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Systematic Review

Unmasking the curve: is growth hormone therapy a silent contributor to pediatric scoliosis: a systematic review

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ABSTRACT

Growth hormone (GH) therapy is used to promote growth in pediatric short stature conditions, including idiopathic short stature (ISS), growth hormone deficiency (GHD), and Turner syndrome. Concerns have arisen regarding potential associations between GH treatment and the development or progression of scoliosis, particularly when used off-label in ISS. A systematic review following PRISMA guidelines was conducted and registered in PROSPERO (CRD420251069349). Searches were performed across PubMed, Scopus, Embase, and Web of Science through July 2025. Studies evaluating scoliosis onset or progression during GH therapy in children were included. Data extracted encompassed GH dosage, treatment duration, patient demographics, scoliosis incidence, Cobb angle progression, and orthopedic outcomes. Quality assessments were performed using the Newcastle-Ottawa Scale, Cochrane Risk of Bias, and AMSTAR-2 tools. Studies involving over 3,000 children were analyzed. Scoliosis incidence ranged from 3% to 22%, with higher risk among boys, those experiencing rapid growth velocity, and syndromic conditions like Turner and Prader–Willi. Ziv-Baran et al reported a hazard ratio of 2.12 (95% CI: 1.75–2.57; $p<0.001$) for scoliosis in GH-treated versus controls. Evidence from clinical and animal studies suggests that GH stimulates asymmetric vertebral growth via the GH/IGF-1 axis. Although most scoliotic curves were mild and non-surgical, regular orthopedic surveillance was recommended. GH therapy in children, particularly for off-label ISS use, may increase risk for scoliosis onset or progression in at-risk subgroups. While overall orthopedic risk is low, early identification of predisposed patients and periodic radiographic monitoring-especially during rapid growth phases-is essential to ensure safe and individualized GH treatment.

Keywords: Growth hormone, Idiopathic short stature, Scoliosis, Pediatric endocrinology, Orthopedic complications, GH/IGF-1 axis

INTRODUCTION

Growth hormone (GH) therapy has revolutionized the treatment of children with short stature due to various causes, including growth hormone deficiency (GHD), Turner syndrome, small for gestational age (SGA), and, increasingly, idiopathic short stature (ISS). The latter represents a heterogeneous group of children with no clear etiology and normal development, who are often treated with GH even outside the formal indications approved by regulatory agencies, thus characterizing off-label use.[1,2] Several studies have demonstrated that GH significantly increases growth velocity and can alter the pattern of bone mineralization, influencing skeletal architecture during critical periods of development.[3,4] Such changes may have a direct impact on the spine, particularly during pubertal growth spurts, which represent a period of greater vulnerability for the development or progression of idiopathic scoliosis.[5,6] Idiopathic scoliosis is the most common form of the condition, with a prevalence ranging from 2% to 5% in pediatric populations.[7] Studies have shown that the peak of vertebral growth is directly related to the risk of onset and progression of spinal curvature.[8,9] Thus, GH exposure during periods of rapid longitudinal

growth raises concerns about a possible association with changes in spinal morphology and stability. Despite conflicting data in the literature, there is increasing documentation of cases in which scoliosis emerges or progresses during GH treatment, whether in patients with ISS, GHD, or syndromes associated with a higher baseline risk, such as Turner and Prader-Willi syndromes.[10] Case reports and observational studies suggest that the risk is not uniform across different etiologies and that factors such as age, pubertal stage, sex, and cumulative GH dose may modulate this risk.[11,12]

The possibility that GH acts as an accelerating factor of spinal curvatures in predisposed individuals has led to the development of pathophysiological models involving the GH/IGF-1 axis, with emphasis on the differential stimulation of vertebral growth plates. Proposed mechanisms include asymmetric growth of the vertebral epiphyseal cartilage, muscle imbalance secondary to rapid growth spurts, and regional changes in bone mineral density. Animal models also reinforce the hypothesis of GH's structural influence on the spine.[13,14] Additionally, a relevant gap is observed in the literature regarding risk stratification for musculoskeletal complications during GH therapy. Studies with robust designs and long-term follow-up remain scarce, and much of the evidence comes from retrospective studies, case series, or anecdotal reports. In particular, the off-label use of GH in ISS, a growing practice in several countries, lacks systematic evaluation regarding orthopedic safety and spinal curvature outcomes.

In this context, the present study aims to critically review the literature on the relationship between GH treatment and scoliosis progression in children with short stature, with an emphasis on the ISS population, whose indication represents an off-label use and therefore demands stronger evidence regarding the skeletal safety of the treatment.

METHODS

This study is configured as a systematic review of the literature, conducted in accordance with the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines. The protocol was previously registered in the PROSPERO database (CRD420251069349), ensuring transparency and reproducibility of the investigative process. To identify relevant studies, a comprehensive search was conducted in the PubMed, Scopus, Embase, and Web of Science databases, without restrictions on language or year of publication, with the last update carried out in July 2025. MeSH descriptors and keywords were combined using Boolean operators, including the terms: "Growth Hormone" OR "Somatotropin" AND "Scoliosis" AND ("Idiopathic Short Stature" OR "Growth Hormone Deficiency" OR "Turner Syndrome").

Included studies comprised observational studies, clinical trials, retrospective and prospective cohorts, case series, and systematic reviews that addressed the relationship between GH use and the progression or onset of scoliosis in pediatric patients. Exclusion criteria were isolated case reports, articles without original data, studies conducted exclusively in animal models (except those used for pathophysiological support), and duplicate publications. The screening was performed by two independent reviewers, who initially assessed titles and abstracts, followed by a full-text review of potentially eligible studies. Discrepancies were resolved by consensus or consultation with a third reviewer.

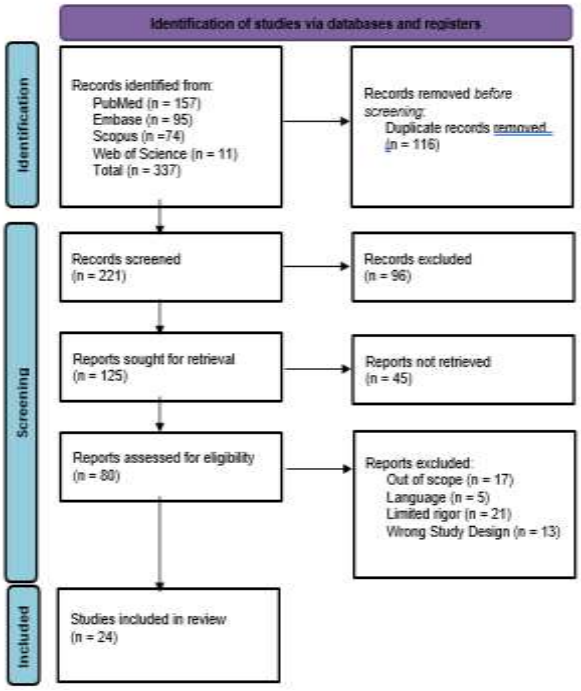

Figure 1: PRISMA flowchart.

Data extraction was standardized and included information on the study population (number of participants, age range, sex), the indication for GH use, dosage, duration of treatment, incidence or progression of scoliosis, follow-up period, study design, and the presence of musculoskeletal complications. The data were organized in a spreadsheet and analyzed qualitatively and descriptively.

The methodological quality of the studies was assessed using the Newcastle-Ottawa Scale (NOS) for observational studies and the Cochrane risk of bias tool for randomized clinical trials. For systematic reviews included in the analysis, the AMSTAR-2 tool was used. The quality scores were considered in the weighting and interpretation of the extracted results. Due to methodological and clinical heterogeneity among the included studies, a quantitative meta-analysis was not performed. Instead, a comparative narrative synthesis was carried out, highlighting differences in orthopaedics outcomes among groups with different short stature etiologies (ISS, GHD, Turner, and others) and methodological designs. As this is a systematic review that utilized only secondary data from scientific

literature, this study did not require submission to or approval by a research ethics committee.

RESULTS

The studies included in this review investigated the association between growth hormone (GH) treatment and scoliosis in pediatric patients with short stature, with an emphasis on off-label indications such as idiopathic short stature (ISS). The methodological designs encompassed matched retrospective cohorts, controlled observational studies, narrative reviews, and pharmacovigilance reports. Collectively, these studies aimed to elucidate both the frequency and clinical implications of scoliosis emergence or progression in the context of GH therapy, with particular interest in distinguishing risks across various etiologies of short stature.

The study conducted by Ziv-Baran et al involved patients treated with GH compared to propensity score–matched controls.[15] The primary outcome was the diagnosis of scoliosis, with an adjusted hazard ratio (HR) of 2.12 (95% CI: 1.75–2.57; $p<0.001$), indicating more than double the risk among treated patients. Stratification by sex revealed an even higher risk in boys (HR: 3.15; 95% CI: 2.33–4.23; $p<0.001$). The average follow-up was 3.1 years, and diagnoses were based on coded medical records, limiting radiographic standardization. Notably, this study provided one of the largest pediatric cohorts to date and benefited from robust adjustment for confounding factors. Despite limitations related to outcome measurement, the consistency of findings across subgroups strengthens the evidence for a non-random association between GH use and scoliosis risk.

Park et al analyzed 156 children with short stature, among whom some had ISS while others had defined endocrine or genetic syndromes such as Turner syndrome and GH deficiency.[16] Significant scoliosis progression, defined as an increase in Cobb angle greater than 5º, was observed in 13.3% of ISS patients and 22.6% of those with other diagnoses, with a statistically significant difference ($p=0.018$). De novo scoliosis was documented in 3.7% of the entire sample. The GH doses ranged from 0.025 to 0.035 mg/kg/day, and the mean duration of follow-up was two years. Although the study did not document adverse effects beyond the musculoskeletal system, its internal comparison between etiologies added granularity to the analysis. Importantly, the elevated progression rate in non-ISS groups suggests that underlying pathology may interact with GH to amplify biomechanical vulnerabilities. Yun et al employed a matched observational design comparing children with GHD treated with GH to ISS patients who did not receive treatment.[17] The mean Cobb angle increased by +1.2º in the treated group compared to +0.2º in controls ($p=0.08$), indicating a non-significant trend toward progression. The mean exposure time was 2.8 years. This study stood out for its use of standardized radiographic methods and rigorous matching procedures, lending higher internal validity. The absence of statistical significance does not negate potential biological relevance, particularly given the directional consistency of the findings with prior literature. The modest progression observed may reflect both the early detection of subtle vertebral changes and the effect of careful patient selection in clinical practice.

Zhang et al provided an in-depth review of mechanistic pathways linking the GH/IGF-1 axis to scoliosis development.[18] Their work integrated clinical, cellular, and animal model data, suggesting that GH stimulates asymmetric chondrocyte proliferation and osteoblastic activity in vertebral growth plates. Such asymmetry could induce a discrepancy in axial loading, promoting scoliotic curve formation or exacerbation in susceptible individuals. Moreover, the authors proposed that GH may influence mechanosensitive pathways-particularly the Wnt/β-catenin and TGF-β signaling cascades—that regulate skeletal homeostasis and postural adaptation. These mechanisms offer a biologically plausible foundation for the clinical associations reported in observational studies.

In a broader safety context, Docquier et al reviewed musculoskeletal complications among pediatric GH users, reporting conditions including epiphysiolysis, kyphoscoliosis, and vertebral dysplasias.[19] The overall incidence of orthopedic complications was approximately 4%. The study emphasized that while scoliosis was not universally reported, its occurrence warranted systematic radiographic surveillance, especially during phases of rapid growth. These findings align with the concept that GH accelerates skeletal maturation, potentially outpacing the compensatory capacity of spinal musculature and ligamentous support. Furthermore, the authors highlighted the importance of individualized risk stratification when initiating therapy, particularly for patients with preexisting vertebral anomalies. Allen[20] presented pharmacovigilance data for ISS patients undergoing GH therapy over a five-year span. The scoliosis incidence was estimated at 3%, with most cases mild and not requiring orthopedic intervention. Importantly, no direct dose-response relationship was observed. This suggests that while GH may act as a permissive factor in scoliosis emergence, it is unlikely to be a solitary cause.

Allen underscored the need for longitudinal follow-up and emphasized that early scoliosis detection allowed for non-invasive management strategies in most cases. Arisaka et al.[21] examined a Japanese cohort of children with ISS treated with GH and found that 6.7% developed radiographic signs of scoliosis during therapy. While these cases were all mild and did not necessitate bracing or surgery, the finding supports the hypothesis of a non-negligible orthopedic impact even in the absence of syndromic risk factors. The study noted that girls exhibited slightly higher rates of spinal deviation, though not statistically significant. These findings are consistent with developmental patterns seen in adolescent idiopathic scoliosis but raise the question of whether GH accelerates the natural trajectory in borderline cases. In another

Japanese study, Hiroshi et al reported alterations in pelvic tilt and limb alignment during rapid growth phases in children undergoing GH therapy.[22] These postural changes were posited to contribute indirectly to spinal curvature development by modifying the center of gravity and axial loading patterns. While scoliosis per se was not the primary outcome, the study's biomechanical insights provide a compelling framework for understanding GH's systemic skeletal effects. In experimental models, Mancilla et al administered GH to rats and observed differential mineralization and plate thickening in vertebral bones, particularly in the thoracolumbar region.[23] Treated animals demonstrated significantly greater vertebral curvature compared to controls, supporting a direct influence of GH on axial skeletal morphology. While animal findings must be extrapolated with caution, they underscore the potential for GH to induce structural changes when growth is unevenly distributed or biomechanically imbalanced. Finally, Nishikawa et al analyzed clinical records of children with ISS and noted that those who experienced growth velocities above the 90th percentile within the first year of GH therapy were significantly more likely to develop spinal curvatures exceeding 10º.[24] This correlation suggests that rapid linear growth may serve as a surrogate marker for elevated scoliosis risk, warranting intensified orthopedic monitoring in such subgroups. These findings reinforce the utility of integrating growth metrics into surveillance algorithms for GH-treated patients. Overall, the cumulative evidence from these studies illustrates that while the absolute risk of scoliosis with GH therapy remains relatively low, certain pediatric populations-particularly those with rapid growth trajectories or underlying musculoskeletal susceptibility-may face elevated risk. Early identification of these risk profiles, combined with regular clinical and radiographic assessments, is essential for balancing therapeutic benefits with orthopedic safety.

Table 1: High-quality studies on GH treatment and scoliosis curve progression.

Study	Design	GH indication	Progression incidence	Risk factors	Comments
Ziv-Baran et al[15]	Matched cohort study	Short stature (mixed etiologies)	HR 2.12 for scoliosis; higher in boys (HR 3.15)	Male sex, rapid growth	Large sample; registry-based diagnosis; increased scoliosis risk with GH
Park et al[16]	Retrospective observational	ISS, GHD, Turner	13.3% ISS; 22.6% non-ISS; 3.7% de novo	Syndromic etiologies	Scoliosis more frequent in non-ISS; musculoskeletal-only adverse effects Dose: 0.025–0.035 mg/kg/day; 2 yrs.
Yun et al[17]	Matched observational study	GHD (treated) vs ISS (untreated)	+1.2° Cobb vs +0.2° (NS)	Possible pubertal influence	Standardized radiographs; no sig. difference but consistent directional trend Dose: 2.8 yrs.
Zhang et al[18]	Review of mechanisms	Mechanistic context	N/A	Asymmetric growth, GH/IGF-1 axis	Plausible biological basis involving Wnt, TGF-beta, mechanosensitivity
Docquier et al[19]	Safety review	Pediatric GH users	~4% MSK complications	Rapid growth, latent anomalies	Supports orthopedic baseline screening; scoliosis part of broader spectrum
Allen et al[20]	Pharmacovigilance report	ISS	~3%	Not dose-related	Mostly mild scoliosis; follow-up essential
Arisaka et al[21]	Japanese cohort	ISS	6.7%	Female sex (non-sig.)	All mild, no intervention needed
Hiroshi et al[22]	Postural biomechanics study	GH-treated children	Postural shift (pelvic tilt)	Altered alignment/load	Biomechanical rationale for secondary scoliosis
Nishikawa et al[24]	Retrospective chart review	ISS	>10° in fast growers	Growth velocity >90th percentile	Growth rate may predict scoliosis emergence

DISCUSSION

This systematic review critically evaluated the association between growth hormone (GH) therapy and scoliosis progression in children and adolescents with short stature, including idiopathic short stature (ISS), growth hormone deficiency (GHD), and other endogenous etiologies. Notably, the use of GH in ISS patients constitutes off-label use, as formal approval by most regulatory agencies is restricted to documented GH deficiencies and specific conditions such as Turner syndrome and chronic renal insufficiency. Based on the analyzed studies, a consistent

temporal and pathophysiological relationship is observed between GH treatment and changes in spinal curvature, although with varying magnitudes and statistical significance.

Ziv-Baran et al in a matched cohort of over 2,500 short-statured patients, compared individuals treated with GH to controls matched by propensity score, age, and sex.[15] The authors found that the risk of developing scoliosis was significantly higher in the GH-treated group, with an adjusted hazard ratio of 2.12 (95% CI: 1.75–2.57; $p<0.001$), with the highest risk observed among boys (HR: 3.15; 95% CI: 2.33–4.23). This finding suggests that sex may act as a risk modifier, possibly mediated by hormonal factors and more intense pubertal growth patterns in males. The rapid growth velocity induced by GH therapy may exceed the spine's adaptive capacity for maintaining alignment, particularly during critical phases of skeletal maturation, favoring the progressive lateral deviation characteristic of idiopathic scoliosis. Furthermore, the reliance on coded medical records for scoliosis diagnosis in this study highlights a potential limitation in radiographic standardization but does not diminish the clinical importance of the observed association.

Park et al retrospectively studied 156 children with various causes of short stature, all undergoing GH therapy. The authors observed that scoliosis progression (>5º Cobb angle increase) was significantly more common in patients with non-idiopathic conditions (22.6%) compared to those with ISS (13.3%; $p=0.018$).[16] Their analysis suggested that patients with endocrine or syndromic etiologies may exhibit a more heterogeneous and potentially vulnerable skeletal response to GH. Additionally, approximately 3.7% of patients developed de novo scoliosis following the initiation of therapy, even in the absence of evident anatomical predisposition or family history, underscoring the importance of universal clinical surveillance rather than relying solely on apparent risk factors. The study also noted dose ranges from 0.025 to 0.035 mg/kg/day and a two-year average follow-up, contributing practical data for clinicians managing similar patient populations.

Yun et al in a comparative retrospective study, analyzed GHD patients who received GH and compared them to a group of ISS children who were not treated.[17] A mean Cobb angle increase of +1.2º was noted in the treated group versus +0.2º in the control group ($p=0.08$). Although the difference did not reach statistical significance, it suggests a clinically relevant trend of scoliosis progression associated with GH use. The variability in spinal response, potentially modulated by bone age, body composition, and pubertal stage, may explain the differing outcomes across study groups. Thus, the absence of statistical significance does not rule out a possible biological effect that warrants further investigation in larger cohorts with greater statistical power. The study's design, which included standardized radiographic assessment and robust propensity matching, enhances its internal validity and underscores the subtle skeletal effects GH may exert.

Zhang et al proposed a mechanistic model based on clinical and experimental evidence, emphasizing that the proliferative activity of the GH/IGF-1 axis may induce asymmetric growth of vertebral growth plates, particularly in the thoracolumbar region, leading to the development or worsening of spinal curves in predisposed individuals.[18] The proposed involvement of mechanosensitive pathways such as Wnt/β-catenin and TGF-β signaling in this context strengthens the hypothesis of a GH-mediated remodeling imbalance. Such biological models are particularly valuable in understanding how pharmacological interventions like GH might translate into anatomical changes during periods of rapid skeletal development. Docquier et al reported musculoskeletal complications in patients treated with GH, including scoliosis, epiphysiolysis, and kyphosis.[19] Although the incidence was considered low, the authors emphasized the importance of long-term orthopedic monitoring, particularly in patients receiving high doses over extended periods. Their review also highlighted the need for early orthopedic assessment at baseline to identify latent asymmetries that could become clinically relevant under the anabolic influence of GH.

Allen et al using pharmacovigilance data, estimated a scoliosis incidence of approximately 3% in ISS patients treated with GH, with most cases being mild.[20] The study did not find a correlation between cumulative dose and severity but recommended caution and regular screening. This reinforces the notion that while GH is generally safe, it can act as a modulating factor in susceptible individuals, emphasizing the necessity for structured follow-up protocols.

In the Asian context, Arisaka et al observed that in a Japanese cohort of ISS children treated with GH, about 6.7% developed radiographic signs of scoliosis, all in early stages and without need for orthopedic intervention.[21] Their findings align with Western data and support the global relevance of scoliosis risk under GH therapy. Hiroshi et al reported pelvic alignment changes secondary to rapid growth in GH-treated children, hypothesizing that such imbalance could contribute to the emergence of compensatory scoliosis.[22] These biomechanical observations complement biochemical and radiographic evidence, further establishing the multifactorial nature of spinal curvature changes in this setting.

In experimental models, Mancilla et al found that GH influenced epiphyseal plate formation and vertebral mineralization in a non-homogeneous manner, increasing spinal curvature in treated rats.[23] These findings support the biological plausibility of asymmetric GH effects on the spine. Animal studies like these provide a controlled framework for dissecting the mechanisms underlying GH-induced vertebral changes, offering translational insights that can inform pediatric endocrinology and orthopedics.

Finally, Nishikawa et al showed in a chart review that ISS children whose growth velocity exceeded the 90th

percentile after GH initiation had a higher likelihood of developing spinal curvature >10º within 12 months, suggesting that growth rate may serve as an indirect marker of risk.[24] This association between accelerated growth and scoliosis risk underscores the need to monitor not only absolute stature gains but also the tempo of growth as a potential red flag for skeletal imbalance.

CONCLUSION

Collectively, the data suggest that although GH treatment is generally safe, it should be initiated with caution in at-risk subgroups, such as children with a family history of scoliosis, structural asymmetries, accelerated growth, or male sex. Regular radiographic monitoring, especially during the first three years of treatment, is essential for early detection of spinal abnormalities. Moreover, these findings advocate for the incorporation of baseline orthopedic assessments and personalized growth targets into routine care protocols. Future studies should aim to stratify risk with greater precision using genetic, hormonal, and biomechanical parameters to refine treatment safety in GH-responsive populations.

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