

Osteoarthritis and Hypothyroidism: What's the Association? A Literature Review

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Abstract

Osteoarthritis (OA) and hypothyroidism are two prevalent conditions with interrelated mechanisms suggesting a potential connection between them. Several studies have indicated that individuals with hypothyroidism are twice as likely to develop OA in their knees compared to those without hypothyroidism. Furthermore, individuals with hypothyroidism are more prone to experiencing joint pain and stiffness, possibly attributed to the effect of thyroid hormone on bone and cartilage proliferation and differentiation, thus increasing the risk of OA. Although the precise relationship between OA and hypothyroidism remains incompletely understood, research underscores a significant association, suggesting a heightened risk of OA development in individuals with hypothyroidism. This research aims to elucidate the mechanisms underlying this relationship and its implications for managing OA in patients with hypothyroidism.

Keywords: Relationship, Osteoarthritis, Hypothyroidism, Mechanism.

INTRODUCTION

Osteoarthritis (OA) is a disease that affects the articular cartilage, subchondral bone, synovium, capsule, and ligaments. The cartilage degenerates, resulting in fibrillation, fissures, ulceration, and loss of full thickness on the joint surface (Knecht et al., 2006). Risk factors for OA include age, obesity, history of joint injury, and genetic factors. Hypothyroidism occurs when the thyroid gland does not produce enough thyroid hormone (Hasan, 2016). Risk factors for hypothyroidism include autoimmune thyroid disease, iodine deficiency, and radiation treatment to the thyroid gland. Thyroid hormone plays an important role in maintaining bone health by regulating bone formation and breakdown. Thyroid hormone deficiency can disrupt this balance, causing decreased bone density and increasing the risk of cartilage damage (Wojcicka et al., 2013a).

Although the relationship between OA and hypothyroidism is not fully understood, research suggests a link between these two conditions. People with hypothyroidism may have a higher risk of developing OA and may experience more severe OA symptoms. The specific issue addressed in this research is the urgent need to better understand the mechanisms underlying the relationship between OA and hypothyroidism, which has significant implications for the prevention and management of these conditions (Oo et al., 2018).

Previous research has explored the individual impacts of hypothyroidism on bone health and the progression of OA, but comprehensive studies integrating these aspects are limited. This research aims to fill that gap by investigating the specific mechanisms and risk factors contributing to OA in individuals with hypothyroidism (Medici et al., 2017). The novelty of this study lies in its integrated approach to examining the intersection of OA and hypothyroidism, providing new insights that could lead to improved prevention strategies and enhanced quality of life for affected individuals.

This research aims to discuss the relationship between OA and hypothyroidism, focusing on the mechanisms underlying this relationship and the risk factors for developing OA in people with hypothyroidism (Mohajer et al., 2023a). Understanding this relationship is crucial for developing more effective prevention strategies for both conditions. The results of this study may help improve the quality of life for people with OA and hypothyroidism.

RESEARCH METHODS

The literature review method for investigating the relationship between osteoarthritis (OA) and hypothyroidism involved analyzing multiple studies to explore the prevalence and mechanisms linking these conditions (B. Li et al., 2024a). Several studies were reviewed to determine the incidence of OA in individuals with hypothyroidism and vice versa. For instance, a comprehensive review by Moisescu-Pop et al. (2020) analyzed data from 11 studies involving 4,659 participants, revealing a higher prevalence of hypothyroidism in OA patients compared to the general population. Similarly, a retrospective cohort study by Wang et al. (2021) examined a large healthcare database, finding a significantly higher incidence of OA in patients with hypothyroidism. Mechanistic studies highlighted that thyroid hormone deficiencies disrupt bone metabolism, increase inflammation, and alter joint biomechanics, contributing to OA development. The review incorporated findings on the impact of thyroid hormones on cartilage metabolism and the role of inflammatory mediators in exacerbating OA (F.-X.-Z. Li et al., 2020). This methodical examination of epidemiological data, clinical studies, and mechanistic insights provided a comprehensive understanding of the potential link between OA and hypothyroidism, emphasizing the need for further research to elucidate the underlying pathways and improve clinical management strategies.

RESULTS AND DISCUSSION

Osteoarthritis (OA) is a degenerative joint disease marked by the breakdown of joint cartilage, predominantly affecting older and middle-aged individuals. It impacts people across all ethnicities, with a higher prevalence in women, and often leads to chronic disability in those over 65 (Warner & Brown, 2011). The condition, characterized by joint deterioration, primarily manifests in the hands, hips, feet, and spine, although it can affect any synovial joint.

Hypothyroidism arises when the thyroid gland fails to generate adequate levels of thyroid hormone (Dunn & Turner, 2016). This condition, characterized by insufficient hormone production, can occasionally be triggered by an enlarged thyroid gland, though this is uncommon. Various external factors can also lead to hypothyroidism, including past thyroid treatments, neck radiation, specific medications, and genetic disorders (Chiovato et al., 2019).

A comprehensive review by Moisescu-Pop et al. (2020) investigated the occurrence of hypothyroidism among individuals with osteoarthritis (OA). Analyzing 11 studies encompassing 4,659 participants, the review revealed that 12.1% of OA patients had hypothyroidism (95% CI: 8.8-16.4%), a notably higher rate than the general population's prevalence of 2-5% (Chen et al., 2022). The researchers concluded that OA patients face a significantly elevated risk of developing hypothyroidism, with an odds ratio of 1.55 (95% CI: 1.20-2.01).

Similarly, a retrospective cohort study by (Tagoe et al., 2021) analyzed data from a large healthcare database in the United States. They found that the incidence of OA was significantly higher in patients with hypothyroidism compared to those without the condition, with a hazard ratio of 1.24 (95% CI: 1.21-1.27). This study provided further evidence for the potential association between hypothyroidism and the development of OA (Mohajer et al., 2023b).

While these studies suggest a link between OA and hypothyroidism, the underlying mechanisms and the directionality of this relationship are not yet fully understood. More research is needed to elucidate the epidemiological patterns, risk factors, and potential causal pathways that may connect these two conditions (Penninx, 2017).

The Relationship Between Osteoarthritis (Oa) And Hypothyroidism

Osteoarthritis (OA) and hypothyroidism are prevalent conditions that are interconnected beyond mere statistics, involving complex mechanisms (Sobieh et al., 2023). Thyroid hormones are crucial in bone metabolism regulation, particularly the bone remodeling process. A deficiency in these hormones, like triiodothyronine (T3) and thyroxine (T4), disrupts the balance between bone resorption and formation, reducing bone density. Chen et al. explored the link between thyroid hormone sensitivity and OA prevalence using NHANES 2007-2010 data. They found that both peripheral and central thyroid resistance indices were significantly correlated with OA prevalence, with higher FT3/FT4 levels and lower TSHI, TT4RI, and TFQI levels being associated with increased OA risk. Kim et al. also found that hypothyroid conditions were linked to a higher incidence of knee osteoarthritis. Devrimsel et al. noted that hypothyroid patients had thinner femurs and a greater OA risk compared to healthy individuals across all measured sites (B. Li et al., 2024b). Additionally, another study reported that FT3/FT4 levels declined as OA risk increased. Collectively, these findings underscore the potential role of thyroid hormone sensitivity in predicting and assessing OA.

Thyroid hormone deficiency can interfere with the production of collagen and proteoglycans, this is important components of cartilage (Wojcicka et al., 2013b). This can cause the cartilage to become brittle and easily damaged, triggering the development of OA. Hypothyroidism can increase inflammation throughout the body, including in the joints (Antonelli et al., 2015). This can lead to increased inflammatory cytokines and immune cell infiltration. Thyroid hormone deficiency can increase the production of inflammatory cytokines, such as IL-1 β and TNF- α , which can damage cartilage and other joint tissue. Thyroid hormone deficiency can increase the infiltration of inflammatory cells into the joints, exacerbating inflammation and accelerating joint damage (Figus et al., 2021).

This hormone helps regulate the immune system and suppress inflammation. A lack of thyroid hormone (hypothyroidism) can disrupt this balance and increase inflammatory activity, including in the joints. Hypothyroidism can increase the infiltration of inflammatory cells such as macrophages and lymphocytes into the joints (Luty et al., 2019). These cells release enzymes and chemicals that can damage cartilage and other joint tissue, accelerating joint damage and the development of OA. And the other study, thyroid hormone deficiency can cause decreased protein synthesis and increased protein breakdown in muscles, leading to decreased muscle mass. This can weaken the strength and stability of the joint, making it more susceptible to injury and worsening OA pain and stiffness (Hafez et al., 2014).

Thyroid hormones, such as thyroxine (T4) and triiodothyronine (T3), play a crucial role in the maintenance and repair of articular cartilage. Thyroid hormones are involved in regulating the metabolism and structure of cartilage, as well as the balance between anabolic and catabolic processes within the joint (Mohammed & Al-Sari, n.d.).

Hypothyroidism, characterized by a deficiency of thyroid hormones, can lead to alterations in cartilage metabolism, increased inflammation, and ultimately, cartilage damage (Chen et al., 2022). This is because thyroid hormones are essential for the proper functioning of chondrocytes, the cells responsible for the production and maintenance of cartilage matrix.

Pathogenesis of OA in the Context of Hypothyroidism

The underlying mechanisms by which hypothyroidism may contribute to the development and progression of OA are not fully elucidated, but several potential pathways have been proposed (Mohammed & Al-Sari, n.d.):

- 1. Impaired cartilage metabolism: Hypothyroidism can disrupt the balance between anabolic and catabolic processes in cartilage, leading to increased catabolism and decreased synthesis of extracellular matrix components, such as collagen and proteoglycans. This can result in the gradual deterioration of articular cartilage.
- 2. Heightened inflammation: Hypothyroidism is linked to elevated levels of inflammatory mediators like cytokines and chemokines. These inflammatory agents can accelerate the onset of osteoarthritis by fostering cartilage breakdown and joint inflammation.
- 3. Altered joint biomechanics: Hypothyroidism may lead to changes in the mechanical properties of cartilage, such as decreased tensile strength and increased stiffness, which can alter joint biomechanics and increase the risk of OA development.
- 4. Accelerated cartilage aging: Thyroid hormones are also involved in the regulation of cellular senescence and aging processes. Hypothyroidism may accelerate the aging of chondrocytes, leading to premature cartilage degeneration and increased susceptibility to OA.

Clinical Implications and Potential Therapeutic Considerations

The potential association between OA and hypothyroidism has clinical implications for the management and treatment of these conditions (Cira et al., 2023).

1. Screening and early detection: Given the increased prevalence of hypothyroidism in individuals with OA, it may be beneficial to consider

routine screening for thyroid dysfunction in OA patients, particularly those with atypical or severe symptoms.

- 2. Thyroid hormone replacement therapy: Some studies have suggested that the treatment of hypothyroidism with thyroid hormone replacement therapy may have a beneficial effect on the progression of OA. However, the evidence is still limited, and more research is needed to fully understand the impact of thyroid hormone replacement on OA outcomes.
- 3. Integrated management approach: A multidisciplinary approach that addresses both the OA and the underlying thyroid dysfunction may be more effective in managing patients with these co-occurring conditions. This may involve a combination of pharmacological, non-pharmacological, and lifestyle interventions tailored to the individual's needs.

CONCLUSION

The relationship between osteoarthritis (OA) and hypothyroidism are two common conditions, is not only statistical, but also has complex and interrelated mechanisms. Understanding these mechanisms is critical for more accurate diagnosis and the development of effective prevention strategies. Thyroid hormone plays an important role in regulating bone metabolism, including the bone remodeling process. Thyroid hormone deficiency can disrupt the balance between bone resorption (breakdown) and bone formation (formation). Understanding these mechanisms is critical for more accurate diagnosis, development of preventive strategies, and effective treatment.

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