International Journal of Medicine and Medical Research
2021, Volume 7, Issue 2, p. 66-75

DOI 10.11603/ijmmr.2413-6077.2021.2.12426

BONE TISSUE METABOLISM AND CHANGES IN THE ORAL CAVITY IN REDUCED FUNCTIONAL ACTIVITY OF THE THYROID GLAND (literature review)

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Background. *Decreased functional activity of the thyroid gland leaves affects many organs and systems as well as bone tissue, pathological changes of which in the oral cavity are most often observed in periodontitis. However, the relationship between thyroid hypofunction and periodontitis or other inflammatory diseases of the oral cavity is still not confirmed.*

Objective. *The aim of the review was to study the published information and analyse bone metabolism and its relationships between autoimmune thyroiditis and oral diseases.*

Methods. *The articles in foreign periodicals on endocrinology, pathophysiology, dental surgery and therapy were the scientific sources for research.*

Results. *Understanding the mechanisms of bone metabolism under the action of thyroid hormones is an important aspect of treatment and diagnostic process, as local treatment of dental pathology without reducing the impact on systemic factors ultimately does not have any positive result. Decreased functional activity of the thyroid gland leads to homeostasis imbalance in the body. The thyroid hormones are important for bone metabolism, publications on periodontitis incidence in cases of autoimmune pathology of the thyroid gland are the most common. However, despite the number of studies, most authors agree that they are currently insufficient to clearly establish a causal relationship between autoimmune thyroid disease and maxillofacial disorders.*

Conclusions. *The study expands our knowledge, but there is still a need for further detailed studies that would clearly define the mechanisms of development of the disorders of the oral bone tissues and its relationships with autoimmune pathology of the thyroid gland.*

KEYWORDS: periodontitis; thyroid gland.

Introduction

Increased attention to the diseases of the thyroid gland is caused by challenging statistics on the increase in its incidence according to the Ministry of Health of Ukraine [1]. This trend is traced in the recent studies [2], their results show that the incidence of thyroid pathology has increased and is 46% of the total endocrinological morbidity. According to the author, during the last 5 years in the western region of Ukraine the incidence of hypothyroidism increased by 28.4%, the increase of thyrotoxicosis – by 8%, and the prevalence of thyroiditis – by 12.7% [2]. These indicators are higher than the national average indicators and that of the north-eastern regions.

According to official WHO data, about 1.5 billion people suffer from thyroid disease at present. However, despite the effective treatment of endocrine pathology, the tendency to its reduce in the world is not observed [3].

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Review

Hypothyroidism, as the most common pathology of the thyroid gland, surely affects the functions and morphology of all tissues and organs, the maxillofacial area as well. It is established that insufficient thyroid hormones adversely affect both tooth mineralization, bone mineral density and calcium-phosphorus metabolism that is clinically observed in the oral cavity as periodontitis, gingivitis or lesions of the tooth hard tissues [4,5]. Understanding the mechanisms of bone metabolism under the action of thyroid hormones is an important aspect of treatment and diagnostic, as local treatment of dental pathology without reducing the impact on systemic factors does not have any positive result [6,7].

It is established that bone tissue is a constantly renewing tissue where remodeling processes take, place i.e. the processes of formation and destruction of osteotissue, provided by osteoblasts, osteoclasts and osteocytes, which functional activity depends on exogenous and endogenous factors, some of which are

thyroid hormones. Although, according to Allen M.R. et al. [8] there are 2 types of remodeling. The first type – targeted remodeling, in which a specific local signal directs the osteoclast to a specific location to begin remodeling (e.g., in the areas of microdamage); the second – chaotic remodeling, a random process in which osteoclasts begin remodeling without any signaling. This type of recovery is significant in calcium homeostasis. At the cellular level, both types of remodeling are equal [8]. According to present scientists, the remodeling cycle has 5 stages (Fig. 1) [9].

In the *first* stage, the stage of activation, the stimulating signals by osteocytes and their transmission to cells of osteoclastic diferon are recognized. In response to this process, monocytic-macrophage cells are attracted to the bone surface, stimulated, proliferated and differentiated into osteoclasts. At the heart of the molecular understanding of regulation, RANKL (receptor activator of nuclear factor kappa B ligand) is a transmembrane ligand of the nuclear factor activator receptor produced by osteoblasts that activates lymphocytes and macrophages. RANKL molecules may remain attached to the surface of osteoblasts or stromal cells for some time. RANK is a transmembrane receptor of nuclear factor activator. RANKL interacts with RANK, which is accompanied by the fusion of several osteoclast progenitor cells into one large structure and mature multinucleated osteoclasts are formed. Thus, osteoblasts regulate formation of osteoclasts [11]. OPG-osteoprotegerin is a protein synthesized by osteoblasts and bone marrow stromal cells. It is proved that at the stage of osteoclast formation, the process can be blocked by the protein OPG-osteoprotegerin, which can bind to RANKL, that prevents formation of the RANKL/RANK complex and thus stops resorption processes [12].

According to recent studies [13], not only in osteoblasts but also in osteocytes, most of the recently synthesized RANKL form a protein complex with OPG and is selectively directed to lysosomes. Only a small fraction of newly synthesized RANKL, which does not form a complex with OPG, is transported to the cell surface. Subsequently, transmembrane RANKL is delivered to the surface of osteoclast precursors to stimulate RANK, and induce activation of the subsequent signaling pathway. According to the authors, the ability of osteocytes to support formation of mature osteoclasts probably depends on the number of RANKL molecules present on their cell surfaces. However, the way in which osteocytes embedded in the bone matrix deliver transmembrane RANKL to the cell surfaces of osteoclast precursors that are localized in the bone marrow cavity should be elucidated.

The *second* stage is the stage of resorption – due to production of enzymes osteoclasts destroy the bone matrix. According to some scientists, at this stage, activated osteoclasts are phagocytes for bone. Lysosomal collagenase is synthesized in large quantities that leads to disruption of the order in the structure of collagen. The products of hydrolysis enter the

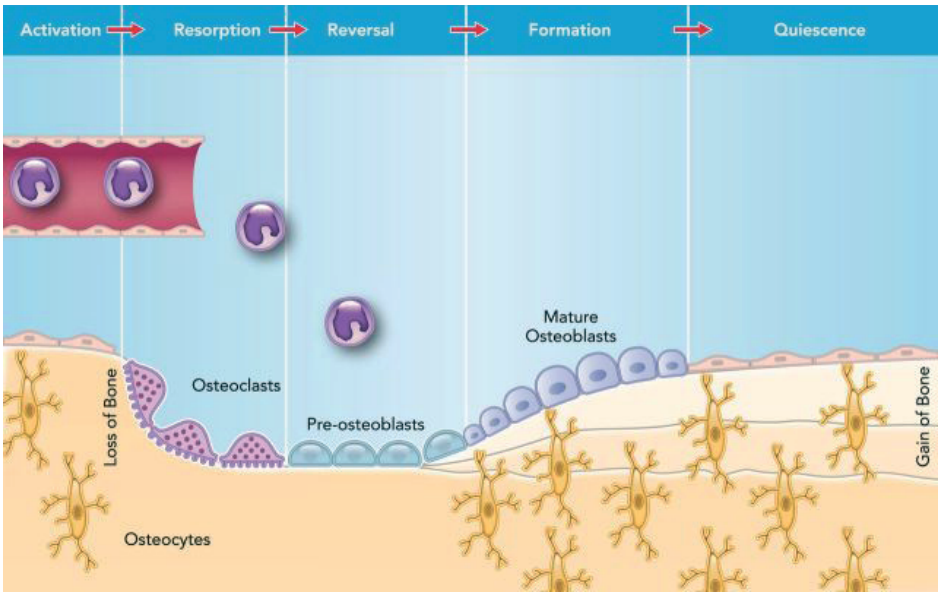

Fig. 1. *Physiological bone remodeling, J.A. Siddiqui and N.C. Partridge [10].*

osteoblast from the "corrugated bristles" by endocytosis, and are released from the basement membrane, which is in contact with the blood vessel. The acidic environment in the area of resorption promotes leaching of calcium from apatites; the so-called "resorptive bone gaps" are formed; as a result, calcium and phosphates get into the blood.

The *third* stage, the stage of reversion, osteoclasts undergo apoptosis, they are replaced by mesenchymal cells, which differentiate into osteoblasts [15]. Active osteoclasts synthesize and secrete acid phosphatase, which dephosphorylates osteopontin, a sialoprotein that attaches to cells in the resorption zone. The connection with the bone surface becomes weak, so the resorption gradually decreases. A cementing line (a layer of secretory glycoproteins) is formed on the resorbed surface, which is able to hold colonies of osteoblasts, an additional prerequisite for this is the availability of local osteoprogenitors [16].

The *fourth* stage, the stage of bone formation, osteoblasts synthesize the main organic substance of the bone matrix – collagen and substances that regulate mineralization (osteocalcin, osteonectin, etc.) [17]. The "matrix bubbles" of osteoblasts are significant in the process of mineralization. Amorphous $Ca_3(PO_4)_2$ is formed first in them, and later – hydroxyapatite: "matrix bubbles", which enter the extracellular space, contain high concentrations of calcium ions, according to Vavilova T.P., in 25-50 times more, than in osteoblasts, as well as enzymes: alkaline phosphatase, pyrophosphatase. In the intercellular matrix, membrane vesicles are destroyed with the release of calcium ions. Due to the influence of alkaline phosphatase, Ca^{2+} ions combine with PO_4^{3-}, resulting in the formation of amorphous calcium phosphate. At the same time, Ca^{2+} and PO_4^{3-} ions bind to collagen and non-collagen proteins and matrix is formed, which is accompanied by the formation of nuclei. On the formed nucleus there are spiral structures, the growth of which takes place on the principle of adding new ions. The step of such a spiral is equal to the height of one structural unit of the crystal. Crystal formation leads to the appearance of other crystals, this process is called epitaxis or epitaxic enucleation [18].

Thus, mineralization occurs through the formation of calcium phosphate compounds that enter the bloodstream and their subsequent crystallization into hydroxyapatite followed by deposition of calcium hydroxyapatite along the collagen fibers [18]; osteoblasts play the main role in collagen synthesis [10].

The *fifth* stage, the final stage, is characterized by differentiation of osteoblasts into osteocytes. After the bone formation stage is finished, the resting stage takes place, the osteoblasts are walled up in the matrix created in them, they lose activity and transform into osteocytes [19], which even recently were considered low metabolically active cells. But, as has been discovered, due to the mechanosensory properties that occur through the induced flow of fluid through the lacuno-tubular system, they regulate remodeling processes [20] and, as studied by Cappuli [21], regulation is carried out by sclerostin protein (SOST), which inhibits osteoblast differentiation.

The process of remodeling is regulated by numerous hormonal and local factors, neuroendocrine and metabolic (Fig. 2) [10]: glucocorticoids reduce synthesis of osteoblasts, which slows down formation of bone tissue; the influence of thyroid hormones (thyroxine and triiodothyronine) increases activity of osteoclasts, which contribute to catabolism of bone tissue. At the same time, sex hormones, especially estrogens, have antiresorptive properties. Somatotropic hormone stimulates proliferation of osteoblasts and growth factors. It is established that with a decrease of the concentration of Ca^{2+} ions in the blood the secretion of parathyroid hormone (PTH) increases, which is produced by cells of the parathyroid glands, and under its influence activates osteoclasts in bone tissue that increases bone resorption. As Ca^{2+} ions increase, the hormone calcitonin is secreted in the blood, which is produced by parafollicular thyroid cells and which bone mineralization increases and the number of osteoclasts reduces, i.e. resorption processes inhibits and, consequently, bone formation accelerates.

Vitamin D is important in regulation of concentration of Ca^{2+} ions in the blood, which are involved in the biosynthesis of Ca^{2+}-binding proteins required for intestinal calcium absorption, renal reabsorption and mobilization of calcium from bones. Recently, evidence has emerged that vitamin D is involved in development of many autoimmune diseases, including patients with autoimmune thyroid disease (AITD) [22].

It is established that thyroid hormones directly affect both remodeling processes, as they activate both osteoblasts and osteoclasts, and the process of calcium-phosphorus me-

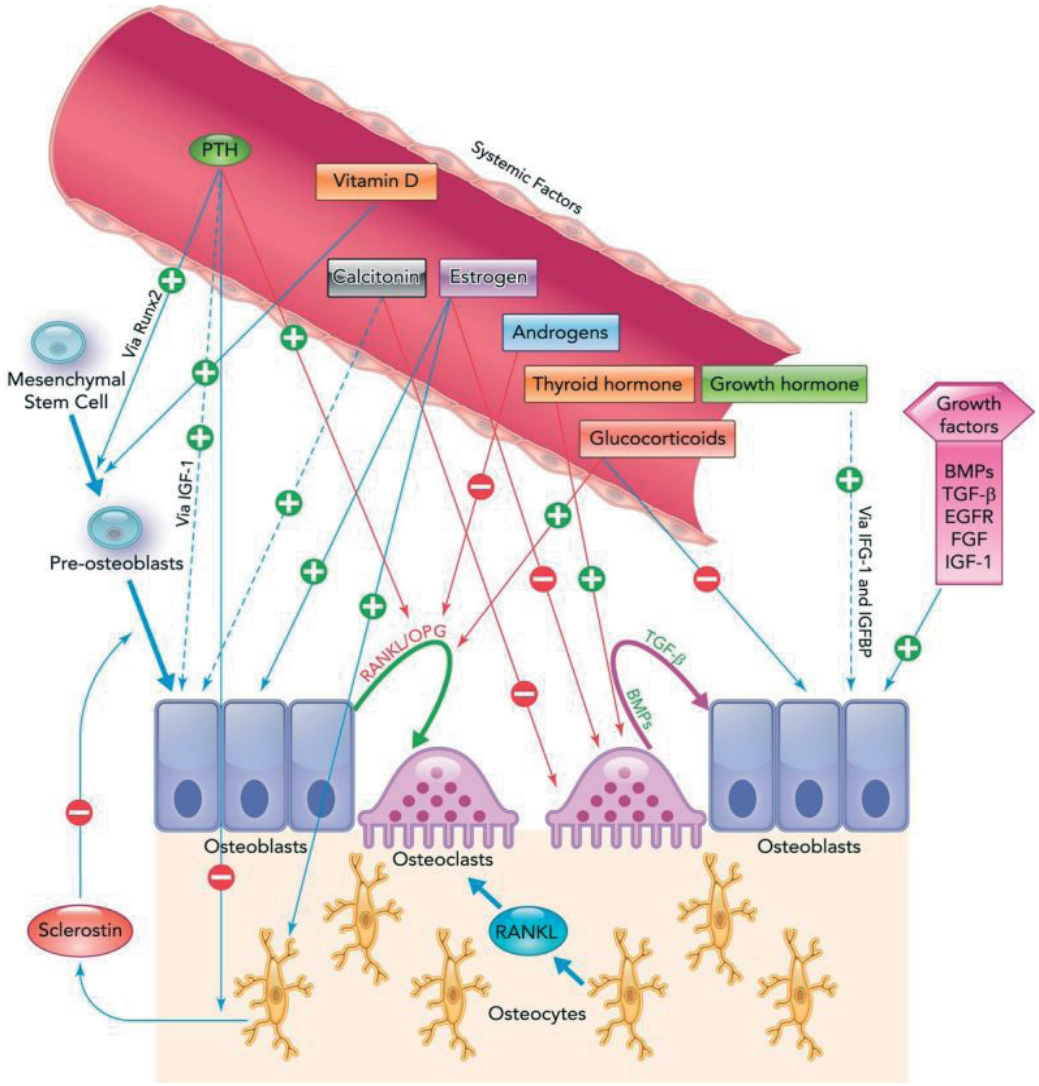

Fig. 2. *Systemic regulation and growth factor of bone remodeling, J. Siddiqui, N. Partridge [10].*

tabolism [24]. The thyroid gland secretes thyroxine (T_4), triiodothyronine (T_3) and calcitonin. The functional activity of the gland is regulated by thyroid-stimulating hormone (TSH), which is synthesized by the pituitary gland in response to the secretion of thyroliberin (TRG) by the hypothalamus. With a decrease in the concentration of thyroid hormones the secretion of TSH increases resulting in an increase in their formation in the thyroid gland, and, vice versa, with an increased concentration of thyroid hormones the formation of TSH decreases [17, 24]. Thus, this cooperation of the main endocrine gland functions on the principle of "negative feedback" that ensures a constant level of hormones. Violation of the concentration of one of the elements of the chain leads to changes in others that ultimately leads to dysfunction of both the endocrine glands and other organs and systems that depend on them.

In the context of bone tissue, according to Pankiv I.V. [4], and as seen at the scheme, thyroid hormones affect bone metabolism by increasing the activity of osteoclasts, which contribute to bone catabolism. There are studies [4, 25, 26], in which the authors indicate of osteoporosis of bone tissue with underlying hypothyroidism. According to [4], in persons with thyroid pathology, changes in bone mineral density were detected in 59 (39.9%) cases: osteopenia – in 45 (30.4%) and osteoporosis – in 14 (9.5%) cases. The incidence of osteopenia and osteoporosis was likely to increase in all groups of patients with thyroid functional disorders. The main factor that leads to decrease in bone strength in patients with thyroid disease is excessive or insufficient production

of thyroid hormones, as well as treatment with suppressive doses of levothyroxine. It is proved that the effect of thyroid hormones on the body cells is caused by the presence of receptors (TR) on their surface (Fig. 3). It is established that there are several types of thyroid receptors: TRα1, TRα2, TRβ1, TRβ2 [10]. It was established that only TRα and TRβ receptors on the surface of osteoblasts and chondrocytes were detected in bone cells [28]. Accordingly, T_3 (triiodothyronine) induces osteogenesis by direct action on osteoblasts. However, recent studies [29] have proved that expression of thyroid receptor (TR) genes α1 and β1 is confirmed in osteoclasts, but it is still indefinite whether triiodothyronine (T_3) stimulates osteoclast activity directly or whether these processes are the result of T_3 action in osteoblasts, osteocytes or other cells.

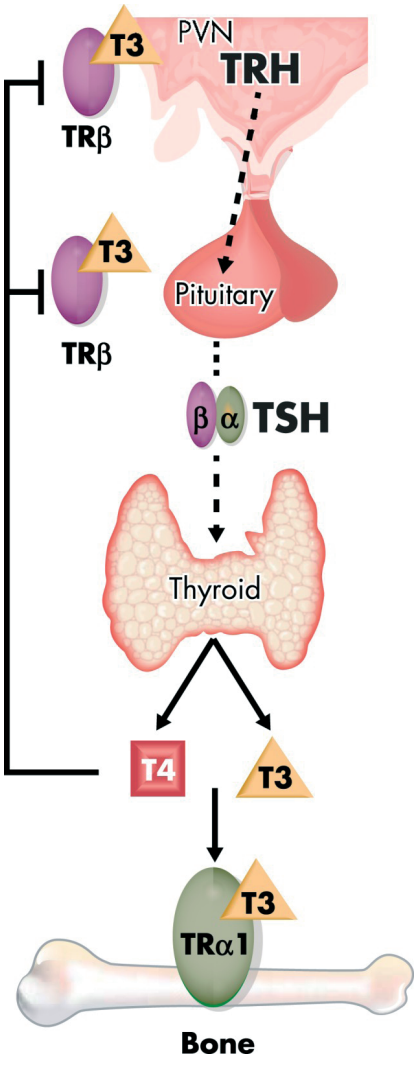

Fig. 3. *The thyroid gland secretes the prohormone T4 and the active hormone T3, and circulating concentrations are regulated by the classical endocrine cycle of negative feedback, which maintains the physiological feedback between TSH and T_4 and T_3. PVN, paraventricular nucleus (Bassett J.H, Williams G.R.) [28]*

As for the TRβ2 receptor, there is evidence that it is associated with the hypothalamus and pituitary gland, where it inhibits the secretion of TRG and TSH, so the hypothalamic-pituitary-thyroid relationship is important in regulation of bone metabolism.

To date, there are still debates on the key role in the functioning of bone tissue:by TSH or thyroid hormones [29]. The recommendations of the American Thyroid Association [30] state that serum TSH levels are one of the most informative indicators of thyroid function, and the Association recommends that all patients have serum TSH levels determined from the age of 35 and monitored every 5 years, which is an important diagnostic aspect.

However, confirmation is found in the literature: hypothyroidism causes general hypometabolism [31], a decrease in osteoblast formation and resorption of osteoclasts, and leads to low bone metabolism or slowing down the remodeling process. According to the author, the processes of osteo formation are slowed down by 50%, the processes of resorption – by 40% [31]. Calciuria decreases, serum concentrations of osteocalcin and alkaline phosphatase decrease, but the concentration of parathyroid hormone and vitamin D in the serum may increase [31].

Analysing this data and drawing parallels with clinical symptoms, scientists pathogenetically distinguish the following types of hypothyroidism:

1. Primary hypothyroidism caused by primary pathology of the thyroid gland, which is divided into hypothyroidism due to a decrease in the amount of functionally active tissue of the gland and impaired biosynthesis of thyroid hormones.

2. Secondary (pituitary) hypothyroidism caused by a decrease in TSH production.

3. Tertiary (hypothalamic) hypothyroidism due to a decrease or production of thyroliberin.

4. Peripheral (tissue) resistance to thyroid hormones [24].

Autoimmune thyroiditis (AIT) is the most common cause of primary hypothyroidism [32]. Taking into account the complex mechanism of metabolic thyroid hormones metabolism, the question is whether thyroid dysfunction affects the course of oral diseases, the development of which is accompanied by destructive processes in bone tissue.

Periodontal diseases are foremost in the structure of dental pathology due to the significant spread among the population of Ukraine [33]. This pathology is characterized by inflammatory-destructive changes of the periodontium, progressive nature of their course and leads to early tooth loss [34]. According to recent literature, the causes for this are both local and systemic factors [35], and sometimes a combination of both. Local factors include microbial film, small orifice of the mouth, pathology of the bridles and the existing strands of the mucous membrane, crowding of the teeth, impaired occlusion. Regarding the systemic factors that contribute to development of periodontal diseases, chronic cardiovascular, digestive and endocrine diseases are important. Most scientists today are inclined to the dominant role of the influence of microorganisms and tissue inflammatory response as a consequence of their activities [36]. The most common microorganisms of the dental microbial film are gram-negative anaerobic bacteria Porphyromonas gingivalis, Tannerella forsythia, Treponema denticola, Prevotellaintermedia [37, 44]. The mechanism of action of microorganisms is explained by their penetration through the connective tissue epithelium of the gingival sulcus that disrupts the integrity of the gingival junction and affects the periodontal tissues located deeper [37]. In cases of the inflammatory process in the periodontium microcirculation is violated that is accompanied by an increase in vascular permeability with insudation of blood plasma proteins into the walls of blood vessels and perivascular tissue. It has been proved that pathology in periodontal vessels is a trigger for progression of periodontitis [38]. In a comparative study of the microcirculation of healthy patients and in the examination of persons with periodontitis of varying severity (1-3 degrees), (127 people), according to laser Doppler flowmetry, it was found that in periodontitis 1st degree severity, in hemodynamics is decreased, compare to the healthy group of people, congestion in the microcirculatory tract is present with further development of rheological disorders. In generalized periodontitis of the 2nd degree, the main indicators of tissue blood flow are reduced, compare to the previous group, and in periodontitis of the 3rd degree microcirculatory disorders worsen with the involvement of all parts of compensatory regulation [38]. Thus, violation of microcirculation in the presence of microbial mechanisms leads to slowing of blood flow, venous stasis, impaired vascular transport [40].

Due to the possible systemic effects of thyroid dysfunction, periodontal tissue microcirculation in this aspect was studied by Scardina G.A., Messina P. (2008) [41], who assessed morphological microcirculation of interdental papillae in patients with Hashimoto's thyroiditis and possible associated periodontal disease. It was emphasized that the group of healthy patients deliberately did not involve the persons with conditions that disrupt microcirculation, such as diabetes or hypertension. All patients did not smoke. Microcirculation was assessed by capillaroscopy. For each patient, visibility, course, tortuosity, average capillary loop size and number of visible capillary loops per square millimeter were investigated. In patients with Hashimoto's thyroiditis, a reduced capillary caliber, as well as a greater number and tortuosity of capillary loops were evidenced. This study showed that changes in the capillaries in patients with Hashimoto's thyroiditis occurred in cases of violation of gums microcirculation that is characteristic of periodontitis.

The opinion that periodontitis is a multifactorial disease with a microbial initiator, the manifestation and progression of which is predisposed by a wide range of factors, one of which is Hashimoto's thyroiditis, is more and more popular [42, 43]. According to Molaris A. et al. [42], after analyzing the data of 30 articles on the relationship between periodontitis and Hashimoto's thyroiditis regarding etiopathogenetic mechanisms, have established that it occurs because some of these mechanisms are accompanied by vascular endothelial dysfunction, microcirculation disorders, as well as due to the impact of hypothyroidism on alveolar protease metabolism, but a causal relationship between the two nosologies requires further research.

According to Patil B., Patil S., Gururaj, T. (2011) [7], their study was initiated due to the lack of effective local therapy of periodontitis in thyropatients. Kothiwale S. et al. [5] presented a very interesting study regarding the impact of thyroid hormone dysfunction on the progression of periodontitis, systemic health of the patient, in which local treatment of periodontal tissues as a complex with endocrine compensation was proved. It was emphasized that the etiotropic phase of dental treatment lasted 8 weeks with the prescribed 150 mg of systemic thyroxine per day. During the dynamic ob-

servation, the hygienic index of the oral cavity improved, but the bleeding did not disappear. After 12 weeks of follow-up, after stabilization of thyroid hormones, a clear decrease in gingival bleeding was evidenced. The need for frequent professional evaluation, training of patients, motivating them to frequent systematic examinations was emphasized, as the treatment of such patients and achieving long-term remission would provide a positive result only in endocrinologist-dentist tandem.

According to Brankhar R.R. et al. (2017) [43] the endocrine system works together with the immune system. Despite the fact that the bilateral effects of systemic diseases on the periodontium are proved, there are a few studies on the effects of periodontal therapy on hormone levels. In the study, the effect of non-surgical periodontal therapy (NSPT) on serum stimulating hormone (TSH) levels in patients with hypothyroidism and periodontitis was assessed. Clinical parameters and serum TSH levels were recorded at baseline in the experimental and control groups and compared with TSH data in 3 months after NSPT in the patients with hypothyroidism. The results of the study showed that NSPT was significant in improving the condition of the periodontium by reducing inflammatory markers and thus affecting thyroid hormone, a significant decrease in TSH in patients with hypothyroidism in 3 months after NSPT.

Chingiz Ragim Ogly Rakhimov, (2020) [44] presented data on the clinical efficacy of hyaluronic acid in the treatment of periodontal disease in patients with hypothyroidism. According to the evaluation of the main hygienic and periodontal indices, it was found that a decrease in the content of thyrohormones led to an increase in the frequency and inflammatory-destructive forms of periodontal disease. In such patients, high-frequency of Porphyromonasgingivalis (25% and 15% in somatically healthy patients) and increased colonization of yeast-like fungi of the genus Candida albicans were evidenced in the oral cavity. After the treatment, there was a positive clinical dynamic, but in patients with impaired gland function had worsening of the oral cavity in a month that confirmed the idea of ineffectiveness of only local dental treatment and the need for dynamic monitoring of hormonal status.

There are studies on effectiveness of intraligamentous administration of vitamin D and calcium in the treatment of chronic periodontitis associated with hypothyroidism. In 3 months, there was a significant decrease in mobility, pocket depth and bleeding in the treatment of chronic periodontitis associated with hypothyroidism. The need for clinical trials with a large sample size and long-term observations was emphasized [45]. The significance of vitamin D in the development of autoimmune thyroiditis was covered by Bizzaro G. et al. [46, 47].

There is no doubt that patients with established hypothyroidism need a replacement therapy, and the study of periodontal status when taking thyroxine is the basis of further research [48]. After analyzing the plaque index, bleeding index, pocket probing depth [PPD], level of clinical attachment [CAL] and radiological parameters, in the study group (52 patients) statistically significantly higher PPD and loss of clinical attachment compare to the control group was established. With the beginning of treatment of periodontitis and hypothyroidism improvement in oral hygiene and a decrease in bleeding gums was evidenced. Regression analysis showed that hypothyroidism and thyroxine replacement therapy were important predictors of PPD and CAL, but it still requires further study. The same opinion is traced in the study by Hajer A. Aldulaijan et al. (2020), who analyzed 847 publications and applied their inclusion and exclusion criteria; thus only 29 publications were selected, which were more critically analyzed. As a result, only four publications were used to further assess the hypothyroidism-periodontitis relationship, including one research note on association between hypothyroidism and periodontitis. Hence, further well-controlled, clinical and immunological studies are needed to confirm this relationship [6].

Conclusions

The results of the studies prove that there is a significant effect of the thyroid gland on the state of the oral cavity that can be manifested by periodontitis, which is accompanied by bone destruction and inflammatory processes in the gum tissue. Local treatment of dental pathology without correction of thyrohormonal status does not provide effective treatment, so the correct diagnosis, selection of treatment and medical cooperation of a dentist and endocrinologist is necessary. Such disorders are common, but there is still a lack of accurate and improved examinations of this problem, which will be the goal of our further research in the future.

Conflict of Interests

Authors declare no conflict of interest.

Authors' Contributions

Olha Skochylo – investigation, formal analysis, writing – original draft. *Svitlana Boitsanyuk* – formal analysis, writing – reviewing and editing, data curation. *Nataliya Tverdokhlib* – conceptualization, methodology, writing – reviewing and editing.

ОСОБЛИВОСТІ МЕТАБОЛІЧНОГО ОБМІНУ У КІСТКОВІЙ ТКАНИНІ ТА ЗМІНИ ЗІ СТОРОНИ РОТОВОЇ ПОРОЖНИНИ НА ГРУНТІ ЗНИЖЕННЯ ФУНКЦІОНАЛЬНОЇ АКТИВНОСТІ ЩИТОПОДІБНОЇ ЗАЛОЗИ (огляд літератури)

***О.В. Скочило, С.І. Бойцанюк, Н.О. Твердохліб**

ТЕРНОПІЛЬСЬКИЙ НАЦІОНАЛЬНИЙ МЕДИЧНИЙ УНІВЕРСИТЕТ ІМЕНІ І.Я. ГОРБАЧЕВСЬКОГО МОЗ УКРАЇНИ, ТЕРНОПІЛЬ, УКРАЇНА

Вступ. *Зниження функціональної активності щитоподібної залози залишає свій слід на багатьох органах та системах. Не виключенням є і кісткова тканина, патологічні зміни якої в ротовій порожнині найчастіше спостерігаємо при пародонтитах. Проте, все ще залишається не підтвердженим взаємозв'язок між гіпофункцією тиреоїдної залози та пародонтитом чи іншими запальними захворюваннями ротової порожнини.*

Метою *нашого огляду власне, і було дослідження опублікованої інформації та її аналіз щодо метаболізму кісткової тканини та її взаємозв'язоку між автоімунним тиреоїдитом та захворюваннями ротової порожнини.*

Методи. *Науковими джерелами були статті у зарубіжних періодичних виданнях з ендокринології, патофізіології, хірургічної, терапевтичної стоматологій.*

Результати. *Розуміння механізмів кісткового метаболізму під дією гормонів щитовидної залози є важливим аспектом лікувально-діагностичного процесу, оскільки місцеве лікування стоматологічної патології без виключення впливу на системні чинники у підсумку не дає позитивного результату. Зниження функціональної активності щитоподібної залози призводить до дисбалансу в гомеостазі організму. Оскільки гормони щитоподібної залози відіграють важливу роль у обміні кісткової тканини, то найчастіше зустрічаються публікації стосовно виникнення пародонтиту на фоні автоімунної патології щитоподібної залози. Проте, незважаючи на існуючі дослідження та зафіксовані зміни, більшість авторів згідні з думкою, що таких досліджень на даний час є недостатньо, щоб чітко встановити причинно-наслідковий взаємозв'язок між автоімунним процесом щитоподібної залози та патологією щелепно-лицевої ділянки.*

Висновки. *Представлені дані розширюють існуючі знання, проте все ще існує потреба в подальших сучасних детальних дослідженнях, які б чітко дали відповідь на механізми виникнення та розвитку взаємозв'язку між автоімунною патологією щитоподібної залози та станом кісткової тканини ротової порожнини.*

КЛЮЧОВІ СЛОВА: пародонтит; щитоподібна залоза.

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Received 19 November 2021; revised 26 November 2021; accepted 7 December 2021.