and abdominal pain (63.6%) were frequent. Six patients (54.5%) had at least one EIM, most commonly hypothyroidism and arthralgia.

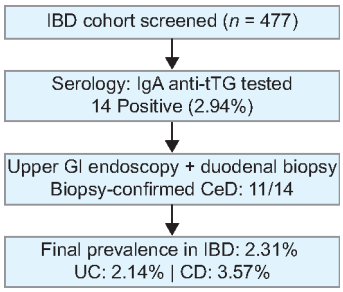

Fig. 1: Screening and diagnostic flowchart for the study

Discussion

In this prospective cohort of 477 Indian IBD patients, the prevalence of histologically-confirmed CeD was 2.31%, compared to the 1.04% population-based prevalence, corresponding to an SPR of 2.22, although this did not reach statistical significance.[3] The prevalence was similar between UC (2.14%) and CD (3.57%) patients. With only 11 biopsy-confirmed CeD cases, our study had limited statistical power to detect small but clinically meaningful differences in prevalence. The non-significant *p*-value (0.655) may reflect a type II error rather than a true absence of association, and these findings warrant validation in larger multicenter studies.

Our findings differ from several Western meta-analyses, which have reported a stronger association. Shah et al. reported an odds ratio of 2.23 (95% CI: 1.99–2.50) for CeD in patients with IBD, while Pinto-Sanchez et al. reported a risk ratio of 3.96 (95% CI: 2.23–7.02) in their 2020 meta-analysis.[11–12] However, these analyses demonstrated substantial heterogeneity (I^2 >70%), with individual study estimates varying widely (0–4.2%). Importantly, several cohort studies from Western populations have reported CeD prevalence rates of 0.5–3.2% in IBD,[19–21] aligning more closely with our findings than the pooled meta-analytic estimates. Thus,

Table 3: Prevalence of celiac disease in inflammatory bowel disease compared with the general population

Group	*n*	*CeD cases*	*Prevalence, % (95% CI)*	*SPR**	*p-value*
All IBD	477	11	2.31 (1.29–4.08)	2.22	0.655
Ulcerative colitis	421	9	2.14 (1.13–4.01)	2.06	0.734
Crohn's disease	56	2	3.57 (0.98–12.12)	3.43	0.607
Seropositive (IgA-tTG)	477	14	2.94 (1.76–4.87)	2.83	0.447

*SPR (standardized prevalence ratio) = Observed prevalence in IBD/expected prevalence in the general population

Table 4: Clinical characteristics: celiac disease positive and celiac disease negative Inflammatory bowel disease patients

Parameter	*IBD with CeD (n = 11)*	*IBD without CeD (n = 466)*	*p-value*
Demographics			
Age (years, mean ± SD)	38.5 ± 11.8	37.8 ± 13.3	0.87
Male:female	6:5 (54.5%:45.5%)	260:206 (55.8%:44.2%)	0.93
IBD type			
Ulcerative colitis	9 (81.8%)	412 (88.4%)	0.59
Crohn's disease	2 (18.2%)	54 (11.6%)	
Laboratory parameters			
Hemoglobin (gm/dL)	9.87 ± 2.21	10.25 ± 2.65	0.65
Platelet count ($\times 10^5/mm^3$)	3.18 ± 1.45	3.08 ± 1.33	0.78
Disease characteristics			
Disease duration (years)	5.2 ± 1.1	4.9 ± 0.9	0.35
UC disease extent (*n* = 9 with CeD, *n* = 412 without)			0.42
Proctitis (E1)	0 (0%)	42 (10.2%)	
Left-sided colitis (E2)	4 (44.4%)	225 (54.6%)	
Pancolitis (E3)	5 (55.6%)	126 (30.6%)	
CD location (*n* = 2 with CeD, *n* = 54 without)			0.88
Ileal (L1)	0 (0%)	4 (7.4%)	
Colonic (L2)	0 (0%)	10 (18.5%)	
Ileocolonic (L3)	2 (100%)	36 (66.7%)	
Upper GI (L4)	0 (0%)	4 (7.4%)	
Clinical features			
Any EIM present	2 (18.2%)	70 (15.0%)	0.72
Hypothyroidism	3 (27.3%)	11 (2.4%)	0.005
Anemia (Hb <12 gm/dL)	9 (81.8%)	348 (74.7%)	0.73
Weight loss	8 (72.7%)	245 (52.6%)	0.18

Table 5: Individual characteristics of inflammatory bowel disease—celiac disease overlap cases (n = 11)

Case	*Age/sex*	*IBD type*	*Duration(years)*	*IBD extent/ Location*	*Marsh grade*	*EIM*	*Presenting symptoms*
1	42/M	UC	6	E3 (pancolitis)	3b	None	Diarrhea, weight loss, and anemia
2	36/F	UC	5	E2 (left-sided)	3a	Hypothyroidism	Diarrhea, fatigue
3	28/M	UC	4	E3 (pancolitis)	3c	Arthralgia	Bloody diarrhea, abdominal pain
4	52/F	UC	7	E2 (left-sided)	3a	Hypothyroidism	Diarrhea, anemia
5	34/M	UC	5	E3 (pancolitis)	3b	None	Diarrhea, weight loss
6	45/F	UC	6	E2 (left-sided)	3a	None	Diarrhea, bloating
7	29/M	UC	3	E3 (pancolitis)	3c	None	Bloody diarrhea, weight loss
8	38/F	UC	5	E2 (left-sided)	3b	Hypothyroidism	Diarrhea, anemia, fatigue
9	58/F	UC	8	E3 (pancolitis)	3b	None	Diarrhea, weight loss
10	32/M	CD	4	L3 (ileocolonic)	3a	Perianal fistula	Diarrhea, abdominal pain, and weight loss
11	22/M	CD	2	L3 (ileocolonic)	3b	Arthralgia	Diarrhea, weight loss, and anemia

methodological and population differences may partly explain the weaker association in our cohort.

Reports from Middle Eastern and Asian countries similarly show modest associations: CeD prevalence of 0.5% in Iran,[22] 2.0% in Turkey,[23] and even lower rates in Japan.[24] Large registry-based studies from Sweden[25] and Israel[26] have shown stronger associations, likely due to larger sample sizes and longer follow-up. Our results are consistent with these Middle Eastern and Asian reports, suggesting potential geographic and ethnic variation in the CeD–IBD association. This geographic heterogeneity likely reflects differences in genetic architecture, particularly HLA-DQ2/DQ8 allele frequencies, environmental factors including dietary gluten exposure, and gut microbiome composition.[7] Interestingly, recent data suggest that IBD treatment with immunosuppressants and biologics may reduce the detection rate of CeD,[27] which could potentially contribute to lower prevalence estimates in treated populations.

In our cohort, demographic and clinical features did not differ significantly between CeD-positive and CeD-negative IBD patients. This contrasts with some Western data suggesting that CeD-IBD overlap is associated with extensive colitis, refractory disease, or primary sclerosing cholangitis.[8,21] The absence of such associations in our study may reflect true population-level differences or limitations imposed by the small number of CeD–IBD overlap cases (n = 11), which limited statistical power for detecting subgroup differences. Among UC patients with CeD, 55.6% had pancolitis compared with 30.6% in those without CeD. Although this difference did not reach statistical significance, likely due to the small number of CeD cases (n = 9), the observed trend may still be clinically relevant. It warrants evaluation in larger cohorts, as more extensive colonic involvement could theoretically be associated with heightened mucosal inflammation, potentially influencing either the true risk of CeD or the likelihood of false-positive serology.

Extraintestinal manifestations were similar between groups. However, autoimmune thyroid disease was observed in 27.3% of CeD–IBD patients versus 2.4% in IBD without CeD, consistent with the well-known association between CeD and autoimmune thyroiditis.[28] Although numbers were small, this signal supports selective screening in IBD patients with autoimmune comorbidities.

The Challenge of False-Positive Serology in IBD

A notable observation in our study was the high false-positive rate of anti-tTG antibodies (21.4%). Several mechanisms may explain this. Anti-tTG antibodies can be produced during intestinal apoptosis and inflammation; transglutaminase overexpression has been documented in CD, and mucosal immune activation may promote low-titer antibody formation independent of gluten exposure.[29] The Japanese experience is instructive: Watanabe et al. reported seropositivity rates of 19.4% in CD and 9.1% in UC, yet found no biopsy-proven CeD in 134 IBD patients.[24] In our study, although anti-tTG sensitivity was 100%, the positive predictive value (PPV) was only 78.6%, substantially lower than the 95–98% PPV observed in general populations. In practical terms, this means approximately 1 in 5 positive anti-tTG results in IBD patients represents a false positive, underscoring the critical importance of histological confirmation before diagnosing CeD to avoid misdiagnosis and unnecessary dietary restrictions.[13]

Given the modest prevalence of CeD (2.31%) and the high false-positive serology rate, routine universal screening of all IBD patients is unlikely to be cost-effective, especially in resource-constrained settings. A selective approach appears more appropriate—targeting patients with unexplained anemia, persistent malabsorption, refractory symptoms despite optimized IBD therapy, or associated autoimmune conditions such as thyroiditis or type 1 diabetes.

This study has several strengths. It is a prospective study with a relatively large, well-characterized IBD cohort. The study employed rigorous diagnostic criteria requiring both serological screening and mandatory histological confirmation in all seropositive patients, thus addressing the limitation of prior studies that relied solely on serology. Additionally, the use of a geographically and genetically comparable population reference enhances the validity of the SPR analysis. Limitations must be acknowledged. As a single-center tertiary-care cohort, referral bias toward complex or severe cases may limit generalizability, not reflecting community IBD epidemiology. The cross-sectional design limits our ability to determine temporal relationships between CeD and IBD. The number of biopsy-confirmed CeD cases was small, leading to wide confidence intervals and limited statistical power for subgroup analyses.

In conclusion, CeD prevalence in Indian IBD patients was numerically higher than the Northern Indian general population but did not reach statistical significance. Routine universal screening is not indicated, but selective testing in high-risk subgroups is advisable. Histological confirmation remains essential in all seropositive cases. Larger, multicenter studies with adequate power are needed to better delineate the CeD–IBD relationship in Indian populations and clarify underlying genetic and environmental contributors.

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