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Thyroid cancer and autoimmune connective tissue disorders

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Abstract

There are substantial data confirming the association between autoimmune disorders, including connective tissue diseases (CTDs), and an increased risk of thyroid malignancy. CTDs and thyroid cancer may co-exist as 2 separate diseases because of their relatively high incidence rates in the population. They can arise from each other due to the increased risk of thyroid cancer in patients with idiopathic inflammatory myositis, rheumatoid arthritis, systemic sclerosis, primary Sjögren's syndrome, and systemic lupus erythematosus. Moreover, in some scarce cases, CTDs may act as the paraneoplastic syndromes of thyroid cancer. The presence of CTDs may impact the diagnostic process, especially distorting the results of imaging tests or falsely indicating the increase of thyroglobulin or calcitonin. Finally, TSH suppression is a crucial element of the treatment of differentiated thyroid cancer, which may decrease bone mineral density and increase the risk of osteoporosis by accelerating bone turnover and shortening the bone remodeling cycle.

The aim of this review is to emphasise the vital aspects of this interrelationship. The authors discuss this phenomenon aiming at the explanation of possible linking mechanisms. The impact of selected CTDs on thyroid cancer management is presented, as well as the possible effects of cancer therapy on skeletal health. **(Endokrynol Pol 2024; 75 (5): 455–460)**

Key words: *connective tissue disorders; thyroid cancer; TSH suppression; rheumatoid arthritis; systemic lupus erythematosus; primary Sjögren's syndrome*

Introduction

Thyroid cancer is the most frequent neoplasm of the endocrine system. Over 95% of thyroid cancer cases constitute differentiated thyroid carcinoma — predominantly papillary thyroid carcinoma and, secondly, follicular thyroid carcinoma [1]. The incidence rates of thyroid cancer are 10 per 100,000 in women and 3 per 100,000 in men, while the mortality rates are 0.5 per 100,000 in women and 0.3 per 100,000 in men [2].

The incidence of connective tissue diseases (CTDs) varies between the geographical region and the examined population. The newest data shows that the age-standardised incidence rate (ASR) of rheumatoid arthritis (RA) increased from 12.21 [95% confidence interval (CI): 11.13–13.38) to 13 (95% CI: 11.83–14.27)] per 100,000 population between 1990 and 2019 [3]. A meta-analysis focused on the prevalence and incidence of systemic sclerosis (SSc) showed that the overall pooled incidence rate of SSc is 1.4 (95% CI: 1.1–1.9) per 100,000 person-years [4]. A review concerning primary Sjögren's syndrome (pSS) showed that the estimated annual incidence of pSS varied from 3.9 per 100,000 (95% CI: 2.8–4.9) in Olmstead County, Minnesota, USA to 5.3 per 100,000 (95% CI: 4.5–6.1) in Greece; however, regardless the country of data origin, the incidence rates were higher in women [5]. The metanalysis from the UK reported that the incidence of systemic lupus erythematosus (SLE) oscillates between 1.4–8.7 per 100,000 persons and is higher in women (4.72 to 14.1 per 100,000 in women and 0.5 to 2.6 per 100,000 in men) [6]. The incidence of idiopathic inflammatory myositis (IIM) is estimated to range from 0.2 to 2 per 100,000 person-years [7].

Because autoimmune rheumatic diseases and thyroid cancer are characterised by relatively high incidence rates in the population, it is obvious that they co-occur. However, we should ask whether this is only a coincidence or whether there is a cause-and-effect connection. We should also consider whether treating one disease can influence the course of another disease or affect the functioning of healthy tissues.

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In this brief literature review, we aim to summarise and present the available knowledge and data from the literature concerning the coexistence of thyroid cancer and autoimmune rheumatic disorders.

Connective tissue diseases as risk factors for thyroid cancer

The published data show the increased risk of malignancy in patients suffering from autoimmune diseases. A large cohort study from China identified the 5 major autoimmune diseases that increase the risk of cancer. These 5 included idiopathic inflammatory myositis (IIM) with a standardised incidence ratio (SIR) of 3.37, rheumatoid arthritis (RA) — SIR of 3.99, systemic sclerosis (SSc) — SIR of 3.77, primary Sjögren's syndrome (pSS) – SIR 2.88, and systemic lupus erythematosus (SLE) — SIR 2.58. The association between autoimmune disease and cancer was more significant in females and younger patients (< 50 years) [8]. The SIR of thyroid cancer in the study cohort was 6.41, while SIRs of thyroid cancer for the RA group, pSS group, and SLE group were 12.58, 8.41, and 4.19, respectively [8].

The results of multiple cohort studies and meta-analyses indicate an increased incidence of thyroid carcinoma among patients with systemic connective tissue diseases. Undoubtedly, the presence of SLE is an important factor in cancer development, including thyroid cancer [9, 10]. In a meta-analysis evaluating the risk of 40 different malignancies, a 50% increase in the risk of developing thyroid cancer was observed in patients suffering from SLE compared to the general population [11]. Research by Chinese scientists confirmed the increased risk of overall cancer and cancer-related death in SLE patients. SLE was indicated as a risk factor for 17 site-specific cancers, including, among others, lung, larynx, vagina, cervix, anus, lymphoma, leukaemia, and thyroid cancer (RR = 2.31; 95% CI: 1.55–3.45) [12].

Patients with pSS are more prone to develop not only haematological malignancies but also solid tumours such as lung cancer or thyroid cancer. The observed number of thyroid cancers is twice as high as expected [13]. The data from French databases confirm a higher incidence rate of haematological malignancies such as Waldenström macroglobulinaemia, lymphomas, leukaemia, as well as thyroid cancers [14].

Korean studies show that RA is associated with a higher incidence of thyroid carcinoma than in the general population. Males, compared to females, were more prone to thyroid cancer (OR = 4.90; 95% CI: 1.49–16.07) and lung cancer [15]. Another study conducted in Korea, which focused only on the female population, showed that RA in women is associated with a higher (by 75%) incidence of thyroid cancer than in the general population [16]. However, while analysing the data from Korea we should be aware of the thyroid cancer "epidemic" in this country due to the specific screening approach resulting in overdiagnosis of this malignancy [17, 18].

Why are connective tissue diseases risk factors for thyroid cancer?

Taking into consideration the increased risk of thyroid carcinoma in the population of patients suffering from CTDs, we should consider the possible causes of this phenomenon.

A study from the UK showed that some autoimmune diseases tend to co-occur. However, this association differs between diseases and is highest among connective tissue diseases, particularly between SLE, pSS, and SSc. In patients with these diseases, the risk of developing a comorbid autoimmune thyroid disease (Graves' disease, Hashimoto's thyroiditis) is approximately 2 times higher compared to the general population [19]. A review and meta-analysis by Sun et al. indicated that the risk of thyroid disease in patients with pSS increased 3-fold compared to controls, while the OR of autoimmune thyroid disease and non-autoimmune thyroid disease were 3.48 (95% CI: 1.59–7.63) and 2.90 (95% CI: 1.51–5.57), respectively [20]. On the other hand, a multicentre cross-sectional study showed that the frequency of another autoimmune disorder is 9.67% in Graves' disease and 14.3% in Hashimoto's thyroiditis. The most common autoimmune disorder coexisting with thyroid autoimmunity was RA, which was found in 3.15% of Graves' disease cases and 4.24% of cases of Hashimoto's thyroiditis [21]. The crucial fact that facilitates understanding the increased incidence of thyroid cancer in patients with connective tissue diseases is that autoimmune thyroid diseases are risk factors for thyroid carcinoma [22, 23]. A review that focused on cytological studies by Boi et al. confirms the association between increased prevalence of papillary thyroid cancer and high titre of antithyroid antibodies and increased TSH concentrations, and is consistent with the hypothesis that autoimmune thyroid diseases promote tumour growth [24]. Another issue is that the oncogenes (*RET/PTC*, *RAS*, and *BRAF*), which are activated in thyroid carcinoma, may evoke a proinflammatory transcriptional reaction in thyroid cells, including cytokines, chemokines, and their receptors [25], especially C-X-C motif chemokine ligand 10 (CXCL10) — a molecule that recruits T1 helper lymphocytes. Lymphocytes express C-X-C chemokine receptor type 3 (CXCR3) and secrete interferone gamma (IFN-γ), which drives the autoimmune inflammation in endocrine glands [26].

When considering the mechanisms of association between autoimmune rheumatic disorders and cancer, we can distinguish 3 groups of patients. In the first group, the rheumatic disorder is the direct result of the tumour itself or its metastases. The second group comprises patients with a diagnosis of rheumatic disease who develop cancer in time due to the higher risk associated with the disease. The third group are patients with paraneoplastic rheumatic disorders, in whom the symptoms of connective tissue diseases precede the diagnosis of the malignancy by months or years [27].

Rheumatic diseases as the paraneoplastic manifestation of differentiated thyroid carcinoma are a rare phenomenon. In the literature we can find a few cases of paraneoplastic syndromes in patients suffering from papillary thyroid carcinoma, such as polymyalgia rheumatica [28], polymyositis [29], dermatomyositis [30], an adult-onset Still's disease [31–33]. A case of hypertrophic osteoarthropathy as a paraneoplastic syndrome of follicular thyroid carcinoma was also reported [34]. Although the correlation between connective tissue diseases and cancer seems obvious, the explanation of this phenomenon remains unclear in many cases. However, there are some hypotheses concerning the mechanism of the rheumatic manifestations of the malignancy. Firstly — one common pathogenetic factor for cancer and rheumatic disorder; secondly — that toxins excreted by tumours evoke tissue inflammation, which manifests as a rheumatic disorder. Finally, according to the third hypothesis, paraneoplastic rheumatic disorders result from the hypersensitivity reaction mediated by the tumoural antigens [27].

The disappearance of symptoms after successful surgery enforces the diagnosis of a paraneoplastic connective tissue disorder. This course of events occurred in the case of a patient diagnosed with vasculitis as a paraneoplastic manifestation of papillary thyroid cancer [35]. Although only a few cases of paraneoplastic vasculitis have been described in patients with papillary thyroid carcinoma, the topic requires more extensive discussion due to the interesting results of Korean researchers [36]. In this publication, thyroid cancer was the most frequent cause of paraneoplastic vasculitis, and the most common types of vasculitis included Behcet disease, granulomatosis with polyangiitis, and Takayasu arteritis. However, when analysing these data, again, it is vital to remember that thyroid carcinoma is the most frequent cancer in the Korean population (its incidence increased more than 5-fold in women and about 8-fold in men between 1999 and 2017) [37].

The influence of rheumatic disorders on diagnostic and therapeutic procedures in thyroid cancer

The next vital aspect is that the presence of CTDs can affect the diagnosis and treatment of thyroid cancer. Firstly, rheumatic diseases may distort the results of imaging tests, especially scintigraphy. A few cases of false positive iodine-131 (^{131}I) uptake in patients with pSS and thyroid carcinoma were reported. The possible mechanism of this phenomenon is the increased permeability of the vessels. It can be observed in patients suffering from pSS with systemic manifestations and vasculitis. This inflammation can result in ^{131}I retention in tissues, which can be falsely interpreted as the presence of cancer metastases [38].

Secondly, an essential fact is that heterophile antibodies can interfere with cancer marker (such as calcitonin and thyroglobulin) assays, falsely indicating the marker's increase [39]. The diagnosis of medullary thyroid carcinoma can be based on serum calcitonin concentration, and it is highly probable if the calcitonin concentration is above 100 ng/L. However, in doubtful cases, we may use the calcitonin stimulation test, which can improve the effectiveness of the preoperative diagnosis [40]. Regular marker assessment — especially thyroglobulin — combined with neck ultrasound, is the basis for monitoring a patient with differentiated thyroid cancer [40]. Therefore, it is crucial to consider interference from heterophile antibodies or rheumatoid factor in the case of discrepancies between the cancer staging, imaging results, and laboratory results to avoid unnecessary escalation of diagnostic tests and even therapy. Moreover, awareness of this problem and quick detection of laboratory bias will reduce the patient's stress resulting from concerns about their health.

Adverse effects of thyroid cancer therapy on connective tissue and bone health

Besides thyroidectomy and radioiodine treatment, a key element of differentiated thyroid cancer therapy is thyroid-stimulating hormone (TSH) suppression to reduce the risk of malignancy recurrence in high-risk patients or in patients who did not demonstrate an excellent response [40]. The suppression can be complete with TSH levels < 0.1 mIU/L or mild — TSH levels 0.1–0.5 mIU/L. Moreover, in patients after the treatment of differentiated thyroid carcinoma, the TSH levels should be kept below 2.0 mIU/L [40].

The concept of this therapy is based on TSH as a factor promoting the growth and proliferation of thyroid cells [40,41]. Suppression of TSH leads to iatrogenic hy-

perthyroidism and is associated with side effects such as osteoporosis, fractures, and atrial fibrillation. The excess of thyroid hormones during the suppression therapy impacts the bones by accelerating bone turnover and shortening the bone remodelling cycle [42]. A meta-analysis from 2021 confirmed a significantly lower bone mineral density of the lumbar spine and femoral neck in postmenopausal women who received TSH suppression therapy after thyroidectomy than in the control group. In contrast, premenopausal women on TSH suppression had significantly higher bone mineral density (BMD) in the lumbar spine and femoral neck than the control group. On the other hand, TSH suppression did not impact BMD in males [43]. The possible explanation for this discrepancy is the protective effect of oestrogen on bones [43].

Another meta-analysis focusing only on postmenopausal women showed that the detrimental impact of THS suppression therapy is more significant in the lumbar spine than in the femoral neck [37]. Moreover, the subgroup analysis showed that the harmful effect of TSH suppression therapy is unrelated to race, body mass index (BMI), and the length of follow-up. However, there was a significant difference in BMD of the femoral neck if the type of thyroid surgery was considered. That is, a deleterious influence on BMD of the femoral neck was observed for total thyroidectomy in contrast to less extensive surgical procedures [44]. This discrepancy can be explained by the structure of the lumbar spine and femoral neck. The lumbar spine is built of a trabecular bone that is more prone to osteoporotic changes than the cortical bone, which is the predominant part of the femoral neck [44].

Another parameter helpful or more appropriate for estimating the bone health of postmenopausal women is the trabecular bone score (TBS). A study from 2021 showed that the TBS is significantly lower in postmenopausal women on TSH suppression therapy than in those without TSH suppression, while BMD did not differ between the groups [45]. The authors of the review from 2022 found that TBS is a new independent predictor of osteoporotic fracture risk in postmenopausal women with thyroid cancer who receive TSH suppression therapy [46].

A Korean study confirmed that the osteoporosis odds ratio (OR) in patients with thyroid cancer was significantly higher (1.41; 95% CI: 1.18–1.70) than in the control group, particularly in women (OR: 1.43; 95% CI: 1.19–1.71). However, in this study, the increased risk of osteoporosis was not accompanied by the increased risk of fractures [47]. On the other hand, a systematic review by Lee et al. showed conflicting results with higher-than-expected fracture SIRs related to TSH suppression, observed mainly in studies including larger numbers of thyroid cancer patients [48].

Because TSH suppression therapy is a vital element of the treatment of thyroid cancer patients, it is necessary to be aware of an increased risk of osteoporosis [49]. When planning therapy, it is crucial to evaluate bone health and to perform densitometry (DXA) in each postmenopausal woman and man aged > 70 years and consider DXA in premenopausal women and men < 70 years with a high risk of osteoporosis and fragility fractures. After this evaluation, proper management with calcium (under the control of calcaemia), vitamin D (usually in dose 2000 IU/d), and antiresorptive therapy (if required) should be introduced [40, 50].

Conclusions

Connective tissue disorders, due to their autoimmune character, increase the risk of thyroid cancer. As paraneoplastic syndromes, they can be the first manifestation of thyroid cancer. Finally, they can impact the diagnosis and treatment. On the other hand, TSH suppression therapy in thyroid cancer patients should be considered as a risk factor for osteoporosis. Increasing the awareness of clinicians managing patients with connective tissue disorders and thyroid carcinomas should improve the quality of provided health care.

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Authors contributions
M.K.C. — study design and manuscript writing, A.H.D. — acceptance of the final version for publication, B.B. — conception of the article and acceptance of the final version for publication, M.T.M.— conception of the article and writing of the manuscript.

Conflict of interests
The authors of this paper report no competing interests.

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References

1. Cabanillas ME, McFadden DG, Durante C. Thyroid cancer. Lancet. 2016; 388(10061): 2783–2795, doi: 10.1016/S0140-6736(16)30172-6, indexed in Pubmed: 27240885.
2. Pizzato M, Li M, Vignat J, et al. The epidemiological landscape of thyroid cancer worldwide: GLOBOCAN estimates for incidence and mortality rates in 2020. Lancet Diabetes Endocrinol. 2022; 10(4): 264–272, doi: 10.1016/S2213-8587(22)00035-3, indexed in Pubmed: 35271818.
3. Cai Y, Zhang J, Liang J, et al. The Burden of Rheumatoid Arthritis: Findings from the 2019 Global Burden of Diseases Study and Forecasts for 2030 by Bayesian Age-Period-Cohort Analysis. J Clin Med. 2023; 12(4), doi: 10.3390/jcm12041291, indexed in Pubmed: 36835827.
4. Bairkdar M, Rossides M, Westerlind H, et al. Incidence and prevalence of systemic sclerosis globally: a comprehensive systematic review

and meta-analysis. Rheumatology (Oxford). 2021; 60(7): 3121–3133, doi: 10.1093/rheumatology/keab190, indexed in Pubmed: 33630060.
5. Patel R, Shahane A. The epidemiology of Sjögren's syndrome. Clin Epidemiol. 2014; 6: 247–255, doi: 10.2147/CLEP.S47399, indexed in Pubmed: 25114590.
6. Fatoye F, Gebrye T, Mbada C. Global and regional prevalence and incidence of systemic lupus erythematosus in low-and-middle income countries: a systematic review and meta-analysis. Rheumatol Int. 2022; 42(12): 2097–2107, doi: 10.1007/s00296-022-05183-4, indexed in Pubmed: 36006459.
7. Khoo T, Lilleker JB, Thong BYH, et al. Epidemiology of the idiopathic inflammatory myopathies. Nat Rev Rheumatol. 2023; 19(11): 695–712, doi: 10.1038/s41584-023-01033-0, indexed in Pubmed: 37803078.
8. Zhou Z, Liu H, Yang Y, et al. The five major autoimmune diseases increase the risk of cancer: epidemiological data from a large-scale cohort study in China. Cancer Commun (Lond). 2022; 42(5): 435–446, doi: 10.1002/cac2.12283, indexed in Pubmed: 35357093.
9. Han JY, Kim H, Jung SY, et al. Increased risk of malignancy in patients with systemic lupus erythematosus: population-based cohort study in Korea. Arthritis Res Ther. 2021; 23(1): 270, doi: 10.1186/s13075-021-02648-y, indexed in Pubmed: 34706772.
10. Hardenbergh D, Naik R, Manno R, et al. The Cancer Risk Profile of Systemic Lupus Erythematosus Patients. J Clin Rheumatol. 2022; 28(1): e257–e262, doi: 10.1097/RHU.0000000000001729, indexed in Pubmed: 33657590.
11. Clarke AE, Pooley N, Marjenberg Z, et al. Risk of malignancy in patients with systemic lupus erythematosus: Systematic review and meta-analysis. Semin Arthritis Rheum. 2021; 51(6): 1230–1241, doi: 10.1016/j.semarthrit.2021.09.009, indexed in Pubmed: 34710720.
12. Zhang M, Wang Y, Wang Y, et al. Association Between Systemic Lupus Erythematosus and Cancer Morbidity and Mortality: Findings From Cohort Studies. Front Oncol. 2022; 12: 860794, doi: 10.3389/fonc.2022.860794, indexed in Pubmed: 35600353.
13. Zhong H, Liu S, Wang Y, et al. Primary Sjögren's syndrome is associated with increased risk of malignancies besides lymphoma: A systematic review and meta-analysis. Autoimmun Rev. 2022; 21(5): 103084, doi: 10.1016/j.autrev.2022.103084, indexed in Pubmed: 35341972.
14. Goulabchand R, Malafaye N, Jacot W, et al. Cancer incidence in primary Sjögren's syndrome: Data from the French hospitalization database. Autoimmun Rev. 2021; 20(12): 102987, doi: 10.1016/j.autrev.2021.102987, indexed in Pubmed: 34718160.
15. Choi HG, Kang HoS, Lim H, et al. Potential Cancer Risk in Patients with Rheumatoid Arthritis: A Longitudinal Korean Population-Based Analysis. J Pers Med. 2022; 12(6), doi: 10.3390/jpm12060965, indexed in Pubmed: 35743750.
16. Lee H. The Risk of Malignancy in Korean Patients with Rheumatoid Arthritis. Yonsei Med J. 2019; 60(2): 223–229, doi: 10.3349/ymj.2019.60.2.223, indexed in Pubmed: 30666845.
17. Miranda-Filho A, Lortet-Tieulent J, Bray F, et al. Thyroid cancer incidence trends by histology in 25 countries: a population-based study. Lancet Diabetes Endocrinol. 2021; 9(4): 225–234, doi: 10.1016/S2213-8587(21)00027-9, indexed in Pubmed: 33662333.
18. Ahn HS, Kim HJ, Welch HG. Korea's thyroid-cancer "epidemic"--screening and overdiagnosis. N Engl J Med. 2014; 371(19): 1765–1767, doi: 10.1056/NEJMp1409841, indexed in Pubmed: 25372084.
19. Conrad N, Misra S, Verbakel JY, et al. Incidence, prevalence, and co-occurrence of autoimmune disorders over time and by age, sex, and socioeconomic status: a population-based cohort study of 22 million individuals in the UK. Lancet. 2023; 401(10391): 1878–1890, doi: 10.1016/S0140-6736(23)00457-9, indexed in Pubmed: 37156255.
20. Sun X, Lu Li, Li Y, et al. Increased risk of thyroid disease in patients with Sjogren's syndrome: a systematic review and meta-analysis. PeerJ. 2019; 7: e6737, doi: 10.7717/peerj.6737, indexed in Pubmed: 30918763.
21. Boelaert K, Newby PR, Simmonds MJ, et al. Prevalence and relative risk of other autoimmune diseases in subjects with autoimmune thyroid disease. Am J Med. 2010; 123(2): 183.e1–183.e9, doi: 10.1016/j.amjmed.2009.06.030, indexed in Pubmed: 20103030.
22. Chen YK, Lin CL, Cheng FTF, et al. Cancer risk in patients with Hashimoto's thyroiditis: a nationwide cohort study. Br J Cancer. 2013; 109(9): 2496–2501, doi: 10.1038/bjc.2013.597, indexed in Pubmed: 24084773.
23. Pazaitou-Panayiotou K, Michalakis K, Paschke R. Thyroid cancer in patients with hyperthyroidism. Horm Metab Res. 2012; 44(4): 255–262, doi: 10.1055/s-0031-1299741, indexed in Pubmed: 22334393.
24. Boi F, Pani F, Mariotti S. Thyroid Autoimmunity and Thyroid Cancer: Review Focused on Cytological Studies. Eur Thyroid J. 2017; 6(4): 178–186, doi: 10.1159/000468928, indexed in Pubmed: 28868258.
25. Liotti F, Visciano C, Melillo RM. Inflammation in thyroid oncogenesis. Am J Cancer Res. 2012; 2(3): 286–297, indexed in Pubmed: 22679559.
26. Rotondi M, Chiovato L, Romagnani S, et al. Role of chemokines in endocrine autoimmune diseases. Endocr Rev. 2007; 28(5): 492–520, doi: 10.1210/er.2006-0044, indexed in Pubmed: 17475924.
27. Racanelli V, Prete M, Minoia C, et al. Rheumatic disorders as paraneoplastic syndromes. Autoimmun Rev. 2008; 7(5): 352–358, doi: 10.1016/j.autrev.2008.02.001, indexed in Pubmed: 18486921.
28. Tabata M, Kobayashi T. Polymyalgia rheumatica and thyroid papillary carcinoma. Intern Med. 1994; 33(1): 41–44, doi: 10.2169/internalmedicine.33.41, indexed in Pubmed: 8180442.
29. Kalliabakos D, Pappas A, Lagoudianakis E, et al. A case of polymyositis associated with papillary thyroid cancer: a case report. Cases J. 2008; 1(1): 289, doi: 10.1186/1757-1626-1-289, indexed in Pubmed: 18973692.
30. Yilmaz U, Ugurlu S. Dermatomyositis Associated With Papillary Thyroid Cancer: Systematic Review of the Literature and a Case Report. J Clin Rheumatol. 2021; 27(4): 168–171, doi: 10.1097/RHU.0000000000001048, indexed in Pubmed: 32803919.
31. Ahn JK, Oh JM, Lee J, et al. Adult onset Still's disease diagnosed concomitantly with occult papillary thyroid cancer: paraneoplastic manifestation or coincidence? Clin Rheumatol. 2010; 29(2): 221–224, doi: 10.1007/s10067-009-1305-x, indexed in Pubmed: 19847590.
32. Inoue R, Kato T, Kim F, et al. A case of adult-onset Still's disease (AOSD)-like manifestations abruptly developing during confirmation of a diagnosis of metastatic papillary thyroid carcinoma. Mod Rheumatol. 2012; 22(5): 796–800, doi: 10.1007/s10165-011-0588-3, indexed in Pubmed: 22270347.
33. Tirri R, Capocotta D. Incidental papillary thyroid cancer diagnosis in patient with adult-onset Still's disease-like manifestations. Reumatismo. 2019; 71(1): 42–45, doi: 10.4081/reumatismo.2019.1118, indexed in Pubmed: 30932443.
34. Tavarelli M, Sarfati J, De Gennes C, et al. Hypertrophic Osteoarthropathy and Follicular Thyroid Cancer: A Rare Paraneoplastic Syndrome. Eur Thyroid J. 2015; 4(4): 266–270, doi: 10.1159/000437052, indexed in Pubmed: 26835431.
35. Guerouaz N, Alaoui M, Raiss M, et al. Systemic paraneoplastic vasculitis secondary to papillary carcinoma of the thyroid. Clin Exp Dermatol. 2016; 41(6): 655–658, doi: 10.1111/ced.12800, indexed in Pubmed: 27416971.
36. Do H, Pyo JY, Ahn SS, et al. Cancer-associated vasculitides: a single-centre case series. Clin Rheumatol. 2023; 42(1): 151–158, doi: 10.1007/s10067-022-06363-0, indexed in Pubmed: 36083400.
37. Hong S, Won YJ, Park YR, et al. Community of Population-Based Regional Cancer Registries. Cancer Statistics in Korea: Incidence, Mortality, Survival, and Prevalence in 2017. Cancer Res Treat. 2020; 52(2): 335–350, doi: 10.4143/crt.2020.206, indexed in Pubmed: 32178489.
38. Bellini P, Albano D, Dondi F, et al. Incidental radioiodine uptake at whole body scan due to Primary Sjogren Syndrome in a patient with differentiated Thyroid cancer. Rev Esp Med Nucl Imagen Mol (Engl Ed). 2022; 41(1): 47–49, doi: 10.1016/j.remnie.2021.01.002, indexed in Pubmed: 34991837.
39. Lupoli GA, Barba L, Liotti A, et al. Falsely elevated thyroglobulin and calcitonin due to rheumatoid factor in non-relapsing thyroid carcinoma: A case report. Medicine (Baltimore). 2019; 98(5): e14178, doi: 10.1097/MD.0000000000014178, indexed in Pubmed: 30702570.
40. Jarząb B, Dedecjus M, Lewiński A, et al. Diagnosis and treatment of thyroid cancer in adult patients - Recommendations of Polish Scientific Societies and the National Oncological Strategy. 2022 Update [Diagnostyka i leczenie raka tarczycy u chorych dorosłych - Rekomendacje Polskich Towarzystw Naukowych oraz Narodowej Strategii Onkologicznej. Aktualizacja na rok 2022]. Endokrynol Pol. 2022; 73(2): 173–300, doi: 10.5603/EP.a2022.0028, indexed in Pubmed: 35593680.
41. Biondi B, Cooper DS. Thyroid Hormone Suppression Therapy. Endocrinol Metab Clin North Am. 2019; 48(1): 227–237, doi: 10.1016/j.ecl.2018.10.008, indexed in Pubmed: 30717904.
42. Cellini M, Rotondi M, Tanda ML, et al. Skeletal health in patients with differentiated thyroid carcinoma. J Endocrinol Invest. 2021; 44(3): 431–442, doi: 10.1007/s40618-020-01359-6, indexed in Pubmed: 32696339.
43. Ku EuJ, Yoo WS, Lee EK, et al. Effect of TSH Suppression Therapy on Bone Mineral Density in Differentiated Thyroid Cancer: A Systematic Review and Meta-analysis. J Clin Endocrinol Metab. 2021; 106(12): 3655–3667, doi: 10.1210/clinem/dgab539, indexed in Pubmed: 34302730.
44. Kwak D, Ha J, Won Y, et al. Effects of thyroid-stimulating hormone suppression after thyroidectomy for thyroid cancer on bone mineral density in postmenopausal women: a systematic review and meta-analysis. BMJ Open. 2021; 11(5): e043007, doi: 10.1136/bmjopen-2020-043007, indexed in Pubmed: 33986046.
45. Chung CW, Choi HS, Kong SH, et al. Measurements of Bone Health after Thyroid-Stimulating Suppression Therapy in Postmenopausal Women with Differentiated Thyroid Carcinoma: Bone Mineral Density versus the Trabecular Bone Score. J Clin Med. 2021; 10(9), doi: 10.3390/jcm10091964, indexed in Pubmed: 34063726.
46. Ran B, Wei F, Gong J, et al. Application and prospect of trabecular bone score in differentiated thyroid cancer patients receiving thyrotropin suppression therapy. Front Endocrinol (Lausanne). 2022; 13: 1004962, doi: 10.3389/fendo.2022.1004962, indexed in Pubmed: 36313757.

47. Jin YJ, Song CM, Park BJ, et al. Analyses of the Association between Thyroid Cancer and Osteoporosis/Fracture Histories: A Cross-Sectional Study Using KoGES HEXA Data. Int J Environ Res Public Health. 2021; 18(9), doi: 10.3390/ijerph18094732, indexed in Pubmed: 33946728.
48. Lee Y, Yoon BH, Lee S, et al. Risk of Osteoporotic Fractures after Thyroid-stimulating Hormone Suppression Therapy in Patients with Thyroid Cancer. J Bone Metab. 2019; 26(1): 45–50, doi: 10.11005/jbm.2019.26.1.45, indexed in Pubmed: 30899724.
49. Notsu M, Yamauchi M, Morita M, et al. Papillary thyroid carcinoma is a risk factor for severe osteoporosis. J Bone Miner Metab. 2020; 38(2): 264–270, doi: 10.1007/s00774-019-01053-5, indexed in Pubmed: 31664535.
50. Hong AR, Kang HC. Evaluation and Management of Bone Health in Patients with Thyroid Diseases: A Position Statement of the Korean Thyroid Association. Endocrinol Metab (Seoul). 2023; 38(2): 175–189, doi: 10.3803/EnM.2023.1701, indexed in Pubmed: 37150514.