International Journal of Health Sciences and Research
[Indexed, Peer-Reviewed Journal]
Volume 16; Issue: 8; August 2026
Website: www.ijhsr.org
Original Research Article *ISSN: 2249-9571*

Prevalence of Celiac Disease in the Pediatric Population with Anemia: A Prospective Observational Study from North India

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DOI: https://doi.org/10.52403/ijhsr.20260801

Received: 05/04/2026; Revised: 25/06/2026; Accepted: 28/07/2026

ABSTRACT

Background & Objectives: Celiac disease is an immune-mediated enteropathy in which iron deficiency anemia may be the only manifestation, and the diagnosis is frequently delayed. This study was undertaken to determine the prevalence of celiac disease among children with anemia and to examine the relationship between hematological parameters and celiac disease.
Materials & Methods: In a hospital-based prospective observational study conducted over twenty-one months at a tertiary care teaching hospital in North India, forty children aged four to eighteen years with anemia, defined by World Health Organization age-specific hemoglobin criteria, were enrolled. All underwent clinical evaluation, complete blood count, iron studies, and serological screening for immunoglobulin A anti-tissue transglutaminase antibodies, followed by upper gastrointestinal endoscopy with duodenal biopsy graded by the modified Marsh–Oberhuber classification. Groups were compared using the Mann–Whitney U test and the Fisher's exact test.
Results: Celiac disease was confirmed in seven of forty children, a prevalence of 17.5% (95% confidence interval 7.3-32.8). Girls comprised 62.5% of participants. Serum iron and transferrin were significantly lower in affected children ($P = 0.018$ and $P = 0.041$, respectively). By the manufacturer's cut-off, all seven biopsy-confirmed cases were antibody negative, and the only positive test occurred in a child without celiac disease. No significant association was found with sex, anemia severity, or Helicobacter pylori status.
Conclusion: The prevalence of celiac disease among children with anemia greatly exceeded that expected in the general population. In this small, single-center cohort, serology was negative in every biopsy-confirmed case; within these limits, the findings suggest that a negative serological test alone may not be sufficient to exclude celiac disease in anemic children when clinical suspicion is high, and that duodenal biopsy retains an important diagnostic role.

Keywords: Anemia, Celiac disease, Children, Duodenal biopsy, Iron deficiency, Tissue transglutaminase antibody.

INTRODUCTION

Celiac disease (CD) is a chronic, immune-mediated systemic disorder triggered by dietary gluten in genetically susceptible individuals. Gluten exposure provokes an inflammatory response in the small-intestinal mucosa that produces villous atrophy, crypt hyperplasia, and intraepithelial lymphocytosis, impairing nutrient absorption and giving rise to a broad range of intestinal and extra-intestinal manifestations. [1,2]

Once regarded as a rare disorder dominated by chronic diarrhea, abdominal distension, and overt malabsorption, CD is now understood as a systemic condition in which atypical and extra-intestinal presentations predominate, particularly in older children and adolescents.[3-5] Among these, anemia is one of the most common and clinically significant presentations, and iron deficiency anemia (IDA) may be the sole manifestation even in the absence of gastrointestinal complaints.[6,7] Persistent or refractory anemia despite adequate iron supplementation should therefore prompt consideration of an underlying malabsorptive disorder such as CD.[8]

India continues to carry a heavy burden of childhood anemia, and nutritional iron deficiency is frequently presumed to be the sole cause, leading to empirical treatment without etiological evaluation.[9] As a result, conditions such as CD remain underdiagnosed in high-risk groups.[10-12] The pathogenesis of anemia in CD is multifactorial: damage to the proximal small intestine impairs iron absorption, while chronic inflammation alters iron handling and erythropoiesis; folate and vitamin B12 deficiencies may coexist in advanced disease.[7,13]

Population- and clinic-based data indicate that the prevalence of CD in children with unexplained anemia is considerably higher than in the general population, [14,15] and early institution of a gluten-free diet can restore mucosal integrity, correct hematological abnormalities, and prevent long-term complications. Despite growing awareness, data on the prevalence of CD among anemic children in North India remain limited. The present study was therefore undertaken to determine the prevalence of CD in children presenting with anemia and to correlate hematological parameters with serological and histopathological findings.

MATERIALS & METHODS

Study design and setting

This was a hospital-based prospective observational study conducted in the Department of Pediatrics, in collaboration with the Departments of Gastroenterology and Pathology, at a tertiary care teaching hospital in North India, over 21 months (June 2024-February 2026).

Participants

Children aged 4-18 years presenting to the pediatric outpatient department or admitted to the pediatric wards with anemia were assessed for eligibility; 40 children fulfilling the inclusion criteria were enrolled. Inclusion criteria were age 4-18 years, anemia by World Health Organization age-specific hemoglobin criteria, written informed parental or guardian consent, and assent from children aged ≥7 years where applicable. Children were excluded for previously diagnosed CD, known hemoglobinopathy, chronic systemic illness (renal, hepatic, or malignant), acute blood loss or transfusion within the preceding three months, or an existing gluten-free diet.

Clinical evaluation and investigations

A structured history (demographic details, gluten intake, gastrointestinal and extra-intestinal symptoms, growth parameters, and family history of CD or autoimmune disease) was recorded on a predesigned proforma, followed by complete physical examination. Investigations comprised hemoglobin, complete blood count, peripheral smear, red-cell indices (MCV, MCH, MCHC), and an iron profile (serum iron, ferritin, transferrin, and total iron-binding capacity).

Serological screening and histopathology

Serological screening for celiac disease used a commercial indirect enzyme-linked immunosorbent assay for IgA antibodies against tissue transglutaminase (Anti-Tissue Transglutaminase ELISA (IgA); EUROIMMUN Medizinische Labordiagnostika AG, Lübeck, Germany; Order No. EA 1910-9601 A). In this system, microplate wells pre-coated with human recombinant tissue transglutaminase captured serum IgA autoantibodies, which were detected with a peroxidase-conjugated anti-human IgA secondary antibody and a tetramethylbenzidine (TMB) chromogen; the enzymatic color reaction was terminated with a sulfuric acid stop solution. In brief, serum diluted in sample buffer was incubated in the antigen-coated wells; after washing to remove unbound immunoglobulin, the wells were incubated with the enzyme conjugate, washed again, developed with TMB substrate, and stopped, and the optical density was read spectrophotometrically at 450 nm against a reference wavelength of 620–650 nm. Each run included the manufacturer's three calibrators (200, 20 and 2 RU/ml) together with positive and negative controls, and results were accepted only when these quality-control criteria were satisfied. Antibody concentrations were expressed in relative units per milliliter (RU/ml); in accordance with the manufacturer, a value <20 RU/ml was regarded as negative and ≥20 RU/ml as positive. All assays were performed strictly according to the manufacturer's instructions, Total serum immunoglobulin A was not routinely measured, and immunoglobulin G-based testing was not systematically performed; interpretation of a negative immunoglobulin A anti-tissue transglutaminase result therefore did not account for possible selective immunoglobulin A deficiency.

All participants underwent upper gastrointestinal endoscopy with multiple duodenal biopsies from the second part of the duodenum; specimens were examined by an experienced pathologist and graded by the modified Marsh–Oberhuber classification (Table 2). Serological results were interpreted together with histopathology, and the final diagnosis of celiac disease rested on characteristic duodenal histopathology in the appropriate clinical and serological context, in keeping with ESPGHAN 2020 guidance [16]; histopathological findings were available for all 40 participants.

Statistical Analysis

No formal a priori sample-size calculation was performed; a convenience sample of 40 eligible children presenting during the study period was enrolled and analyzed. Standardized study proformas and uniform laboratory and histopathological protocols were used to reduce measurement bias, and there were no missing data for the variables reported. Data were entered in Microsoft Excel and analyzed with IBM SPSS Statistics (version 21.0; IBM Corp., Armonk, NY, USA). Categorical variables are summarized as frequencies and percentages, and continuous variables as mean ± standard deviation (SD) with median and interquartile range (IQR). Normality was assessed with the Kolmogorov–Smirnov test; between-group comparisons used the Mann–Whitney U test, and categorical associations the Fisher's exact test. Because several continuous variables were skewed and non-parametric tests were used, group comparisons are interpreted primarily on the basis of the median and interquartile range, with the mean ± standard deviation reported alongside for completeness (Table 3). The prevalence of celiac disease is reported with an exact (Clopper–Pearson) 95% confidence interval (CI). A two-sided $P<0.05$ was considered statistically significant.

Ethical considerations

The study was approved by the University Ethics Committee (Medical), Swami Vivekanand Subharti University, Meerut (Approval Ref. No. SMC/UECM/2024/1386; Committee Registration No. EC/NEW/INST/2023/UP/0358). Written informed consent was obtained from parents

or guardians, and data confidentiality was maintained throughout.

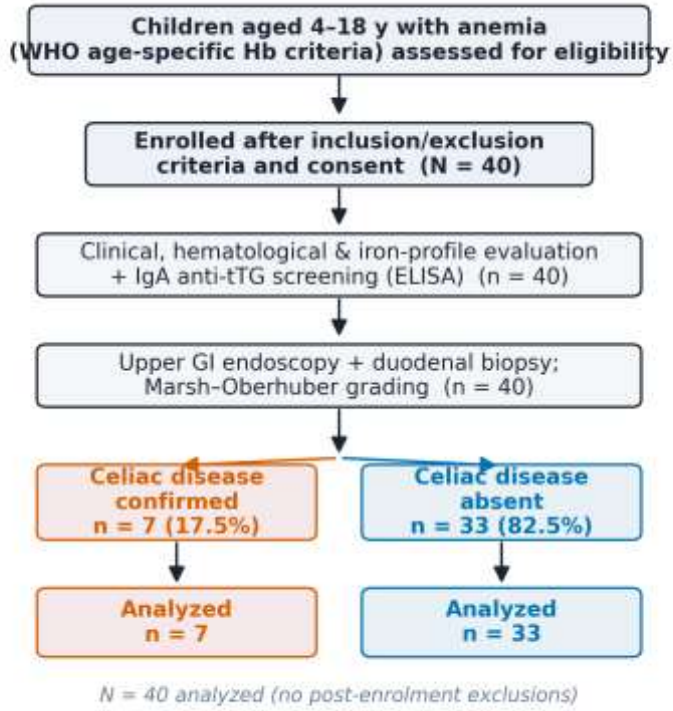

Figure 1: Flow chart of study participants. Of 40 anemic children enrolled and analyzed, 7 (17.5%) had biopsy-confirmed celiac disease and 33 (82.5%) did not. CD, celiac disease; GI, gastrointestinal; anti-tTG, IgA anti-tissue transglutaminase.

RESULT

Baseline characteristics

Forty anemic children were enrolled (Table 1). Twenty-five (62.5%) were female and 15 (37.5%) male (female-to-male ratio ≈ 1.7:1). The mean age was 12.1 ± 3.7 years and mean hemoglobin 10.2 ± 1.7 g/dL. Anemia was mild in 25 children (62.5%), moderate in 10 (25.0%), and severe in 5 (12.5%).

Table 1: Baseline demographics and anemia severity (N = 40).

Characteristic	Category	Frequency (n)	Percent (%)
Sex	Male	15	37.5
	Female	25	62.5
Age (years)	Mean ± SD	12.1 ± 3.7	—
	Median (IQR)	13.0 (10.0–15.0)	—
Severity of anemia	Mild	25	62.5
	Moderate	10	25.0
	Severe	5	12.5
Total		**40**	**100.0**

SD, standard deviation; IQR, interquartile range.

Prevalence of celiac disease and histopathology

CD was confirmed in 7 of the 40 children, a prevalence of 17.5% (95% CI 7.3–32.8%; exact binomial); the remaining 33 (82.5%) showed other duodenal pathologies. On histopathology, chronic active duodenitis was the most frequent finding (23; 57.5%), followed by biopsy-confirmed CD (7; 17.5%) and mild chronic duodenitis (3; 7.5%); active duodenitis and chronic active gastritis occurred in 2 each (5.0%), and non-

tropical sprue, severe active duodenitis, and severe duodenitis in 1 each (2.5%) (Figure 2). A modified Marsh–Oberhuber grade was documented for 8 children: grade 1 in 1 child (a non-celiac child whose biopsy showed non-tropical sprue), grade 3A in 4 children, and grade 3B in 3 children. All 7 biopsy-confirmed celiac cases carried a destructive lesion (grade 3A in 4 and grade 3B in 3). The remaining 32 biopsies were reported under descriptive histopathological categories without an assigned numerical Marsh grade (Table 2).

By the manufacturer's cut-off (≥20 RU/ml), only 1 of 40 children (2.5%) was IgA anti-tTG positive (22.9 RU/ml), and this child had mild chronic duodenitis rather than celiac disease on biopsy. All 7 histopathologically diagnosed celiac cases were anti-tTG negative, with titers ranging from 0.2 to 18.7 RU/ml. Serology therefore did not identify any of the biopsy-confirmed cases.

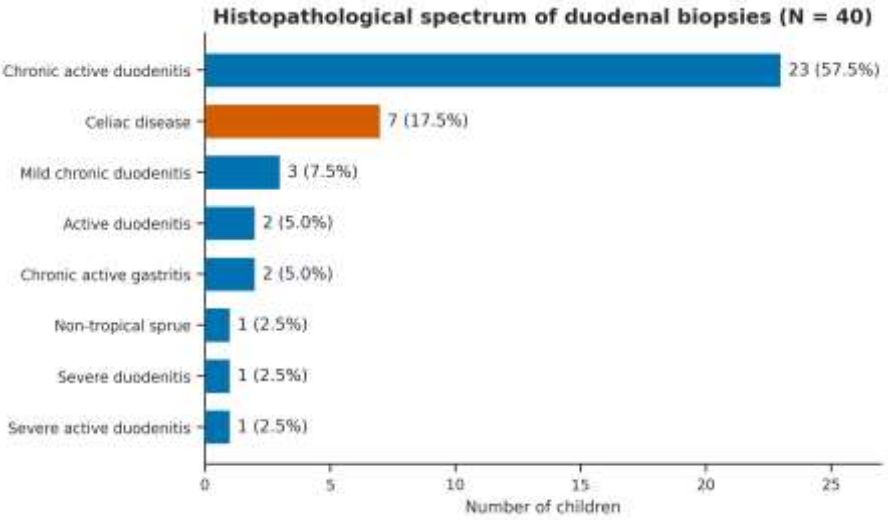

Figure 2: Distribution of duodenal histopathological findings (N = 40). Celiac disease (highlighted) accounted for 7 (17.5%) of biopsies; chronic active duodenitis was the commonest finding (23; 57.5%).

Table 2: The modified Marsh–Oberhuber classification of duodenal mucosal injury.

Type	Lesion	Villous architecture	Crypts	IEL (per 100 enterocytes)
0	Pre-infiltrative (normal)	Normal (villous/crypt ratio 3:1)	Normal	≤25 (normal)
1	Infiltrative	Normal	Normal	>25 (increased)
2	Hyperplastic	Normal	Hyperplastic (reduced mucinous activity, increased mitoses)	>25 (increased)
3A	Destructive	Mild (partial) villous atrophy	Hyperplastic	>25 (increased)
3B	Destructive	Moderate (subtotal) villous atrophy	Hyperplastic	>25 (increased)
3C	Destructive	Total villous atrophy (villi absent)	Hyperplastic	>25 (increased)

Criteria as applied in the study methodology. IEL, intraepithelial lymphocytes. Type 3 lesions additionally show surface enterocytes of reduced height with irregular brush border and cytoplasmic vacuolation. Diagnosis of celiac disease requires these histological features together with compatible clinical and serological data.

Hematological and biochemical comparison by celiac status

Children with CD had lower mean hemoglobin, MCV, and MCH than non-CD children, but these differences were not statistically significant (Table 3). Median serum iron was significantly lower in the CD group (22.0 [IQR 14.0–52.0] vs 74.0 [IQR 38.0–98.0] µg/dL; P = 0.018), as was median transferrin (7.3 [IQR 4.0–17.2] vs 24.0 [IQR 11.9–34.9] mg/dL; P = 0.041) (Figure 3). Ferritin, TIBC, and anti-tTG did not differ

significantly; the higher mean ferritin among non-CD children reflected marked right skew from a few elevated values.

Table 3: Comparison of hematological and biochemical parameters by celiac disease status.

Variable	Celiac present (n = 7) Mean ± SD; Median (IQR)	Celiac absent (n = 33) Mean ± SD; Median (IQR)	P value
Age (years)	9.4 ± 4.2 10.0 (5.0–14.0)	12.6 ± 3.4 13.0 (10.0–15.0)	0.081
Hemoglobin (g/dL)	9.5 ± 2.1 10.4 (7.8–11.3)	10.4 ± 1.6 11.1 (9.6–11.3)	0.246
MCV (fL)	77.1 ± 11.4 83.1 (68.0–85.0)	82.1 ± 13.2 83.9 (74.6–88.8)	0.309
MCH (pg)	25.0 ± 4.5 27.4 (21.2–27.7)	27.1 ± 4.8 28.1 (24.1–29.8)	0.261
MCHC (g/dL)	32.3 ± 1.5 32.4 (31.1–33.7)	32.8 ± 1.3 33.0 (32.6–33.6)	0.442
Ferritin (ng/mL)	59.5 ± 89.1 25.7 (6.9–75.5)	167.1 ± 380.6 34.7 (24.1–93.2)	0.261
TIBC (µg/dL)	350.4 ± 81.7 322.0 (300–422)	305.5 ± 81.7 305.0 (267–369)	0.218
Anti-tTG (U/mL)	4.2 ± 6.7 0.6 (0.2–5.8)	2.4 ± 4.9 0.3 (0.2–0.9)	0.205
Serum iron (µg/dL)	**34.7 ± 27.0** **22.0 (14.0–52.0)**	**74.8 ± 47.7** **74.0 (38.0–98.0)**	**0.018**
Transferrin (mg/dL)	**12.8 ± 12.2** **7.3 (4.0–17.2)**	**26.8 ± 17.3** **24.0 (11.9–34.9)**	**0.041**

Comparisons by the Mann–Whitney U test; statistically significant results (P<0.05) are shown in bold. SD, standard deviation; IQR, interquartile range; MCV, mean corpuscular volume; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; TIBC, total iron-binding capacity; anti-tTG, IgA anti-tissue transglutaminase antibody.

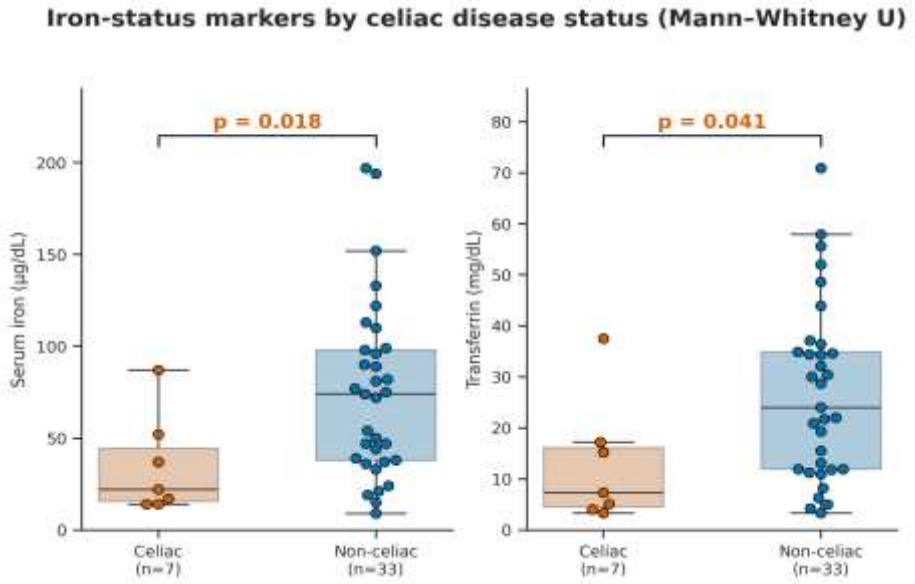

Figure 3: Serum iron and transferrin by celiac disease status (n = 7 celiac, n = 33 non-celiac). Box-and-whisker plots show median and interquartile range with overlaid individual values; serum iron P = 0.018 and transferrin P = 0.041 (Mann–Whitney U test). CD, celiac disease.

Associations with celiac disease

Celiac disease showed no significant association with sex (P = 1.000) or with anemia severity (P = 0.258); moderate-to-severe anemia was present in 57.1% (4 of 7) of children with celiac disease and 33.3% (11

of 33) of those without. Helicobacter pylori–associated pathology was identified in 13 of 40 children (32.5%) overall. None of the 7 children with celiac disease had H. pylori–associated pathology, compared with 13 of 33 (39.4%) of those without celiac disease; this inverse trend did not reach statistical significance (P = 0.074) (Table 4).

Table 4: Association of celiac disease with sex, anemia severity, and Helicobacter pylori status.

Variable	Category	Celiac present (n = 7)	Celiac absent (n = 33)	P value
Sex	Male	3 (42.9%)	12 (36.4%)	1.000
	Female	4 (57.1%)	21 (63.6%)	
Severity of anemia	Mild	3 (42.9%)	22 (66.7%)	0.258
	Moderate	2 (28.6%)	8 (24.2%)	
	Severe	2 (28.6%)	3 (9.1%)	
H. pylori status	Associated	0 (0.0%)	13 (39.4%)	0.074
	Not associated	7 (100.0%)	20 (60.6%)	

Associations tested by the Fisher's exact test; percentages are column proportions within each group. H. pylori, Helicobacter pylori.

Exploratory correlations

In post-hoc analyses across all 40 participants, serum iron correlated positively with transferrin (Figure 4; Pearson r = 0.88, P<0.001) and anti-tTG titer correlated inversely with hemoglobin (Figure 5; Pearson r = −0.42, P = 0.006). These bivariate correlations were exploratory and were not part of the pre-specified analysis.

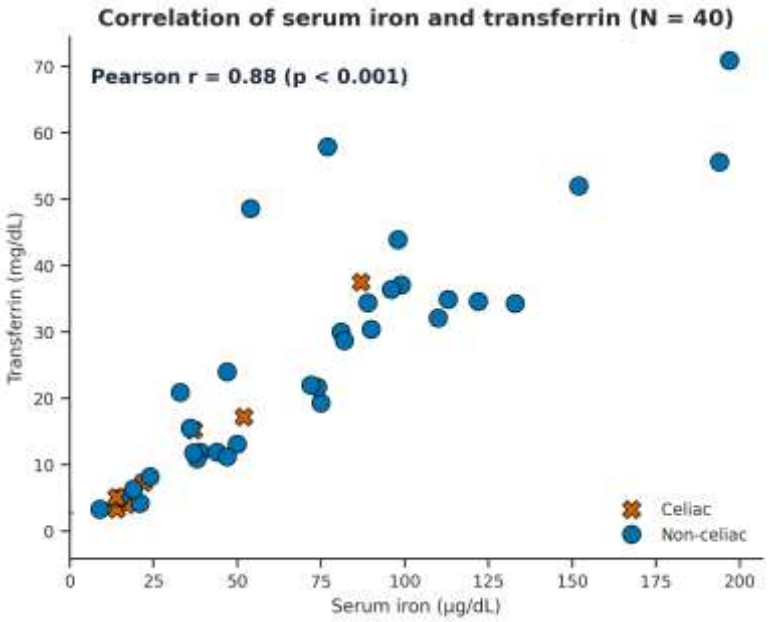

Figure 4: Correlation between serum iron and transferrin (N = 40), color-coded by celiac disease status. Dashed line, least-squares fit with 95% confidence interval; Pearson r = 0.88, P<0.001. CD, celiac disease.

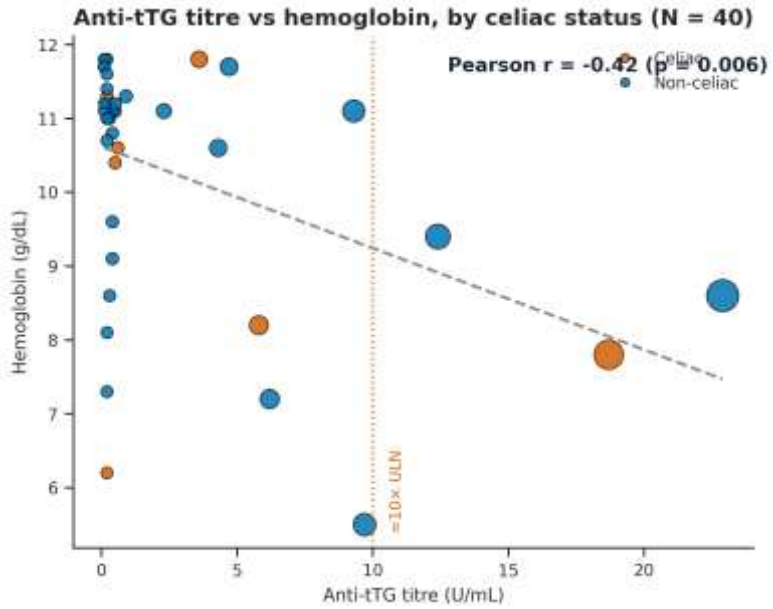

Figure 5: Anti-tissue transglutaminase (anti-tTG) titer versus hemoglobin (N = 40), color-coded by celiac disease status (log-scaled x-axis; Pearson r = −0.42, P = 0.006). Dotted line, manufacturer cut-off (20 RU/ml). CD, celiac disease; RU, relative units.

DISCUSSION

In this prospective study of 40 anemic children at a tertiary care center in North India, CD was identified in 17.5%, far exceeding the ~1% expected in the general population[10,17] and the pooled 3–5.5% reported for biopsy-confirmed CD among children with IDA.[14,15] The higher yield is consistent with a selected, symptomatic tertiary care population in a wheat-consuming region, and with evidence that screening anemic children detects CD at substantially higher rates than population-based screening.[14]

The most robust biochemical finding was significantly lower serum iron and transferrin in children with celiac disease, in the setting of comparable hemoglobin and red-cell indices. This pattern is consistent with impaired iron absorption from villous atrophy of the proximal small intestine, where dietary iron is chiefly absorbed. [7,12,18] That hemoglobin and red-cell indices did not differ significantly probably reflects the small sample and the fact that all participants were anemic by design, which compressed the between-group contrast in hemoglobin. In exploratory analyses, children with celiac disease clustered in the low-iron, low-transferrin region (Figure 4), and those with the highest anti-tTG titers tended to have lower hemoglobin (Figure 5); although hypothesis-generating, these patterns are consistent with a malabsorptive contribution to their anemia.

Females predominated in the cohort; although no significant sex–CD association was observed, a modest female preponderance accords with prior descriptions of pediatric CD. Anemia severity did not differ significantly by CD status, but moderate-to-severe anemia was proportionally more frequent among children with CD, echoing reports that villous-atrophy severity tracks with the degree of hematological derangement. [19,20]

All seven biopsy-confirmed celiac cases showed destructive Marsh grade 3A or 3B lesions, underscoring the value of duodenal biopsy in anemic children, in whom antibody titers are often lower and serology alone less reliable.[21] The most striking observation in this cohort was that IgA anti-tTG was negative in all seven biopsy-diagnosed cases, while the single seropositive child did not have celiac disease. Universal seronegativity across all seven biopsy-confirmed cases is an unusual finding that warrants careful interpretation. Several factors are relevant. First, the assay was performed strictly per the

manufacturer's protocol, with each run validated against three calibrators (200, 20 and 2 RU/ml) and positive and negative controls, and the single seropositive result (22.9 RU/ml) in a non-celiac child indicates that the assay was capable of generating positive values; assay failure is therefore an unlikely sole explanation. Second, and most importantly, the diagnosis in this study did not rest on serology but on characteristic destructive duodenal histopathology - every case showed Marsh grade 3A or 3B villous atrophy - so the celiac diagnoses are histologically secure irrespective of antibody status. Third, seronegative celiac disease is well recognized and may arise from selective immunoglobulin A deficiency - the commonest cause of false-negative immunoglobulin A-based serology - or from the effect of chronic illness and malnutrition on antibody production. A key limitation is that total serum immunoglobulin A was not measured in this cohort, so selective immunoglobulin A deficiency could neither be confirmed nor excluded as the explanation; this is acknowledged as a limitation, and measurement of total immunoglobulin A with immunoglobulin G-based serology is recommended in future work. The coexistence of unequivocal Marsh 3A/3B villous atrophy with negative serology in every case makes this cohort a clear illustration of the phenomenon. Within this small, single-center cohort, reliance on IgA anti-tTG alone would have missed every case identified here. While these numbers are too limited to support broad recommendations, they suggest that, in this setting, a negative serological result should be interpreted with caution and should not by itself exclude celiac disease when clinical suspicion is high.

Helicobacter pylori–associated pathology was present in about one-third of the cohort but in none of the children with celiac disease, an inverse trend that did not reach statistical significance (P = 0.074). H. pylori is itself a recognized contributor to iron deficiency anemia in children,[22] and relationships between H. pylori infection and anemia have been described in pediatric populations.[23] With only seven celiac cases, these numbers are too small to support inference, and the observation should be regarded as hypothesis-generating rather than evidence of a protective effect.

The clinical implication is direct. Where childhood anemia is highly prevalent and commonly treated empirically, a malabsorptive cause such as celiac disease is easily missed. [9,24] Failure to identify it prolongs anemia and risks growth impairment, delayed puberty, reduced bone mineral density, and impaired quality of life.[25] In this cohort a negative IgA anti-tTG result did not exclude celiac disease; where clinical suspicion persists in an anemic child, duodenal biopsy remains the definitive diagnostic step. A suggested pathway that positions endoscopy and biopsy accordingly is outlined in Figure 6. [16,26]

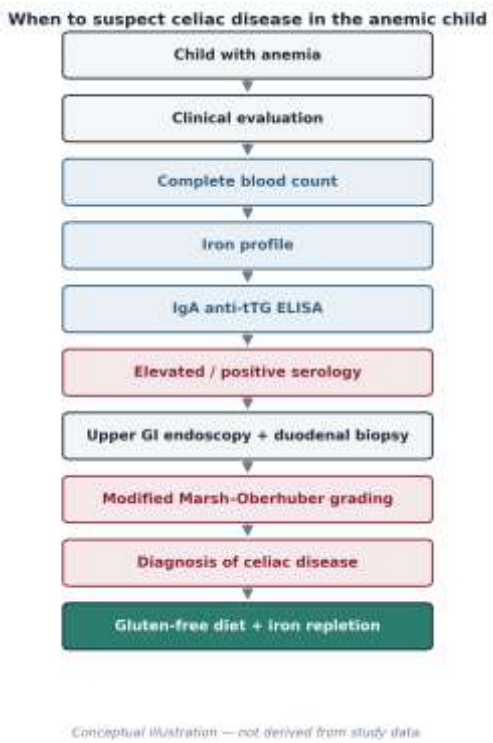

Figure 6: Suggested diagnostic algorithm for celiac disease in the child with anemia (conceptual illustration; not derived from study data). anti-tTG, IgA anti-tissue transglutaminase; GI, gastrointestinal.

Limitations

This study has several limitations. The sample was small, limiting power to detect

associations of modest magnitude, and the single-center, tertiary care design restricts generalizability and may inflate the observed prevalence through referral selection. Total serum IgA was not available, so selective IgA deficiency—a recognized cause of false-negative IgA anti-tTG—could not be confirmed or excluded as the reason for the uniform seronegativity of the biopsy-proven cases; IgG-based serology was likewise not systematically performed. A numerical modified Marsh–Oberhuber grade was assigned in 8 of the 40 biopsies, the remainder being reported descriptively, which limits comparison of mucosal injury across the whole cohort. Finally, there was no long-term follow-up to assess hematological recovery after a gluten-free diet, and micronutrient assessment beyond the iron profile (folate, vitamin B12) was limited.

CONCLUSION

Celiac disease was present in 17.5% of anemic children, well above the general-population estimate. The significantly lower serum iron and transferrin among affected children support intestinal malabsorption as a central mechanism of anemia in pediatric celiac disease. In this small, single-center cohort, IgA anti-tTG was negative in every biopsy-proven case; although the sample is too small for firm recommendations, this observation suggests that a negative serological test alone may be insufficient to exclude celiac disease and supports maintaining a low threshold for duodenal biopsy when clinical suspicion persists. These findings require confirmation in larger, multicenter studies before they can be generalized. Larger multicenter studies with follow-up, incorporating total IgA and IgG-based serology, are needed to define the true burden, clarify the high rate of seronegativity, and chart the trajectory of hematological recovery.

Declaration by Authors

Ethical Approval: Approved by the University Ethics Committee (Medical), Swami Vivekanand Subharti University, Meerut (Approval Ref. No. SMC/UECM/2024/1386)

Acknowledgement: The authors thank the Departments of Pediatrics, Gastroenterology, and Pathology and all participating children and their caregivers.

Source of Funding: None

Conflict of Interest: The authors declare no conflict of interest.

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How to cite this article: Sindhuri TMJ, Dadoria R, Yadav A, Punj A, Gupta A. Prevalence of celiac disease in the pediatric population with anemia: a prospective observational study from North India. *Int J Health Sci Res.* 2026; 16(8):1-11. doi: *10.52403/ijhsr.20260801*

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