Dynamics of endocrine and metabolic changes among patients with coronary artery disease, type 2 diabetes mellitus and metabolic syndrome while treating with telmisartan

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Abstract. Background. The correlation between renin-angiotensin system and hypothalamic-pituitary system is a pathogenetic link leading to many comorbid diseases, particularly type 2 diabetes mellitus (DM) and coronary artery disease (CAD). Several studies have been dedicated to the hormones of the pituitary gland, hypothalamus as well as peripheral organs of the endocrine system. The presence of common links between pathogenesis and regulating factors forces us to search for new methods of treatment which should have an overall effect on comorbid diseases. The use of telmisartan, which is a blocker of angiotensin II receptors, is among various treatment options. Nevertheless, the changes in hormonal status and lipid spectrum, which are characteristic of the patient’s condition in the course of treatment, remain to be insufficiently researched. This is the reason that justifies the expediency of our research. The study is aimed at scrutinizing endocrine and metabolic changes in patients suffering from coronary artery disease, type 2 diabetes mellitus caused by metabolic syndrome (MS) while treating with telmisartan.

Materials and methods. Fifty-one patients (26 female and 25 male patients) suffering from coronary artery disease and type 2 diabetes mellitus triggered by metabolic syndrome were examined in Lviv Regional State Clinical Medical Treatment and Diagnostic Endocrinology Center and CNE “City Clinical Hospital 5 in Lviv”. The patients were divided into two groups: experimental group and comparison group depending on the treatment prescribed. The experimental group consisted of patients (n = 27) suffering from CAD, type 2 DM and MS (women — 14, men — 13) who were prescribed with telmisartan 80 mg/day and standard therapy. The comparison group consisted of 24 patients with CAD and type 2 DM caused by MS (women — 12, men — 12) who were prescribed with standard therapy. The control group consisted of 40 healthy individuals (men — 17 (42.5 %), women — 23 (57.5 %)). The first examination was conducted on admission to the inpatient department and the second one was performed a month after the beginning of treatment. Patients’ levels of prolactin, cortisol, free thyroxine, and thyroid-stimulating hormone as well as lipid spectrum parameters were defined. Results. The dynamics of prolactin, cortisol, free thyroxine, and thyroid-stimulating hormone levels in patients suffering from CAD, type 2 DM caused by MS was studied before and after the start of treatment with telmisartan. The results of the study demonstrated the changes in hormonal spectrum and lipid metabolism after the beginning of treatment with telmisartan. The cortisol level in the experimental group was not significantly different from the control values on admission to the inpatient department. Within a month of treatment, the cortisol level exhibited a tendency to decrease in comparison with its initial level. The cortisol level in the comparison group also tended to reduce in standard therapy if compared to its initial level. Therefore, both treatment with telmisartan and standard therapy contributed to the reduction of the cortisol level. Before the start of treatment in the inpatient department, the prolactin level in women of the experimental group was not significantly different from the control values and kept increasing substantially within a month of treatment, whereas the prolactin level in females of the comparison group exhibited only a growing tendency within the course of treatment. At the beginning of observation, the prolactin level in men of the experimental group was significantly higher than at the control values. While treating with telmisartan, the level of prolactin in males of the experimental group increased significantly, whereas in the comparison group, it did not change dramatically. Therefore, a considerable increase...
of prolactin levels in males and females was observed in telmisartan treatment. The level of thyroid-stimulating hormone in patients of the experimental group was significantly higher if compared with the control values before the beginning of treatment. The level of the above-mentioned hormone kept decreasing considerably within the course of treatment unlike the thyroid-stimulating hormone level in patients of the comparison group which tended to increase at the beginning of observation and did not change dramatically in the course of treatment. The major increase in free thyroxine level was typical for the patients in the experimental group within the course of treatment, while the level of free thyroxine in the patients of the comparison group did not change significantly in the course of treatment. A dramatic increase in levels of triglycerides as well as very-low density lipoprotein cholesterol and a significant decrease in high-density lipoprotein cholesterol were typical for the lipid spectrum in patients of both experimental and comparison groups. The use of telmisartan as a part of standard therapy was accompanied by a significant decrease in total cholesterol (within the reference values), triglycerides, low-density lipoprotein cholesterol and very-low-density lipoprotein cholesterol. **Conclusions.** The use of telmisartan as a part of combined therapy facilitates the reduction of the cortisol level \( (p > 0.05) \) and leads to a significant rise in male and female prolactin levels (within the range of reference values). It triggers an apparent increase in free thyroxine and decrease in thyroid-stimulating hormone. These findings reveal the impact of telmisartan on the correction of metabolic disorders, particularly the effect on the manifestations of subclinical hypothyroidism. Telmisartan has a beneficial effect on the lipid spectrum of blood. It greatly reduces the levels of total cholesterol, triglycerides, low-density lipoprotein cholesterol as well as very-low-density lipoprotein cholesterol.

**Keywords:** telmisartan; cortisol; prolactin; thyroid-stimulating hormone; coronary artery disease; type 2 diabetes mellitus

**Introduction**

The renin-angiotensin system (RAS) has been studied thoroughly as a factor that regulates water-salt metabolism and blood pressure [1]. The systemic effect of RAS can be explained by the location of angiotensin receptors in many organs and systems. RAS homeostasis is maintained with the help of a simultaneous presence of two types of angiotensin II receptors: angiotensin II type 1 receptors, which promote vasoconstriction and have antinatriuretic as well as pro-inflammatory effects; angiotensin II type 2 receptors, which are responsible for vasodilation, natriuresis, anti-inflammatory effect. On the contrary, angiotensin II type 2 receptors perform a protective function in the blood pressure regulation mechanism [2]. Disruptive influence upon these two means of regulation leads to the development of cardiovascular, autoimmune diseases, and systemic inflammation [3].

Angiotensin II is produced by both local, self-sufficient renin-angiotensin systems localized in blood vessels, adipose tissue, pancreas, adrenal cortex, etc., and central renin-angiotensin system [4].

The interconnection between the renin-angiotensin system and the hypothalamic-pituitary system is well studied. Due to the activation of angiotensin II type 1 receptors located within brain structures, the hormones of the hypothalamic-pituitary-adrenal axis are activated and stress as well as inflammatory reactions are regulated [5].

Angiotensin II is known to be involved in the regulation of prolactin secretion. The appearance of the renin-angiotensin system components in adipocytes within adipose tissue explains its pathogenetic link leading to obesity owning to the synthesis of many biologically active substances, especially the synthesis of angiotensinogen by adipocytes, which causes hypertension and metabolic syndrome (MS) [6].

Taking into consideration the remarkable effect caused by blockers of angiotensin II receptors, they are considered to be medications that are applicable within the course of pathogenetic treatment of individuals suffering from type 2 diabetes mellitus (DM) and coronary artery disease (CAD) [7]. The alterations of the hormonal spectrum caused by such treatment are considered to be studied insufficiently. These changes are well worth further study because they indicate the response of the whole body to therapy.

**Objective:** the study is aimed at scrutinizing endocrine and metabolic changes in patients suffering from coronary artery disease, type 2 diabetes mellitus caused by metabolic syndrome within the course of treatment with telmisartan.

**Materials and methods**

After a signed consent for the survey according to the principles stated in the “Declaration of Helsinki. The Council of Europe: Convention on Human Rights and Biomedicine” and relevant Ukrainian laws, a comprehensive study of 51 randomly chosen individuals (26 females, 25 males) was conducted. Prior stratification of patients was performed with a view of diagnosing those suffering from CAD, type 2 DM caused by MS. All patients were treated at Lviv Regional State Clinical Medical Treatment and Diagnostic Endocrinology Center and CNE “City Clinical Hospital 5 of Lviv”.

The survey has been approved by Danylo Halytsky LNMU Ethics Committee concerning the issues of scientific research, experimental development, and scientific work dated 22.05.2019, protocol No 5.

The patients were divided into two groups depending on the treatment prescribed. The first group \((n = 27)\) consists of patients with CAD, type 2 DM caused by MS. It was an experimental group including 14 female and 13 male patients, who receive angiotensin II receptor blocker telmisartan 80 mg/day and standard therapy. Examinations have been performed on admission to the inpatient department and a month after the start of treatment. The comparison group consisted of 24 patients (12 females, 12 males suffering from CAD, type 2 DM triggered by MS) who received standard therapy. The control group includes 40 apparently healthy individuals \((men — 17 (42.5\%), women — 23 (57.5\%))\).
The exclusion criterion is the presence of another co-morbidity, including clinically manifested acute and chronic diseases, oncologic pathology, etc.

The inclusion criteria are diagnosed CAD and type 2 DM triggered by MS (with class II–III obesity).

MS was diagnosed based on the diagnostic criteria of the International Diabetes Federation (IDF Brussels: 2005). The recommendations of the European Association for the Study of Diabetes and the American Heart Association.

The patients who participated in the research were diagnosed with class I–II (the classification provided by Canadian Cardiovascular Society) stable angina (based on the results of exercise tolerance test, particularly cardiac stress test) and class I–II heart failure (by the NYHA criteria).

Type 2 DM was diagnosed in accordance with the corresponding guidelines provided by both the American Diabetes Association and the European Association for the Study of Diabetes regarding the diagnostic criteria for diabetes mellitus, i.e. blood sugar level ≥ 7.0 mmol/l and glycated haemoglobin level > 6.5 %. Glycated haemoglobin was used as the main criterion that enabled us to subdivide DM into compensated and decompensated types according to the recommendations of IDF.

As to analyze lipid metabolism, the values of total cholesterol (TCH), low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, triglycerides (TG) were defined. LDL was calculated applying the formula introduced by W.T. Friedwald: LDL cholesterol = TCH – (TG) measured in mmol/l.

Cortisol, prolactin, free thyroxine (fT₄), and thyroid-stimulating hormone (TSH) levels were measured in patients of all groups.

Cortisol was measured with the help of enzyme-linked immunosorbent assay applying “DS-EIA-Steroid-Cortisol” reagents and monoclonal antibodies. The levels of fT₄ and thyroid-stimulating hormone were studied using solid-phase enzyme-linked immunosorbent assay applying “DS-EIA-Thyroid-TSH” kit reagents. Prolactin was measured using an enzyme-linked immunosorbent assay applying “DS-EIA-Prolactin” kit reagents.

Microsoft Excel was used to conduct statistical analysis. The above-mentioned software was crucial for the creation of a database. The Mann-Whitney U test was used while comparing the parameters of two independent groups. The statistical characteristics were represented by a median, lower, and upper quartiles. Then, the significance level was set based on the p-value < 0.05.

**Results**

The results of the study (Table 1) show characteristic features of the hormonal status of individuals on admission to the inpatient department and a month after the beginning of treatment.

The cortisol level in the experimental group before treatment was 150.93 [123.79; 177.67] ng/ml and did not significantly differ from the control values 159.68 [115.32; 188.51] ng/ml (p = 0.88).

The cortisol level tended to decrease to 142.7 [125.74; 228.72] ng/ml in patients who were undergoing treatment in comparison with its level before receiving therapy (p = 0.82). The comparison group also tended to have the reduction of the cortisol level after therapy (146.0 [133; 147.1] ng/ml) if compared with its level before treatment (156.47 [110.01; 195.87] ng/ml (p = 0.45)).

The prolactin level in women of the experimental group before treatment accounted for 10.96 [6.94; 12.91] ng/ml. It reached 10.9 [7.7; 15.4] ng/ml (p = 0.36) at the control level and increased significantly up to 19.02 [13.64; 19.65] ng/ml (p = 0.007) after treatment. The prolactin level in women of the comparison group after treatment did not change significantly accounting for 11.6 [8.87; 14.42] ng/ml (p = 0.69) compared with its level before the start of therapy 11.07 [9.35; 17.30] ng/ml and compared with the control values.

### Table 1. Dynamics of hormonal parameters before and after a month of treatment with telmisartan

| Indicator                              | Control group (n = 40) | Experimental group (n = 27) | Comparison group (n = 24) |
|----------------------------------------|------------------------|-----------------------------|---------------------------|
|                                        | before treatment with telmisartan | after a month of treatment with telmisartan | before receiving standard therapy | a month after receiving standard therapy |
| Cortisol, ng/ml                         | 159.68 [115.32; 188.51] | 150.93 [123.79; 177.67]   | 142.7 [125.74; 228.72]    | 156.47 [110.01; 195.87]      | 146.0 [133; 147.1] |
| Prolactin (females), ng/ml             | 10.9 [7.7; 15.4]       | 10.96 [6.94; 12.91]       | 19.02 [13.64; 19.65]*     | 11.07 [9.35; 17.30]         | 11.6 [8.87; 14.42]* |
| Prolactin (males), ng/ml               | 7.15 [6.70; 9.50]      | 9.58 [8.16; 13.77]        | 12.42 [11.79; 18.24]*     | 8.04 [7.62; 10.28]          | 8.86 [7.6; 10.97]** |
| Thyroid-stimulating hormone, mU/dl     | 1.91 [0.97; 2.94]*     | 2.92 [1.78; 3.76]*        | 1.96 [1.14; 3.11]*        | 2.61 [1.83; 3.09]           | 2.67 [1.45; 3.94] |
| Free thyroxine, ng/dl                  | 1.38 [1.20; 1.50]*     | 1.16 [1.01; 1.3]*         | 1.39 [1.24; 1.7]*         | 0.93 [0.85; 1.4]            | 1.0 [0.97; 1.07]** |

**Notes:** * — the difference with the control group has been verified (p < 0.05); ** — the difference between the values on admission to the inpatient department and a month after the start of treatment has been verified (p < 0.05); "#" — the difference between the values of the experimental and comparison groups after a month of treatment has been verified (p < 0.05).
The level of thyroid-stimulating hormone in patients of the experimental group before receiving treatment accounted for 3.47 [2.55; 4.09] mU/dl and did not change significantly if compared with its values before treatment, remaining at the level of 1.93 [1.49; 2.12] mmol/l (p > 0.05). It is worth noting that the levels of total cholesterol in patients of the experimental group were significantly lower than in those in the comparison group (p = 0.04).

An increased level of triglycerides in patients of the experimental group accounted for 1.89 [1.62; 2.6] mmol/l (p < 0.05) compared with the control values. The level kept decreasing considerably until it reached 1.57 [1.18; 2.0] mmol/l (p = 0.02) while patients were receiving telmisartan treatment. The level of triglycerides in patients of the comparison group accounted for 1.9 [1.41; 2.47] mmol/l before treatment and did not change while receiving treatment, remaining at the level of 1.93 [1.49; 2.12] mmol/l (p > 0.05).

Table 2 presents the dynamics of the blood lipid spectrum on the background of the treatment with telmisartan.

| Indicator                                      | Control values (mmol/l) | Experimental group (n = 27) | Comparison group (n = 24) |
|------------------------------------------------|-------------------------|-----------------------------|---------------------------|
| before treatment with telmisartan              |                         | after a month of treatment  | before receiving standard therapy | a month after receiving standard therapy |
| Total cholesterol, mmol/l                      | 4.8 [3.89; 5.19]        | 5.2 [4.51; 5.41]            | 5.17 [4.21; 6.2]          | 5.10 [4.05; 6]**                      |
| Triglycerides, mmol/l                          | 0.85 [0.69; 0.94]       | 1.89 [1.62; 2.6]**          | 1.9 [1.41; 2.47]**        | 1.93 [1.49; 2.12]**                   |
| High-density lipoprotein cholesterol, mmol/l   | 1.25 [1.03; 1.54]       | 0.98 [0.55; 1.1]**          | 0.97 [0.805; 1.26]**      | 0.905 [0.79; 1.15]**                 |
| Low-density lipoprotein cholesterol, mmol/l    | 3.24 [1.87; 3.54]       | 3.42 [2.89; 4.04]          | 2.0 [1.5; 2.84]**         | 3.47 [2.55; 4.09]                    |
| Very-low-density lipoprotein cholesterol, mmol/l | 0.39 [0.31; 0.43] | 0.9 [0.74; 1.25]**          | 0.86 [0.64; 1.13]**       | 0.83 [0.68; 0.96]**                  |

Notes: * — the difference with the control group has been verified (p < 0.05); ** — the difference if the indicators are compared while undergoing treatment has been verified (p < 0.05); *** — the difference has been verified (p < 0.05) if the indicators of the experimental and comparison groups are compared after a month of receiving treatment.

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9.0 [7.7; 15.4] ng/ml (p > 0.05). It should be noted that the prolactin level in females of the experimental group after treatment was significantly higher than that in women of the comparison group (p = 0.002). Consequently, a significant increase in prolactin level in females was observed while undergoing treatment with telmisartan.

The prolactin level in men of the experimental group after treatment accounted for 2.61 [1.83; 3.09] mmol/l (p = 0.0029). Subsequently, an apparent increase in prolactin level in males was observed while treating them with telmisartan.

An increased level of free thyroxine was typical for patients in the experimental group after undergoing treatment and accounted for 1.38 [1.20; 1.50] ng/dl (p < 0.05). The level of free thyroxine in the patients of the comparison group did not vary considerably — 8.86 [7.6; 10.97] ng/ml in comparison with its level before treatment 8.04 [7.62; 10.28] ng/dl (p = 0.53). It was significantly lower if compared with the prolactin level after treatment of the experimental group (p = 0.0000065).

An increased level of free thyroxine was typical for patients in the experimental group after undergoing treatment and accounted for 1.38 [1.20; 1.50] ng/dl (p < 0.02), while its level before therapy was 1.16 [1.01; 1.3] ng/dl. The latter was considerably while patients were undergoing telmisartan treatment, namely 2.61 [1.83; 3.09] mmol/l (p = 0.08).

The level of thyroid-stimulating hormone in patients of the comparison group did not vary considerably — 8.86 [7.6; 10.97] ng/ml if compared with the values before treatment 9.58 [8.16; 13.77] ng/ml. The free thyroxine level remained lower after therapy if compared with that in the experimental group (p = 0.001).

Notes: * — the difference with the control group has been verified (p < 0.05); ** — the difference if the indicators are compared while undergoing treatment has been verified (p < 0.05); *** — the difference has been verified (p < 0.05) if the indicators of the experimental and comparison groups are compared after a month of receiving treatment.
The level of LDL cholesterol in patients of the experimental group before treatment with telmisartan reached 3.42 [2.89; 4.04] mmol/l and was not significantly different from the control values, namely 3.24 [1.87; 3.54] mmol/l (p > 0.05). After receiving telmisartan treatment, the level of LDL cholesterol decreased to 2.0 [1.5; 2.84] mmol/l. The level of LDL cholesterol in patients of the comparison group was 3.47 [2.55; 4.09] mmol/l before undergoing treatment with telmisartan. After the treatment, the level of LDL cholesterol in the comparison group patients accounted for 3.44 [2.91; 4.65] mmol/l (p = 0.6). The level of LDL cholesterol in the experimental group after treatment was considerably lower 2.0 [1.5; 2.84] mmol/l versus that in the comparison group after treatment, namely 3.44 [2.91; 4.65] mmol/l (p = 0.001).

The level of very-low-density lipoprotein (VLDL) cholesterol in patients of the experimental group before treatment was 0.9 [0.74; 1.25] mmol/l and was significantly higher compared with the control values — 0.39 [0.31; 0.43] mmol/l (p < 0.05). While the patients were getting telmisartan treatment, the level kept reducing considerably reaching 0.71 [0.54; 0.9] mmol/l, whereas the level of VLDL cholesterol in patients of the comparison group displayed the tendency to decrease ranging from 0.86 [0.64; 1.13] to 0.83 [0.68; 0.96] mmol/l (p = 0.42).

Discussion

While receiving telmisartan treatment, the cortisol level in the patients of the experimental group tended to reduce and the prolactin level tended to increase significantly (within reference values). The following values were typical for both males and females.

The cortisol level displayed a marked tendency to decrease in the comparison group as well. Such positive results indicated changes in response to treatment, whereas telmisartan did not influence the dynamics of the cortisol level.

Our results correspond to the findings presented by other researchers [8].

The interpretation of different values of prolactin [9] in patients suffering from CAD [10] or/and type 2 DM [11] and its impact on lipid and carbohydrate metabolism is controversial in different scientific studies [12]. The existence of a wide range of normal prolactin values (1–25 μg/l) partly explains the controversy over attempts to interpret different indicators of this hormone. Both prolactin and cortisol are considered to be stress hormones that change in acute conditions, particularly in acute coronary syndrome [13]. The action of telmisartan on angiotensin II receptors, which are located at the level of the hypothalamus and pituitary gland, produces a modulating effect on the secretion of pituitary hormones [14].

Telmisartan proved to have a specific effect on the structures of the diencephalon owing to angiotensin II type 1 receptors. Telmisartan selectively blocks the receptors and has a stabilizing influence on the activity ratios of the pituitary hormones, namely adrenocorticotropicin, thyroid-stimulating hormone, prolactin [15].

These regulatory processes directly affect the autonomie functions. Prolactin regulates and maintains metabolic homeostasis simultaneously. While studying obesity, it has been found that prolactin can provide an adaptive response to protect the body from metabolic disorders. It has been found that functional transient increase in prolactin levels is a physiological response to metabolic changes [16]. Therefore, the results of our research suggest that telmisartan facilitates the increase of the prolactin level (within reference values). This action is aimed at correcting the existing metabolic disorders.

The thyroid status of the experimental group was characterized by increased TSH and decreased \( \Gamma \) (within the range of reference values) verifying the results of clinical studies which consider hypothryoidism a key link in the pathogenesis of MS [17]. The disorders of peripheral conversion of thyroid hormones which are accompanied by the non-thyroidal illness syndrome have been studied in patients with MS [18].

Decreased TSH and increased \( \Gamma \) (within the range of reference values) are interpreted as means of subclinical hypothyroidism correction within the scope of our study.

A study of the lipid spectrum during the treatment with telmisartan revealed positive changes due to the reduction of the total cholesterol level (within the range of reference values), triglycerides, LDL cholesterol, and VLDL cholesterol. This is due to the effect of telmisartan on the expression of PPAR-\( \gamma \) target genes [19], the nuclear receptor of which can increase insulin sensitivity, the levels of HDL cholesterol, reduce systemic inflammation, symptoms of oxidative stress, fatty acids and triglyceride levels [20]. Consequently, the administration of telmisartan as a part of a comprehensive treatment approach has a positive impact on the condition of RAS which, in turn, facilitates the functions of the hypothalamo-pituitary system, the endocrine organs (the thyroid gland, the adrenal glands) and stabilizes lipid metabolism.

Conclusions

The use of telmisartan as a part of a comprehensive treatment approach promotes the reduction of cortisol level (p > 0.05), significantly increases prolactin level in males and females (within reference values). It triggers a considerable increase in the level of \( \Gamma \), and a decrease in the level of TSH. The data prove that telmisartan has a pronounced positive effect and corrects metabolic disorders, in particular the manifestations of subclinical hypothryoidism. Telmisartan has a positive impact on the blood lipid spectrum and significantly reduces the levels of total cholesterol, triglycerides, LDL cholesterol, and VLDL cholesterol.

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Динаміка ендокринних та обмінних зрушень у пацієнтів з ішемічною хворобою серця, цукровим діабетом 2-го типу і метаболічним синдромом при лікуванні телмісартаном

Резюме. Актуальность. Взаємозв’язок ренін-ангіотензинової та гіпоталамо-гіпофізарної систем — патогенетична ланка багатьох коморбідних захворювань, зокрема цукрового діабету (ЦД) 2-го типу та ішемічної хвороби серця (ІХС). Зацікавленість гормональних структур як гіпоталамус, гіпофіза, так і периферичних органів ендокринної системи підтверджена в багатьох роботах. Наявність спільних ланок гіпоталамо-гіпофізарної системи з спектовим стресом та патогенезу й регулюючих чинників змушує шукати нові методи патогенетичного лікування, які б мали комплексну дію на коморбідну патологію. Одним із таких напрямків лікування є застосування телмісартану — блокатора рецепторів ангіотензину II. Однак зміни гормонального статусу, ліпідного спектру, що характеризують стан пацієнта в процесі лікування, залежать від вивчених недостатньо, що й зумовлює до
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цільність проведення цього дослідження. Мета дослідження: вивчення динаміки ендокринних та обмінних зрушень у пацієнтів з ішемічною хворобою серця та цукровим діабетом 2-го типу на тлі метаболічного синдрому (МС) при лікуванні телмісартаном. Матеріал і методи. Обстежено 51 пацієнта (26 жінок, 25 чоловіків) з ІХС та ЦД 2-го типу, який виник на тлі метаболічного синдрому. Дослідження проводилось в умовах Львівського обласного державного клінічного лікувально-діагностичного ендокринологічного центру та КНП «П'яті міська клінічна лікарня м. Львова». Пацієнтів поділено на дві групи — дослідну та групу порівняння залежно від призначеного лікування. До дослідної групи (n = 27) увійшли особи з ІХС та ЦД 2-го типу (на тлі МС) (жінок — 14, чоловіків — 13), які отримували телмісартан 80 мг/добу та стандартну терапію. Групу порівняння становили 24 пацієнти з ІХС та ЦД 2-го типу (на тлі МС) (жінок — 12, чоловіків — 12), які отримували стандартну терапію. До контрольної групи ввійшли 40 практично здорових осіб (чоловіків — 17 (42,5 %), жінок — 23 (57,5 %)). Обстеження проведене при надходженні до стаціонару й через один місяць після початку лікування. У пацієнтів визначали рівень пролактину, кортизолу, вільного тироксину та тиреотропного гормона, а також показники ліпідного спектра.

Результати. У роботі досліджено динаміку рівнів пролактину, кортизолу, вільного тироксину й тиреотропного гормона в пацієнтів з ІХС та ЦД 2-го типу, який виник на тлі МС, до лікування та через один місяць після початку лікування. Згідно з результатами дослідження, виявлені такі зміни гормонального спектра та ліпідного обміну. У дослідній групі при надходженні до стаціонару рівень кортизолу був вірогідно вищим від контрольних величин, і через один місяць лікування відзначалась тенденція до його зниження порівняно з вихідним показником. У групі порівняння також була вірогідна тенденція до зниження рівня кортизолу впродовж лікування порівняно з початковим показником. Отже, як лікування телмісартаном, так і стандартна терапія сприяли зниженню рівня кортизолу.

Висновки. Застосування телмісартану в складі комплексної терапії сприяло зниженню відсотка вартів від кортизолу (р > 0,05), вірогідному зниженню рівня пролактину в жінок та чоловіків (у межах референтних значень), вірогідному зниженню рівня тиреотропного гормона (р > 0,05), вірогідному зниженню загального холестерину, тригліцеридів, холестерину ліпопротеїнів низької щільності та ліпопротеїнів високої щільності. Застосування в складі комплексної терапії телмісартану сприяло статистично зниженню рівня холестерину ліпопротеїнів низької і дуже низької щільності. Телмісартан сприйняв значну частину з таких асоцій сальво-метаболітичних. Ефект телмісартану у процесі лікування вірогідно не змінювався вірогідно і вірогідно знижувався вірогідно.