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Original Article

Diagnosis and Follow-up of Children with Potential Celiac Disease on a Gluten-Containing Diet: A Retrospective Cohort Study

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Abstract

Background and objectives: Potential celiac disease (PCD) is defined as elevated celiac serology with a preserved small intestinal mucosa. This study aimed to identify baseline characteristics and the outcomes of children with PCD consuming a gluten-containing diet.

Methods: This was a retrospective cohort study of pediatric PCD patients diagnosed between 12/2018 and 10/2024. Baseline data included demographics, anthropometrics, clinical symptoms and signs, celiac serology, and biopsy results. Follow-up data included repeat serology and biopsy results when performed.

Results: PCD was diagnosed in 75/517 (14.5%) children undergoing upper endoscopy for suspected celiac disease (CeD). Baseline anti-transglutaminase IgA (TTG) was above 10× the upper limit of normal (ULN) in 18 (24%), between 3–10× ULN in 52 (69.3%), and <3× ULN in five (6.6%). Anti-endomysial antibody was positive in 57 (76%). Among 48 children (64%) with at least one year of follow-up, TTG normalized in 26 (54.1%), decreased to <3× ULN in 13 (27.1%), was between 3–10× ULN in six (12.5%), and was above 10× ULN in three (6.3%). Nine children had a repeat endoscopy, and six (66.7%) were diagnosed with CeD, while three remained PCD. Among the 11 children with TTG >10× ULN and at least one year of follow-up, TTG normalized in three, declined in five, and increased or remained above 10× ULN in three.

Conclusions: PCD is common and may be found in children with TTG above 10× ULN; approximately half will normalize TTG. The omission of biopsies may result in an erroneous diagnosis of CeD.

Introduction

Celiac disease (CeD) is a systemic immune-mediated disorder caused by the ingestion of gluten-containing grains in genetically susceptible individuals. The diagnosis of CeD can be made by positive celiac serology [anti-transglutaminase IgA (TTG) antibodies] and endoscopy showing an increase in intra-epithelial lymphocytes, crypt hyperplasia, and villous atrophy in duodenal biopsies (Marsh 2–3), or by a "no-biopsy" approach dependent on a high level of TTG antibodies (above 10 times the upper limit of normal (ULN)) and anti-endomysial antibody (EMA) positivity in a second serum sample.[1] Patients with positive celiac serology who undergo an upper gastrointestinal endoscopy due to suspected celiac, and whose duodenal biopsies demonstrate preserved small intestinal mucosa in the absence of villous atrophy (Marsh 0–1), are diagnosed with potential celiac disease (PCD).[1] Previous studies have shown that PCD is found in approximately 10–20% of patients undergoing endoscopic investigation for CeD, and that it is more common in younger children.[2–5]

Several studies have examined the rate at which PCD progresses to CeD over various time periods. Lionetti *et al*.[6] followed 23 pediatric patients with PCD who continued consuming a gluten-containing diet (GCD) for more than 10 years and found that only three (13%) developed CeD. In a recently published meta-anal-

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ysis,[7] which included 17 studies comprising 1,010 patients with PCD, the prevalence of PCD was 16% (95% confidence interval (CI) 10% to 22%). Villous atrophy, confirming CeD, developed over time in roughly a third of the patients with PCD, while a similar proportion of patients showed normalization of serology despite remaining on a GCD.

Predictive factors for the development of villous atrophy have been previously reported; however, they are still not well defined. Auricchio *et al*.[8,9] found that the amount of γδ intraepithelial lymphocytes, age (older than three years), and HLA DQB1*02 homozygosity are the strongest predictors for villous atrophy development. However, γδ staining and HLA testing are not available for most patients. The most recent study by Auricchio *et al*.[10] showed that asymptomatic children with PCD who are likely to progress to mucosal damage and develop CeD present markers of immune response activation at diagnosis: increased serum expression of inflammatory proteins involved in lymphocyte activation, JAK-STAT signaling, and intestinal stem cell proliferation.

There is still controversy among physicians about the best way to follow patients with PCD. When the decision to continue a GCD is made, it is still unknown how often serology should be tested and when a repeat intestinal biopsy should be obtained. These questions remain unanswered.

In our previous study on pediatric patients with PCD,[11] we showed that after a mean follow-up time of 18 months, CeD was diagnosed in 12/90 (13.3%) pediatric patients, based on repeated serology tests and repeated biopsies obtained in 20/90 patients. Although older age and elevated TTG titers were found to be predictors of CeD development, 38% of patients normalized their TTG levels, including 29% of those with initial TTG levels over 10× ULN.

In this study, we continued the follow-up of patients previously diagnosed with PCD from our medical center and included newly diagnosed patients up to 10/2024. Our primary aim was to characterize the outcomes of PCD in patients consuming a GCD with a longer follow-up time. Our secondary aim was to study the prevalence of PCD and the characteristics of patients in whom TTG normalized over time, versus patients whose TTG levels remained high or were later diagnosed with CeD.

Materials and methods

Study design

A retrospective cohort study reviewing the medical records of pediatric patients diagnosed with PCD at Kaplan Medical Center in Israel, ages one to eighteen years old, consuming a GCD between December 2018 and October 2024.

Inclusion and exclusion criteria

Inclusion criteria

(1) Children who underwent endoscopy while on a GCD. The pediatric gastroenterologists confirmed with the child and family before the endoscopy and at each follow-up that they were consuming a GCD. (2) In families with other members diagnosed with CeD, the child was eating at least one portion of gluten daily (above 15 g per day). (3) The availability of six duodenal biopsies taken at the time of endoscopy, at least one of which was obtained from the duodenal bulb. Pathology findings compatible with Marsh score 0 or 1. (4) Confirmation of the diagnosis of PCD was done by an unblinded review of all biopsies by a pediatric gastroenterologist and by an experienced pathologist.

Exclusion criteria

(1) Missing follow-up testing. (2) IgA deficiency. (3) Patients who initiated a gluten-free diet (GFD). (4) Children diagnosed with CeD with a no-biopsy diagnosis.

Data collection and study variables

Data at baseline

Presenting signs and symptoms, CeD family history, gender, and age. Serology levels (TTG IgA antibody) were tested using the Multiplex TTG (Bioplex 2200) assay.[12] Maximal levels of TTG were defined according to the manufacturer's guidelines as > 250 μ/mL, while normal levels were defined as 0–15 U/mL. TTG levels were collected as absolute values but presented as relative multiples to the ULN (1–3× ULN, 3–10× ULN, or above 10× ULN).[13] Deamidated gliadin peptide (DGP) antibody testing was not available and was not performed. EMA assay results are presented as positive or negative, omitting titer or extent of elevation. This is due to the fact that not all tests underwent the same EMA assay in a single laboratory. The initial serological assessments were conducted less than three months prior to the endoscopy.

Follow-up data

Collected at six- and twelve-month intervals while patients remained on a GCD. Data collected included repeat serology and information on the type of diet consumed, confirming that the child was consuming gluten. In patients who had a second endoscopy, data were collected on the indication for endoscopy, TTG levels at the time of endoscopy, and the pathology results.

Outcomes

The primary outcome was the change in TTG levels over time in patients who continued a GCD, while the secondary outcome was the proportion of patients who were diagnosed with CeD during follow-up. To measure these outcomes, we collected follow-up data at six- and twelve-month intervals, including repeat TTG serology tests and confirmation of gluten consumption. For patients who underwent a second endoscopy, data were also collected on the indication for the procedure, TTG levels at the time of endoscopy, and the results of the pathology report.

Statistical analysis

Since no comparison between groups was done, only descriptive statistics were utilized in the study. Continuous variables were summarized using means and ranges, while categorical variables were summarized as counts and percentages. An exploratory stratified analysis of baseline EMA positivity across TTG categories and by sex and family history was conducted. Cross-tabulations were summarized with counts and row percentages. We applied chi-square tests (or Fisher's exact when expected counts < 5) and, for ordered TTG categories, a linear-by-linear trend test. Analyses were descriptive.

Ethical approval

The research was conducted in accordance with the Declaration of Helsinki and received approval from the Regional Ethics Committee at Kaplan Medical Center (Protocol Number: KMC-0041-21). Due to the retrospective nature of the study and the use of anonymized patient data, the ethics committee waived the need for informed consent.

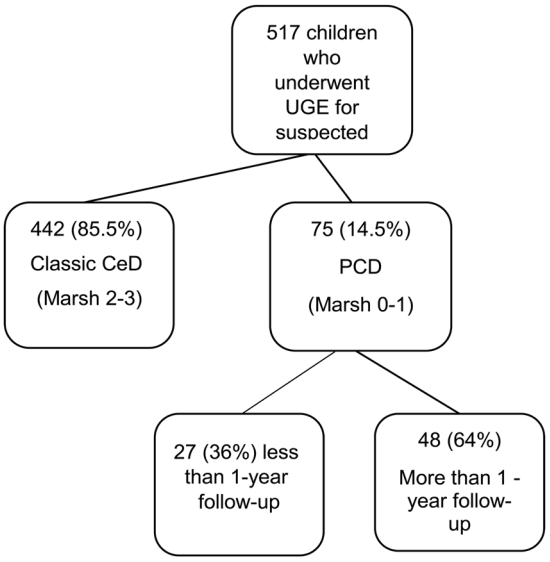

Fig. 1. Flow chart of 517 children undergoing UGE for suspected CeD. CeD, celiac disease; PCD, potential celiac disease; UGE, upper gastrointestinal endoscopy.

Results

Baseline characteristics and clinical presentation

During the study period, 517 children had an upper gastrointestinal endoscopy for suspected CeD, of whom 75 (14.5%) were diagnosed with PCD (Fig. 1). The mean age at diagnosis was 7.0 years (range 1.3–16.4 years), with 53 (70.6%) females. Clinical symptoms at the time of PCD diagnosis included gastrointestinal symptoms in 26 (34.6%), growth delay (postponed onset of puberty, short stature, inadequate weight and height gain) in 20 (26.6%), and iron-deficiency anemia in five (6.6%). Eighteen patients (24%) were asymptomatic. Among the asymptomatic patients, 10/18 (55.6%) were from high-risk groups: nine with first-degree relatives with CeD and one with insulin-dependent diabetes mellitus. Baseline clinical and laboratory data are presented in Table 1. EMA was available in 68/75 children (57/68, 83.8% positive; 11/68 negative; seven not tested). EMA positivity increased across TTG categories: 50% for <3× ULN (2/4), 80% for 3–10× ULN (36/45), and 100% for ≥10× ULN (19/19) ($\chi^2(2) = 7.53$, $p = 0.023$; linear by linear trend $p = 0.007$). No significant differences were observed by sex (EMA: Fisher $p = 0.486$; TTG: χ^2 $p = 0.262$) or by family history (EMA: Fisher $p = 0.137$; TTG after combining first/second degree: χ^2 $p = 0.894$).

Histological findings were consistent with Marsh 0 in 39 (52%) and Marsh 1 in 36 (48%) cases.

Follow-up and outcome

During the follow-up period, all children included in the study remained on a GCD and had at least one TTG test performed. The mean follow-up time was 29.1 months (range 5–72). Forty-eight children (64%) had at least one repeat TTG level at the 12-month follow-up period (Fig. 1). At this time, TTG normalized in 26 (54.1%), declined to <3× ULN in 13 (27.1%), was between 3–10× ULN in six (12.5%), and was above >10× ULN in three (6.3%) (Fig. 2). At 12 months, among 37 children with data on TTG levels and EMA positivity, TTG was <3× ULN in 59.5%, 3–10× in 29.7%, and ≥10× in 10.8%.

Table 1. Baseline characteristics of 75 patients with potential celiac disease

Baseline clinical and laboratory data - 75 patients	
Age (years) (mean, range)	7.0 (1.3–16.4)
Sex (female)	53 (70.6%)
Symptoms leading to diagnosis	
Gastrointestinal	26 (34.6%)
Growth delay	20 (26.6%)
Iron-deficiency anemia	5 (6.6%)
No symptoms	18 (24%)
Family history of CeD	
1st degree	14 (18.6%)
2nd degree	9 (12%)
Baseline laboratory values	
Hemoglobin level (g/dL (mean, range)	12.32 (6.8–15.8)
Ferritin (ng/mL) (mean, range)	27.3 (2.8–176)
Positive anti-endomysial antibody	57 (76%)
Anti-TTG titer (before endoscopy)	
>10 ULN	18 (24%)
3–10 ULN	52 (69.3%)
<3 ULN	5 (6.66%)

CeD, celiac disease; TTG, transglutaminase; ULN, upper limit of normal.

Among the whole cohort, nine children underwent a repeat endoscopy: five due to TTG elevation and the remaining four because of clinical symptoms requiring a repeat endoscopic evaluation. In 6/9 (66.7%) children, the repeat biopsies exhibited a Marsh 2–3 lesion, and therefore CeD diagnosis was established. In four of these patients, EMA was positive. In three patients, the repeat biopsy demonstrated Marsh 0–1; two continued a GCD, and one started a GFD due to clinical symptoms. Thus, at the end of follow-up, 7/75 patients (9.3%) converted to a GFD, with CeD confirmed by repeat endoscopy and biopsy in six patients. Among these six children, baseline TTG was greater than 10× ULN in two (33.3%) and 3–10× ULN in four (66.6%).

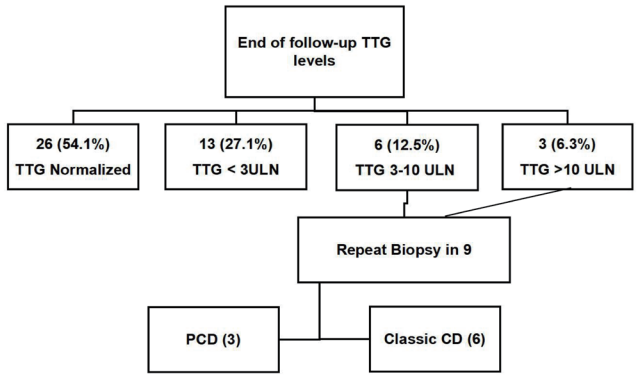

Fig. 2. TTG levels of 48 patients with PCD and at least 12 months' follow-up. CD, celiac disease; PCD, potential celiac disease; TTG, tissue transglutaminase; ULN, upper limit of normal.

There were 18 children with an initial TTG > 10× ULN and EMA positivity. These patients may have been diagnosed with CeD according to the no-biopsy approach as stated by the latest ESPGHAN guidelines.[7] However, following parental preference (after an explanation of the no-biopsy versus biopsy approach by the case-managing gastroenterologist), endoscopy was performed. Eleven of the 18 children had at least a one-year follow-up. Among them, TTG normalized in three (27.2%), declined in five (45.4%), and increased or remained >10× ULN in three (27.2%). Two of these children underwent repeat endoscopies after 33 and 41 months; however, only one was diagnosed with CeD on repeat biopsy.

Discussion

The risk and predictors of PCD progressing to CeD, and the optimal way to follow patients with PCD, are still areas of debate.[6–9,14,15] In our cohort, which analyzed the baseline characteristics of 75 children with PCD, of whom two-thirds had at least 12 months of follow-up, CeD was diagnosed in less than 10%. The diagnosis of CeD was made based on a significant rise in TTG levels during follow-up and repeat biopsies done in nine children. The biopsies were consistent with CeD in only six. During follow-up, TTG normalized in more than half of the patients, and in almost 30%, it decreased to below 3× ULN. In these two subgroups of patients, a repeat biopsy was not performed, as there was no indication, due to the very low risk of villous atrophy with normal or low levels of TTG. However, we recommended that all patients with PCD continue clinical and serological follow-up.

Based on our current and previous studies, and other studies on the natural history of PCD patients, we believe that an anticipatory approach is justified, particularly in asymptomatic patients.[3,7,16–19] In the most recent and largest systematic review and meta-analysis on PCD,[7] which included 17 studies and 1,010 patients, the authors showed that 16% of patients with positive celiac serology were diagnosed with PCD. About one-third of patients with PCD who consume a GCD were subsequently diagnosed with CeD during follow-up. However, a similar proportion of patients normalized their TTG levels. Many of the studies included in the meta-analysis had a relatively small sample size. Eight out of 17 studies were from Italy, and ten were pediatric studies. The rate of progression to villous atrophy was lower in children (28%; 95% CI 10% to 45%) than in adult patients (39%; 95% CI 13% to 65%). Nevertheless, the variation was not statistically significant ($p = 0.50$).

The ability to differentiate and predict at diagnosis which patients are likely to develop villous atrophy, as opposed to those who are unlikely to progress to CeD, would be extremely helpful in making decisions concerning the initiation of the GFD. Previously described predictors included the amount of γδ intraepithelial lymphocytes, older age, and HLA DQB1*02 homozygosity.[8,9] Recently, proteomic biomarkers in the serum of children with PCD have been helpful in predicting the development of villous atrophy at diagnosis.[10] Our study did not aim to identify predictors of progression to CeD, but only to describe the natural history in children remaining on a GCD. However, using a descriptive analysis, we confirmed the expected concordance between higher TTG levels and EMA positivity, while showing no detectable differences by sex or family history. Interpretation is limited due to small cell counts and missing EMA in seven children, so these findings should be viewed as exploratory.

In recent years, the diagnosis of CeD based on elevated celiac serology (the no-biopsy approach) has become more prevalent. The current ESPGHAN guidelines[1] enable the diagnosis of CeD in both asymptomatic and symptomatic children with TTG levels equal to or above 10 times the ULN, performed twice, with EMA positivity confirmed in a second test. Due to this approach, PCD may be diagnosed as CeD, wrongly prescribing patients to a GFD for life. Though rare, false-positive celiac serology may be caused by *Giardia lamblia*, which may be an incidental finding in children investigated for CeD or may be the cause of elevated TTG levels, which normalize after treatment with metronidazole.[20] False-positive TTG elevation has also been associated with other infections, such as *Helicobacter pylori* and HIV.[21,22] There are rare reports of TTG elevation in immune-proliferative small-intestinal disease.[23–25]

It is well known that while TTG levels increase, the risk of diagnosing PCD decreases[9,26–30]; yet, patients with TTG levels above 10× ULN without diagnostic mucosal damage are still found. In our current cohort, among 11 children with an initial TTG > 10× ULN and at least one year of follow-up, TTG normalized in three (27.2%), declined in five (45.4%), and increased or remained above 10× ULN in three (27.2%). The omission of biopsies in these patients would have misdiagnosed CeD in at least three children.

The strengths of our study include the comparatively large number of pediatric patients with PCD at our center, as well as the ability to establish the proportions of patients with PCD in relation to the total number of CeD patients diagnosed by endoscopy during the study period. The majority of children had one or more follow-up visits that included serology levels 12 months after diagnosis. This enabled the evaluation of changes in CeD serology and the natural history of PCD among our cohort who continued consuming gluten.

Our real-world retrospective study has several limitations that need to be addressed. The follow-up period of one to five years is relatively short. In addition, the retrospective design is a notable limitation. In the study, only TTG antibodies (Bioplex 2200) were used, as DGP antibodies were unavailable. HLA testing for DQ2/DQ8 positivity was not analyzed in our patients. The lack of data on infection screening for patients with very high TTG levels remains an additional gap. The confirmation of gluten consumption at endoscopy and follow-up was done by questioning the child and family at limited times. In addition, repeated routine biopsies were not executed in all patients. However, in more than 80% of patients, TTG normalized or declined to <3× ULN, a level at which the risk of CeD is considered low. Thus, we believe there was no justification for performing additional biopsies. Repeat endoscopy should be performed during follow-up if indicated clinically or when TTG levels remain greater than 3× ULN over time.

Future follow-up of PCD patients and future studies on PCD could benefit from including DGP antibody testing, especially for children under two years, where TTG sensitivity is lower. Incorporating infection screening data for patients with very high TTG (>10× ULN) would improve diagnostic accuracy and understanding of false positives.

Conclusions

PCD is common and occurs in approximately 15% of children undergoing upper endoscopy for suspected CeD. It may occur in patients with serology levels above 10× ULN. Pediatric patients with PCD can and should be followed while consuming a GCD, given that the majority of patients will not progress to CeD. Approximately 50% of PCD patients will normalize TTG levels, and close

to 30% will decrease levels to below 3× ULN. All PCD patients should have regular serological testing and clinical follow-up by a pediatric gastroenterologist, with the option of repeated biopsy when indicated. CeD may be overdiagnosed if endoscopy and biopsy are not performed at the time of diagnosis.

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Conflict of interest

All authors declare no conflict of interest related to this publication.

Author contributions

Data collection and analysis, assistance with statistical analysis, interpretation of data, approval of the final manuscript (BL), data analysis, assistance with statistical analysis, interpretation of data, approval of the final manuscript (AH), conceptualization and design of the study, data analysis and interpretation, drafting and critical review of the manuscript, and approval of the final manuscript (MK). All authors have read and agreed to the published version of the manuscript.

Ethical statement

The research was conducted in accordance with the Declaration of Helsinki (as revised in 2024) and received approval from the Regional Ethics Committee at Kaplan Medical Center (Protocol Number: KMC-0041-21). Due to the retrospective nature of the study and the use of anonymized patient data, the ethics committee waived the need for informed consent.

Data sharing statement

The dataset used in support of the findings of this study are available from the corresponding author at korifamily@yahoo.com upon request.

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