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Growth Hormone Deficiency Associated with Celiac Disease Revealed by Persistent Growth Failure Despite a Gluten-Free Diet: A Retrospective Case Series

Gorgi K[1] and Chaouche M[2]*

[1]*Department of Endocrinology-Diabetology, Ibn Sina University Hospital, Rabat, Morocco*

[2]*Department of Dermatology, Mohammed VI University Hospital, Agadir, Morocco*

Abstract

Introduction: Celiac disease (CD) is an autoimmune enteropathy frequently associated with growth failure. A gluten-free diet (GFD) usually allows catch-up growth. Lack of response suggests an associated condition, particularly growth hormone (GH) deficiency.

Objective: To describe the clinical, biological, and outcome characteristics of patients with CD associated with GH deficiency.

Methods: This retrospective study included patients with confirmed CD and GH deficiency documented by dynamic testing, with failure of catch-up growth after at least 12 months of strict GFD.

Results: Seven patients (6 males, 1 female), with a mean age of 14 years (range 7–19 years), were included. Severe growth failure (< −3 SDS) was present in 71% of cases. No catch-up growth occurred despite adherence to a strict GFD. GH deficiency was partial in 6 cases and complete in 1 case. Pituitary MRI was normal in all patients, consistent with idiopathic GH deficiency. GH therapy resulted in a mean height gain of +1.5 SDS.

Conclusion: GH deficiency should be investigated in patients with persistent growth failure despite a gluten-free diet.

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Correspondence:
Khaoula Gorgi, Department of Endocrinology-Diabetology, Ibn Sina University Hospital, Rabat, Morocco, Tel: 0615591874; E-mail: khaoulagorgi@gmail.com

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Introduction

Celiac disease is an immune-mediated disorder triggered by gluten ingestion in genetically predisposed individuals, characterized by intestinal mucosal atrophy [1].

Growth failure may be the only clinical manifestation in children, even in the absence of gastrointestinal symptoms [2].

A strict gluten-free diet usually leads to rapid catch-up growth, particularly within the first year after diagnosis [3]. However, persistent growth failure despite good adherence and serological normalization should prompt investigation for associated conditions, particularly growth hormone deficiency (GH) [4].

Materials and Methods

Study design

This was a retrospective monocentric descriptive study.

Study population

Patients followed for growth failure with:

- Confirmed CD (serology ± histology).
- Confirmed GH deficiency by dynamic testing.

Inclusion criteria

- Height < −2 SDS.
- Positive anti-transglutaminase antibodies.

- No catch-up growth after ≥12 months of strict GFD.
- Serological normalization.

Endocrine evaluation

GH deficiency was assessed using:

- Insulin tolerance test.
- Glucagon–propranolol test.
- Pituitary MRI performed in all patients.

Diagnosis was established according to Growth Hormone Research Society guidelines.

Data collected

- Anthropometric data.
- Severity of growth failure.
- Type of GH deficiency.
- Treatment response.

Results

Seven patients were included (6 males, 1 female), with a mean age of 14 years (range 7–19 years).

Severe growth failure (< −3 SDS) was observed in 5 patients (71%).

All patients were on a strict GFD with confirmed serological normalization.

No catch-up growth was observed.

GH deficiency was:

- Partial in 6 cases.
- Complete in 1 case.

Pituitary MRI was normal in all patients, confirming idiopathic GH deficiency.

GH therapy resulted in a mean height gain of +1.5 SDS.

See Figures 1-2 and Table 1.

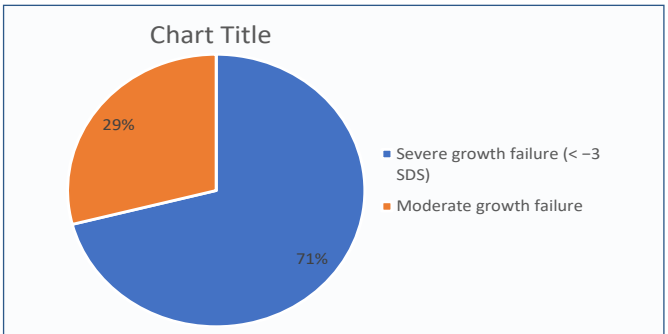

Figure 1: Severity distribution of growth failure in patients.

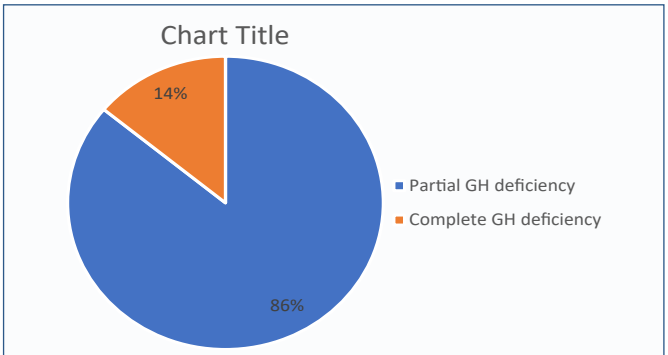

Figure 2: Distribution of GH deficiency type.

Table 1: Individual response to GH therapy.

Patient	SDS before GH	SDS after GH	Gain (SDS)
P1	-3.5	-2.0	+1.5
P2	-3.2	-1.8	+1.4
P3	-3.8	-2.1	+1.7
P4	-3.6	-1.9	+1.7
P5	-3.4	-2.0	+1.4
P6	-2.8	-1.3	+1.5
P7	-2.6	-1.2	+1.4

Discussion

Growth failure is one of the most common extraintestinal manifestations of celiac disease (CD), reported in 10–40% of children at diagnosis [2]. It mainly results from chronic malabsorption, nutritional deficiencies, and persistent systemic inflammation.

A strict gluten-free diet (GFD) usually leads to significant improvement in growth, with catch-up growth typically observed within 6–12 months after diagnosis [3]. Therefore, absence of growth recovery beyond this period should be considered abnormal and requires further investigation.

In our series, all patients showed persistent growth failure despite strict adherence to a GFD and normalization of anti-transglutaminase antibodies, suggesting an additional underlying condition independent of intestinal malabsorption.

Several mechanisms may explain the association between CD and impairment of the GH–IGF-1 axis. Chronic malnutrition and intestinal inflammation may reduce IGF-1 production and induce peripheral GH resistance, leading to a functional and reversible deficiency under dietary treatment [5]. Conversely, true organic GH deficiency may coexist, related to hypothalamic–pituitary abnormalities or autoimmune mechanisms that are not yet fully understood [6].

In our study, dynamic hormonal evaluation confirmed GH deficiency in all patients, and pituitary MRI was normal in all cases. These findings support a diagnosis of idiopathic GH deficiency, excluding structural pituitary abnormalities.

This association remains rare but is likely underdiagnosed in pediatric populations [7]. Growth failure attributed solely to CD may mask an underlying endocrine disorder, delaying diagnosis.

We observed a predominance of partial GH deficiency, consistent with published data. This form may represent an intermediate stage between functional impairment related to CD and true hormonal deficiency.

Recombinant GH therapy resulted in significant improvement in growth velocity and height SDS in all patients, with a mean gain of approximately +1.5 SDS. These findings are consistent with previous reports showing the efficacy and safety of GH therapy in

CD-associated GH deficiency without worsening disease activity [8].

This rare but clinically important association highlights the need to:

- Closely monitor growth after initiation of GFD.
- Confirm dietary adherence and serological normalization.
- Perform endocrine evaluation in case of growth failure.
- Avoid attributing growth failure solely to CD.

Conclusion

Growth hormone deficiency should be investigated in patients with celiac disease presenting persistent growth failure despite a well-conducted gluten-free diet.

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