ORIGINAL ARTICLE

CONTENTS

Alterations of thyroid and adipose tissue hormones in acute and convalescent COVID-19 patients

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ABSTRACT

Aim: To study and analyze studies data of thyroid function and adipose tissue hormones changes in patients with coronavirus infection.

Materials and Methods: The study was conducted on the basis of the Clinical Hospital «CMCH», Uzhhorod, outpatient department. The clinical presentation of the patients with Covid-19 verified diagnosis of was represented by damage to the upper respiratory tract, in particular nasopharyngitis. The age of the studied patients was 18±2.15 years.

Results: All thyroid status parameters varied within the reference values, but it is noteworthy that after treatment, a significant increase in TSH levels was observed both after treatment and in comparison with the control group ($p_1<0.01$; p3<0.01) and after 6 months шт 3.1 times ($p_2<0.01$; $p_4<0.01$). Representative dynamic values of ATPO were identified both after treatment and in comparison with the control group ($p_1<0.01$; $p_3<0.01$) and after 6 months. A significant decrease in ATPO was observed in 4.7 times ($p_2<0.01$; $p_4<0.01$). All levels of the studied adiponectin, leptin and C-peptide parameters varied within the reference range. Adiponectin values significantly decreased after treatment and after 6 months ($p_4<0.01$) in 4 times; Leptin levels significantly decreased after treatment in 2.8 times and after 6 months - in 3 times ($p_4<0.01$). C-peptide values ($p_4=0.12$) reached the level of the control group.

Conclusions: Investigation of thyroid function and adipose tissue hormones changes in patients with Covad-19 was conducted. All thyroid status parameters and adipose tissue hormones varied within the reference values with different variations.

KEY WORDS: Covid-19, upper respiratory tract infection, nasopharyngitis, thyroid hormones, adipose tissue hormones, patient

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INTRODUCTION

The response of each organism to a viral factor is unique and can be considered as a set of contributing factors for the development of certain clinical presentations. The persistence of the virus or viral fragments determines have triggered to the immune response, the duration of the disease and the possibility of developing complications. [1] From 20% to 60% of patients with COVID-19 have some endocrine disorders during the acute phase of the disease or during the convalescence period, according to numerous studies. Moreover, some hormonal disorders can persist for months after recovery, forming part of the symptom complex of "post-COVID syndrome" or "long COVID". Understanding the relationship between COVID-19 and the endocrine system is important for clinical practice, timely diagnosis, adequate treatment and prevention of long-term complications. [2]. If the immune response is ineffective in the first phase of SARS-CoV-2 infection, a second or late phase develops, which is based on large-scale virus replication and a "cytokine storm". This is accompanied by the generation of a large number of virions, which leads to massive damage of target tissues, with including lung tissue. Affected ACE2-expressing cells produce pro-inflammatory cytokines that recruit effector cells (macrophages, neutrophils) and release alarmins that induce the activity of inflammasomes (inflammasome from the English inflammation – inflammation) – a multi-protein oligomeric complex responsible for activating the inflammatory response. The functioning of the latter is accompanied by the release of a significant number of pro-inflammatory cytokines and, as result, the "cytokine storm"will developed, which enhance the participation of the above-mentioned macrophages and neutrophils, creating an extremely high level of inflammatory reaction [3].

The SARS-CoV-2 virus enters cells through the angiotensin-converting enzyme 2 (ACE2) receptor, which is widely expressed not only in the lungs, but also in many endocrine organs: the pancreas (islets of Langerhans), thyroid gland, adrenal glands, pituitary gland, testes, ovaries. The presence of these receptors makes the endocrine glands vulnerable to direct viral damage.

The virus can caused, after entering the cell[4]:
-direct cytopathic cell damage;
-local inflammatory reaction:
-glandular dysfunction;
-small vessel thrombosis with organ ischemia;
-autoimmune reactions against glandular tissues [5,6].

The coronavirus attacks fat cells and certain immune cells in fat tissue, creating an immune response that can lead to serious damage, according to the studies. This finding may explain why people, who are overweight or obese have an increasing risk in severe illness and death from COVID-19 [7].

AIM

The aim was to study and analyze studies data of thyroid function and adipose tissue hormones changes in patients with coronavirus infection

MATERIALS AND METHODS

The study was conducted on the basis of the Clinical Hospital «CMCH», Uzhhorod, outpatient department (2019-2021years). The clinical presentation of the patients with Covid-19 verified diagnosis of was represented by damage to the upper respiratory tract, in particular nasopharyngitis. The age of the studied patients (n=60) was 18±2.15 years(67,0±1,8% women, 33,0±2,6% men).

The developed treatment regimen was applied according to the treatment protocol of Ministry of Health of Ukraine [8] The Covid-19 strain named "Omicron" was detected in the studied contingent

Criteria for including of the examined contingent with diagnosed COVID-19: patients, aged 18,00±2.15 years, with a diagnosis of COVID-19, with clinical manifestations of confirmed results infection by the subjective and objective investigation, with laboratory data and the presence of informed consent to diagnostic and treatment measures conducting.

Exclusion/non-inclusion criteria: pztients before 18,00±2.15 years of age; oncological, autoimmune diseases; laboratory detection of TORCH infection, human immunodeficiency virus (HIV); viral diseases (rubella, syphilis, hepatitis, chickenpox), gonococcal infection; tuberculosis; malaria; severe defects.

A control group (n=28) was identical in age and sex, without clinical and laboratory manifestations of coronavirus infection (CI), was also formed.

Blood serum studies were performed on the ELISA analyzer VER-2000 (Siemens) (thyroid hormones by immunochemiluminescent method; leptin, adiponectin, C-peptide - by enzyme-linked immunosorbent assay). The study algorithm included observation for 6 months. All studies performed and treatment methods applied were carried out with the voluntary written consent of the patients, in compliance with all requirements for ensuring the anonymity and confidentiality of the results obtained. The methodology of scientific and practical monitoring was carried out in accordance with the criteria of the Helsinki Declaration of Human Rights of 1975 and its revision of 1983, the norms of the Council of Europe Convention on Human Rights and Biomedicine, as well as the current norms of Ukrainian legislation and the requirements of the local medical commission of the State Higher Educational Institution «Uzhhorod National University».

RESULTS

A very limited number of studies and reports describe the association between SARS-CoV-2 infection and thyroid diseases in children [7,8].

McCowan et al. [7] had studied 244 children with thyroid abnormalities, hypothyroidism or hyperthyroidism at a tertiary pediatric endocrine center in the United Kingdom before and after COVID-19 for identification any changes in their clinical presentation. They suggested that this finding may be related to the development of thyroiditis secondary to SARS-CoV-2 infection, which regressed before requiring treatment. This condition was associated with a significantly higher risk of death and severe inflammation (detected by high levels of IL-6), highlighting the importance of monitoring thyroid function test results in patients with severe COVID-19 infection [9]. The higher reported the thyroid dysfunction incidence where in the majority of analyzed patients. These patients had a mild course of COVID-19, which suggests a disease severity correlation with thyroid dysfunction [10,11].

A retrospective study of patients referred to a research center with a diagnosis of MIS-C showed that over 90% of patients had nonthyroidal illness syndrome (NTIS) [12]. Among the laboratory variants of NTIS, the most common was an isolated decrease in T3 free [12]. TG balance was restored in 100% of patients after twenty months[13].

Although data on thyroid function impairment during COVID-19 infection in the patients are still limited, the available evidence supports the theory that the virus

Table 1. Dynamic parameters of thyroid status in children with diagnosed coronavirus infection

Parameters	Control group N=28, M± m	1 group (n=60) , M± m		
		Before treatment	After treatment	After 6 month
TSH (0,4-4,0, мкIU/ml)	1.87 ± 0.46	0.41 ± 0.03	0.73 ± 0.19 (p_1<0.01; p_3<0.01)	1.25 ± 0.40 (p_2<0.01; p_4<0.01)
Free triiodothyronine (1,2-2,8, nmol/l)	1.33 ± 0.08	1.30 ± 0.28	1.40 ± 0.21 (p_1=0.11; p_3=0.18)	1.20 ± 0.31 (p_2=0.004; p_4=0.12)
Free thyroxine (12,5-21,0, nmol/l)	14.22 ± 0.49	15.00 ± 2.10	11.10 ± 1.42 (p_1<0.01; p_3<0.01)	13.84 ± 1.09 (p_2<0.01; p_4=0.08)
ATPO (< 35, IU/ml)	5.69 ± 0.11	6.10 ± 4.58	2.80 ± 1.86 (p_1<0.01; p_3<0.01)	1.32 ± 1.18 (p_2<0.01; p_4<0.01)

Notes: p_1 - significance of differences between the values of indicators before and after treatment; p_2 - significance of differences between the values of indicators after treatment and after 6 months; p_3 - significance of differences between the values of indicators after treatment and the parameters of the control group; p_4 - significance of differences between the values of indicators after 6 months and the parameters of the control group
Source: compiled by the authors of this study

Table 2. Dynamic characteristics of adipose tissue hormones in children with diagnosed coronavirus infection

Parameters	Control group N=28, M± m	1 group (n=60) , M± m		
		Before treatment	After treatment	After 6 month
Adiponectin (5-37, mkg/ml)	7.73 ± 0.86	31.20 ± 10.11	8.12 ± 1.65 (p_1<0.01; p_3<0.01)	8.34 ± 0.79 (p_2=0.22; p_4<0.01)
leptin (2,05-11,09, ng/ml)	6.97 ± 0.32	11.97 ± 2.28	3.81 ± 1.29 (p_1<0.01; p_3<0.01)	4.11 ± 0.55 (p_2=0.41; p_4<0.01)
C-Peptide (0,81-3,85, ng/ml)	1.43 ± 0.08	4.71 ± 1.52	2.28 ± 0.79 (p_1<0.01; p_3<0.01)	1.48 ± 0.49 (p_2<0.01; p_4=0.12)

Notes: p_1 - significance of differences between the values of indicators before and after treatment; p_2 - significance of differences between the values of indicators after treatment and after 6 months; p_3 - significance of differences between the values of indicators after treatment and the parameters of the control group; p_4 - significance of differences between the values of indicators after 6 months and the parameters of the control group
Source: compiled by the authors of this study

plays a role in thyroid dysfunction, which in most cases is transient[14]. Autoimmune thyroiditis and primary adrenal insufficiency have also been identified in the study. The role of COVID-19 in the etiopathogenesis of APS2 is unclear, but it may be a trigger for the rapid progression of both adrenal insufficiency and hypothyroidism [15]. A third potential mechanism is selective transient pituitary dysregulation, as secondary to either the direct cytotoxic effects of the virus on the pituitary or the indirect effects of the "cytokine storm" that can induce NTIS [16-19].

It should also be considered that the COVID-19 pandemic has limited access to healthcare services for the general population worldwide, which may have affected the severity of thyroid disease at diagnosis in adults [20]. It has been hypothesized that SARS-CoV-2 may enter thyroid cells via ACE2 and the transmembrane serine protease (2TMPRSS2), which is highly expressed in this gland [21,22]. The "cytokine storm" induced by the virus is characterized by a hyperactive Th1/Th17 immune response with overexpression of pro-inflammatory cytokines such as IL-6, which has been shown to be closely associated with thyroiditis [23]. Cases of undiagnosed subacute thyroiditis have been described in patients with COVID-19 with typical clinical presentation and onset within 5–30 days of illness onset, which may be explained by the use of glucocorticoids in COVID-19, which may mask cases of destructive thyroiditis. Subacute thyroiditis was most often associated with mild COVID-19.[24]. All of the above leads to pantropism – damage to all parenchymal organs, as well as mucous membranes.

We will consider the levels of thyroid status parameters (Table 1).

According to the table, all thyroid atatus indicators varied within the reference values, but it is noteworthy that after treatment, a significant increase in TSH levels was observed both after treatment and in comparison with the control group (p1<0.01; p3<0.01) and after 6 months in 3.1 times (p2<0.01; p4<0.01). Representative dynamic data of the ATPO indicator were identified, both after treatment and in comparison with the control group (p1<0.01; p3<0.01) and after 6 months a ATPO significant decrease in 4.7 times (p2<0.01; p4<0.01).

Adipose tissue has attracted considerable attention due to its role in immune response and inflammation through the secretion of adipokines. Adipokines, such as leptin and adiponectin, are secreted by adipose tissue and are involved in various physiological processes, with particular emphasis on their role in modulating immune responses and inflammation. Leptin and adiponectin are the most abundant adipokines in humans, playing crucial roles in regulating cardiac function, skeletal muscle, growth, and inflammation. Leptin, a pro-inflammatory adipokine, is involved in the control of food intake and energy expenditure, and also influences immune cell activation and cytokine production. In contrast, adiponectin, an anti-inflammatory adipokine, circulates at high levels in plasma and modulates immune cell function, counteracting the effects of leptin. Here, we provide an overview of the role of adipokines in immune response and inflammation. Furthermore, the leptin-adiponectin ratio (Adpn/Lep) has become an important indicator of various metabolic diseases and conditions. Further studies are needed to fully elucidate the mechanisms by which adipokines influence immune responses and to identify potential therapeutic targets for inflammatory and metabolic disorders[25].

We also have investigated adipose tissue hormones and the role of the metabolic component in the development of inflammation.(Table 2).

All levels of the studied indicators of adiponectin, leptin and C-peptide varied within the reference range. Adiponectin values significantly decreased after treatment and after 6 months (p4<0.01) in 4 times, when compared with the initial indicators. The value of the leptin level significantly decreased after treatment in 2.8 times and after 6 months in 3 times (p4<0.01). The value of C-peptide (p4=0.12) reached the level of the control group. Considering that C-peptide has a significant role in the secretion of inflammatory factors, including IL-6, IL-8 and correction of adhesion to endothelial cells of human vessels, the organism, after suffering from Covid-19, does not need the above-mentioned effects. Obesity has been identified as a risk factor for progression to severe COVID-19 [26].

The mechanisms underlying the association between obesity and disease severity following SARS-CoV-2 infection remain unclear. In principle, obesity may contribute to infection in several ways:

(a) down-modulation of antiviral responses [27];

(b) release of lipids that promote endothelial dysfunction and support intravascular coagulation [28];

(c) disruption of leptin and insulin signaling, thereby enhancing the inflammatory response [29];

(d) promotion of enhanced expression of SARS-CoV-2 receptors [30];

(e) representation of a large reservoir for viral replication with increased secretion of virus and inflammatory mediators [31]

Obesity is an independent risk factor for severe COVID-19, but there is still a lack of consensus on the mechanisms underlying this association. A hypothesis that has attracted considerable attention suggests that SARS-CoV-2 disrupts adipose tissue function either through direct infection or through indirect mechanisms. [32] Furthermore, the study suggested that healthcare providers should consider patient weight and body fat when administering COVID-19 vaccines and treatments. [33]

DISCUSSION

Despite less severe manifestations in the acute phase, two major long-term complications of COVID-19 have been reported in children: multisystem inflammatory syndrome in children (MIS-C) and prolonged COVID. Since symptoms usually begin four to six weeks after initial infection, it has been hypothesized that the virus remains in the intestine of children, causing irritation of its mucosa. For this reason, viral antigens have time to cross the intestinal barrier into the bloodstream, reaching other organs and causing a significant inflammatory response [34]. Prolonged COVID is a heterogeneous multisystem condition characterized by the persistence of signs and symptoms that occur three months after the onset of COVID-19 and persist for at least two months, and cannot be explained by an alternative diagnosis [35]. Lopez-Leon et al. [36] conducted a systematic review, showing a prevalence of prolonged COVID of 25.24%. The most frequently reported symptoms were mood changes (16.50%), fatigue (9.66%), sleep disturbances (8.42%), headache (7.84%), and respiratory symptoms (7.62%). The identified risk factors for developing prolonged COVID are older age, female gender, severe COVID-19, overweight/obesity, concomitant allergic diseases, and other long-term comorbidities [8]. There are no recommendations

for the diagnosis and treatment of prolonged COVID. The underlying pathogenetic mechanisms of these patients have not yet been identified [37].

SARS-CoV-2 is known to interact with host cells through its spike protein, binding to the membrane enzyme angiotensin-converting enzyme 2 (ACE2). After the virus enters the cells, the STAT3/NF-kB pathway is activated, which causes the production of pro-inflammatory cytokines and chemokines, leading to systemic hyperinflammation known as a "cytokine storm" [38]. Adipokines have been shown to influence inflammatory and immune responses in a variety of diseases. ACE2 is not only expressed in lung cells: it is ubiquitous, which explains the multiorgan involvement commonly seen in COVID-19. Further studies are needed to elucidate the mechanisms by which adipokines exert their immunomodulatory effects on all cells of the immune system[38].

CONCLUSIONS

1. All thyroid status parameters varied within the reference values, but it is noteworthy that after treatment, a significant increase in TSH levels was observed both after treatment and in comparison with the control group (p1<0.01; p3<0.01) and after 6 months by 3.1 times (p2<0.01; p4<0.01). Representative dynamic values of ATPO were identified both after treatment and in comparison with the control group (p1<0.01; p3<0.01) and after 6 months. A significant decrease in ATPO was observed in 4.7 times (p2<0.01; p4<0.01).
2. All levels of the studied adiponectin, leptin and C-peptide parameters varied within the reference range. Adiponectin values significantly decreased after treatment and after 6 months (p4<0.01) in 4 times when compared with the initial indicators. Leptin levels significantly decreased after treatment in 2.8 times and after 6 months - in 3 times (p4<0.01). C-peptide values (p4=0.12) reached the level of the control group. Considering that C-peptide has a significant role in the secretion of inflammatory factors, including IL-6, IL-8 and correction of adhesion to endothelial cells of human vessels, after suffering from Covid-19, does not need the above-mentioned effects.

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CONFLICT OF INTEREST
The Authors declare no conflict of interest

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