

# The impact of cholecalciferol on cognitive performance in patients with hypothyroidism and autoimmune thyroiditis



**I. I. Kamyshna**

*Ivan Horbachevsky Ternopil National Medical University  
of the Ministry of Health of Ukraine*

Hashimoto's thyroiditis is a chronic autoimmune thyroiditis characterized by lymphocytic infiltration that may result in the gradual loss of thyroid tissue [1]. According to a study, even with normal thyroid function, autoimmune thyroiditis can cause neuroinflammation, leading to emotional changes and mental disorders [2].

Although the mechanism is unclear, some research indicates that vitamin D has a beneficial effect on autoimmune thyroid diseases [3]. A case-control study found that patients with HT had lower levels of vitamin D [4]. Another meta-analysis has linked vitamin D deficiency with autoimmune thyroid disease as well [5]. Clinical studies have indicated a strong correlation between serum vitamin D levels and cognitive impairment in patients with autoimmune thyroiditis [6]. Vitamin D is regarded as a neurosteroid that regulates immunomodulation, brain development, and function in adulthood [7, 8].

The vitamin D receptor (VDR) and the enzyme that transforms 25(OH)D to the active form of the vitamin, 1,25-dihydroxyvitamin D, are expressed in all organs, including the brain [9]. According to a recent study conducted by the Institute of Medicine, only individuals with 25(OH)D levels below 10 ng/mL are at a permanent risk of cognitive decline, regardless of other factors [10, 11].

**Objective** — to investigate the impact of cholecalciferol on cognitive function in patients with hypothyroidism and autoimmune thyroiditis in the Western Ukrainian population.

## MATERIALS AND METHODS

Our research was conducted at Bukovinian State Medical University, Chernivtsi Regional Endocrinology Center, and I. Horbachevsky Ternopil National Medical University, Ukraine. The prospective cohort study design included 56 patients with hypothyroidism (H) caused by autoimmune thyroiditis (AIT). These patients were distributed into two groups. Patients in the Group 1 ( $n = 28$ ) received cholecalciferol at a dose of 4.000 IU/day (28.000 IU/week) and L-thyroxine ( $88.39 \pm 12.70 \mu\text{g/day}$ ). Patients in the Group 2 ( $n = 28$ ) were prescribed only L-thyroxine ( $87.50 \pm 12.73 \mu\text{g/day}$ ). During the treatment, all patients were visited and interviewed about possible side effects, and to determine the degree of compliance Examinations were performed at the beginning and end of the 12-week treatment. After 12 weeks of treatment, all laboratory tests and clinical evaluations were repeated as per the initial visit.

To diagnose hypothyroidism, we were guided by recommendations required by the American Association of Clinical Endocrinologists 2012. The corresponding clinical features were considered when verifying AIT, namely the results of a sonogram of the thyroid gland (reduced echogenicity) and circulating antibodies to thyroid antigens were detected [12].

Blood samples from patients and controls were taken in the morning (8 to 10 am) after a night fast. Using STAT FAX303/Plus analyzer (Awareness Technology Inc, USA),

we determined levels of free thyroxine ( $fT_4$ ), normal range 6.0—13.0 pmol/L for males and 7.0—13.5 pmol/L for females, thyroid-stimulating hormone (TSH, normal range 0.3—4.0 mIU/mL), anti-thyroid peroxidase (anti-TPO, normal range 0—30 IU/mL) and anti-thyroglobulin (anti-TG, normal range 0—65 IU/mL) in each individual who participated in the study.

Study exclusion criteria were the following: less than 18 years of age, malignancy, inflammation resulting from rheumatic diseases or acute/chronic infection, diabetes mellitus, vascular, chronic diseases of liver and kidneys, and pregnancy. Individuals administering drugs that could influence thyroid function were also ruled out from the study.

We detect a decline in cognitive function using the Mini-Mental State Examination (MMSE), which has been the most used screening instrument throughout decades [13, 14].

When determining 25-OH Vitamin D levels in the serum of the patients and healthy individuals, we applied the ELISA using the 25-OH Vitamin D Total (Vit D-Direct) Test System ELISA Kit (Monobind Inc., United States, Product Code: 9425-300) on E.I.A. Reader Sirio S (Seac, Italy).

Statistical analysis. Quantitative variables were assessed for normality using the Shapiro-Wilk test (when the number of subjects was less than 50) or the Kolmogorov-Smirnov test (when the number of subjects was more than 50). Quantitative variables following non-normal distribution were described using median (Me) and lower and upper quartiles ( $Q_1$  —  $Q_3$ ). Comparisons of three or more groups on a quantitative variable whose distribution differed from normal were made using the Kruskal-Wallis test and Dunn's criterion with Holm correction as a post-hoc method. A comparison of frequencies in the analysis of multifield contingency tables was performed using Pearson's chi-square test (for expected values greater than 10).

Ethical approval. The study fully ensured the standards described in the 1975 Helsinki Declaration of Human Rights (amended in 2008). The participants completed and signed a written informed consent before enrolling voluntarily in the research.

## RESULTS

We performed the analysis of TSH and  $fT_4$  levels before and after vitamin D treatment (Table 1).

Table 1 presented in the previous context suggests that there was a statistically significant decrease in TSH levels in both groups of patients after therapy

Table 1

### Analysis of TSH levels before and after treatment

Group	Value	
<b>Level of TSH, mIU/mL</b>		
1 (n = 28)	Before treatment	7.10 (6.80—7.60)
	After treatment	4.00 (3.90—4.10)*
2 (n = 28)	Before treatment	7.10 (6.80—7.40)
	After treatment	3.90 (3.60—4.10)*
<b>Level of <math>fT_4</math>, pmol/L</b>		
1 (n = 28)	Before treatment	4.10 (3.40—4.80)
	After treatment	7.30 (7.10—7.50)*
2 (n = 28)	Before treatment	4.00 (3.70—4.60)
	After treatment	7.35 (7.10—8.10)*

Note. Data are presented as median and lower and upper quartiles (Me ( $Q_1$  —  $Q_3$ )).

\* The difference to the value before treatment is statistically significant ( $p < 0.001$ ).

( $p < 0.001$ ). Additionally, there were statistically significant differences in  $fT_4$  levels depending on their initial level ( $p < 0.001$ ). The table also indicates that after treatment,  $fT_4$  levels were normalized in both patient groups. We performed the analysis of anti-TPO and anti-TG levels before and after treatment (Figure 1, 2).

According to the data obtained when comparing anti-TPO statistically significant differences were revealed depending on the level of anti-TPO ( $p < 0.001$ ).

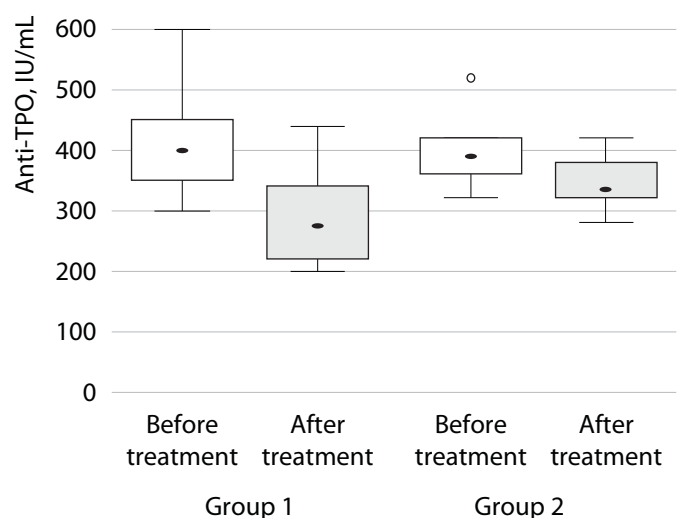


Figure 1. Analysis of anti-TPO levels before and after treatment

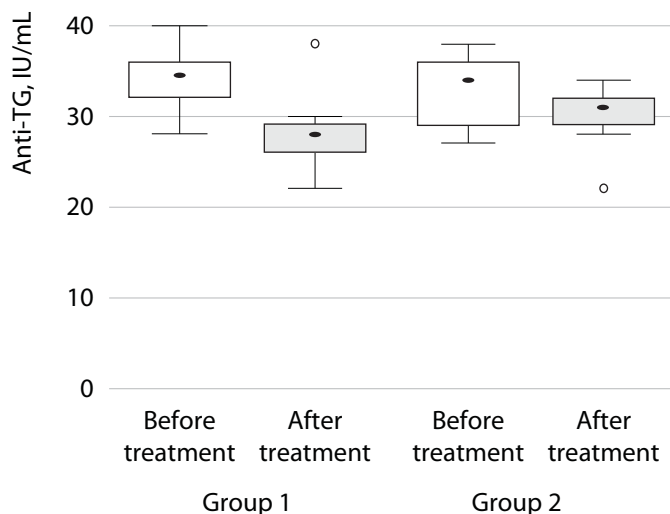


Figure 2. Analysis of anti-TG levels before and after treatment

In this study, the group of patients who received cholecalciferol and L-thyroxine showed a greater decrease in anti-TPO levels compared to the group of patients who received only L-thyroxine. Specifically, the group that received cholecalciferol and L-thyroxine showed a decrease of 31.25 % in anti-TPO levels after treatment, while the group that received only L-thyroxine showed a decrease of only 14.1 %.

Based on the data analysis, it was found that there was a potential reduction in anti-TG levels in patients who received both cholecalciferol and L-thyroxine by 18.84 % after treatment. On the other hand, in Group 2 patients who only received L-thyroxine, anti-TG levels decreased by only 8.82 % after treatment (see Figure 2).

Analysis of 25(OH)D levels was performed before and after treatment.

Table 2 shows that after treatment in patients of Group 1 there was a normalization of the level of 25(OH) D, at the same time in patients of Group 2 level of 25(OH) D decreased compared to its level before treatment.

Analysis of MMSE was performed conditioning on MMSE test (Table 3).

After treatment, there is a statically significant improvement in cognitive function according to the MMCE test in a group of patients who took cholecalciferol and L-thyroxine compared with patients who received only L-thyroxine ( $p = 0.011$ ) (applied method: The Kruskal-Wallis test).

Statistically significant differences were revealed when comparing Cognitive impairment depending on the MMSE test ( $p < 0.001$ ) (applied method: Pearson's chi-square test).

Table 2

Analysis of 25(OH)D levels before and after treatment, ng/mL

Group		Value
1 (n = 28)	Before treatment	18.0 (16.0—20.0)
	After treatment	40.0 (38.0—42.0)*
2 (n = 28)	Before treatment	19.5 (18.0—22.0)
	After treatment	18.0 (16.0—20.0)*#

Note. Data are presented as median and lower and upper quartiles (Me ( $Q_1 - Q_3$ )).

\* The difference to the value before treatment is statistically significant ( $p < 0.001$ ).

# The difference to the indicator value of Group 1 after treatment is statistically significant ( $p < 0.001$ ).

Table 3

Analysis of MMSE test after treatment

Group		Value
1 (n = 28)	Before treatment	26.0 (24.0—27.0)
	After treatment	27.0 (27.0—28.0)*
2 (n = 28)	Before treatment	26.0 (24.0—26.0)
	After treatment	26.5 (25.0—27.0)#

Note. Data are presented as median and lower and upper quartiles (Me ( $Q_1 - Q_3$ )).

\* The difference to the value before treatment is statistically significant ( $p < 0.001$ ).

# The difference to the indicator value of Group 1 after treatment is statistically significant ( $p < 0.05$ ).

After the course of treatment, patients showed improvement in cognitive function. Thus, in both groups, according to the MMSE test, patients with mild dementia improved cognitive performance to moderate (pre-dementia) cognitive impairment. In Group 1 patients who received cholecalciferol and L-thyroxine after treatment, the percentage of patients with moderate (pre-dementia) cognitive impairment decreased from 50.0 % to 21.4 % (Table 4). At the same time, no statistically significant changes were found in the group of patients taking L-thyroxine alone. Normalization of cognitive functions according to the MMCE test increased from 28.6 % to 78.6 % in the first group, and from 21.4 % to 50.0 % in patients of Group 2. Thus, treatment with cholecalciferol supplementation was statistically more effective than L-thyroxine alone.

Table 4

## Analysis of Cognitive impairment conditioning on MMSE test

Impairment	Group 1		Group 2	
	Before treatment	After treatment*	Before treatment	After treatment <sup>#</sup>
Mild dementia	6 (21.4 %)	0	2 (7.1 %)	0
Moderate (pre-dementia) cognitive impairment	14 (50.0 %)	6 (21.4 %)	20 (71.4 %)	14 (50.0 %)
No cognitive impairment	8 (28.6 %)	22 (78.6 %)	6 (21.4 %)	14 (50.0 %)

Note.\* The difference to the value before treatment is statistically significant ( $p < 0.01$ ).

<sup>#</sup> The difference to the indicator value of Group 1 after treatment is statistically significant ( $p < 0.001$ ).

## DISCUSSION

Thyroid disease is a prevalent endocrine disorder, and autoimmune thyroid disease (AITD) is thought to be the most common autoimmune disease [15]. Vitamin D, as an immunomodulator, plays a role in the development and progression of AITD [16]. Due to the increasing evidence of the association between vitamin D deficiency and thyroid disorders, there is a growing interest in using vitamin D supplementation for the prevention and treatment of thyroid disorders.

These are some examples of studies that have investigated the relationship between vitamin D and thyroid function. The first study, conducted by F. Ucar et al. in Turkey, found lower levels of 25(OH)D in elderly patients with subclinical hypothyroidism compared to healthy individuals [17]. A case-control study by N. J. Aljohani et al. found an inverse relationship between vitamin D status and free triiodothyronine levels [18]. A Polish pilot study that monitored vitamin D status during summer months in patients on L-thyroxine treatment reported that vitamin D sufficiency did not improve even during the summer. These studies suggest that there may be a relationship between vitamin D deficiency and thyroid dysfunction, but further research is needed to fully understand this relationship [19].

The study by I. M. B. Botelho et al. suggested that a decreased level of  $fT_4$  could be a predictor of vitamin D deficiency in patients with Hashimoto's thyroiditis, which is in line with the findings of the current study that suggest that thyroid hormone may play a role in regulating autoimmune thyroid function in the presence of sufficient vitamin D levels [20]. Another study reported a positive interaction between  $fT_4$  and 25(OH)D levels, suggesting that supplementation with either vitamin D or thyroid hormones could help regulate the balance between these two parameters [21].

Our study showed a statistically significant decrease in anti-TPO levels in patients who received both cholecalciferol and L-thyroxine, with a reduction of 31.25%, compared to patients who only received L-thyroxine, where anti-TPO levels decreased by 14.1%. Additionally, we observed a decrease in anti-TG levels in patients receiving cholecalciferol and L-thyroxine by 18.84%, while patients in Group 2 who only received L-thyroxine experienced a decrease in anti-TG levels by only 8.82%.

Vitamin D plays an important role in regulating various vital physiological processes in the central nervous system, including cell differentiation, neurotransmitter biosynthesis, and neurotrophic release [22, 23]. Studies have shown that vitamin D supplementation can prevent cognitive impairment and improve hippocampal plasticity in aged rats by increasing the expression of potential key genes involved in neuroplasticity or enhancing the function of major neurotransmitter receptors such as dopamine, serotonin, and glutamate [24].

According to our study, treatment with cholecalciferol and L-thyroxine resulted in a significant improvement in cognitive function, as assessed by the MMCE test, compared to patients who received only L-thyroxine. In the group that received cholecalciferol and L-thyroxine, there was a decrease in the percentage of patients with moderate cognitive impairment (pre-dementia) from 50.0% to 21.4% after treatment. In contrast, no significant changes were observed in the group that received L-thyroxine alone. The percentage of patients with normalized cognitive function, as assessed by the MMCE test, increased from 28.6% to 78.6% in the cholecalciferol and L-thyroxine group and from 21.4% to 50.0% in the L-thyroxine alone group. Thus, our findings suggest that cholecalciferol supplementation combined with L-thyroxine treatment is more effective in improving cognitive function than L-thyroxine alone.

The results mentioned earlier suggest that vitamin D may have a positive effect on the connectivity of certain neural circuits involved in reward-dependent and motor behavior, such as the ventral tegmental area-accumbens *nucleus*-prefrontal cortex circuit and the nigro-striatal circuit. Additionally, the presence of vitamin D receptors and enzymes involved in vitamin D metabolism in brain regions involved in cognitive processes, such as complex planning and memory formation, suggests a potential role for vitamin D in neuro-cognition [10].

### CONCLUSIONS

Based on the results of our study, which showed a significant improvement in cognitive function and a reduction in thyroid autoantibodies in patients receiving vitamin D supplementation, it can be concluded that vitamin D may have a positive influence on improving cognitive function in patients with AIT and hypothyroidism. Additionally, our findings suggest that vitamin D therapy may be a safe and effective treatment option for these conditions, particularly in patients with vitamin D deficiency.

**Ethics approval.** Our study was conducted according to the Declaration of Helsinki adopted in 1975 and revised in 2008, and the ethical principles were entirely respected.

**Consent to participate.** Written informed consent was obtained from the participants.

**Data availability.** The data of this study is available by request.

**Conflicts of interest:** none.

### ЛІТЕРАТУРА/REFERENCES

1. Wang S, Liu Y, Zhao N, et al. IL-34 expression is reduced in Hashimoto's thyroiditis and associated with thyrocyte apoptosis. *Front Endocrinol (Lausanne)*. 2018 Oct 23;9:629. doi: 10.3389/fendo.2018.00629. PMID: 30405534; PMCID: PMC6206842.
2. Cai YJ, Wang F, Chen ZX, et al. Hashimoto's thyroiditis induces neuroinflammation and emotional alterations in euthyroid mice. *J Neuroinflammation*. 2018 Oct 29;15(1):299. doi: 10.1186/s12974-018-1341-z. PMID: 30373627; PMCID: PMC6206655.
3. Pankiv I. Vitamin D: new aspects of application, effective doses. The current state of the problem. *International Journal of Endocrinology*. 2021;17(1): 38-42. <https://doi.org/10.22141/2224-0721.17.1.2021.226430>. Ukrainian.
4. Ma J, Wu D, Li C, et al. Lower serum 25-hydroxyvitamin D level is associated with 3 types of autoimmune thyroid diseases. *Medicine (Baltimore)*. 2015 Sep;94(39):e1639. doi: 10.1097/MD.0000000000001639. PMID: 26426654; PMCID: PMC4616844.
5. Wang J, Lv S, Chen G, et al. Meta-analysis of the association between vitamin D and autoimmune thyroid disease. *Nutrients*. 2015 Apr 3;7(4):2485-98. doi: 10.3390/nu7042485. PMID: 25854833; PMCID: PMC4425156.
6. Xu J, Zhu XY, Sun H, et al. Low vitamin D levels are associated with cognitive impairment in patients with Hashimoto thyroiditis. *BMC Endocr Disord*. 2018 Nov 26;18(1):87. doi: 10.1186/s12902-018-0314-7. PMID: 30477467; PMCID: PMC6260768.
7. Sharif K, Sharif Y, Watad A, et al. Vitamin D, autoimmunity and recurrent pregnancy loss: More than an association. *Am J Reprod Immunol*. 2018 Sep;80(3):e12991. doi: 10.1111/aji.12991. Epub 2018 Jun 19. PMID: 29923244.
8. AlJohri R, AIOkail M, Haq SH. Neuroprotective role of vitamin D in primary neuronal cortical culture. *eNeurological Sci*. 2018 Dec 17;14:43-8. doi: 10.1016/j.ensci.2018.12.004. PMID: 30619951; PMCID: PMC6312860.
9. Eyles DW, Liu PY, Josh P, Cui X. Intracellular distribution of the vitamin D receptor in the brain: comparison with classic target tissues and redistribution with development. *Neuroscience*. 2014 May 30;268:1-9. doi: 10.1016/j.neuroscience.2014.02.042. Epub 2014 Mar 6. PMID: 24607320.
10. Laughlin GA, Kritz-Silverstein D, Bergstrom J, et al. Vitamin D insufficiency and cognitive function trajectories in older adults: The Rancho Bernardo Study. *J Alzheimers Dis*. 2017;58(3):871-83. doi:10.3233/JAD-161295.
11. Kamyshna II. Vitamin D in the treatment of anxiety in patients with autoimmune thyroiditis and hypothyroidism in the West-Ukrainian population. *Clinical Endocrinology and Endocrine Surgery*. 2022; 3(79):20-4. DOI: 30978/CEES-2022-3-20.
12. Garber JR, Cobin RH, Gharib H, et al. Clinical practice guidelines for hypothyroidism in adults: cosponsored by the American Association of Clinical Endocrinologists and the American Thyroid Association. *Endocr Pract*. 2012;18(6):988-1028. doi: 10.4158/EP12280.GL.
13. Batty GD, Li Q, Huxley R, Zoungas S, Taylor BA, Neal B, de Galan B, Woodward M, et al.; VANCE Collaborative group. Oral disease in relation to future risk of dementia and cognitive decline: prospective cohort study based on the Action in Diabetes and Vascular Disease: Preterax and Diamicon Modified-Release Controlled Evaluation (ADVANCE) trial. *Eur Psychiatry* 28(1), 49-52, 2013.
14. Bos D, Vernooij MW, de Bruijn RF, et al. Atherosclerotic calcification is related to a higher risk of dementia and cognitive decline. *Alzheimers Dement*. 2015 Jun;11(6):639-47.e1. doi: 10.1016/j.jalz.2014.05.1758. Epub 2014 Aug 20. PMID: 25150731.
15. Pankiv I. Prevalence of autoimmune thyroiditis among women with vitamin D deficiency. *International Journal of Endocrinology (Ukraine)*. 2017;13(5):336-9. <https://doi.org/10.22141/2224-0721.13.5.2017.110023>.
16. Sulejmanovic M, Begić A, Mujarić-Bousbia F, Salkić S, Ramas A. The relationship between thyroid antibodies and vitamin D level in primary hypothyroidism. *Med Arch*. 2020 Oct;74(5):359-62. doi: 10.5455/medarch.2020.74.359-362. PMID: 33424090; PMCID: PMC7780811.
17. Ucar F, Akyol S, Ozturk G, et al. Evaluation of serum vitamin D levels in elderly patients with subclinical hypothyroidism. *J Exper Clin Med*. 2014, 31:77-80. DOI: 10.5835/jecm.omu.31.02.003.

18. Aljohani NJ, Al-Daghri NM, Al-Attas OS, et al. Differences and associations of metabolic and vitamin D status among patients with and without sub-clinical hypothyroid dysfunction. *BMC Endocr Disord*. 2013 Aug 20;13:31. doi: 10.1186/1472-6823-13-31. PMID: 23962199; PMCID: PMC3751774.
19. Kmieć P, Minkiewicz I, Rola R, Sworczak K, Żmijewski MA, Kowalski K. Vitamin D status including 3-epi-25(OH)D3 among adult patients with thyroid disorders during summer months. *Endokrynol Pol*. 2018;69(6):653-60. doi: 10.5603/EP.a2018.0065. Epub 2018 Sep 27. PMID: 30259504.
20. Botelho IMB, Moura Neto A, Silva CA, Tambascia MA, Alegre SM, Zantut-Wittmann DE. Vitamin D in Hashimoto's thyroiditis and its relationship with thyroid function and inflammatory status. *Endocr J*. 2018 Oct 29;65(10):1029-37. doi: 10.1507/endocrj.EJ18-0166. Epub 2018 Jul 27. PMID: 30058600.
21. Chao G, Zhu Y, Fang L. Correlation between Hashimoto's thyroiditis-related thyroid hormone levels and 25-hydroxyvitamin D. *Front Endocrinol (Lausanne)*. 2020 Feb 14;11:4. doi: 10.3389/fendo.2020.00004. PMID: 32117049; PMCID: PMC7034299.
22. Gil Á, Plaza-Díaz J, Mesa MD. Vitamin D: classic and novel actions. *Ann Nutr Metab*. 2018;72(2):87-95. doi: 10.1159/000486536. Epub 2018 Jan 18. PMID: 29346788.
23. Máčková L, Bičíková M, Ostatníková D, Hill M, Stárka L. Vitamin D, neurosteroids and autism. *Physiol Res*. 2017 Sep 26;66(Suppl 3):S333-S340. doi: 10.33549/physiolres.933721. PMID: 28948817.
24. Latimer CS, Brewer LD, Searcy JL, et al. Vitamin D prevents cognitive decline and enhances hippocampal synaptic function in aging rats. *Proc Natl Acad Sci U S A*. 2014 Oct 14;111(41):E4359-66. doi: 10.1073/pnas.1404477111. Epub 2014 Sep 29. PMID: 25267625; PMCID: PMC4205629.

## ABSTRACT

Hashimoto's thyroiditis is a chronic autoimmune thyroiditis characterized by lymphocytic infiltration that may result in the gradual loss of thyroid tissue. According to a study, even with normal thyroid function, autoimmune thyroiditis can cause neuroinflammation, leading to emotional changes and mental disorders. Although the mechanism is unclear, some research indicates that vitamin D has a beneficial effect on autoimmune thyroid diseases. The relationship between serum vitamin D levels and cognitive impairment in patients with autoimmune thyroiditis has been established in clinical studies.

**Objective** — to investigate the impact of cholecalciferol on cognitive function in patients with hypothyroidism and autoimmune thyroiditis in the Western Ukrainian population.

**Materials and methods.** The study involved 56 patients diagnosed with hypothyroidism resulting from autoimmune thyroiditis. These patients were divided into two groups. Group 1 (n = 28) received cholecalciferol at a dose of 4000 IU/day (28.000 IU/week) and L-thyroxine

(88.39 ± 12.70 µg/day), while Group 2 (n = 28) received only L-thyroxine (87.50 ± 12.73 µg/day). Assessments were conducted at the start and end of a 12-week treatment period, and cognitive function was measured using the Mini-Mental State Examination. The results showed a decline in cognitive function.

**Results.** Following the treatment course, patients demonstrated an improvement in cognitive function. In Group 1, where patients were treated with cholecalciferol and L-thyroxine, the proportion of patients with moderate (pre-dementia) cognitive impairment decreased from 50 % to 21.4 %. However, there were no significant changes observed in the group that received only L-thyroxine. Normalization of cognitive function as determined by the MMCE test increased from 28.6 % to 78.6 % in Group 1, and from 21.4 % to 50 % in Group 2 patients.

**Conclusions.** Based on our findings, vitamin D supplements should be given to patients diagnosed with autoimmune thyroiditis and hypothyroidism in order to enhance cognitive function.

**Keywords:** autoimmune thyroiditis, hypothyroidism, cognitive function, cholecalciferol.

## РЕЗЮМЕ

### Вплив холекальциферолу на когнітивну діяльність у хворих на гіпотиреоз і автоімунний тиреоїдит

I. I. Камишна

Тернопільський національний медичний університет імені І. Я. Горбачевського

Автоімунний тиреоїдит характеризується лімфоцитарною інфільтрацією, що може призвести до поступової втрати тканини та погіршення функціонального стану щитоподібної залози. За даними досліджень, навіть за нормальної функції щитоподібної залози автоімунний тиреоїдит може спричинити нейрозапалення, що призводить до емоційних змін і психічних розладів. Хоча механізм повністю не з'ясований, деякі дослідження показують, що вітамін D має сприятливий вплив на автоімунні захворювання щитоподібної залози. У клінічних дослідженнях встановлено зв'язок між рівнем вітаміну D у сироватці крові та порушенням когнітивної функції у пацієнтів з автоімунним тиреоїдитом.

**Мета роботи** — дослідити вплив холекальциферолу на когнітивну функцію у пацієнтів з гіпотиреозом на тлі автоімунного тиреоїдиту у західноукраїнській популяції.

**Матеріали та методи.** У дослідженні взяли участь 56 пацієнтів з гіпотиреозом, який виник унаслідок аутоімунного тиреоїдиту. Пацієнтів розподілили на дві групи. Хворі першої групи (n=28) отримували холекальциферол у дозі 4000 МО/добу (28 000 МО/тиж) і левотироксин ((88,39 ± 12,70) мкг/добу), хворі другої групи (n=28) — лише левотироксин ((87,50 ± 12,73) мкг/добу). Когнітивні функції оцінювали за допомогою Mini-Mental State Examination (MMSE) на початку і в кінці 12-тижневого курсу лікування.

**Результати.** За даними тесту MMSE, у пацієнтів з легким ступенем деменції когнітивні показники поліпшилися до помірних (переддементних) порушень. У групі пацієнтів, які отримували холекаль-

циферол і левотироксин, після лікування частка хворих з помірними (переддементними) когнітивними порушеннями зменшилася з 50,0 до 21,4%, тоді як у групі пацієнтів, які приймали лише левотироксин, статистично значущих змін не відзначено. Нормалізацію когнітивної функції за результатами тесту MMSE зареєстровано у 78,6% пацієнтів першої групи (до лікування — у 28,6%) та 50,0% — другої групи (до лікування — у 21,4%).

**Висновки.** Пацієнтам з гіпотиреозом на тлі аутоімунного тиреоїдиту слід призначати додаткові дози вітаміну D для поліпшення когнітивної функції.

**Ключові слова:** аутоімунний тиреоїдит, гіпотиреоз, когнітивна функція, холекальциферол.

*Received* • Отримано 16/01/2023  
*Peer-reviewed* • Рецензовано 17/02/2023  
*Accepted* • Прийнято до друку 10/03/2023