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Applying the Functional Medicine Model to Idiopathic Scoliosis Management: A Narrative Mini-Review

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Abstract

Addressing the underlying components of idiopathic scoliosis, including hormone and neurotransmitter imbalances, has the potential to improve scoliosis treatment outcomes. While the precise relationship between these imbalances and scoliosis development is not fully understood, emerging evidence suggests that correcting these imbalances may positively influence the progression and management of idiopathic scoliosis. The functional medicine model of patient evaluation and treatment may offer some insight as to more robustly, and possibly effectively, manage the entire idiopathic scoliosis condition. This review describes many of the known underlying abnormalities associated with idiopathic scoliosis and offers perspective and ideas for future research into comprehensive idiopathic scoliosis management.

Keywords: Scoliosis; Chiropractic Scoliosis; Melatonin; Osteoblasts; Osteoclasts

Introduction

The intricate relationship between the spinal cord and the vertebral column during periods of rapid growth is a critical factor influencing the development of scoliosis. Discrepancies in growth rates between these two structures can result in a disrupted equilibrium, leading to the manifestation of scoliosis. Understanding the pathophysiological mechanisms underlying this phenomenon is paramount for early detection, appropriate intervention, and effective management of scoliosis during periods of accelerated growth.

The growth discrepancy between the spinal cord and the vertebral column plays a pivotal role in scoliosis etiology. It primarily arises from variations in longitudinal growth rates, leading to an incongruity in size and shape between the spinal cord and the vertebral elements. Consequently, this incongruity engenders a lateral deviation of the spinal column, giving rise to the characteristic curvature observed in scoliosis.

Disproportionate growth patterns precipitate "relative spinal cord tethering," an anatomical phenomenon wherein the accelerated growth of the vertebral column exerts tensile forces on the spinal cord. This tethering effect elicits a subsequent malalignment of the spinal cord within the spinal canal, inducing a complex, multiplanar deformity of the vertebral column. Consequently, the asymmetric loading forces exerted by the deformed spinal column result in muscular, ligamentous, and bony imbalances, contributing to the manifestation of scoliosis. This concept has been previously described by Stokes et al [1].

The association between rapid growth phases, particularly during adolescence, and the onset of scoliosis is well-documented. The heightened physiological demands of accelerated growth exert additional strain on the already vulnerable spinal structures, exacerbating the growth discrepancy-induced scoliotic deformity. Consequently, individuals undergoing rapid growth are predisposed to an augmented risk of scoliosis development due to the pronounced growth incongruity during these critical periods.

Although recent attempts have been made to account for non-musculoskeletal signs and symptoms in idiopathic scoliosis, such as the double neuro-osseous theory put forth by Burwell et al. [2], there have been no attempts to incorporate this information into clinical practice beyond typical musculoskeletal treatment modalities.

The Functional Medicine Model of Care

The functional medicine model of patient care is an approach that seeks to address the root causes of illness and promote optimal health and well-being. It recognizes that each individual is

unique and treats patients as whole persons, taking into account their genetics, environment, lifestyle, and personal history.

In the functional medicine model, the focus is on understanding and addressing the underlying imbalances and dysfunctions that contribute to disease. This is done by evaluating and addressing key factors such as nutrition, stress, physical activity, sleep, toxin exposure, and social connections. By identifying and addressing these factors, functional medicine aims to restore balance and support the body's innate healing abilities.

A key principle of functional medicine is the understanding that different individuals may have different underlying causes for their health conditions, even if the symptoms appear similar. This personalized approach allows for targeted interventions and treatments tailored to each patient's specific needs.

Functional medicine practitioners often utilize a variety of diagnostic tools and assessments, including comprehensive health histories, laboratory tests, and advanced imaging techniques, to gain a deeper understanding of the patient's health status. Treatment plans typically involve a combination of lifestyle modifications, dietary interventions, supplementation, stress management techniques, and other therapies that support the body's natural healing processes.

The functional medicine model also emphasizes patient education and empowerment, encouraging individuals to take an active role in their own health and well-being. By addressing the underlying imbalances and working collaboratively with patients, functional medicine aims to not only alleviate symptoms but also promote long-term health and vitality.

This model of care could address the concurrent associated abnormalities in idiopathic scoliosis, such as the hormone and neurotransmitter imbalances, as well as the micronutrient deficiencies. Specific treatment interventions for idiopathic scoliosis patients can be specific to their symptoms and will be discussed in this review.

Current Idiopathic Scoliosis Management

The management of scoliosis resulting from disproportionate growth centers around addressing the underlying growth discrepancy and restoring optimal spinal alignment. Treatment modalities encompass a spectrum of interventions, including orthotic bracing, physiotherapy, tailored exercise regimens, and other multidisciplinary approaches aimed at rectifying muscular imbalances, enhancing flexibility, and improving postural control. In severe cases characterized by progressive curvature, surgical interventions may be warranted to achieve spinal correction and stabilization. A review of the 2016 Society on Scoliosis Orthopedic and Rehabilitation Treatment (SOSORT) guidelines shows that all treatment is musculoskeletal-based, and does not take any metabolic, genetic, or lifestyle factors into account when managing idiopathic scoliosis [**3**].

Idiopathic Scoliosis and Nutrient Deficiencies

Nutrient deficiencies, including vitamin D, selenium, and manganese, have been implicated in the development of idiopathic scoliosis, and it has been observed that these deficiencies are more prevalent in children with idiopathic scoliosis. Adequate nutrition is crucial for proper skeletal development, and imbalances in essential nutrients can impact bone health and contribute to the onset and progression of scoliosis.

Vitamin D is essential for calcium absorption and utilization and plays a critical role in bone mineralization. Insufficient levels of vitamin D have been associated with impaired bone health and increased risk of skeletal deformities, including scoliosis. Studies have found a higher prevalence of vitamin D deficiency in children with idiopathic scoliosis compared to the general population [4], suggesting a potential link between vitamin D deficiency and scoliosis development. Other studies, such as one by Ahuja et al. [5], showed that IS patients were prone to vitamin D deficiency and poor calcium balance, as well as high bone turnover and low bone density.

A previous review by Normand et al. [6] showed that idiopathic scoliosis patients had not only lower levels of vitamin D, but also calcitonin and parathyroid hormone. These findings offer potential explanations as to why idiopathic scoliosis patients also have lower average bone density than their peers [7]. Selenium is an essential trace mineral that serves as a cofactor for several enzymes involved in antioxidant defense mechanisms and bone metabolism. Selenium deficiency has been associated with impaired bone development and increased susceptibility to skeletal abnormalities. Dastych et al reported lower selenium levels in individuals with idiopathic scoliosis, indicating a possible role of selenium deficiency in scoliosis pathogenesis [8]. Manganese is another essential trace mineral that plays a role in bone formation and development. Research has shown a correlation between low manganese levels, a specific genetic variant, and the prevalence of idiopathic scoliosis [9]. Manganese deficiency may affect skeletal growth and cartilage formation, potentially influencing the progression of scoliosis. Addressing nutrient deficiencies in individuals with idiopathic scoliosis is an important aspect of comprehensive treatment. Supplementation or dietary modifications to ensure adequate intake of vitamin D, selenium, and manganese may be considered in conjunction with other treatment strategies.

Idiopathic Scoliosis and Hormone Imbalances

Hormone imbalances have been implicated as potential factors in the development and progression of idiopathic scoliosis. One hormone that has received significant attention in relation to scoliosis is melatonin. Melatonin is a hormone produced by the pineal gland in the brain, primarily involved in regulating sleep-wake cycles. It also plays a role in skeletal growth and development. Studies have shown that individuals with idiopathic

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scoliosis often have lower levels of melatonin or altered melatonin signaling. Melatonin influences bone metabolism, including the regulation of osteoblasts and osteoclasts, the cells responsible for bone formation and resorption [10]. Disruptions in melatonin production or signaling may impact bone growth and contribute to the progression of scoliosis. Melatonin has been used in animal studies to improve bone mass [11,12].

Other hormones, such as growth hormone, estrogen, and progesterone, have also been studied in relation to idiopathic scoliosis. Growth hormone is essential for skeletal growth and development. Imbalances in growth hormone levels or signaling may affect the normal growth of the spine, potentially contributing to scoliosis development. Estrogen and progesterone, which increase during puberty, may also play a role in scoliosis. Changes in hormone levels during this period may impact bone density, bone remodeling, and the overall growth of the spine.

Growth hormone is thought to be a contributor to scoliosis progression. Multiple studies [13-16] have shown that patients with idiopathic scoliosis have elevated growth hormone levels [15,16], and that administration of growth hormone in studies on children with short stature showed increased incidence of scoliosis progression [13,14].

Previous research suggests a correlation between a higher risk of idiopathic scoliosis in females and delayed onset of menstruation, suggesting that reduced estrogen levels may contribute to the onset of idiopathic scoliosis [17]. Another study revealed significantly lower serum estradiol levels in IS patients compared to non-IS patients, suggesting that an estrogen deficiency during the developmental phase of may be implicated in the emergence and progression of scoliosis [18]. Impacts of estrogen receptor polymorphisms [19], as well as estrogen's role in bone growth on idiopathic scoliosis [20], have also been detailed.

Finally, Morningstar and Strauchman [21] observed that female patients without idiopathic scoliosis had 49% more salivary progesterone than that of their scoliosis peers. When examining across the lifespan, their levels remained different until post-menopause.

Idiopathic Scoliosis and Neurotransmitter Imbalances

Serotonin, a neurotransmitter primarily synthesized in the brain, is involved in mood regulation, sleep, and other physiological functions. It also plays a role in bone development and remodeling. Imbalances in serotonin signaling have been implicated in scoliosis, with studies suggesting potential alterations in serotonin receptor expression in individuals with idiopathic scoliosis. Other studies have demonstrated a link between frank serotonin deficiency and idiopathic scoliosis [22]. Multiple neurotransmitter levels differ among patients with idiopathic scoliosis when compared to their peers [23]. This is not a new concept [24]. However, few advances in clinical application have been made regarding this topic.

Evidence for a Functional Medicine Model of Idiopathic Scoliosis Management

Recent evidence points to the ability of functional medicine strategies to improve the clinical outcomes associated with idiopathic scoliosis treatment. In a study by Herdea et al. [25], 26 children with mild idiopathic scoliosis were given melatonin, vitamin D3, and calcium as part of their typical observation-only management and compared them with 25 children who were only being observed. In that treatment group, their curves were significantly less likely to increase to a threshold where conventional treatment was recommended.

In another study by Morningstar et al, children participating in a short-term intensive outpatient scoliosis-specific rehabilitation program were prescribed various nutrients for observed neurotransmitter imbalances [26]. These children's results were compared to other children who completed the same physical treatment but did not take the nutrient therapies. At 6-month follow-up, radiographic curve measurement improvements were much more stable in the children taking the nutrients compared to the physical treatment-only group.

In a study of adult patients with lumbar scoliosis and rotary olisthesis, patients given estrogen replacement therapy saw improvements in their olisthesis and curve measurements after starting the hormone replacement [27]. This study suggests that hormone therapy may reduce the effects of abnormal biomechanical factors contributing to scoliosis curve progression in adulthood.

Considering that idiopathic scoliosis patients have lower bone density [7], the synergistic effects of melatonin, strontium, vitamin D, and vitamin K2 have been shown to increase bone density by improving osteoblastic signaling [28]. This is yet another potential role for functional medicine therapies at improving the underlying components of the idiopathic scoliosis cascade.

Given the vast array of personalized treatment options, the functional medicine model of care may have extensive and important contributions to the comprehensive management of idiopathic scoliosis.

Conclusion

It is important to note that while addressing micronutrient deficiencies, hormone imbalances, and neurotransmitter imbalances may hold promise, their precise role in scoliosis management and the optimal approaches for intervention require further investigation. Scoliosis treatment is typically multifaceted and individualized, incorporating various modalities such as bracing, exercise programs, physical therapy, and, in severe cases, surgical intervention. Integrating functional medicine interventions that target the underlying genetic, environmental, and metabolic factors of idiopathic scoliosis alongside conventional treatments may offer a more comprehensive approach to scoliosis

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management, potentially yielding improved treatment outcomes. However, further research is needed to establish the specific protocols, timing, and effectiveness of such interventions in the context of idiopathic scoliosis.

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