Scientific Reports in Medicine, 2025; 2(3): 120-126

Scientific Reports in Medicine

Research Article

Clinical characteristics and treatment outcomes of growth hormone therapy in pediatric patients: a single-center experience and review of current literature

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DOI: 10.37609/srinmed.55

Abstract

Objective: Recombinant human growth hormone (rhGH) became available for clinical trials, and growth hormone deficiency has been treated more safely and effectively since 1985. Growth hormone therapy enables patients to attain an adult height consistent with their genetic target height.

The present study was undertaken to evaluate patients' characteristics, their response to therapy, factors influencing outcome, and side effects of treatment.

Method: A retrospective file review was conducted. Out of 149 patients followed up with a GHD diagnosis, 92 patients whose files were accessible and who received treatment for at least 1 year were included in the study. Patients' chronological ages, ages at diagnosis, sex, pretreatment bone ages (BA), pubertal stages, annual growth velocities, auxological data during treatment, and side effects were recorded.

Results: Age at diagnosis, sex, etiologic distributions, and auxological data were found to be similar to the results of Western countries. Height gain was found to be significant in the first year of treatment, and then growth velocity declined gradually. Age at the initiation of GH treatment has been shown to be negatively correlated with the response to therapy, emphasizing the need for early diagnosis and treatment of the condition. No significant adverse effects were observed throughout the 16-year follow-up period.

Conclusion: Early detection of pathological short stature, particularly Growth Hormone Deficiency (GHD), and the initiation of treatment at an early stage, lead to a superior final height outcome. Patients with GHD can be treated both effectively and safely with recombinant growth hormone therapy.

Keywords: Growth hormone, child, growth hormone replacement therapy.

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Recieved: 27.10.2025
Accepted: 15.12.2025

INTRODUCTION

Growth represents one of the most distinctive features of childhood. Evaluating growth is important as it is a sensitive indicator of a child's health, nutrition, and genetic makeup (1). Short stature is a common problem encountered in clinical practice. The prevalence of short stature associated with Growth Hormone Deficiency (GHD) is estimated to be approximately 1:4000 to 1:10000. In studies conducted in various clinics regarding the etiology of short stature, GHD constitutes 7-23% of cases presenting with short stature (2). GHD should be considered particularly in children with severe short stature (height below 3 SD or the 1st percentile for age and sex, those with significantly low growth velocity (below 5-10th percentile), those whose height is between 2 and 3 SD but whose growth velocity has decreased, children with hypoglycemia, micropenis, a history of Central Nervous System (CNS) tumors and radiation or surgery in its treatment, and the presence of congenital or acquired other pituitary hormone deficiencies (1).GHD may occur as an isolated problem or accompany other pituitary hormone deficiencies (most frequently TSH deficiency, less frequently prolactin, gonadotropin, and ACTH deficiencies) (3). According to data from approximately 100,000 patients from 4 major growth hormone study groups worldwide (National Cooperative Growth Study of Genentech (NCGS), Kabi Farmacia International Growth Study (KIGS), Australasian Pediatric Endocrine Group Database (OZGROW), International Cooperative Growth Study in Japan (ICGS)) between 1997-1999, 40-61% of patients using GH treatment had idiopathic GHD, 13-16% had organic causes (multiple pituitary hormone deficiency), 14-40% had idiopathic short stature, 4-18% had Turner syndrome, 1-6% had chronic kidney failure (CKF), and 1-4% had other causes (4). The 2006 data from KIGS, comprising 55,000 patients, reported idiopathic GHD as 51%, organic GHD as 36%, and other causes as 13% (5). The clinical characteristics of patients with GHD are quite heterogeneous, depending on the etiology, the age of onset of the deficiency, and accompanying other hormone deficiencies and diseases. However, GH treatment can ensure that these patients reach adult height consistent with their target height (6). GH treatment has a history of 70 years, In 1985, biosynthetic GH with a polypeptide structure obtained by recombinant DNA technology was introduced (7,8). Due to its safer and more effective nature, the use of GH has become increasingly widespread. In addition to other diseases that cause growth retardation even without GH deficiency, such as idiopathic short stature, Turner syndrome, chronic kidney failure (CKF), and intrauterine growth retardation, the use of GH in adults with GHD has also become relevant due to its metabolic effects. However, significant differences in practice regarding the diagnosis and treatment of GHD have been observed, leading to the publication of various national and international consensus reports. In our country, a consensus report has also been prepared by the National Pediatric Endocrinology Society for clinical and auxological findings in the diagnosis of GHD, appropriate laboratory tests, GH doses used in treatment, factors affecting treatment, aspects to consider during follow-up, and indications for discontinuing GH treatment (9).Our study aimed to evaluate the demographic, clinical characteristics, adherence and response to treatment, and side effects of patients diagnosed with GHD and treated with GH replacement therapy in our clinic.

METHOD

Ethical permission was obtained from the Akdeniz University, Medical Faculty Research Ethics Committee for this study with date 2007/number 183, Medical records of patients receiving GH treatment at Akdeniz University Faculty of Medicine Pediatric Endocrinology Clinic were retrospectively scanned. Data collected included patients' chronological ages (CA), ages at diagnosis, sex, pretreatment bone ages (BA), height measurements, parental heights, pubertal stages, pretreatment annual growth velocities, bone ages and auxological data during and after treatment, and any arising side effects and dose changes were recorded. Between 1991 and 2007, 149 patients followed up with a GHD

diagnosis were identified, and 92 patients whose files were accessible and who received treatment for at least 1 year were included in the study.

Statistical Method: Results were expressed as mean ± SD. Statistical analyses were performed using SPSS version 14.0 (SPSS Inc., Chicago, IL, USA). One Way ANOVA (parametric) and Kruskal-Wallis (non-parametric) tests were used for comparison of three groups, while Independent samples t-test and Mann-Whitney U test were used to show pairwise differences between 3 groups and/or compare two groups, and Wilcoxon Signed-Rank test was used for paired comparison. $P<0.05$ was considered a significant difference, but due to the changing alpha value in pairwise comparisons, Bonferroni correction was applied, and alpha=0.017 was accepted as significant. Spearman correlation test was used for evaluating correlations.

RESULTS

Of the 92 patients included in the study, 30 were female (33%), 52 were male (56%), and 10 patients (11%) were diagnosed with Turner syndrome. According to the etiologies of GHD, 73% of the patients were evaluated as idiopathic GHD, while 27% were found to have organic GHD. 64 cases (69%) were diagnosed with isolated GHD (IGHD) (89.1% idiopathic, 10.9% organic), while 18 cases (20%) were diagnosed with multiple pituitary hormone deficiency (MPHD) (9.1% idiopathic, 89% organic). Among the 64 cases with IGHD, pituitary hypoplasia was detected in the MRI of only 7 (10.9%), while no pathology was found in the pituitary MRIs of the others. Among the 18 patients with MPHD, pituitary MRI was normal in 2 (11%), while 1 patient had partial empty sella, 3 cases had empty sella, 2 had ectopic neurohypophysis, and 10 cases had pituitary hypoplasia. PIT-1 mutation was detected in two siblings, and pituitary hypoplasia was also present in these patients. Magnetic Resonance Imaging (MRI) findings according to the patients' diagnoses are shown in Figure 1. The mean age at initiation of treatment was 10.4 ± 3.59 years, and the mean duration of treatment was 2.8 ± 1.9 years. Pretreatment auxological data of the patients are summarized in Table 1. While the pretreatment annual growth velocity of all cases was 3.47 ± 1.7 cm, it was found to be 8.88 ± 3.69cm, 5.89 ± 1.99 cm, and 4.75±1.09 cm in the 1st, 2nd, and 3rd years of treatment, respectively, with the fastest growth occurring in the 1st year of treatment. The growth velocities of the patients during treatment are shown in Figure 2. In Turner syndrome patients, the age at initiation of treatment was 9.78 ±3.7 years, the mean duration of treatment was 3.4 ± 2.3 years, and while the pretreatment annual growth velocity was 2.57 ±1.7 cm, the fastest growth after treatment was 7.86 ± 2.39cm, again in the 1st year. The mean duration of treatment for the 10 patients (1 with Turner syndrome) who reached final height was 5.09 ±2.94 years. The pretreatment height SDS of the patient with Turner syndrome reached -2.1 after treatment, from -4.05. The pretreatment height SDS of the other patients reached -1.30 ± 0.86, from -4.35 ±2.1. The characteristics of the patients who reached final height are shown in Table 2. Side effects developed in only 2 of the 92 patients; 1 patient had elevated liver transaminases (5 times the normal limit), and 1 patient had insulin resistance, leading to temporary discontinuation of treatment and subsequent continuation.

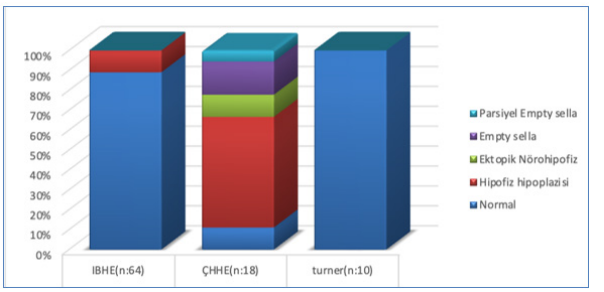

Figure 1. Magnetic Resonance Imaging (MRI) findings according to the patients' diagnose

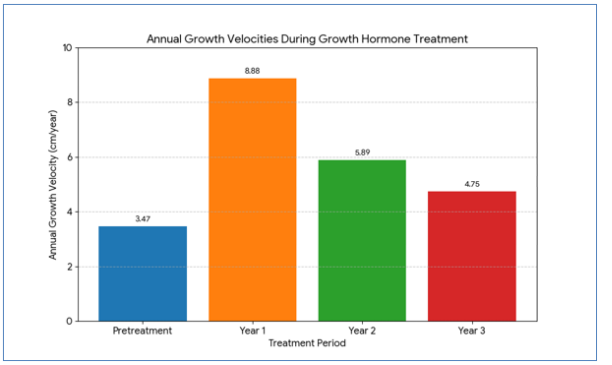

Figure 2. Growth velocities of the patients during treatment

DISCUSSION

In our study, the characteristics of 92 patients who had been receiving regular treatment for at least 1 year with a diagnosis of GHD were evaluated. When patients were evaluated according to sex, the male/female ratio was 1.73. The male/female ratio for patients with GHD was reported to be 1.3 in the USA, 2.1 for idiopathic GHD according to KIGS, and 1.3 for organic causes (10). The higher prevalence observed in males has been attributed to both genetic factors and sociocultural influences. However, the view that social acceptability, rather than genetic predisposition, creates the male sex tendency is dominant (5). Our male/female ratio was found to be higher than in the USA but similar to European countries. According to the etiologies, 73% of the patients were evaluated as idiopathic GHD, while 27% were found to have organic GHD. 64 cases (69%) were diagnosed with isolated GHD (IGHD) (89.1% idiopathic, 10.9% organic), while 18 cases (20%) were diagnosed with multiple pituitary hormone deficiency (MPHD) (11% idiopathic, 89% organic), and 10 cases (11%) were diagnosed with Turner syndrome (3 of whom had mosaic Turner). A multicenter study conducted in our country with data from 70 patients with GHD who reached final height found that 26% of the patients had MPHD (72.2% organic, 27.8% idiopathic), and 74% had IGHD (7.7% organic, 92.3% idiopathic) (11). A study conducted in Canada in 2006 found idiopathic GHD to be 65.6% and organic GHD to be 34.4% (12). In England, idiopathic GHD was reported to constitute 68% of patients treated for GHD (13). Our diagnostic distribution was found to be similar to studies in our country as well as in Canada and England. In our study, pituitary pathology (pituitary hypoplasia) was detected in the MRI of only 7% (10.9%) of cases with IGHD, while pituitary pathology (1 patient partial empty sella, 3 cases empty sella, 2 ectopic neurohypophysis, 10 cases pituitary hypoplasia) was detected in 16% (89%) of the 18 patients with MPHD. Published reports regarding this issue show that pituitary pathology is found in 10-50% of patients with IGHD in MRI, while pituitary pathology is present in 52-93% of patients with MPHD (14-16). Our MRI findings were consistent with these studies. The mean age at diagnosis of our cases was 10.40±3.59 years. The mean age at diagnosis of cases with MPHD (8.38 ± 3.55) was found to be smaller than that of patients with IGHD (10.1± 2.15), although this was not statistically significant (p=0.11). The mean height of our patients at the start of treatment was 119.3 ± 18.53 cm (height SDS -3.42±1.29). When growth velocities were evaluated as a response to treatment, it was found that the mean growth velocity in the first year with GH treatment was 9.88 ±2.66 cm, which was about 2.5 times higher than the pretreatment growth velocity, and then gradually decreased in subsequent years. First-year growth velocity is important because it is a determinant of final height and better treatment outcomes. According to the literature, first-year growth velocity is reported to be between 8-13 cm/year, and growth velocity decreases in subsequent years (17,18). This decrease in growth velocity is independent of the treatment dose and can be shown to be due to the resolution of the GHD condition and adaptation to normal growth velocity, not insufficient treatment (19). In this study, the mean duration of treatment for the 10 patients (8 IGHD, 2 MPHD) who reached final height was found to be 5.09 ± 2.94 years. While their pretreatment mean height was 114.5 ± 22.78 cm, their posttreatment final height was 162 ± 17.99 cm (final height SDS -1.30 ± 0.86), and the Deltaheight SDS was 2.49 ±1.20. The data from the KIGS and NCGS study groups reported a treatment duration of 5-8.1 years, final height SDS of -1.3, and Deltaheight SDS of +1.4-1.7 (20). Our findings are seen to be similar. The key indicator of successful GH therapy is the final height achieved by the patients. Reaching the final height within the target height range is possible with GH replacement therapy (9,21). Factors affecting final height have been evaluated in various studies in the literature. A positive correlation was reported between height at the start of treatment, patients' genetic height potential (target height), duration of treatment, and first-year growth velocities with

final height. The major determinant of final height is stated to be the child's genetic potential, i.e., target height, and that a longer pretreatment height and longer duration of treatment may also provide a taller final height (10,12,22).Starting treatment early, administering treatment for a longer period, and having a high height gain in the first year lead to a better final height (9). The most important proven side effect of GH treatment so far is Creutzfeldt-Jakob disease observed in those treated with GH prepared with human pituitary extract. This side effect has been eliminated with the use of recombinant GH. Numerous review studies have been conducted on potential serious side effects of GH treatment in terms of safety and efficacy, such as malignancy development and recurrence, intracranial hypertension (IH), metabolic side effects, orthopedic problems, stroke, and increased mortality rate (23-27). According to the 2022 KIGS data consisting of approximately 84,000 patients, the most frequently observed side effects in cases receiving GH were non-specific findings such as upper respiratory tract infection, fever, gastroenteritis, headache, and muscle pain, while scoliosis was reported as the second most frequent. It was thought that GH and IGF-1 could increase the risk of malignancy due to their growth-promoting and mitogenic effects. Recurrences have been observed in patients with craniopharyngioma, but a direct causal relationship with treatment could not be demonstrated (24). In patients with intracranial tumors, relapses were reported in 5-10% with an average of 2 years of GH treatment, but the risk was not observed to be increased compared to patients not using GH. It was reported that the incidence of type 1 diabetes was not higher than in the general population, and although the incidence of type 2 diabetes was higher than expected, it was thought to be probably due to previous patient predisposition (24,28). Slipped capital femoral epiphysis, increased intracranial pressure, convulsions, and gynecomastia have been reported. However, the direct relationship of these findings with GH has not been definitively demonstrated (9). In our study, insulin resistance was detected for the first time in only 2 of the 92 patients (treatment was restarted after insulin resistance returned to normal limits after a 1-month break), and no other serious side effect was observed.

CONCLUSION

Early detection of pathological short stature, especially GHD, and the initiation of early treatment, resulting in a longer duration of treatment, lead to a better final height outcome. Recombinant growth hormone therapy is both an effective and safe treatment option for patients with growth hormone deficiency.

Table 1. Pretreatment Auxological Data of the Patients

Data	Total (n=92)	Male	Female	Turner	p
Age(year)	10.43±3.59 (1.5-17.6)	10.61±3.91 (1.5-17)	10,35±3.02 (5-17.6)	9.78±3.7 (4.7-14.9)	0.794
Height (cm)	119.3±18.53 (62-147)	120.76±19.95 (62-147)	118.87±16.96 (90-145)	112.94±15.19 (90.5-134)	0.269
Height SDS	-3.42±1.29	-3.45±1.18	-3.34±1.53	-3.52±1.18	0.973
PH (cm)	157.14±8.68 (140-177)	161.33±6.64 (150-177)	152.8±8.24 (140-169)	151.29±7.63 (142.2-168)	<0.01*
TH (cm)	162.1±7.36 (148-177)	166.42±5.09 (155.3-177)	156±6.2 (148-175.1)	157.8±5.1 (152-166)	<0.01*
BA	7.87±3.55	7.6±3.6	8.3±3.4	7.8±3.73	0.707

*p < .005, SDS = standard deviation score, PH=Predicted Height, TH=Target Height, BA=Bone Age

Table 2. Characteristics of Patients at Final Height								
Male (6)					**Female (4)**			
	Mean	**SDS**	**Median**	**Range**	**Mean**	**SDS**	**Median**	**Range**
Age(years)	9.97	4.02	10.5	1.6-16.6	9	3.92	8.5	5-14
Treatment (years)	5.09	2,94	4	2-11	5.5	2.08	5.5	3-8
Pretreatment Height (cm)	114.5	22.78	120	62-140	117	21.02	117.5	94-139
Pretreatment HSDS	-4.35	2.1	-3.33	-9.67-2.65	-3.52	1.18	-3.33	-5.13-2.29
Final Height (cm)	166.6	17.99	168	157-175	158.5	6.03	158,5	152-165
Final HSDS	-1.3	0,86	-1,81	-2.10 -0.23	-1.17	0.75	-1.15	-2.1-0.3
ΔHSDS	2.49	1.2	2.46	0.92- 3.93	2.22	1.42	1.85	1-4.2
SDS = standard deviation score, PH=Predicted Height, TH=Target Height								

Acknowledgement

Peer-Review

Double blind both externally and Internally Peer Reviewed

Conflict of Interest

The authors declare that they have no conflict of interest regarding content of this article.

Financial Support

The Authors report no financial support regarding content of this article.

Ethical Declaration

Ethical permission was obtained from the Akdeniz University, Medical Faculty Research Ethics Committee for this study with date 2007/number 183, and Helsinki Declaration rules were followed to conduct this study.

Authorship Contributions

Concept: ÖU, Design: ÖU, BI, Supervising: BI, Data collection and entry: ÖU, Analysis and interpretation: ÖU, Literature search: ÖU, Writing: ÖU, Critical review: ÖU

Thesis?

This study was prepared by rearrangement of the specialty thesis by Ulaş Özdemir, entitled as "Akdeniz Üniversitesi Tıp Fakültesi Pediatrik Endokrinoloji Bilim Dalında 1991-2007 Yılları Arasında Büyüme Hormonu Replasman Tedavisi Alan Hastaların Değerlendirilmesi".

Is previously presented?

Some part of this study was presented as oral/poster presentation at "Congress of 18th International Eastern Mediterranean Family Medicine Congress 25 – 28 April, 2019 – Adana Divan Hotel, Türkiye"

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