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THE INFLUENCE OF THYROID PATHOLOGY ON THE COURSE OF PERIODONTAL DISEASE IN CHILDREN AND WAYS TO CORRECT IT (REVIEW)

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Iodine deficiency diseases constitute an important medical and social problem, since a significant part of the territory of Ukraine is characterized by biogeochemical indicators of low iodine content in the environment, which is the cause of hypothyroidism development. At the same time, there are many other factors that can trigger the thyroid disorders in children. Epidemiological studies carried out in the Carpathian region have shown that iodine deficiency of varying degrees is specific to this area. Professor M.M. Kovalev's in-depth study of the causes of goitre in Bukovyna showed that along with iodine deficiency, macro- and micronutrient deficiencies associated with certain regions play an important role. Iodine deficiency promotes goitrogenesis and decreases the secretory capacity of the thyroid gland. As a result of reduced intrathyroid iodine in the gland, diiodothyrosine synthesis decreases and monoiodothyrosine increases, the hormone ratio increases due to an increase in the more biologically active triiodothyronine. This compensates for the euthyroid state against the background iodine deficiency. An adaptive manifestation of iodine deficiency is goitre, considered being the main sign of iodine deficiency; as iodine deficiency or thyroid hormone requirements increase, goiter size increases and more severe forms of thyroopathology develop. Iodine deficiency is common in many countries of the world. The analysis of the research data has shown the multifactorial nature of the etiology and pathogenesis of periodontal diseases in children, as well as the significant impact of thyroid disorders on the condition of the human dentition. In this regard, the study of cause-effect relationships between the initial manifestations of thyroid pathology and periodontal tissue changes in children seems relevant and promising in terms of improving methods of diagnosis, prevention and treatment.

Keywords: *periodontium, diffuse non-toxic goitre, children.*

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Iodine deficiency diseases constitute an important medical and social problem, since a significant part of the territory of Ukraine is characterized by biogeochemical indicators of low iodine content in the environment, which is the cause of hypothyroidism development. At the same time, there are many other factors that can trigger the thyroid disorders in children [1; 2].

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Epidemiological studies carried out in the Carpathian region have shown that iodine deficiency of varying degrees is specific to this area [3–6]. Professor Kovalev's M.M. in-depth study of the causes of goitre in Bukovyna showed that along with iodine deficiency, macro- and micronutrient deficiencies associated with certain regions play an important role [7].

Iodine deficiency promotes goitrogenesis and decreases the secretory capacity of the thyroid gland. As a result of reduced intrathyroid iodine in the gland, diiodothyrosine synthesis decreases and monoiodothyrosine increases, the hormone ratio increases due to an increase in the more biologically active triiodothyronine. This compensates for the euthyroid state against the background iodine deficiency. An adaptive manifestation of iodine deficiency is goitre, considered being the main sign of iodine deficiency; as iodine deficiency or thyroid hormone requirements increase, goiter size increases [8–10] and more severe forms of thyroopathology develop. Iodine deficiency is common in many countries of the world.

The development of any disease in children, especially of a chronic nature, with grade 1 goiter increases by more than 20%, grades 2 and 3 of goiter by more than 40% [11; 12]. Predominant are the processes of stunting of growth and development of children's body, while in modern children there is a tendency to acceleration. Iodine deficiency leads to disorders in the physical development of children due to decreased body weight, reduced growth, and a sharp disharmony of development; in girls, there is delayed sexual development and menstrual disorders, and delayed puberty in boys; in children, there is a decrease in immunological reactivity and changes in the cytokine profiles [13–16].

Immunocompetent cell imbalance and activation of the humoral immune system are observed in children affected with DIC [16]. Thyroid hormones have been shown to enhance the immune response by influencing lymphocytes directly or by stimulating metabolic processes in lymphoid organs [17]. At the same time, the expression of IL-1, IL-2, IL-4, IL-6, IL-10, TNF-α and IFN-γ has been established in thyroid cells. However, it is known that gene expression does not necessarily correlate with protein production itself and depends on many factors, in particular cytokines, thyrotropic hormone, iodine, complement components, etc. [16].

In schoolchildren aged 13–17 years with mild iodine deficiency and euthyroidism, a probable decrease in the total T-lymphocyte population, an increase in CD8+ cells and a decrease in the immunoregulatory index have been found [17–18].

Fundamental epidemiological studies by Kerimov E.A., Gorzov I.P. and Politun A.M. have shown that periodontal tissue disease and the intensity of dental caries lesions are often intertwined with thyreopathology, which is explained by a natural deficiency of fluoride and iodine [18], as confirmed by other authors [19; 20].

According to the research data [21], the occurrence of periodontal disease in children with comorbid endemic goitre is 68.8%. In 17.8% of these children, teething disorders occur.

In 1997, Bidenko N.V. [19] examined children with diffuse euthyroid goiter at stages Ia, Ib and II, and found a high occurrence and intensity of dental caries, as well as a significant gingivitis incidence (41.0%) in them. In 29.75% of the children, serum calcium and inorganic phosphate levels were found to be different from normal values. Deficiencies in specific and non-specific local immunity were observed.

Studies conducted in our country have shown that one-third of children with endemic goiter show signs of various forms of dental hypoplasia, half of them have decompensated forms of caries; and more than 20% of those examined exhibited

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a simultaneous presence of caries and hypoplasia. In the pathogenesis of hard tissue lesions of teeth, the authors emphasize a decrease in local non-specific defense reactions, in particular the levels of lysozyme and sIgA activity [23].

Enamel hypoplasia has been found to occur more frequently in children with endocrine pathology (diffuse non-toxic goiter (DNG), thyroiditis) and gastrointestinal pathology. Deficiencies of hard dental tissues in these children are widespread (40.82%), which coincides with the prevalence of non-carious dental lesions in children living in the plain zone of Ukraine (48.95%), and almost 1.5 times higher than this figure in children living in conditions of iodine deficiency in the Carpathian region (27.5%). Hypoplasia of dental tissues in children is also seen against the background of hyperthyroidism, if the time of action of hormones coincided with the periods of dental formation [24].

In children with DNG, there are disorders of dental hard tissues and periodontal tissues mineralization due to changes in hormonal regulation of bone remodeling. In the oral cavity, there can be also observed imbalance in the pro-oxidant-antioxidant system, inactivation of protective mechanisms, increasing levels of dysbiosis, reducing the demineralizing potential of the oral fluid, as well as increasing local inflammatory processes [25].

Epidemiological studies conducted in Bukovina revealed high prevalence rates of major dental diseases in children with comorbid thyroid diseases: caries lesions in 91.19% of examined, periodontal pathology in 85.69%. Dental caries was observed in 94.29% of children with concomitant hypothyroidism, in 91.84% of children with DNG, and in 89.23% of cases of thyroiditis. Periodontal disease was registered in 86.73% of children with DNG, in 85.71% of children with hypothyroidism, and in 84.62% with thyroiditis.

Chronic catarrhal gingivitis (CCG) prevails in the structure of periodontal diseases [26].

Children with chronic thyroid disease were found to have significantly higher occurrence (89.9%) and intensity (5.02±1.25) of periodontal tissue diseases compared to practically healthy children, in whom these indicators were 10.7% and 1.36±0.44, respectively [27]. The authors believe that one of the pathogenetic mechanisms of periodontal tissue damage in such children may be changes in the immunological reactivity of the organism, namely, the predominance of changes in the humoral link of systemic as well as local protection of the oral cavity over cellular protection mechanisms, a significant increase of pro-inflammatory cytokines in blood serum, especially TNF-α and IL-6, compared to the anti-inflammatory link, which is proved by a reduced IL-10 content.

Published data indicate that body tissues, including periodontal tissues, are damaged frequently in thyroid dysfunction, with the degree and severity of the pathological processes depending on the severity and duration of hypothyroidism [27].

Almost 80% of young people with diffuse enlargement of the thyroid are diagnosed with CCG. Bleeding segments outnumber tartar segments in the structure of periodontal tissue disease [28].

Primary hypothyroidism is characterized by a latent course of chronic generalized periodontitis with simultaneous pronounced clinical manifestations, in particular gingival recession, loss of attachment, changes in immune homeostasis, decreased phagocyte activity, cellular immunity deficiency. Thus, the decrease in activity of macrophage/monocyte element of immunity leads to the decrease in activity of T-lymphocytes, the disturbance of phagocytosis, the decrease in concentration of IgM and increase in IgA and IgG level in the oral liquid, the disturbance of

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formation and circulation of immune complexes. Reduced levels of IgA and IgG in saliva lead to an imbalance of local immunity in children.

Cytokine status is characterised by an increased concentration of IL-1 in the oral fluid, the increased serum IL-8 and the decreased serum IL-4 in serum [28].

Hypothyroidism is known to cause dystrophic inflammatory periodontal disease, while hyperthyroidism induces primary inflammatory processes in the periodontium. At the same time, the structural and functional state of the bone system changes, as indicated by reduced forearm and alveolar mineral density in both diseases, indicating impaired bone metabolism [29].

In primary hypothyroidism, there is a significant increase in bone metabolism as evidenced by an increase in resorption markers, namely an increase in bone alkaline phosphatase isoenzyme activity and thyroid hormone levels, against the background of a decrease in bone formation markers, which is a valid predictor of osteodystrophy [30].

According to Rozhko O.P., hypothyroidism is associated with disorders in the structure of the dentoalveolar system, reduced mineralizing capacity of the pulp and jawbone, which leads to the development of atrophic processes in the periodontium and dental caries lesions. The author also notes that the incidence of inflammatory periodontal disease in children with DNG is twice as high as in healthy children [27].

The development of inflammatory diseases of periodontal tissues against the background of primary hypothyroidism is accompanied by a decrease in the level of the cellular element of immunity, an increase in IgA, IgG and IgM content, a decrease in lysozyme activity and an increase in sIgA content, and intensified processes of connective tissue destruction and osteoresorption of interalveolar septum tissue, which in turn dramatically exceeds the intensity of osteosynthesis and formation of connective tissue [32]. The author also notes a decrease in general immune resistance, which, in his opinion, is associated with the occurrence of thyroid hormone deficiency in hypothyroidism and, as a consequence, significant disorders of metabolic processes in the body involving a decrease in non-specific resistance, resulting in the development of inflammatory and inflammatory dystrophic changes in the periodontal tissues [33].

Generalised periodontitis with concomitant thyroid disease is characterised by a more severely damaged immune humoral and cellular response, with increased levels of certain populations of T-lymphocytes and B-lymphocytes, and decreased immunoglobulin concentrations [33].

In the case of endemic euthyroid goiter, inflammatory-dystrophic disease of the periodontal tissues manifests itself mainly in a moderate form of severity, with a third of cases diagnosed as severe disease. In goiter that has lasted for more than 5 years, severe periodontitis is more frequently detected, indicating the influence of the duration of thyroid disease on the course of dental pathology [34].

Besides, periodontal diseases in the presence of concomitant thyroid pathology are characterized by metabolic disturbances, in particular metabolic processes, increased blood proteolytic activity, and increased excretion of mineral components. Disturbance of homeostatic equilibrium in the body leads to destructive and inflammatory changes in periodontal tissues [27; 28; 30].

In summary, some correlations between dental pathology and thyroid pathology have been established. There are more in the case of thyroid dysfunction, but such changes are also observed in the case of the euthyroid state. However, not every aspect of the development and course of periodontal diseases in children

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against the background of thyroid diseases has been studied yet. In particular, it would be relevant to investigate changes in immunological and carbohydrate parameters in the oral fluid of children with concomitant DNG.

A review of the research data has shown that the scientific pursuit for the treatment of periodontal disease in children is aimed at optimising the treatment of CCG in children by means of a combined pathogenic therapy [32].

A complex of therapeutic measures for children with CCG living in environmentally unfavourable and fluorine- and iodine-deficient conditions was developed, including professional oral hygiene, traditional anti-inflammatories, Holisal gel applications, the use of Lakalut® activ herbal toothpaste and Lakalut® activ mouthwash. For general treatment, Kinder Biovital Lecithin Gel to enhance immune system function and Ascorutin to reduce capillary permeability and fragility were suggested [33].

Supieva E.T. (1996) suggests that children in endemic goitre areas should be treated with local remineralising agents and iodine-containing toothpastes [18].

A differentiated approach to solving the problem of treatment and prevention of dental diseases in children with DNG was implemented by Rozhko A.P. [26]. To this end, she developed a staged therapeutic and preventive complex, including elimination of iodine deficiency (Iodine-active), enhancement of immunity, regulation of thyroid hormonal activity (Selenium-active), use of adaptogens and antioxidants (Lecithin, Biotrit), remineralising agents (R.O.C.S. rinse and toothpaste) and osteogenesis stimulating agents (Calcicor), and anti-inflammatory agents (Lysodent). This enabled the author to obtain the prophylactic efficacy of 55.3% against caries and to significantly improve oral hygiene and periodontal indices in the patients.

According to the research data [32], in case of periodontal tissue diseases in children with DNG, it is advisable to correct macro- and microelement metabolism, if possible, systemically and locally. For this purpose, R.O.C.S. Bionica healing and prophylactic toothpaste with the active fractions of thyme and kelp, and R.O.C.S.-School mouthwash containing kelp extract, xylitol, calcium and magnesium should be used. The use of Teraflex in the complex treatment of CCG in the conditions of DNZ results in the restoration of the dentoalveolar structures built of the connective tissue.

In children with thyreopathology, mouthwashes with a 1–2% Achilles salt solution are recommended to increase the protective properties and remineralising function of the oral fluid [34].

Bidenko N.V. [19] recommends a combination of measures, including dietary adjustments, preventive examinations, the use of remineralising solutions, and rinsing the mouth with Diuciphonum, which greatly potentiate the benefits of caries and gingivitis treatment in children with diffuse euthyroid goitre.

According to Sarafanova A.B. [23], individualised iodine prophylaxis should be given as part of the treatment package to improve CCG management in young patients with thyroid hyperplasia.

It has been found that the combined use of Diplen-F film, Calcinova granulate and Iotrin in children with concomitant hypoplasia against the background of hypothyroidism improves local immunity [29].

Kolesnyk K.A. [22] recommends that in the course of orthodontic treatment of children with DNG, a complex of preparations with adaptogenic, osteotropic and prebiotic effects be used to restore microcirculation in the periodontal tissues.

In children affected by chronic thyroiditis, the use of a common regimen for the treatment and prevention of dental caries and gingivitis, including remineralising,

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antimicrobial and immunocorrective agents (Belagel Ca/P, Paragel) is recommended [35].

To manage generalised periodontitis in adolescents with thyreopathology, treatment and prophylactic measures with Erbisol have been recommended, which demonstrated a positive effect and elimination of the inflammatory process in the periodontal tissues [33].

In pubertal children, the treatment of CCG with the use of Echiposol oral gel, Colgate® Herbal Healing toothpaste and Colgate® Plax Total Herbal Rinse has proven effective [14].

Diseases of periodontal tissues caused by endocrine-immune pathology associated with iodine deficiency are effectively treated with Iodis-calendula and Iodis-concentrate in combination with Calcium-D3 Nycomed, which helps to normalise thyroid function and improve bone tissue metabolic parameters [35].

In patients affected by chronic generalised periodontitis against the background of primary hypothyroidism, the immunomodulator Galavit, organosilicon gel-based magnetophoretic compositions, Alloplant graft and autoplasma combinations are recommended [29].

Antioxidants in the complex of medical therapy for patients with concomitant thyroid pathology significantly increase the effectiveness of treatment and stabilize the pathological process in periodontal tissues.

According to the research data [34], patients affected by chronic generalised periodontitis with concomitant thyreopathology should be given a full range of preventive and curative measures, including the use of Elam-Dent iodine-containing toothpaste, at least four times a year.

To normalise bone metabolism in generalised periodontitis with concomitant thyroid pathology, the use of Calcium-D3 Nicomed and Fitor is recommended [28].

A treatment and prophylactic scheme for periodontal disease on the background of primary hypothyroidism, which includes the combined use of the antiseptic Holisal and the immunomodulatory complex Immunovel, was developed and introduced into dental practice with regard to the basic periodontal therapy and the correction of thyroid pathology [35].

Thus, practical dentistry has developed many approaches to the treatment and prevention of periodontal diseases in concomitant thyroid pathology in children and adults. However, the issues of improvement of treatment and prophylactic measures with regard to individual factors of the child's body and regional peculiarities remain topical. In particular, the study and correction of immunological changes and oral glycoprotein level abnormalities in children with CCG against the background of DNG are required, and that will constitute the subject of the study in our work.

The analysis of the research data has shown the multifactorial nature of the etiology and pathogenesis of periodontal diseases in children, as well as the significant impact of thyroid disorders on the condition of the human dentition. In this regard, the study of cause-effect relationships between the initial manifestations of thyroid pathology and periodontal tissue changes in children seems relevant and promising in terms of improving methods of diagnosis, prevention and treatment.

Conflict of interest: none declared.

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Кіцак Т.С.

ВПЛИВ ПАТОЛОГІЇ ЩИТОВИДНОЇ ЗАЛОЗИ НА ПЕРЕБІГ ЗАХВОРЮВАНЬ ПАРОДОНТУ У ДІТЕЙ ТА ШЛЯХИ ЇЇ КОРЕКЦІЇ

Хвороби щитоподібної залози займають чільне місце в структурі дитячої захворюваності. Йододефіцитні захворювання становлять важливу медико-соціальну проблему, оскільки значна територія України, за біогеохімічними показниками, характеризується низьким вмістом йоду у навколишньому середовищі, що є причиною розвитку гіпотиреозу. Водночас є чимало інших чинників, які впливають на організм дитини і призводять до розвитку захворювань щитоподібної залози. Епідеміологічні дослідження, проведені в Карпатському регіоні, показали, що йому притаманна різного ступеня йодна недостатність. Поглиблене вивчення причин виникнення зобу на Буковині професором Ковальовим М.М. показало, що разом із нестачею йоду, важливу роль відіграє дефіцит макро- і мікроелементів, що супроводжує певні регіони. Нестача йоду сприяє зобогенезу і зменшенню секреторної здатності щитоподібної залози. Внаслідок зменшення інтратиреоїдного йоду в залозі зменшується синтез дийодтирозину і зростає кількість монойодтирозину, збільшується співвідношення гормонів внаслідок зростання біологічно активнішого трийодтироніну. Цим компенсується еутиреоїдний стан на тлі нестачі йоду. Адаптивним проявом дефіциту йоду є зоб, який вважають основною ознакою йодної недостатності; зі зростанням дефіциту йоду чи потреби в тиреоїдних гормонах розміри зоба збільшуються. Виникають більш тяжкі форми тиреопатології. Йододефіцит притаманний багатьом країнам світу.

Ключові слова: *пародонт, дифузний нетоксичний зоб, діти.*

Кицак Т.С.

ВЛИЯНИЕ ПАТОЛОГИИ ЩИТОВИДНОЙ ЖЕЛЕЗЫ НА ТЕЧЕНИЕ ЗАБОЛЕВАНИЙ ТКАНЕЙ ПАРОДОНТА У ДЕТЕЙ И ПУТИ ЕЕ КОРРЕКЦИИ (ОБЗОР)

Болезни щитовидной железы занимают главное место в структуре детской заболеваемости. Йододефицитные заболевания представляют важную медико-социальную проблему, поскольку значительная территория Украины, по биогеохимическим показателям, характеризуется низким содержанием йода в окружающей среде, что является причиной развития гипотиреоза. В то же время есть немало других факторов, которые влияют на организм ребенка и приводят к развитию заболеваний щитовидной железы. Эпидемиологические исследования, проведенные в Карпатском регионе, показали, что ему присущая разной степени йодная недостаточность. Углубленное изучение причин возникновения зоба на Буковине профессором Ковалевым М.М. показало негативное влияние дефицита других макро- и микроэлементов в некоторых регионах Украины, что сочетается с недостатком йода. Недостаток йода способствует развитию зоба и уменьшению секреторной способности щитовидной железы. В результате уменьшения интратиреоидного йода в железе уменьшается синтез дийодтирозина и растет количество монойодтирозина, увеличивается соотношение гормонов вследствие роста биологически более активного трийодтиронина. Этим компенсируется эутиреоидное состояние на фоне недостатка йода. Адаптивным проявлением дефицита йода является зоб, считающийся основным признаком йодной недостаточности; с ростом дефицита йода и потребности в тиреоидных гормонах размеры зоба увеличиваются. Возникают более тяжелые формы тиреопатологии. Йододефицит присущ многим странам мира.

Ключевые слова: *пародонт, диффузный нетоксичный зоб, дети.*

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