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ASSESSMENT OF THYROID STATUS IN WOMEN WITH MENSTRUAL FUNCTION DISORDERS AFTER COVID-19

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Abstract

Introduction. During the COVID-19 pandemic, an increase in menstrual irregularities, decreased fertility, and a higher incidence of gynecological pathology was observed. COVID-19 is a systemic immunosuppressive disease capable of activating autoimmune processes, particularly in the thyroid gland. Due to the expression of ACE2 receptors, SARS-CoV-2 can induce thyrotoxicosis or thyroiditis. Despite the well-known interrelation between thyroid and reproductive systems, the mechanisms of their reciprocal changes after COVID-19 and the role of virus-induced thyroiditis in menstrual disorders remain insufficiently studied.
Aim. To analyze the functional and structural state of the thyroid gland in women with menstrual cycle disorders after COVID-19.
Materials and methods. A prospective study was conducted involving 171 women aged 18-40 years with menstrual dysfunction after COVID-19. Group 1 (n = 82) included patients with post-acute COVID (1-3 months after infection); Group 2 (n = 89) included patients with long COVID (3-6 months). All participants underwent thyroid ultrasonography and laboratory assessment of TSH, free T4, LH, FSH, estradiol, progesterone, and prolactin levels.
Results. In Group 1, a significant decrease in TSH (0.58 ± 1.04 μIU/mL; $p < 0.05$) was observed, corresponding to transient thyrotoxicosis associated with post-viral thyroiditis. In Group 2, elevated TSH levels (6.91 ± 2.09 μIU/ mL; $p < 0.05$) indicated manifest hypothyroidism, most likely of autoimmune or post-viral origin – a common complication of the long COVID phase. Women with long COVID showed reduced estradiol and progesterone levels, elevated prolactin, and ovulatory dysfunction (amenorrhea, oligomenorrhea, menometrorrhagia).
Conclusions. Thyroid dysfunction after COVID exhibits a phase-dependent pattern. Subclinical thyrotoxicosis predominates in the post-acute COVID, whereas primary autoimmune hypothyroidism prevails in long COVID. These phases have specific menstrual disturbances- cycle shortening with subclinical thyrotoxicosis (post-acute COVID) and oligomenorrhea or amenorrhea with dysfunctional bleeding in autoimmune thyroid processes (long COVID). Early screening of thyroid and sex hormones in reproductive-aged women presenting with post-COVID menstrual changes is recommended as an intervention that would assist timely support of recovery of reproduction.

Keywords: **COVID-19 morbidity, long COVID, post-COVID syndrome, menstrual cycle, reproductive health, endocrine pathology, gynecological care organization**

INTRODUCTION

The female reproductive system is one of the most sensitive and responsive systems to various external influences [1]. It is therefore logical that during the COVID-19 pandemic, under viral stress on the female body, the reproductive system is among the first to be affected. Women often experience menstrual irregularities, reduced fertility, an increased incidence of gynecological disorders, and a higher risk of pregnancy complications, spontaneous abortions, and stillbirths [2].

COVID-19 is a severe immunosuppressive disease with a multifaceted impact on the entire body and the potential to exacerbate pre-existing autoimmune or chronic diseases. The thyroid gland remains one of the most vulnerable organs. The most common thyroid pathology associated with COVID-19 is the onset or exacerbation of autoimmune thyroiditis, which may lead not only to structural changes within the gland but also to various functional impairments [3, 4].

Due to the expression of angiotensin-converting enzyme 2 (ACE2) receptors in thyroid tissue, which facilitate viral entry into cells, and the action of integrins, there is a high probability of developing thyrotoxicosis as well as subacute and chronic thyroiditis after SARS-CoV-2 infection [5, 6].

It should be noted that, despite the existing evidence of the physiological interdependence between the thyroid gland and the female reproductive system, data on their mutual changes after COVID-19 remain insufficient. In particular, the mechanisms linking thyroid dysfunction and menstrual disorders, as well as the principles of early diagnosis of virus-induced menstrual disorders, are still not sufficiently elucidated.

AIM

To analyze the functional and structural state of the thyroid gland in women with menstrual cycle disorders after COVID-19.

MATERIALS AND METHODS

A clinical prospective study of 171 females complaining of menstrual dysfunction post-COVID-19 was initiated at the gynecology department within an 18-month time frame (January 2023 – June 2024). This study has been approved by the Local Ethics Committee at The State Institution of Science «Center of innovative healthcare technologies» State Administrative Department. Written informed consent from all participants in this study was a prerequisite for enrollment. The research adhered to the principles of bioethics and the relevant legal and ethical standards, including the Declaration of Helsinki (1964-2013), EEC Directive 86/609 (1986), ICH-GCP Guidelines (1996), the Ministry of Health of Ukraine Guideline 42-7.0:2005 «Medicinal Products. Good Clinical Practice» (2005), the Regulation on Ethics Committees (Order No. 690, Ministry of Health of Ukraine, 2009), the Constitution of Ukraine, the Civil Code of Ukraine (2006), and the Fundamentals of Health Legislation of Ukraine (1992).

Patients were stratified according to the onset time of menstrual disorders following recovery from acute COVID-19:

- **Group 1 (n = 82):** women of reproductive age whose menstrual disorders appeared within 1-3 months after the acute phase – classified as the *post-acute COVID-19 phase*.
- **Group 2 (n = 89):** women whose menstrual disorders developed 3-6 months after infection without an alternative diagnosis – classified as *long COVID* or *post-COVID condition*.

The definition of long COVID/post-COVID condition was based on WHO (World Health Organization), NICE (National Institute for Health and Care Excellence, UK), and CDC (Centers for Disease Control and Prevention, USA) recommendations, requiring symptoms persisting > 12 weeks after infection and not explained by another pathology.

Inclusion criteria. Women aged 18-40 years with confirmed previous COVID-19 infection and newly developed menstrual disorders within 1-6 months after recovery were included. Additional criteria: non-pregnant, generally healthy, without severe gynecologic disease, and with regular menstrual cycles prior to COVID-19.

Exclusion criteria. Women with organic reproductive tract pathology, severe somatic or endocrine disorders unrelated to COVID-19, those planning pregnancy, or those using hormonal contraceptives within 9 months before the study were excluded.

Clinical and laboratory assessment. All participants underwent comprehensive evaluation including detailed medical history, anthropometric measurements (height, weight), general and gynecological examinations, and thyroid ultrasonography. In cooperation with an endocrinologist, all subjects received a complete thyroid assessment. Laboratory evaluation included measurement of serum luteinizing hormone (LH), follicle-stimulating hormone (FSH), estradiol, progesterone, prolactin, thyroid-stimulating hormone (TSH), and free thyroxine (T4).

Statistical analysis. Data were analyzed using standard descriptive and inferential statistical methods. Quantitative parameters were expressed as mean ± standard deviation (SD). Differences were considered statistically significant at $p < 0.05$.

RESULTS

The mean age of participants was 31.4 ± 6.8 years in Group 1 and 32.1 ± 7.2 years in Group 2 ($p = 0.523$). The mean body mass index (BMI) was 24.6 ± 4.2 kg/m^2 in Group 1 compared with 25.1 ± 4.8 kg/m^2 in Group 2 ($p = 0.467$) (Table 1).

Table 1

Baseline Characteristics of the Study Participants according to the Post-Acute COVID-19 Phase (Group 1) and Long COVID-19 Phase (Group 2)

Parameter	Group 1 (n=82)	Group 2 (n=89)	p-value
Age, years (M±SD)	31,4±6,8	32,1±7,2	0,523
Body mass index, kg/m^2 (M±SD)	24,6±4,2	25,1±4,8	0,467

The percentage of vaccinated patients against SARS-CoV-2 was 19.1% in Group 1 and 11.2% in Group 2 (*p* = 0.179; not statistically significant).

During thyroid palpation and ultrasonographic screening, 37 patients (42.0%) in Group 2 demonstrated diffuse thyroid enlargement and parenchymal heterogeneity with hypoechoic areas lacking clear borders. These findings suggest thyrocyte destruction due to immune dysregulation and impaired compensatory mechanisms, indicating early structural changes consistent with autoimmune thyroiditis (AIT) [2, 8].

In contrast, 28 women (34.1%) in Group 1 exhibited palpable thyroid tenderness and heterogeneity. Ultrasonography in this cohort revealed focal hypoechoic regions within the parenchyma, interpreted as sonographic signs of subacute thyroiditis, likely associated with immune-cell infiltration of thyroid tissue in response to viral aggression (Table 2).

Table 2

Thyroid Screening Results and Menstrual Cycle Abnormalities in Study Participants

Parameter	Group 2 (n=89 (100%))		Group 1 (n=82 (100%))	
	n	%	n	%
Presence of BTP and MD	45	50,6*	36	44.0
Absence of BTP and MD	44	49,4	46	56,0*

Note: * – statistically significant differences between groups (p < 0.05).

Analysis of thyroid screening results and menstrual cycle abnormalities demonstrated that a higher percentage of menstrual dysfunction (MD) occurred in the presence of benign thyroid pathology (BTP) in both study groups (Table 2).

The structure of menstrual cycle disorders between Group 1 (post-acute COVID-19) and Group 2 (long COVID-19) was further analyzed. In Group 1, the cases of polymenorrhea and hypomenorrhea indicated that pathological factors mainly affected the rhythmicity and volume of menstrual bleeding in the early post-COVID period. The leading type of menstrual dysfunction in Group 2 was oligo-opsomenorrhea, which already indicated a predominant dysregulation of the hypothalamic–pituitary–ovarian axis. There were also cases of amenorrhea, hypomenorrhea, and menometrorrhagia reported; this variation already proves more variability and severity of endocrine disturbance during the post-COVID condition phase. This variation proves a shift from functional to endocrine dysregulation.

Table 3

Structure of Menstrual Cycle Disorders in Study Groups

Type of menstrual disorder	Group 1 (n=82)	Group 2 (n=89)	p-value
Polymenorrhea	28(34,1%)	7(7,0%)	0,05*
Hypomenorrhea	25(30,5%)	8 (9,0%)	0,05*
Oligo-opsomenorrhea	-	37(42.0%)	
Menometrorrhagia	12(14,7%)	13(15.0%)	
Metrorrhagia	17(20.7%)	-	
Amenorrhea	-	24(27.0%)	

Note: * – statistically significant differences between groups (p < 0.05).

Subsequent analysis assessed the functional state of the thyroid gland and evaluation of sex hormone levels to offer a clinical characterization of various types of menstrual dysfunction after COVID-19 infection.

Examination of the hormonal profile in women during the post-acute and long COVID-19 phases indicated statistically significant differences between key endocrine parameters for these two groups, thus confirming different hormonal patterns for each phase (Table 4).

The hormonal picture speaks of signs of thyroid dysfunction associated with post-COVID syndrome in both groups. In the patients who were in the post-acute phase of COVID-19, a clear tendency toward lower levels of thyroid-stimulating hormone (TSH) was observed (0.58 ± 1.04 µIU/mL; p < 0.05). This fact, together with characteristic sonographic findings, allows us to state that it is a question of the development of virus-associated thyroid disease – namely subacute thyroiditis accompanied by manifest thyrotoxicosis.

Conversely, in the long COVID-19 group, there was a marked worsening of thyroid dysfunction, manifested by a significant shift in TSH distribution toward elevated values (6.91 ± 2.09 µIU/mL; p < 0.05), consistent with manifest hypothyroidism of likely autoimmune or post-viral origin – one of the common complications observed in this phase.

Gonadotropic and steroid hormones participating in the regulation of menstruation revealed significant differences between the groups. In the long COVID-19 phase, alongside elevated TSH levels, LH concentrations were shifted toward the upper limit of normal, while FSH was within the normal range compared to Group 1. Low progesterone accompanied both thyrotoxicosis and hypothyroidism. In long COVID, the shift was statistically significantly below normal (p < 0.05). This finding does explain menstrual pathology in this group – all forms of amenorrhea and opsoligomenorrhea – more pronounced in long COVID conditions that develop due to anovulation and absence or dysfunction of the corpus luteum, as well as insufficiency of the luteal phase. It does not, however, occur in post-acute COVID.

Table 4

Hormonal Profile of Women during Post-Acute and Long COVID-19 Phases

Parameter	Reference ranges	Group 1 (n=82)	Group 2 (n=89)
Follicular phase of the natural menstrual cycle or conditional menstrual cycle			
Luteinizing hormone (LH), IU/L	1,9-12,5	2,07 ± 1,35*	10,02 ± 3,26#
Follicle-stimulating hormone (FSH), IU/L	3,0-8,78	5,21 ± 1,69	9,57 ± 3,13$
Thyroid-stimulating hormone (TSH), μIU/mL	0,4-4,78	0,58 ± 1,04*	6,91 ± 2,09$
Prolactin, ng/mL	3,34-26,72	35,56±8,05#	72,29±7,67$
Estradiol, pg/mL	22.4-115	98,5 ± 26,24#	21,42 ± 5,66&
Progesterone, ng/mL	0,31-1,52	0,34±0,16*	0,25 ± 0,14&

Notes: * – significant deviation of hormone level distribution toward the lower limit of the normal range according to Pearson's $\chi 2$ test, compared with the clinically expected distribution (15% / 70% / 15%), $p < 0.05$;
– significant deviation of hormone level distribution toward the upper limit of the normal range according to Pearson's $\chi 2$ test, compared with the clinically expected distribution (70% / 15% / 15%);
& – significant deviation of hormone level distribution below the normal range according to Pearson's $\chi 2$ test, compared with the clinically expected distribution (15% / 70% / 15%), $p < 0.05$;
$ – significant deviation of hormone level distribution above the normal range according to Pearson's $\chi 2$ test, compared with the clinically expected distribution (70% / 15% / 15%), $p < 0.05$.

Women with long COVID presented significantly reduced estradiol levels, due to progressive neurohormonal dysfunction in the pituitary–thyroid–ovarian regulatory axis. This precipitates inadequate luteal phase formation and consequently progesterone deficiency and chronic anovulation which ultimately lowers endometrial receptivity. Also, prolactin was much higher in long COVID patients than in the post-acute group. This can be due to a hyperprolactinemic state from disease in the hypothalamic–pituitary–gonadal axis as well as from overt hypothyroidism. All of these conditions lead to unmasked anovulation, follicular persistence, and hyperestrogenism together with the development of anovulatory menstrual pattern.

DISCUSSION

This study confirms the results of international studies that have previously established a link between COVID-19 and thyroid dysfunction. COVID-19 triggers thyroid dysfunction, ranging from destructive thyroiditis with transient thyrotoxicosis to autoimmune hypothyroidism. These trends confirm the specificity of endocrine disorders in women of reproductive age depending on the phase of the post-COVID period. While previous studies described such changes in generalized terms, this study compared two different phases of the post-COVID period: post-acute COVID-19 (Group 1) and long COVID-19 (Group 2). It is these two periods that revealed the different nature of thyroid dysfunction and associated menstrual disorders.

In the post-acute phase of COVID-19 (Group 1), subacute virus-associated thyroiditis was predominant: ultrasound findings showed focal hypoechoic areas in the thyroid gland in 34.1% of patients, accompanied by painful palpation [13]. Biochemically, this phase was characterized by a reduced TSH level (mean value 0.58 ± 1.04 μIU/ml) – thyrotoxicosis, which is probably transient due to subacute thyroiditis. The clinical picture described above is consistent with the suppression of the central hypothalamic-pituitary-thyroid axis due to neuroendocrine exhaustion caused by acute infection [6]. SARS-CoV-2 is able to penetrate the hypothalamus and induce a cytokine storm (mainly through increased IL6 levels) [6]. The effects of stress and glucocorticoid effects are manifested in reduced secretion of thyrotropin-releasing hormone (TRH) and, thus, TSH production by the pituitary gland [6]. Therefore, in the early period after COVID, there is insufficient stimulation of structurally intact thyroid glands. Our study confirms the mechanism of central thyroid dysfunction in post-COVID syndrome, which may later first clinically manifest itself in specific menstrual cycle disorders. Polymenorrhea was 34.1%, and hypomenorrhea – 30.5% of patients in Group 1, which describes a shortening of the cycle and a decrease in menstrual bleeding volume disorders. This means that pathological factors mainly affect the rhythm and volume of bleeding during the early recovery period, most likely due to thyrotoxicosis and the associated luteal insufficiency.

In the long COVID phase, thyroid pathology shifts towards overt hypothyroidism. In 42% of patients, ultrasound examination showed diffuse enlargement of the thyroid gland with pronounced hypoechogenicity, i.e. signs of autoimmune thyroiditis [4]. Similarly, significantly elevated TSH values indicate the development of primary hypothyroidism due to autoimmune post-viral changes. Similar effects have been described by other authors in viral infections [6]. Our contribution links this pathology to specific menstrual disorders characteristic of the prolonged post-COVID period. Oligopsomenorrhea, i.e. long and infrequent cycles, was the leading type of menstrual disorder in Group 2, registered in 42% of cases; finally, amenorrhea and menometrorrhagia were also quite common in 27% and 15% of cases, respectively [4]. This distribution suggests anovulatory disorders in patients with long COVID due to hypothyroidism and secondary hyperprolactinemia (prolactin levels in Group 2 were on average twice as high as in Group 1). It is well known that hypothyroidism can cause hyperprolactinemia and simultaneously suppress or inhibit ovarian function through its effects on the hypothalamic-pituitary-ovarian axis; which in turn causes chronic anovulation [6]. This leads to estrogen deficiency (estradiol levels in Group 2 were significantly lower than normal) with relative endometrial hyperestrogenism, since the cyclical progesterone phase is absent. This may lead to concomitant oligomenorrhea/ amenorrhea, a tendency to uterine bleeding due to irregular desquamation of the hyperplastic endometrium. Thus, the deeper endocrine disorders caused by COVID-19 progress from functional (central) to structural (autoimmune). Our study clearly shows how these hormonal changes manifest in menstrual cycle problems – from less pronounced cycle disorders with short-term high thyroid hormone levels to severe problems with lack of ovulation with low thyroid hormone levels.

CONCLUSIONS

In women, during the early post-Covid (post-acute) phase, subclinical thyrotoxicosis caused by subacute thyroiditis is more common, while in the long COVID phase, autoimmune primary hypothyroidism is more common. Various menstrual disorders have been described with different phases of the post-Covid syndrome. In the post-acute phase, reduction or polymenorrhea and metrorrhagia predominate; in the long COVID phase, oligomenorrhea or amenorrhea due to anovulation, which is also associated with dysfunctional uterine bleeding, is more common. Changes in the menstrual cycle detected in the early stages of recovery indicate autonomic destabilization and hormonal imbalance initiated by SARS-CoV-2. In these patients, menstrual dysfunction best describes disorders of the hypothalamic-pituitary-thyroid-ovarian axis, but it may be a sign of deeper but latent endocrine disorders.

In women of reproductive age with menstrual disorders after COVID-19,, screening of thyroid function and sex hormones is recommended, depending on the type of menstrual disorder. This will allow for timely initiation of therapeutic treatment (hormone replacement therapy or correction of hypothyroidism), as well as treatment to restore reproductive health during the recovery period after COVID-19.

Prospects for further research. In view of the above findings, further studies are recommended to better elucidate the mechanisms of paths by which COVID-19 runs in its effects on the thyroid gland and the female reproductive system. It will then be possible to generalize an effective diagnostic, therapeutic, and preventive algorithm in management for these disorders. This will not only ensure timeliness in detection and treatment of dysfunction but also in lowering both the incidence and degree of abnormality regarding menstruation – hence improving women's health in terms of reproduction.

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The authors declare no conflict of interest regarding this publication.

COMPLIANCE WITH ETHICAL REQUIREMENTS

This review article is based on an analysis of publicly available scientific data from peer-reviewed journals, clinical guidelines, and databases. No data containing patient identifiers were used during the study, which did not include new clinical interventions or the primary collection of patient information, so approval from the ethics committee was not necessary. The authors adhered to the ethical principles of the Helsinki Declaration of the World Medical Association and international standards for medical journal publications. No element of the work contains plagiarism or data fabrication. All sources of information are cited appropriately and are properly formatted.

Artificial intelligence (AI) was not used in the preparation of this work for text generation, data analysis, or image creation. All content was prepared exclusively by the authors, who bear full responsibility for the accuracy, originality, and adherence to ethical standards.

AUTHOR CONTRIBUTIONS

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REFERENCES

1. Kuan, K. K. W., & Saunders, P. T. K. (2022). Female reproductive systems: Hormone dependence and receptor expression. In M. J. Campbell & C. L. Bevan (Eds.), Nuclear receptors in human health and disease (Vol. 1390). Springer, Cham. https://doi.org/10.1007/978-3-031-11836-4_2
2. Wang, M., Zhang, B., & Jin, L. (2021). Female fertility under the impact of COVID-19 pandemic: A narrative review. Expert Reviews in Molecular Medicine, 23, e15. https://doi.org/10.1017/erm.2021.19
3. Ostapchuk, V. O., & Ostapchuk, V. A. (2022). Funktsional′ni ta strukturni zminy shchytopodibnoyi zalozy khvorykh na avtoimunnyy tyreoyidyt z hipotyreozom u pislyakovidnomu periodi [Functional and structural changes in the thyroid gland in patients with autoimmune thyroiditis with hypothyroidism in the postpartum period]. Infektsiyni khvoroby, 1(107), 40-45. Available from: https://ojs.tdmu.edu.ua/index.php/inf-patol/article/download/13018/12195/44334
4. Inaba, H., & Aizawa, T. (2021). Coronavirus disease 2019 and the thyroid: Progress and perspectives. Frontiers in Endocrinology, 12, 708333. https://doi.org/10.3389/fendo.2021.708333
5. Kostitska, I., Skrypnyk, N., Zherdova, N., Cherniavska, I., Vyshyvanyuk, V., Tymochko, N., & Gerych, O. (2023). Koronavirusna khvoroba (COVID-19) – potentsiynyy tryher tyreoyidytu de Kervena: klinichnyy vypadok ta ohlyad literatury [Coronavirus disease (COVID-19) – a potential trigger for de Quervain's thyroiditis: a clinical case and literature review]. Terapevtyka, 3(4), 44-55. Available from: https://terapevtyka.com.ua/index.php/journal/article/view/121
6. Ruggeri, R. M., Campennì, A., Deandreis, D., Siracusa, M., Tozzoli, R., Ovčariček, P., & Giovanella, L. (2021). SARS-CoV-2-related immune-inflammatory thyroid disorders: Facts and perspectives. Expert Review of Clinical Immunology, 17(7), 737-759. https://doi.org/10.1080/1744666X.2021.19324
7. Wang, Q., Zhao, R., Han, C., et al. (2024). Correlation between thyroid hormone sensitivity and the risk of polycystic ovary syndrome. BMC Endocrine Disorders, 24, 76. https://doi.org/10.1186/s12902-024-01607-3
8. Singh, S., Singh, V., Tiwari, H., & Mehta, A. (2024). Evaluation of thyroid dysfunction in reproductive-age women with menstrual disorders: A case-control study. European Journal of Cardiovascular Medicine, 14(1), 794-800.
9. Brancatella, A., Ricci, D., Viola, N., Sgrò, D., Santini, F., & Latrofa, F. (2020). Subacute thyroiditis after SARS-CoV-2 infection. The Journal of Clinical Endocrinology & Metabolism, 105(7), 2367-2370. https://doi.org/10.1210/clinem/dgaa276
10. Muller, I., Cannavaro, D., Dazzi, D., Covelli, D., Mantovani, G., Muscatello, A., et al. (2020). SARS-CoV-2-related atypical thyroiditis. The Lancet Diabetes & Endocrinology, 8(9), 739-741. https://doi.org/10.1016/S2213-8587(20)30266-7
11. Pal, R., & Banerjee, M. (2020). COVID-19 and the endocrine system: Exploring the unexplored. Journal of Endocrinological Investigation, 43, 1027-1031. https://doi.org/10.1007/s40618-020-01276-8
12. Krassas, G. E., Poppe, K., & Glinoer, D. (2010). Thyroid function and human reproductive health. Endocrine Reviews, 31(5), 702-755. https://doi.org/10.1210/er.2009-0041
13. Naguib R. (2022). Potential relationships between COVID-19 and the thyroid gland: an update. The Journal of international medical research, 50(2), 3000605221082898. https://doi.org/10.1177/03000605221082898
14. Piekarska, A., Góral, M., Kozula, M., Jawiarczyk-Przybyłowska, A., Zawadzka, K., & Bolanowski, M. (2023). The Influence of SARS-CoV-2 Infection on the Thyroid Gland. Biomedicines, 11.

Резюме

ОЦІНКА ТИРЕОЇДНОГО СТАНУ У ЖІНОК З ПОРУШЕННЯМИ МЕНСТРУАЛЬНОЇ ФУНКЦІЇ ПІСЛЯ COVID-19

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Вступ. Під час пандемії COVID-19, спостерігалися порушення менструальної функції, зниження фертильності та зростання частоти гінекологічної патології [1, 2]. COVID-19, – системне імуносупресивне захворювання, здатне активувати автоімунні процеси, зокрема в щитоподібній залозі. Завдяки експресії рецепторів АПФ2 SARS-CoV-2 може викликати тиреотоксикоз або тиреоїдит [3-6]. Попри відомий зв'язок між тиреоїдною та репродуктивною системами, механізми їх взаємних змін після COVID-19, та роль вірус-індукованого тиреоїдиту в менструальних порушеннях залишаються недостатньо дослідженими.

Мета. Проаналізувати функціональний та структурний стан щитоподібної залози у жінок з порушеннями менструального циклу після перенесеного COVID-19.

Матеріали та методи. Проспективне дослідження 171 жінки віком 18-40 років із менструальними порушеннями після перенесеного COVID-19. Група 1 (n=82) – post-acute COVID (1-3 місяці після COVID-19); група 2 (n = 89) – long COVID (3-6 місяців). Проведено УЗД щитоподібної залози, визначення рівнів ТТГ, вільного Т4, ЛГ, ФСГ, естрадіолу, прогестерону та пролактину.

Результати. У групі 1 спостерігалося достовірне зниження ТТГ (0,58 ± 1,04 мкМО/мл; $p < 0,05$), що відповідало транзиторному тиреотоксикозу на тлі поствірусного тиреоїдиту. У групі 2 зафіксовано підвищення ТТГ (6,91 ± 2,09 мкМО/мл; $p < 0,05$), характерне для маніфестного гіпотиреозу, ймовірно аутоімунного або післявірусного походження, що є одним із частих ускладнень фази long COVID. Для жінок із long COVID відзначено нижчі рівні естрадіолу й прогестерону, підвищений пролактин і порушення овуляції (аменорея, олігоменорея, менометрорагії).

Висновки. Дисфункція щитоподібної залози після COVID демонструє фазозалежну картину. Субклінічний тиреотоксикоз переважає в постгострому періоді COVID, тоді як первинний аутоімунний гіпотиреоз – при тривалому COVID. Ці фази мають специфічні менструальні порушення – вкорочення циклу при субклінічному тиреотоксикозі (постгострому COVID) та олігоменорею або аменорею з дисфункціональними кровотечами при аутоімунних процесах щитоподібної залози (тривалий COVID). Раннє скринінгування гормонів щитоподібної залози та статевих гормонів у жінок репродуктивного віку, що звертаються зі змінами менструального циклу після COVID, рекомендується як втручання, яке сприятиме своєчасній підтримці відновлення репродукції.

Ключові слова: **захворюваність на COVID-19, Long COVID, постковідний синдром, менструальний цикл, репродуктивне здоров'я, ендокринна патологія, організація гінекологічної допомоги**

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