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(REVIEW ARTICLE)

GROWTH HORMONE DEFICIENCY AND CRANIOFACIAL DEVELOPMENT: IMPLICATIONS FOR ORTHODONTIC TREATMENT TIMING - A NARRATIVE REVIEW

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ABSTRACT

Background: Growth hormone deficiency (GHD) is the most common pituitary hormone deficiency in children and a principal cause of short stature. Because it manifests during the period of active craniofacial growth, it is directly relevant to orthodontic and dentofacial orthopedic decision-making.

Objective: This narrative review synthesises evidence published between 2016 and 2026 on craniofacial and dental characteristics in children with GHD and evaluates the effect of recombinant human growth hormone (rhGH) replacement on craniofacial morphology and its implications for the timing of orthodontic intervention.

Methods: Secondary data were obtained from PubMed, Scopus, ScienceDirect, and Google Scholar using Boolean combinations of terms relating to GHD, GH therapy, craniofacial and mandibular growth, skeletal maturation, dental development, malocclusion, and orthodontic timing. English-language articles published from 2016 to 2026 were considered.

Results and Discussion: GH acts principally through IGF-1 on the mandibular condylar secondary cartilage, producing effects that are dimension-specific: sagittal and vertical mandibular deficiency, with transverse and arch-length involvement of the maxilla. GHD is associated with reduced cranial base length, a shortened and retropositioned mandible, reduced ramus height, and altered facial height proportions, with Angle Class II malocclusion reported in up to 31% of patients in a systematic review covering 465 children with GHD. Dental maturation and eruption are delayed, and bone age lags further still. rhGH partially but incompletely normalises this phenotype, the mandible responding preferentially in the sagittal and vertical planes while dental maturation and transverse dimensions lag behind.

Conclusion: Chronological and dental age are unreliable timing criteria in GHD. Orthodontic and dentofacial orthopedic treatment in this population should instead be timed according to individualised skeletal-maturation assessment, coordinated with the child's ongoing endocrine treatment.

Keywords: Growth Hormone Deficiency; Craniofacial Growth; Mandibular Condyle; Skeletal Maturation; Orthodontic Treatment Timing; Recombinant Human Growth Hormone.

1 INTRODUCTION

Growth hormone deficiency, also known as GHD, is an endocrine disorder and is the most commonly found pituitary hormone deficiency, which contributes as one of the main causes of short stature in pediatric populations due to an insufficient production of the growth hormone (GH) which directly causes an impaired overall growth and musculoskeletal development[1,2]. GHD is considered a rare disorder, although published estimates of its frequency vary considerably. A commonly cited figure places the worldwide prevalence at approximately 1 in 4,000 to 1 in 10,000 live births[2], whereas the only systematic review of pediatric GHD epidemiology published to date reported a wider prevalence range of 1:1,107 to 1:8,646 across nine studies, with mean incidence estimates ranging from 1:28,800 to 1:46,700 cases per year[1]. This discrepancy is attributable largely to heterogeneity in diagnostic criteria, case definitions, and study populations, and to the fact that the available data are drawn predominantly from European cohorts; no single global estimate can therefore be regarded as definitive at present. Irrespective of the exact figure, GHD remains the most common pituitary hormone deficiency in children and is consistently identified in pediatric endocrinology clinics worldwide[1]. This population is simultaneously undergoing active craniofacial growth and development, a period highly relevant to orthodontic clinical decision-making.

The GH/IGF axis, which constitutes the regulation of GH, plays a strong influence in the growth and metabolism of both craniofacial bone and dental tissues. Those with disturbances in the GH/IGF axis, namely patients with dwarfism or acromegaly, have been reported to exhibit dysmorphology in their teeth and craniofacial bone structures. These findings are also in line with the claim that GH is closely associated with dental maturity, alterations in tooth and root dimensions, as well as mandibular morphogenesis. Studies regarding genetic polymorphisms in GH receptors are also shown to contribute to developmental defects of dental enamel[3].

Clinical evidence shows that children with GHD exhibit craniofacial morphology that consistently differs from healthy children, in which all craniofacial structures (such as cranial base length, maxillary and mandibular corpus length, as well as both anterior and posterior facial height), had a reduced length, as well as a tendency towards retrognathic facial type[4]. It has been observed that the combination of these characteristics contributes to a high prevalence of Angle Class II malocclusion within the GHD population, which was reported to affect up to 31% of cases in one large systematic review encompassing 465 patients[5]. Regarding conditions of GH excess such as acromegaly, an inverse craniofacial phenotype is produced that is dominated by mandibular overgrowth, prognathism, and disruptions in the relationship between dental arches, which may lead to open bites or crossbites[6]. These findings reinforce the claim that GH activity shapes the maxillomandibular relationship in a bidirectional manner as deviations from normal GH secretion or receptor function are understood as a legitimate cause of malocclusion.

Furthermore, recent studies have also shown that delayed dental maturity is frequently exhibited by GHD patients, which complicates how dental age indicators are interpreted by orthodontists when determining optimal treatment timing[5]. Contemporary orthodontic and dentofacial orthopedic practice depends heavily on correctly timing intervention relative to a patient's individual biological growth status rather than the patients' chronological age alone, since functional and orthopedic appliances are reliably effective only when applied during specific pre-pubertal, pubertal, or post-pubertal growth windows[7,8]. As a result, typical protocols used for the general population may not be applicable to people with GHD, and clinicians risk either missing the true growth window or misjudging a patient's remaining growth potential if only chronological age is used as a treatment-timing criterion.

Therefore, this narrative review aims to: (1) synthesize current evidence (papers published from 2016–2026) on craniofacial growth characteristics in children with GHD, and (2) evaluate evidence on the effects of GH replacement therapy on craniofacial morphology and its implications for orthodontic treatment timing.

2 METHODS

The type of research used was a narrative literature review. The data for this study were sourced from several online scientific journal articles obtained from databases such as PubMed, Scopus, ScienceDirect, and Google Scholar. Search terms were combined using Boolean operators (AND/OR) and included variations and combinations of: "growth hormone deficiency", "growth hormone therapy", "craniofacial development", "craniofacial growth", "mandibular growth", "GH/IGF-1 axis", "skeletal maturation", "dento-alveolar", "tooth development", "malocclusion", "functional appliances", "orthodontic treatment", and "orthodontic timing".

Inclusion criteria were also applied in selecting the literatures, including: (a) were published in English between 2016 and 2026, (b) involved studies related to populations diagnosed with GHD or hypopituitarism, (b) reported outcomes related to craniofacial morphology, cephalometric measurements, dental development, or orthodontic treatment considerations, and (d) were published as a systematic review, cohort, case-control, cross-sectional study, or case series or report providing clinically relevant detail.

In addition, several exclusion criteria were also applied, including: (a) any article published before 2016, (b) any article published in a language other than English, (c) were conducted exclusively on animal or in vitro models without translational discussion relevant to human craniofacial or dental development, (d) reported GH therapy outcomes with no relevance to craniofacial structures, dental development, or growth timing, (e) were conference abstracts, editorials, or commentaries without original data or substantive review content, or (f) were not accessible in full text.

3 RESULTS AND DISCUSSION

3.1 Growth Hormone and Its Mechanistic Role in Craniofacial Growth

GH primarily influences overall skeletal development indirectly through the synthesis of IGF-1. However, direct GH-receptor-mediated actions on peripheral tissues have also been described as GH plays a role in bone remodelling and maturation, as well as linear bone growth[9]. Within the mandible, the condylar cartilage is a secondary cartilage that continues to proliferate throughout childhood and adolescence and is under substantial IGF-1 control. An experimental work using local IGF-1 delivery into the temporomandibular joint of juvenile rats results in measurable increases in mandibular length and bone quality. An experimental study delivering IGF-1 locally into the temporomandibular joint of juvenile rats produced measurable increases in mandibular length and bone quality[10]. Although species differences prevent direct application to humans, these findings explain how IGF-1 promotes postnatal mandibular elongation. This mechanism helps clarify why individuals with GH or IGF-1 deficiency often experience mandibular deficiency.

This condyle-specific sensitivity to IGF-1 offers a biological explanation for why the mandible is typically the structure most affected rather than the maxilla, and later during treatment, most responsive in states of GH excess or deficiency[5]. A single case report described enhanced mandibular growth and a more prognathic mandibular position when fixed functional appliance therapy was combined with GH administration in an adolescent without GHD[11]. This finding is consistent with a broader body of evidence reviewing the effects of hormonal regulation towards the development of mandibular condylar cartilage, in which GH and IGF-I, alongside sex steroids and other endocrine mediators, were found to greatly influence the rate and magnitude of condylar growth response to functional appliance therapy[12].

Beyond the mandible, GH and IGF-1 also regulate the growth of the dental arches and the mineralized structures of the teeth. Adequate GH and IGF-1 signaling directly influences arch dimensions and root and crown formation, where deficient signaling was found to cause measurable reductions in arch size[5]. These mechanisms give rise to an important distinction. The lower jaw and the midface not only exhibit different levels of sensitivity to GH, but also grow through different mechanisms and are therefore affected in different ways. The length of the mandible and the height of the ramus depend on endochondral proliferation in the secondary condylar cartilage, which is a process under the direct control of IGF-1[10,12], whereas the dimensions of the maxilla and dental arch depend heavily on suture apposition and surface remodeling[3,5].

These distinctions become crucial in understanding how skeletal and dental responses differ among GHD patients. Collectively, the various mechanisms that contribute to craniofacial growth, growth hormone release, condylar cartilage development, and dentoalveolar development are all disrupted by GHD, which explains why the craniofacial structure is significantly impacted.

3.2 The Craniofacial and Dental Phenotype of Growth Hormone Deficiency

Among the various causes of short stature, consistent craniofacial patterns have been reported in GHD. Patients with GHD often exhibit a reduced cranial base length, a smaller and more posteriorly positioned lower jaw with reduced ramus height, an obtuse gonial angle, and a reduction in facial height in both the anterior and posterior regions[13]. Cephalometric studies also found children with GHD with a smaller and more retrusive mandible with a retrognathic maxilla, reduced anterior facial height, and a generally underdeveloped facial skeleton. Additionally, lower facial height is increased relative to posterior facial height, showing a consistent pattern of craniofacial features regardless of the cause of short stature[14].

Consistent with this dimension-specific pattern, the picture at the dental arches differs from that of the sagittal skeletal profile. In adults with severe congenital isolated GH deficiency (IGHD), all maxillary arch measurements were reduced relative to controls, whereas among mandibular arches only canine width and arch length were affected, suggesting that the maxillary arch is disproportionately vulnerable to chronic GH/IGF-I insufficiency[15]. Furthermore, studies found that in adults with severe congenital isolated GH deficiency (IGHD), all maxillary arch measurements were reduced relative to controls, whereas among mandibular arches only canine width and arch length were affected, suggesting that the maxillary arch is disproportionately vulnerable to chronic GH/IGF-I insufficiency despite the mandibular predominance seen in cephalometric studies of children with GHD[13,14].

Functional consequences of these skeletal changes extend to the airway and to speech production, where adults with lifelong untreated isolated GHD demonstrate altered vowel formant frequencies that correlate more strongly with cephalometric craniofacial measurements than with pharyngeal airway width itself, indicating that the reduced size of the oral cavity, rather than airway narrowing, underlies the observed voice changes[16]. At the level of occlusion and dental development, children with GHD commonly show a discrepancy between chronological, skeletal, and dental age, with dental maturation typically delayed relative to chronological age and bone age delayed even further, alongside a higher prevalence of malocclusion traits such as increased overjet, Class II, and crowding tendencies[17].

Tooth eruption itself also appears to be affected, in which a recent case-control study found a significant delay in the eruption of incisors and first permanent molars during the early mixed-dentition stage in GHD and idiopathic short-statured children prior to treatment, compared with healthy controls of matched age and sex[18]. Beyond timing, structural dental anomalies, including developmental enamel defects and disturbances of tooth number and size (such as microdontia and hypodontia), have also been documented at a higher frequency among GHD patients than among healthy peers, suggesting that GH influences not only the pace of dental development but also aspects of tooth morphogenesis itself[19].

3.3 Effect of Growth Hormone Replacement Therapy on Craniofacial Development

Recombinant human growth hormone (rhGH) replacement therapy has been shown to partially reverse the craniofacial deficits associated with GHD, although the degree and pattern of catch-up growth vary depending on the timing, duration, and dose of treatment as well as the underlying aetiology of short stature. Comparative cephalometric studies show that children receiving GH treatment tend to display more favorable craniofacial dimensions, exhibiting decreased facial convexity, increased mandibular length and posterior facial height compared with untreated children, suggesting a genuine treatment effect on skeletal and dental development[13]. It has also been found that rhGH therapy produced clinically meaningful increases in mandibular length, ramal length, anterior facial height, and anterior cranial base length, while transverse maxillary and mandibular arch dimensions changed comparatively little, indicating that rhGH's craniofacial effect is most pronounced in the sagittal and vertical dimensions of the mandible rather than in arch width[20].

Direct clinical evidence of GH's local skeletal effect comes from a case report combining fixed functional appliance therapy with GH administration in an adolescent without GHD, in which the mandible showed substantial growth during combined treatment that remained stable through nearly three years of retention, illustrating that exogenous GH can meaningfully augment the endochondral growth response of the mandibular condyle beyond what functional appliances alone typically achieve[11]. This observation also aligns with a broader review of nutritional and hormonal influences on functional appliance outcomes, which concludes that GH and other hormonal factors modulate the magnitude of condylar and mandibular growth response to treatment, and that hormonal status should be considered when counseling patients on expected outcomes of growth-modification therapy[12]. Notably, the degree of craniofacial catch-up appears to depend on treatment duration. Children who have received prolonged rhGH therapy show more pronounced increases in craniofacial skeletal dimensions, particularly of the maxilla and mandibular ramus, than those treated for shorter periods, supporting the view that GH's craniofacial effect accumulates progressively rather than occurring as an early, one-off catch-up[5].

Dental age and tooth eruption, in contrast, appear to be less responsive to rhGH compared with skeletal parameters. Several studies found that only slight improvement is present in dental maturation compared with relatively substantial gains seen in mandibular length[20]. Taken together, this body of evidence points that rhGH therapy partially, but incompletely, normalizes the craniofacial skeleton in GHD, with the mandible responding preferentially in the sagittal and vertical dimensions, while dental maturation and transverse jaw dimensions lag behind. Such asymmetry in responsiveness to rhGH treatment is directly relevant to orthodontic planning, because it implies that a GHD patient's occlusal and skeletal relationships may continue to change meaningfully for as long as rhGH therapy continues, independent of the patient's chronological age.

3.4 Implications for Orthodontic Treatment Timing

Clinical evidence regarding GH and craniofacial growth has direct implications for determining the optimal timing of orthodontic treatment, particularly in cases of skeletal Class II malocclusion with mandibular hypoplasia. In the general growing population, the timing of functional and interceptive orthodontic treatment is understood to depend more on the growth phase than on chronological age. Correction of mandibular hypoplasia is generally most effective when functional therapy or growth modification is initiated during the pubertal growth spurt, and accurate identification of this growth peak through indicators of skeletal maturity rather than based solely on chronological age is crucial for maximizing treatment efficiency, as the rate of mandibular growth, along with its sex-specific patterns, varies significantly among individuals[21].

Reviews of growth-indicator methodology emphasize that relying solely on chronological age can be misleading given the wide interindividual variation in the timing of the pubertal growth spurt, and that indicators of skeletal maturity

such as hand-wrist radiography and cervical vertebral maturation (CVM) as well as methods such as third-finger middle phalanx maturation (MPM) should instead be used to identify the pre-pubertal, circumpubertal, and post-pubertal growth phases relevant to treatment planning[8]. Comparative studies have found that the MPM method acts as a valid, lower-radiation alternative to CVM staging for identifying the peak of pubertal growth, and a series of cases applying MPM-based timing to Class II functional treatment have reported favorable and stable mandibular elongation when treatment was initiated at the correctly identified growth stage[22,23]. However, it should be noted that the hand-wrist, CVM, and MPM staging methods were developed and validated in populations with normal endocrine status. Their application to children with GHD, in whom skeletal maturation is delayed and the onset of puberty may be delayed, constitutes a reasonable extrapolation rather than a validated protocol.

These timing principles are particularly important in children with GHD, as GHD is characterized by a systematic mismatch between chronological age, bone age, and dental age, accompanied by a delayed and sometimes attenuated pubertal onset. Therefore, a clinician who relies solely on chronological age in a GHD patient risks initiating functional or orthopedic treatment before the patient has actually entered the rapid growth phase, given that skeletal maturation may lag one year or more behind chronological age[17]. Furthermore, because rhGH therapy itself measurably increases mandibular growth, and because this craniofacial effect appears to increase with the duration of treatment rather than reaching a plateau early on[5], the growth window available for orthodontic correction in GHD patients may not align with the same stage of skeletal maturation, nor occur within the same timeframe, as in an endocrinologically normal peer.

These considerations raise several practical implications regarding the timing of orthodontic treatment in children with GHD. First, chronological age should not be used in isolation to schedule functional or interceptive treatment in this population. Instead, skeletal-maturation staging (hand-wrist radiography, CVM, or MPM) should be used to determine the patient's actual growth phase, in accordance with general principles of orthodontic timing, but applied with greater caution given the known age dissociation in GHD[8]. Second, because rhGH therapy itself appears to create or prolong a period of increased mandibular growth potential, close coordination between the treating orthodontist and a pediatric endocrinologist is strongly recommended, so that orthodontic or orthopedic intervention can be scheduled to coincide with the child's actual growth spurt[24]. Third, given that dental maturation and tooth eruption lag behind skeletal maturation to varying degrees in this population[17,18], the stage of dental development alone should not be used as the primary criterion for treatment timing. Finally, because the mandible responds preferentially to GH stimulation in the sagittal and vertical dimensions, while transverse dimensions change comparatively little[20], the residual transverse discrepancy in GHD patients may still require a separate treatment strategy specific to that dimension, even after a successful period of sagittal correction.

4 CONCLUSION

GH is a key regulator of craniofacial growth, acting primarily by stimulating the mandibular condylar cartilage through IGF-1, with genetic variation in the GH receptor further modulating the potential for mandibular growth in each individual. GHD produces a consistent, though variably severe, craniofacial and dental phenotype characterized by a retrognathic, under-grown mandible, delayed and dissociated bone and dental age, and an increased tendency toward Class II malocclusion as well as crowding.

Recombinant human GH replacement therapy partially, but incompletely, corrects this phenotype, with the mandible showing the most consistent sagittal and vertical catch-up growth. Because standard, chronological-age-based orthodontic timing protocols assume a growth trajectory that does not reliably hold in GHD, orthodontic and dentofacial orthopedic treatment in this population should instead be timed according to individualized skeletal-maturation assessment, ideally coordinated with the child's ongoing endocrine treatment, rather than according to age or dental development alone.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

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