Vol.20, No.2, Page 530- 538, 2026 http://dx.doi.org/10.59628/jchm.v20i2.2238 ISSN 2958-7476 JMCH

Sana'a University Journal of Medical and Health Sciences

Seroprevalence of Celiac Disease Among Children with Short Stature in Sana'a City, Yemen

Ahlam Abdulmalik Esmail[1], Ahmed S. Hudna[1*], Sulaiman A. Al-Haiga[2], Tawfiq M. Al-Hammadi[2], Hamza A. Abdullah[2], Abdulrahman Y. Khamis[3], Kamal W. Yousef[3] and Anas Alshomi [2]

[1]**Department of Pediatrics, Faculty of Medicine and Health Sciences, University of Science and Technology, Sana'a, Yemen,**
[2]**Department of Pediatrics, University of Science and Technology Hospital, Sana'a, Yemen,**
[3]**Department of Medicine, University of Science and Technology Hospital, Sana'a, Yemen.**

*Corresponding author: Email: drahmedhudna@gmail.com

Abstract

Background: Celiac disease (CD) is an immune-mediated enteropathy in individuals genetically susceptible to gluten and its related proteins. It is an important but often under-recognized cause of short stature in children, frequently presenting without gastrointestinal symptoms. This study aimed to determine the seroprevalence of CD among children with short stature in Sana'a City, Yemen.

Methods: A retrospective cross-sectional study was conducted at the main Pediatric Endocrinology Center in Sana'a, from August to November 2024. Medical records of 385 children with short stature aged $<$20 years were reviewed. Data on demographic, clinical, and laboratory characteristics were extracted, and anti-tissue transglutaminase immunoglobulin A (anti-tTG IgA) was used to determine the prevalence of CD. Data were described using frequency and percentage.

Results: The mean age of children was 9.8 $\pm$ 4.4 years, with males comprising 58.7%. More than half of the children (51.4%) were older than 10 years. Gastrointestinal manifestations were uncommon, with only three children (0.8%) presenting with diarrhea. Seropositivity for anti-tTG IgA was found in 6% of the tested children, while growth hormone deficiency and low IGF-1 levels were detected in 27% and 41% of the cases, respectively. More than half of the children (51.6%) had a vitamin D deficiency. Concurrent chronic diseases were identified in 17.9% of children, most commonly type 1 diabetes mellitus (6.2%), while Turner syndrome was the most frequent associated genetic disorder (1.8%).

Conclusion: CD is relatively common among children with short stature in Yemen, even in the absence of gastrointestinal symptoms. Growth failure in children with short stature is likely to result from a combination of endocrine, nutritional, and immunological factors. Routine screening for CD should be incorporated into the diagnostic evaluation of all children with an unexplained short stature.

Article Info

Keywords:
short stature, celiac disease, prevalence, children, Yemen.

Article History:
Received: 10-September-2025,
Revised: 12-September-2025,
Accepted: 5-March-2026,
Published: 28-April-2026.

1. INTRODUCTION

Celiac disease (CD), also called gluten-sensitive enteropathy or nontropical sprue, is an immune-mediated disorder elicited by the consumption of wheat gluten and gluten-related proteins in other types of cereals, such as barley and rye, in genetically susceptible individuals and is characterized by chronic enteritis [1–3]. A recent systematic review revealed a pooled global seroprevalence of 1.4%, with a biopsy-confirmed prevalence of 0.7% [4]. The incidence is higher in women (17.4 per 100,000 person-years) than in men (7.8 per 100,000 person-years) [4]. Early studies have demonstrated a strong genetic association between CD and human leukocyte antigen (HLA) alleles [5–7]. It involves both innate and adaptive immune responses that damage

the small intestinal mucosa, produce disease-specific anti-transglutaminase 2 (anti-TG2) autoantibodies, and manifest with both intestinal and extraintestinal complications [8]. The autoimmune response in CD also involves antibodies against other transglutaminase family members, such as TG3 and TG6 [9].

In addition to genetics, environmental risk factors may contribute to the risk and timing of CD development [10]. These factors include delivery methods, gluten exposure, infant feeding practices, childhood infections, and gut microbiota composition [10–12]. However, the timing of gluten introduction and breastfeeding duration were not been found to be significantly associated with CD risk [11]. Understanding these environmental factors is essential for elucidating CD pathogenesis and guiding potential prevention strategies, particularly for at-risk populations [10–12].

A large Norwegian birth cohort study found that introducing gluten after six months and breastfeeding beyond 12 months were both associated with an increased risk of developing CD in children [13]. However, it can present at any age, with diagnostic peaks during early childhood and adulthood [14]. The disease shows a female predominance in diagnosis despite similar autoantibody prevalence between sexes, with age- and sex-related differences in presentation indicating that males, particularly in early adulthood, are underdiagnosed and warrant greater diagnostic attention [15]. The clinical manifestations differ by age, with young children typically presenting with diarrhea and growth failure, whereas older children and adults may exhibit a broader spectrum of gastrointestinal and extraintestinal symptoms [16]. CD is associated with various complications, including nutritional deficiencies, poor growth, anemia, osteoporosis, infertility, cardiovascular and neurological disorders, renal and hepatic involvement, psychiatric diseases, increased risk of autoimmune diseases and malignancies, and digestive complications such as microscopic colitis and refractory sprue [17, 18]. Rare but serious complications include refractory CD, enteropathy-associated T-cell lymphoma, and small bowel adenocarcinoma [19, 20].

The diagnosis of CD relies on serologic testing, primarily by the detection of IgA anti-TG2, followed by duodenal biopsy to confirm characteristic mucosal changes, such as intraepithelial lymphocytosis and villous atrophy, with symptomatic and histologic improvement after gluten withdrawal [21, 22]. Serological tests for pediatric celiac disease demonstrate high diagnostic accuracy; however, improved standardization and further research are needed to enhance follow-up strategies and broaden the application of no-biopsy diagnostic criteria [23]]. A lifelong gluten-free diet (GFD) remains the primary treatment for CD because it can alleviate symptoms and trigger mucosal recovery [24]. However, adherence to a GFD is often difficult because of the risk of accidental gluten exposure, potential nutrient deficiencies, and the higher cost and lower nutritional quality of gluten-free products compared with their gluten-containing counterparts [24, 25]. Long-term GFD adherence may lead to nutritional deficiencies, particularly in fiber, B vitamins, iron, and trace minerals [26, 27].

Short stature in children is a common concern that can indicate various underlying health issues, including nutritional deficiencies, endocrine abnormalities, genetic disorders, and chronic illnesses [28]. It is the most common reason for referral to pediatric endocrinologists [29]. Common causes include growth hormone deficiency, malnutrition, familial short stature, hypothyroidism, and Turner syndrome [30]. The prevalence of short stature among children ranges from 4.3% in certain outpatient settings [31] to as high as 33.7% in specific regions, such as Taif City in Saudi Arabia [32]. CD is an important cause of short stature in children, often presenting without gastrointestinal symptoms and with varying prevalence rates across different regions [33].

In Yemen, a relatively high prevalence of 9.2% for CD was reported among patients with gastrointestinal symptoms in Sana'a City [34]. However, a lower prevalence of 0.22% was later reported among outpatients with gastrointestinal complaints in Sana'a [35]. A recent five-year retrospective study in Sana'a revealed a higher-than-global prevalence of 3.4% for CD among Yemeni children [36]. However, no study has investigated the prevalence of CD among Yemeni children with short stature. Therefore, this study aimed to determine the prevalence of CD among this group in Sana'a, Yemen.

2. METHODS

2.1. Study design, population and setting

A retrospective, cross-sectional study was conducted using medical records of children with short stature attending the Pediatric Endocrinology Center in Sana'a City, from August to November 2024. Short stature was operationally defined as height-for-age below the 3^{rd} percentile [37], which corresponds to a height-for-age Z-score (HAZ) < -2 standard deviations (SD). Due to the retrospective record-based nature of the study, anthropometric measurements were not performed by the research team. A total of eligible 385 records of children with short stature aged < 20 years were included. Records with incomplete data were excluded from the study.

2.2. Data collection

Data were extracted from patients' medical records using a data extraction sheet developed by the researchers based on the data available after reviewing relevant litera-

Table 1. Characteristics of children with short stature included in the study (N = 385).

Characteristics	*n*(%)
Age (years)	
Mean ± SD (range): 9.8 ± 4.4 (0.8 – 19.8)	
≤9	157(**36.3**)
>9	228(**63.7**)
Gender	
Male	226(**58.7**)
Female	159(**41.3**)

ture that captured demographic characteristics (age and gender), and concurrent chronic diseases and genetic syndromes. In addition, relevant laboratory investigations for CD and short stature were reviewed, particularly anti-tTG IgA antibodies, insulin-like growth factor 1 (IGF-1) levels, and growth hormone (GH) stimulation test results.

2.3. Data analysis

Data were analyzed using IBM SPSS Statistics, version 21 (IBM Corp., Armonk, NY, USA). Categorical data were described using frequencies and percentages. Growth assessment at the center was carried out using routinely conducted internationally accepted World Health Organization (WHO) standards, including the WHO Child Growth Standards (2006) for children aged <5 years [38] and the WHO Growth Reference (2007) for those aged ≥ 5 years [39]. HAZ were generated using the WHO Anthro software for children < years and WHO AnthroPlus software for those aged ≥ 5 years. Categorization was based on standard WHO cut-off points, where normal stature was considered at HAZ values ≥ −2 SD and stunting at HAZ values < −2 SD.

2.4. Ethical considerations

Ethical approval for the study was obtained from the Research Ethics Committee of the Faculty of Medicine and Health Sciences, University of Science and Technology, Sana'a, Yemen. In addition, permission to access the children's records was obtained from the center's administration. Given the retrospective design of this study, the requirement for informed consent was waived.

3. RESULTS

3.1. Demographic characteristics of the study population

Table 1 shows that the mean age of children with short stature was 9.8 ± 4.4 years (range: 0.8–19.8 years), with the majority being older than nine years (63.7%) and males (58.7%).

3.2. Clinical presentation of children at admission

Table 2 shows that more than half of children with short stature (51.4%) were presented after the age of 10 years. Gastrointestinal manifestations were uncommon, with constipation reported in seven children (1.8%), diarrhea in three (0.8%), abdominal pain in two (0.5%), and abdominal distension in one (0.3%).

3.3. Seroprevalence of CD and laboratory findings

Table 3 shows that CD was detected among 6% (13/228) of children with short stature based on the positivity of anti-tTG IgA.

3.4. Laboratory findings of CD

Hemoglobin levels were ≤10 mg/dl in only 6% of cases. Thyroid-stimulating hormone (TSH) levels were elevated in 16.6% of patients, while free thyroxine (FT4) levels were low in 8.5% of patients. More than half of the children (51.6%) had vitamin D deficiency, 27% had GH deficiency (GHD), 41% had low IGF-1 levels, and 30% had an elevated erythrocyte sedimentation rate (ESR). However, only 1.6% of the children had abnormal serum creatinine levels, 11.8% had elevated alanine aminotransferase (ALT) levels, and 7% had elevated random blood sugar (RBS) levels.

3.5. Concurrent chronic diseases and syndromes among children with short stature

Table 5 shows that 17.9% of the children with short stature had at least one chronic disease. Neurological conditions were uncommon, including cerebral palsy in three cases (0.8%), epilepsy in two cases (0.5%), and brain tumors in one case (0.3%). On the other hand, only one child (0.3%) had congenital heart disease (CHD). Among musculoskeletal diseases, osteogenesis imperfecta was observed in five cases (1.3%), whereas

Table 2. Clinical presentation of children with short stature at admission (N = 385).

Variable	*n* (%)
Age at presentation (years)	
≤10	187 (**48.6**)
>10	198 (**51.4**)
Diarrhea	
Yes	3 (**0.8**)
No	382 (**99.2**)
Constipation	
Yes	7 (**1.8**)
No	378 (**98.2**)
Vomiting	
Yes	0 (**0.0**)
No	385 (**100.0**)
Abdominal pain	
Yes	2 (**0.5**)
No	383 (**99.5**)
Abdominal distention	
Yes	1 (**0.3**)
No	384 (**99.7**)

Table 3. Seroprevalence of CD children with short stature attending the Pediatric Endocrinology Center in Sana'a City (2024).

Laboratory finding	*n* (%)
Anti-tTG IgA (N = 228)	
Normal	215 (**94.0**)
High	13 (**6.0**)

juvenile idiopathic arthritis (JIA), Duchenne muscular dystrophy, and rheumatoid arthritis (RA) were each observed in one child (0.3% each). Concurrent endocrine and metabolic disorders included type 1 diabetes mellitus (T1DM) in 6.2% of children, followed by congenital adrenal hyperplasia (CAH) in six cases (1.6%) and obesity in eight cases (2.1%). Other conditions, including central diabetes insipidus, Addison's disease, hypoparathyroidism, and congenital hypothyroidism, were rare (0.3% each). Regarding renal diseases, chronic kidney disease (CKD) was observed in two cases (0.5%), with single cases of nephrotic syndrome and renal tubular acidosis (RTA) of the Fanconi type (0.3% each). Other chronic conditions included thalassemia, sickle cell anemia (SCA), and glucose-6-phosphate dehydrogenase (G6PD) deficiency, each observed in two cases (0.5%). Overall, concurrent syndromes were present in 19 children (4.9%), with Turner syndrome being the most frequent (1.8%), followed by 3M syndrome (0.8%), achondroplasia, and Neger syndrome (0.5% each). Single cases of Down syndrome, Russell-Silver syndrome, Klinefelter syndrome, Hurler syndrome, and Klippel syndrome were also documented.

4. DISCUSSION

To the best of our knowledge, this study is the first to investigate CD among children with short stature in Yemen. The seroprevalence of CD in the present study was 6% based on positive anti-tTG IgA results, which is almost double the 3.4% reported in a five-year retrospective study among children admitted to Al-Sabeen Maternity Hospital in Sana'a [36]. However, the latter study examined all pediatric admissions rather than focusing on children with short stature. The higher prevalence among children with short stature highlights that CD is a risk factor for impaired growth. The prevalence of CD among children in the present study closely mirrors the 6.7% prevalence observed among short-statured children attending endocrinology clinics in Karachi, Pakistan [40]. A higher prevalence of 8.1% was reported for CD among Iranian children with idiopathic short stature [41] and 9.9% for asymptomatic CD was reported in a prospective study among children with proportionate short stature in Lahore, Pakistan [42]. These findings reinforce that children with short stature represent a high-risk group for CD and should undergo routine serological screening regardless of symptoms. In comparison with the present study, a higher prevalence of CD was reported among children with short stature in India (11.8%) [43], Saudi Arabia (13.8%) [44], and Erbil, Iraq (13.9%) [45], and an even greater CD prevalence of 33.3% was reported among children with short stature in Peshawar, Pakistan. [46]. The variation in CD prevalence among children with short stature across studies in different countries can be explained by differences in study design, diagnostic methods, sample size, and criteria for defining short

Table 4. Laboratory findings among children with short stature attending the Pediatric Endocrinology Center in Sana'a City (2024).

Laboratory finding	*n* (%)
Hemoglobin (*N* = 272)	
≤10 mg/dl	17 (**6.0**)
>10 mg/dl	255 (**94.0**)
Thyroid-stimulating hormone (*N* = 278)	
Normal	221 (**79.4**)
Low	3 (**1.0**)
High	55 (**19.6**)
Free thyroxine (*N* = 283)	
Normal	256 (**90.4**)
Low	24 (**8.5**)
High	3 (**1.1**)
Vitamin D (*N* = 31)	
Normal	15 (**48.4**)
Low	16 (**51.6**)
Serum creatinine (*N* = 257)	
Normal	253 (**98.4**)
Abnormal	4 (**1.6**)
Alanine aminotransferase (*N* = 220)	
Normal	194 (**88.2**)
High	26 (**11.8**)
Random blood sugar (*N* = 44)	
Normal	41 (**93.0**)
High	3 (**7.0**)
GH stimulation test (*N* = 96)	
Normal	70 (**73.0**)
Low	26 (**27.0**)
IGF-1 (*N* = 214)	
Normal	126 (**59.0**)
Low	88 (**41.0**)
Erythrocyte sedimentation rate (*N* = 148)	
Normal	103 (**70.0**)
High	45 (**30.0**)

IgA, immunoglobulin A; anti-tTG, anti-tissue transglutaminase; GH, growth hormone; IGF-1, insulin-like growth factor 1.

Table 5. Concurrent chronic diseases and syndromes among children with short stature attending the Pediatric Endocrinology Center in Sana'a City, Yemen in 2024 (*N* = 385).

Associated disease/syndrome	***n* (%)**
Presence of chronic diseases	69 (**17.9**)
CNS diseases	
Cerebral palsy	3 (**0.8**)
Epilepsy	2 (**0.5**)
Brain tumor	1 (**0.3**)
Cardiovascular diseases	
Congenital heart disease	1 (**0.3**)
Musculoskeletal diseases	
Osteogenesis imperfecta	5 (**1.3**)
Rheumatoid arthritis	1 (**0.3**)
Duchene muscular dystrophy	1 (**0.3**)
Juvenile idiopathic arthritis	1 (**0.3**)
Endocrine and metabolic disorders	
Type 1 diabetes mellitus	24 (**6.2**)
Obesity	8 (**2.1**)
Congenital adrenal hyperplasia	6 (**1.6**)
Central diabetes insipidus	1 (**0.3**)
Familial hypercholesterolemia	1 (**0.3**)
Addison's disease	1 (**0.3**)
Hyperparathyroidism	1 (**0.3**)
Congenital hypothyroidism	1 (**0.3**)
Renal diseases	
Chronic kidney disease	2 (**0.5**)
Renal tubular acidosis (Fanconi syndrome)	1 (**0.3**)
Nephrotic syndrome	1 (**0.3**)
Other concurrent chronic diseases	
Thalassemia	2 (**0.5**)
Sickle cell anemia	2 (**0.5**)
Glucose-6-phosphate dehydrogenase deficiency	1 (**0.3**)
Neurofibromatosis type 1	1 (**0.3**)
Immune thrombocytopenic purpura	1 (**0.3**)
Acute lymphoblastic leukemia	1 (**0.3**)
Presence of concurrent syndromes	**19** (**4.9**)
Turner Syndrome	7 (**1.8**)
3 M syndrome*	3 (**0.8**)
Achondroplasia	2 (**0.5**)
Neger syndrome	2 (**0.5**)
Down Syndrome	1 (**0.3**)
Russell-Silver Syndrome	1 (**0.3**)
Klinefelter syndrome	1 (**0.3**)
Hurler syndrome	1 (**0.3**)
Klippel syndrome	1 (**0.3**)

* Miller, McKusick and Malveaux syndrome.

stature, as well as population characteristics, genetics, and environmental influences.

The majority of children with short stature in the present study did not present with classical gastrointestinal symptoms. This finding is in agreement with several reports indicating that growth failure may be the only or the predominant manifestation of CD in children. For instance, none of the Pakistani children with short stature presented with any gastrointestinal symptoms in Karachi [40]. Similarly, most short-statured children who tested positive for anti-tTG antibodies had no gastrointestinal symptoms in India and Saudi Arabia [43, 44]. These findings suggest that short stature may be an extraintestinal manifestation of CD. Nutrient malabsorption due to CD may manifest as short stature, often without overt gastrointestinal symptoms [33]. These studies indicate that short stature, even without digestive symptoms, should prompt screening for CD in children. In addition, the present study is consistent with international evidence, emphasizing the need to include CD testing in the routine screening of all children with an unexplained short stature. In contrast, 75.2% of children with idiopathic short stature in Indiana, United States, had one or more gastrointestinal tract diseases [47].

Apart from gastrointestinal symptoms, a substantial proportion of children with short stature in the present study presented with several endocrinological and metabolic abnormalities, particularly low IGF-1 levels and GHD. In this context, 41% of the children had low IGF-1 levels and 27% had GHD, suggesting that impaired growth in this population may result from complex hormonal dysregulation in addition to unrecognized CD. In contrast, a lower IGF-1 deficiency of 20% was reported in a European study of prepubertal children with isolated short stature [48], and a lower GHD of 13.9% was reported among children with short stature in a hospital-based survey in Rawalpindi, Pakistan [49]. These discrepancies may be explained by differences in study design, population genetics, environmental exposures, nutritional status, and the co-existence of chronic conditions such as malnutrition, which are more common in conflict-affected settings such as Yemen. Genetic factors, such as IGF1 haploinsufficiency, have been identified as causes of short stature, often presenting with microcephaly and SGA birth history [50]. Furthermore, therapeutic advances, such as the use of recombinant human IGF-1, have shown promise in managing children with primary IGF-1 deficiency who do not respond adequately to growth hormone therapy [51]. These findings suggest that growth impairment in children with short stature is multifactorial and that comprehensive endocrine evaluation should be an integral part of their diagnostic workup, along with routine celiac disease screening.

The role of vitamin D deficiency in exacerbating growth failure was evident in our study, where it affected more than half of the children. Similarly, in a study of idiopathic short stature, vitamin D deficiency and insufficiency were observed in the majority of children and were accompanied by alterations in GH and IGF-1 dynamics [52]. Notably, vitamin D deficiency is associated with impaired height growth in young children, even in those without short stature [53]. These findings emphasize the importance of assessing vitamin D status in children with growth issues and suggest that vitamin D supplementation may be beneficial for improving growth outcomes in affected children.

In our study, concurrent chronic diseases and genetic syndromes were found in 17.9% and 4.9% of children with short stature, respectively, with T1DM (6.2%) and Turner syndrome (1.8%) being the most common. CD is known to cluster with other autoimmune diseases and chromosomal abnormalities, highlighting the complex interplay between genetic and immunological factors. Research indicates that over 60% of CD-associated susceptibility loci overlap with those of other autoimmune conditions, suggesting shared pathogenic mechanisms [54]. Greco et al. (2011) found that autoimmune conditions, like T1DM and thyroid disease, are significantly more prevalent among CD patients in the Mediterranean region, especially in pediatric populations [55]. The association with Turner syndrome is well established, as this chromosomal disorder frequently presents with short stature and has a higher risk of CD [55]. However, other syndromes, such as Down syndrome (0.3%), and rare genetic conditions were less commonly identified in our study, although they can further exacerbate growth problems.

This study provides insights into the seroprevalence of CD among children with short stature in Sana'a City, thereby addressing a critical gap in knowledge within a resource-limited and conflict-affected setting. The relatively large sample size and inclusion of detailed clinical, laboratory, and comorbidity data strengthen the findings of this study. However, this study has several limitations. The retrospective nature of the study and reliance on medical records may have introduced information bias and missing data into the study. Not all children underwent complete laboratory evaluation, including anti-tTG IgA testing and vitamin D assessment, which may have led to an underestimation or overestimation of the true prevalence. In addition, the specific commercial kits and cutoff values for serological assays were not consistently documented in the medical records. Although all results were interpreted according to laboratory-established reference ranges and internal quality control procedures, potential inter-assay variability cannot be excluded. Furthermore, the diagnosis of celiac disease was based solely on serological testing without histopathological confirmation. However, reliance on serology aligns with established practices in resource-limited settings, particularly where biopsy is not readily available. Finally, the study was conducted in a single tertiary referral center,

making it difficult to generalize its findings to all children with short stature in the country. Future studies should adopt a prospective multicenter design with larger and more diverse populations to improve generalizability and reduce bias. In addition, whenever possible, serological findings should be complemented with duodenal biopsy to strengthen the diagnostic accuracy.

5. CONCLUSION

CD is relatively common among children with short stature, as indicated by anti-tTG IgA positivity, despite the almost absence of gastrointestinal manifestations. GHD, IGF-1 deficiency, and vitamin D deficiency are also frequent, along with chronic diseases and genetic syndromes, suggesting that growth failure arises from a combination of endocrine, nutritional, and immunological factors. These findings highlight that short stature in children is a heterogeneous condition with diverse underlying etiologies, and routine investigation for CD in conjunction with other systemic disorders is essential for timely diagnosis and appropriate management.

CONFLICT OF INTEREST

No conflict of interest.

FUNDING

None

ACKNOWLEDGMENTS

The authors thank the administration and staff of the Main Pediatric Endocrinology Center in Sana'a City for their cooperation and help during data collection.

REFERENCES

[1] C. Briani, D. Samaroo, and A. Alaedini. "Celiac disease: from gluten to autoimmunity". In: *Autoimmun. Rev.* 7 (2008), pp. 644–650. DOI: 10.1016/j.autrev.2008.05.006.

[2] A. Fasano and C. Catassi. "Celiac disease". In: *New Engl. J. Med.* 367 (2012), pp. 2419–2426. DOI: 10.1056/NEJMcp1113994.

[3] M. Malalgoda and S. Simsek. "Celiac disease and cereal proteins". In: *Food Hydrocoll.* 68 (2017), pp. 108–113. DOI: 10.1016/j.foodhyd.2016.09.024.

[4] G. K. Makharia et al. "Epidemiology of coeliac disease". In: *Aliment. Pharmacol. & Ther.* 56 (2022), S3–S17. DOI: 10.1111/apt.16787.

[5] Z. M. Falchuk, G. N. Rogentine, and W. Strober. "Predominance of histocompatibility antigen HL-A8 in patients with gluten-sensitive enteropathy". In: *J. Clin. Investig.* 51 (1972), pp. 1602–1605. DOI: 10.1172/jci106958.

[6] P. L. Stokes et al. "Histocompatibility antigens associated with adult coeliac disease". In: *The Lancet* 2 (1972), pp. 162–164. DOI: 10.1016/S0140-6736(72)91330-X.

[7] R. Tosi et al. "Evidence that celiac disease is primarily associated with a DC locus allelic specificity". In: *Clin. Immunol. Immunopathol.* 28 (1983), pp. 395–404. DOI: 10.1016/0090-1229(83)90106-X.

[8] K. Lindfors, K. Kaukinen, and M. Mäki. "A role for anti-transglutaminase 2 autoantibodies in the pathogenesis of coeliac disease?" In: *Amino Acids* 36 (2009), pp. 685–691. DOI: 10.1007/s00726-008-0127-5.

[9] S. Caja et al. "Antibodies in celiac disease: implications beyond diagnostics". In: *Cell. Mol. Immunol.* 8 (2011), pp. 103–109. DOI: 10.1038/cmi.2010.65.

[10] M. Sarno et al. "Risk factors for celiac disease". In: *Italian J. Pediatr.* 41 (2015), p. 57. DOI: 10.1186/s13052-015-0166-y.

[11] E. Lionetti and C. Catassi. "The role of environmental factors in the development of celiac disease: what is new?" In: *Diseases* 3 (2015), pp. 282–293. DOI: 10.3390/diseases3040282.

[12] G. M. Pes, S. Bibbò, and M. P. Dore. "Coeliac disease: beyond genetic susceptibility and gluten". In: *Ann. Med.* 51 (2019), pp. 1–16. DOI: 10.1080/07853890.2019.1569254.

[13] K. Størdal et al. "Early feeding and risk of celiac disease in a prospective birth cohort". In: *Pediatrics* 132 (2013), e1202–e1209. DOI: 10.1542/peds.2013-1752.

[14] N. R. Reilly, A. Fasano, and P. H. R. Green. "Presentation of celiac disease". In: *Gastrointest. Endosc. Clin. North Am.* 22 (2012), pp. 613–621. DOI: 10.1016/j.giec.2012.07.008.

[15] R. Dixit et al. "Celiac disease is diagnosed less frequently in young adult males". In: *Dig. Dis. Sci.* 59 (2014), pp. 1509–1512. DOI: 10.1007/s10620-014-3025-6.

[16] S. Vivas et al. "Age-related differences in celiac disease: specific characteristics of adult presentation". In: *World J. Gastrointest. Pharmacol. Ther.* 6 (2015), pp. 207–212. DOI: 10.4292/wjgpt.v6.i4.207.

[17] J. Cosnes and I. Nion-Larmurier. "Complications of celiac disease". In: *Pathol. Biol.* 61 (2013), e21–e26. DOI: 10.1016/j.patbio.2011.03.004.

[18] J. F. Ludvigsson et al. "Coeliac disease: complications and comorbidities". In: *Nat. Rev. Gastroenterol. & Hepatol.* 22 (2025), pp. 252–264. DOI: 10.1038/s41575-024-01032-w.

[19] S. Ondrejka and D. Jagadeesh. "Enteropathy-associated T-cell lymphoma". In: *Curr. Hematol. Malig. Reports* 11 (2016), pp. 504–513. DOI: 10.1007/s11899-016-0357-7.

[20] M. Tinguria. "Rare complications of celiac disease: clinicopathologic features". In: *Med. Res. Arch.* 11 (2023). DOI: 10.18103/mra.v11i4.3763.

[21] P. H. Green, K. Rostami, and M. N. Marsh. "Diagnosis of coeliac disease". In: *Best Pract. & Res. Clin. Gastroenterol.* 19 (2005), pp. 389–400. DOI: 10.1016/j.bpg.2005.02.006.

[22] C. P. Kelly et al. "Advances in diagnosis and management of celiac disease". In: *Gastroenterology* 148 (2015), pp. 1175–1186. DOI: 10.1053/j.gastro.2015.01.044.

[23] M. Repo et al. "Serological testing for celiac disease in children". In: *Expert Rev. Gastroenterol. & Hepatol.* 19 (2025), pp. 155–164. DOI: 10.1080/17474124.2025.2462245.

[24] A. M. Mazzola et al. "Gluten-free diet and other celiac disease therapies: current understanding and emerging strategies". In: *Nutrients* 16 (2024). DOI: 10.3390/nu16071006.

[25] I. Parzanese et al. "Celiac disease: from pathophysiology to treatment". In: *World J. Gastrointest. Pathophysiol.* 8 (2017), pp. 27–38. DOI: 10.4291/wjgp.v8.i2.27.

[26] T. G. Theethira and M. Dennis. "Celiac disease and the gluten-free diet: consequences and recommendations for improvement". In: *Dig. Dis.* 33 (2015), pp. 175–182. DOI: 10.1159/000369504.

[27] M. Ghunaim et al. “Impact of a gluten-free diet in adults with celiac disease: nutritional deficiencies and challenges”. In: *Cureus* 16 (2024), e74983. DOI: 10.7759/cureus.74983.

[28] N. Polidori et al. “Deciphering short stature in children”. In: *Ann. Pediatr. Endocrinol. & Metab.* 25 (2020), pp. 69–79. DOI: 10.6065/apem.2040064.032.

[29] P. F. Collett-Solberg et al. “Growth hormone therapy in children; research and practice – A review”. In: *Growth Horm. & IGF Res.* 44 (2019), pp. 20–32. DOI: 10.1016/j.ghir.2018.12.004.

[30] K. Angadi et al. “A study of clinical profile of children with short stature”. In: *Indian J. Child Health* 8 (2021), pp. 228–230.

[31] M. Shrivastava and A. S. Shrivastava. “A cross-sectional study of the prevalence of short stature among children visiting the outpatient department of a tertiary care center”. In: *Indian J. Child Health* 11 (2024), pp. 82–87.

[32] A. Al Ghamdi et al. “Prevalence of short stature among children aged 5–12 years old in Taif City, Saudi Arabia”. In: *J. Healthc. Sci.* 2 (2022), pp. 192–196. DOI: 10.52533/JOHS.2022.2808.

[33] C. Meazza et al. “Celiac disease and short stature in children”. In: *Expert Rev. Endocrinol. & Metab.* 9 (2014), pp. 535–542. DOI: 10.1586/17446651.2014.932248.

[34] O. A. I. Al-Dossary et al. “Celiac disease among gastrointestinal patients in Yemen: its prevalence, symptoms and accompanying signs, and its association with age and gender”. In: *Univers. J. Pharm. Res.* 6 (2021), pp. 1–6. DOI: 10.22270/ujpr.v6i5.665.

[35] A. A. Esmail et al. “Celiac disease among outpatient attendees with gastrointestinal complaints at a tertiary care hospital in Sana’a City, Yemen: a four-year retrospective study”. In: *UST J. Med. Sci.* 3 (2025). DOI: 10.59222/ustjms.3.3.

[36] S. A. Alsofi et al. “Prevalence and clinical profile of celiac disease in Yemeni children: a five-year retrospective study at Al-Sabeen Hospital”. In: *Cureus* 17 (2025), e82824. DOI: 10.7759/cureus.82824.

[37] C. Barstow and C. Rerucha. “Evaluation of short and tall stature in children”. In: *Am. Fam. Physician* 92 (2015), pp. 43–50.

[38] World Health Organization. *WHO child growth standards: length/height-for-age, weight-for-age, weight-for-length, weight-for-height and body mass index-for-age: methods and development*. Geneva: World Health Organization, 2006.

[39] M. de Onis et al. “Development of a WHO growth reference for school-aged children and adolescents”. In: *Bull. World Health Organ.* 85 (2007), pp. 660–667. DOI: 10.2471/blt.07.043497.

[40] M. Jalil et al. “Celiac disease in short stature pediatric population presenting to endocrinology clinics: a cross-sectional study from a tertiary care hospital in Karachi”. In: *Liaquat National J. Prim. Care* 5 (2023), pp. 22–25.

[41] J. Hashemi et al. “Prevalence of celiac disease in Iranian children with idiopathic short stature”. In: *World J. Gastroenterol.* 14 (2008), pp. 7376–7380. DOI: 10.3748/wjg.14.7376.

[42] M. Javed et al. “Short stature as a major manifestation of celiac disease in children 1–14 years of age at Shaikh Zayed Hospital Lahore”. In: *Pak. J. Med. Health Sci.* 17 (2023), p. 12. DOI: 10.53350/pjmhs202317912.

[43] F. Ahmad et al. “IgA anti-tTG antibodies in children with severe short stature without gastrointestinal manifestations”. In: *Indian J. Gastroenterol.* 31 (2012), pp. 32–33. DOI: 10.1007/s12664-011-0160-2.

[44] O. I. Saadah et al. “Celiac disease in Saudi children with isolated short stature: is it rare or are we not screening rigorously enough?” In: *J. Pediatr. Endocrinol. Metab.* 33 (2020), pp. 89–93. DOI: 10.1515/jpem-2019-0348.

[45] D. D. Abdullah and M. S. Mahmood. “The prevalence of celiac disease among children with short stature”. In: *Adv. Med. J.* 9 (2024), pp. 146–154.

[46] A. Muhammad et al. “Short stature and celiac disease in children (5 to 16 years) presenting at a tertiary care hospital in Peshawar”. In: *Cureus* 14 (2022), e26099. DOI: 10.7759/cureus.26099.

[47] R. Stawerska et al. “Frequency of oligosymptomatic gastrointestinal tract diseases and its relation to insulin-like growth factor I in idiopathic (non-GH-deficient) short stature children”. In: *Arch. Med. Sci.* 17 (2021), pp. 1663–1671. DOI: 10.5114/aoms.2020.93809.

[48] T. Edouard et al. “Prevalence of IGF1 deficiency in prepubertal children with isolated short stature”. In: *Eur. J. Endocrinol.* 161 (2009), pp. 43–50. DOI: 10.1530/eje-08-0964.

[49] T. M. Awan, A. Sattar, and E. G. Khattak. “Frequency of growth hormone deficiency in short statured children”. In: *J. Coll. Physicians Surg. Pak.* 15 (2005), pp. 295–298.

[50] W. Staels et al. “IGF1 haploinsufficiency in children with short stature: a case series”. In: *Eur. J. Endocrinol.* 185 (2021), pp. 323–332. DOI: 10.1530/eje-20-1463.

[51] P. F. Collett-Solberg and M. Misra. “The role of recombinant human insulin-like growth factor-I in treating children with short stature”. In: *J. Clin. Endocrinol. & Metab.* 93 (2008), pp. 10–18. DOI: 10.1210/jc.2007-1534.

[52] M. O. Ryznychuk. “Vitamin D status in children with idiopathic stunting”. In: *Mod. Pediatr. (Ukraine)* 5 (2024), p. 141. DOI: 10.15574/SP.2024.5(141).4954.

[53] S. Kuraoka et al. “Impaired height growth associated with vitamin D deficiency in young children from the Japan Environment and Children’s Study”. In: *Nutrients* 14 (2022). DOI: 10.3390/nu14163325.

[54] R. Troncone and V. Discepolo. “Celiac disease and autoimmunity”. In: *J. Pediatr. Gastroenterol. Nutr.* 59 (2014), S9–S11. DOI: 10.1097/01.mpg.0000450394.30780.ea.

[55] L. Greco et al. “Burden of celiac disease in the Mediterranean area”. In: *World J. Gastroenterol.* 17 (2011), pp. 4971–4978. DOI: 10.3748/wjg.v17.i45.4971.