

Mental health and sleep disorders in obese patients, their relationship with clinical and pathogenetic changes in the organism and impact on quality of life

T.O. Bagro, V.I. Tkachenko

Shupyk National Healthcare University of Ukraine, Kyiv

In the world the problem of obesity is an important aspect in the non-infectious diseases progression. It is known, that 95% of the pathogenic factor for the obesity development depends on the changes caused by neurochemical, hormonal and metabolic mechanisms that occur by depression, anxiety, eating disorders, circadian rhythms and sleepiness.

The objective: to determine the psychosocial features and their relationship with clinical and pathogenetic changes in obese patients of working age.

Materials and methods. 75 patients with obesity of the 1st and 2d degrees (39.03 ± 0.93 years old) and 75 practically healthy persons of the corresponding age (36.84 ± 0.96 years old) were examined. Waist circumference (WC), hip circumference (HC), body surface area (BSA), waist/hip ratio (WHR), conicity index (ConI), a body shape index (ABSI), abdominal volume index (AVI), blood pressure, blood levels of glucose, insulin, index HOMA, cholesterol, lipidogram indicators, serotonin, and leptin were determined in the patients.

The psychological status was assessed by Hospital Anxiety and Depression Scale (HADS), Hamilton Anxiety Scale (HAM-A), the Dutch Eating Behavior Questionnaire (DEBQ), Epworth Sleepiness Scale (ESS), Pittsburgh Sleep Quality Index (PSQI), the patient's quality of life – by 36-Item Short Form Survey (SF-36). The statistical analysis was conducted by IBM SPSS Statistics, Statistica 12, descriptive statistics Excel 2010.

Results. The patients of the studied group, unlike the control group, had significantly higher indices of WC, HC, BMI, WHR, ConI, ABSI, AVI, HOMA index, blood pressure, glucose, insulin, total cholesterol, lipidogram, leptin and the lower level of serotonin. This was accompanied with clinically expressed anxiety of depression which led to eating disorders (a tendency “to eat emotions”, to overeat when food is available, a habit to eat without restrictions), sleep disorders (excessive day time sleepiness, low quality of sleep) and reduced quality of life.

The results of correlation analysis indicate a strong or moderate positive correlation between obesity indices, glucose level, lipidogram, atherogenicity index, HOMA index, scores of depression and anxiety scales, as well as strong negative correlation with ABSI index, high density lipoproteins, serotonin. In addition, a positive correlation was found between leptin level and scores of depression and anxiety scales, scores of eating behavior, sleepiness, and a negative correlation was determined between these indices and serotonin level.

Conclusions. The patients of the studied group (100.0 %) had abdominal obesity. Among the anthropometric indices ABSI and AVI ones were the most significant and informative for determination of abdominal obesity in gender aspect, this can be an alternative for MRI diagnosis of visceral obesity at the level of primary medical care.

A close relationship between abdominal obesity with psycho-emotional disorders, disorders of sleep and eat behavior, metabolic disorders, leptin and serotonin levels was determined. Taking into account these interrelationships in a patient-centered management in persons with obesity will improve the quality of medical care.

Keywords: abdominal obesity, anxiety, depression, quality of sleep, sleepiness, eating behavior, serotonin, leptin, primary medical care.

Порушення психічного здоров'я та сну у пацієнтів з ожирінням, їх взаємозв'язок з клініко-патогенетичними змінами організму та вплив на якість життя

T.O. Bagro, V.I. Tkachenko

Проблематика ожиріння у світі відіграє важливу роль у прогресуванні неінфекційних захворювань. Відомо, що 95% патогенетичного фактора розвитку ожиріння залежить від змін, які обумовлені нейрохімічними, гормональними та метаболічними механізмами, що виникають при депресії, тривозі, порушенні харчової поведінки, циркадних ритмів та сонливості.

Мета дослідження: визначення психосоціальних особливостей та їх взаємозв'язок з клініко-патогенетичними змінами у пацієнтів працездатного віку з ожирінням.

Матеріали та методи. Обстежено 75 пацієнтів з ожирінням 1-го та 2-го ступеня ($39,03 \pm 0,93$ року) та 75 практично здорових осіб відповідного віку ($36,84 \pm 0,96$ року). Пацієнтам визначали об'єм талії (ОТ) та стегон (ОС), площу поверхні тіла (BSA), співвідношення талії/стегон – WHR, індекс конусності (ConI), індекс форми тіла (ABSI), індекс абдомінального об'єму (AVI), артеріальний тиск, рівні в крові глюкози, інсуліну, індексу НОМА, холестерину, показники ліпідограми, серотоніну та лептину.

Психосоціальний статус оцінювали за допомогою госпітальної шкали тривоги та депресії HADS, шкали Бека, шкали Гамільтона (HAM-A), Голландського опитувальника харчової поведінки (DEBQ), шкали сонливості Епворта (Epworth Sleepiness Scale (ESS)), Пітсбурзького опитувальника якості сну (PSQI), якості життя – SF-36. Статистичний аналіз здійснювали за допомогою IBM SPSS Statistics, Statistica 12, описова статистика Excel 2010.

Результати. Пацієнти дослідної групи, на відміну від контрольної, мали достовірно вищі показники ОТ, ОС, ІМТ, WHR, BSA, індексів ConI, ABSI та AVI, індексу НОМА, АТ, глюкози, інсуліну, загального холестерину, ліпідограми, лептину і нижчий рівень серотоніну. Це супроводжувалось клінічно вираженою тривогою чи депресією, що вплинуло на появу змін харчової поведінки (схильність «заїдати емоції», переїдати при доступності їжі, звичка до харчування без обмежень), порушення сну (надмірна денна сонливість, низька якість сну) та зниження якості життя.

Кореляційний аналіз продемонстрував сильний або середньої сили позитивний кореляційний зв'язок між індексами ожиріння, рівнями глюкози, ліпідограми, індексом атерогенності, індексом НОМА, лептину, балами шкал депресії і тривоги, а також сильний негативний з індексом ABSI, ЛПВЩ, серотоніном. Крім того, позитивний кореляційний зв'язок спостерігався між рівнем лептину та балами шкал депресії та тривоги, балами харчової поведінки, сонливості та негативний кореляційний зв'язок цих показників з рівнем серотоніну.

Висновки. Пацієнти дослідної групи (100%) мали абдомінальне ожиріння. Серед антропометричних індексів найбільш достовірними та інформативними для визначення абдомінального ожиріння в гендерному аспекті є індекси ABSI та AVI, що може бути альтернативою МРТ діагностики вісцерального ожиріння в первинній медичній допомозі. Визначено тісний взаємозв'язок абдомінального ожиріння з психоемоційними розладами, порушеннями сну, харчової поведінки, метаболічними порушеннями та рівнями лептину і серотоніну. Врахування цих взаємозв'язків при застосуванні пацієнт-орієнтованого підходу ведення пацієнтів з ожирінням дозволить покращити якість медичної допомоги.

Ключові слова: абдомінальне ожиріння, тривога, депресія, якість сну, сонливість, харчова поведінка, серотонін, лептин, первинна медична допомога.

The problem of obesity in the world plays a significant role in the progression of non-infectious diseases [1, 2]. Obesity, as a chronic disease, requires significant attention from family doctors, at primary and preventive examinations, in order to prevent a number of complications and help them modify their lifestyle [3].

It is known that 95% of the pathogenetic circle of the development of obesity depends on changes caused by neurochemical, hormonal and metabolic mechanisms that occur in depression, anxiety, eating disorders and circadian rhythms, drowsiness [4]. Human response to stress is regulated by neuroendocrine mechanisms of the hypothalamic-pituitary-adrenal system; disturbances that occur in it contribute to the emergence of anxiety and deterioration of the emotional state, which leads to the development of depression and a decrease in the concentration of serotonin [5, 6]. Serotonin affects the regulation of circadian rhythms and food intake through direct and indirect effects on adipose tissue, eating behavior, sleep, and appetite [7–17]. Therefore, in patients with psychoemotional disorders, overeating is the main mechanism of providing the body with such neurotransmitters as serotonin and dopamine [18, 19]. The central regulation of energy balance and eating behavior occurs in the ventromedial nuclei of the hypothalamus (satiety center) and in the lateral hypothalamus (hunger center) [20]. Activation of the hypothalamic-pituitary-adrenal system not only activates the ventromedial nuclei of the hypothalamus but also increases the circulation of corticosteroids in the blood [21, 22]. The increased level of cortisol and insulin in the circulating blood provokes not only the utilization of glucose but also the accumulation of adipose tissue, which, under such stimulation, secretes adipokines, including leptin [23]. Leptin is a hormone that stimulates corticotropin-releasing hormone, proopiomelanocortin, and melanocortin mRNA in the aquatic nuclei of the hypothalamus. According to the principle of feedback, this leads to a decrease in food consumption and loss of body weight [24–26]. Leptin, insulin and other mediators of energy metabolism affect the limbic system and the hy-

pothalamus, where receptors found for them control behavior, emotions, motivation, and its transformation into action [27, 28].

The formation of precisely the abdominal type of obesity closes the pathogenetic circle since visceral fat is a hormonally active tissue that deepens hormonal imbalance, causes increased anxiety and depression, which in turn affects sleep disorders and additional risks of non-infectious diseases [5, 29–32]. Research on the relationship between abdominal obesity, mental disorders, circadian rhythm disorders, and the hormonal background will improve a patient-oriented approach and management of obese patients.

The objective: to determine psychosocial features and their relationship with clinical-pathogenetic changes in obese patients of working age.

MATERIALS AND METHODS

We examined 75 patients with obesity of the I and II degree (body weight 96.12 ± 1.60 kg, BMI = 33.66 ± 0.37 kg/m², I degree – 51 patients, II degree – 24 patients), aged 39.03 ± 0.93 years, including 39 women (weight 90.98 ± 10.51 kg, age 37.74 ± 4.36) and 36 men (body weight 101.69 ± 11.74 kg aged 40.42 ± 4.67 years), who made up the study group. As a control group, 75 practically healthy people of the appropriate age (36.84 ± 0.96 years) with normal body weight (73.33 ± 0.73 kg, BMI 23.50 ± 0.15 kg/m²) took part in the study, including 39 women (weight 70.29 ± 8.12 kg, age 35.44 ± 4.09 years), 36 men (weight 76.62 ± 8.85 kg, age 38.36 ± 4.43 years).

The next indexes were determined – waist circumference (WC) and hip circumference (HC), calculated anthropometric indicators – body surface area (BSA), Waist-to-Hip Ratio (WHR), Conicity Index (ConI), A body shape index – (ABSI), Abdominal Volume Index (AVI). Clinical and laboratory examination included measurement of blood pressure, blood levels of glucose, insulin, HOMA index, total cholesterol, lipidogram indicators, serotonin and leptin. Psychosocial status was assessed using the Hospital Anxiety and Depression Scale (HADS), the Beck Scale,

the Hamilton Scale (HAM-A), the Dutch Eating Behavior Questionnaire (DEBQ), the Epworth Sleepiness Scale (ESS), the Pittsburgh Sleep Quality Questionnaire (PSQI), quality of life – SF-36. Statistical analysis was performed using IBM SPSS Statistics, Statistica 12, and descriptive statistics Excel 2010.

RESULTS

The detailed analysis of the anthropometric indicators of patients of both groups with the calculation of BSA, WHR, ConI, ABSI and AVI indices allowed to assess the peculiarities of the distribution of subcutaneous adipose tissue and determine the presence of abdominal obesity in the patients participating in the study. According to the obtained results, the patients of the study group had

significantly higher indicators of WC, HC, BMI, WHR, BSA, ConI, ABSI and AVI indices, in contrast to the control group (Table 1). This indicates the presence of abdominal obesity in most patients of the research group (n=75, 100%).

When analyzing anthropometric parameters in terms of gender, no significant difference was found in the body shape of men with and without obesity according to WHR (p(m/m)=0.21) and the ConI index (p(m/m)=0.37), although it was present among women (respectively, WHR p(w/w)=1.76E-36 and ConI p(w/w)=4.32E-25). In addition, no significant difference was found when comparing the indicated indices between men and women of the experimental group (WHR (p(w/m)=0.11); ConI (p(w/m)=0.94)), in that while it was present in the control

Table 1

Results of examinations of patients of study and control groups

| Indicator | Research group (n=75), M±m | Control group (n=75), M±m | p | |
|--|----------------------------|---------------------------|---------------------|---------------------|
| BMI, kg/m ² | 33,66±0,37 | 23,5±0,15 | p<0,05 (p=4,84E-56) | |
| WC, m | 1,04±0,12 | 0,79±0,01 | p<0,05 (p=5,85E-24) | |
| HC, m | 1,15±0,02 | 1,04±0,01 | p<0,05 (p=1,80E-09) | |
| BSA, м2 | 2,16±0,02 | 1,99±0,01 | p<0,05 (p=8,21E-09) | |
| WHR | 0,91±0,01 | 0,77±0,02 | p<0,05 (p=4,26E-13) | |
| ConI, м3/2/кг1/2 | 1,27±0,01 | 1,13±0,02 | p<0,05 (p=1,22E-08) | |
| ABSI, м5/3·кг-2/3 | 0,0772±0,0089 | 0,0829±0,0096 | p<0,05 (p=3,18E-03) | |
| AVI | 22,15±0,60 | 13,51±0,43 | p<0,05 (p=8,47E-23) | |
| BPs, mmHg | 135,87±1,75 | 119,12±1,55 | p<0,05 (p=3,88E-11) | |
| BPd, mmHg | 86,67±1,19 | 69,35±1,31 | p<0,05 (p=1,16E-17) | |
| Glucose, mmol/l | 6,31±0,11 | 4,80±0,06 | p<0,05 (p=1,68E-22) | |
| Insulin, μIU/ml | 17,08±0,53 | 11,61±0,20 | p<0,05 (p=1,15E-17) | |
| HOMA index, μmol·μl·ml ² | 4,9±0,21 | 2,45±0,03 | p<0,05 (p=2,56E-23) | |
| Total cholesterol, mmol/l | 5,44±0,12 | 4,14±0,10 | p<0,05 (p=2,8E-14) | |
| HDL, mmol/l | 1,56±0,03 | 1,59±0,04 | p=0,604 | |
| LDL, mmol/l | 3,79±0,11 | 1,79±0,12 | p<0,05 (p=4,13E-24) | |
| VLDL, mmol/l | 0,81±0,03 | 0,75±0,05 | p=0,344 | |
| Atherogenic index | 2,67±0,14 | 1,75±0,11 | p<0,05 (p=5,71E-07) | |
| Serotonin, μg/l | 154,91±2,49 | 175,73±5,80 | p<0,05 (p=1,29E-03) | |
| Leptin, ng/ml | 11,72±0,74 | 6,02±0,25 | p<0,05 (p=2,13E-11) | |
| Hamilton anxiety scale, points | 11,76±0,58 | 6,67±0,32 | p<0,05 (p=1,73E-12) | |
| HADS, points | anxiety | 10,15±0,54 | 6,63±0,30 | p<0,05 (p=4,37E-09) |
| | depression | 12,04±0,57 | 6,36±0,37 | p<0,05 (p=3,99E-14) |
| Beck's (depression scale), points | 13,12±0,80 | 6,87±0,41 | p<0,05 (p=1,14E-10) | |
| DEBQ, points | Emotional type | 2,94±0,11 | 2,32±0,12 | p<0,05 (p=1,19E-04) |
| | External type | 3,53±0,10 | 2,16±0,16 | p<0,05 (p=4,22E-11) |
| | Restrictive type | 3,80±0,09 | 3,32±0,11 | p<0,05 (p=6,43E-04) |
| Sleep quality (PSQI Global score), points | 8,44±0,34 | 5,72±0,31 | p<0,05 (p=2,75E-08) | |
| Drowsiness (ESS), points | 8,49±0,46 | 6,09±0,33 | p<0,05 (p=3,58E-05) | |
| SF-36 – physical component of health, points | Physical functioning (PF) | 66,1±1,61 | 85,13±1,5 | p<0,05 (p=3,67E-15) |
| | Role functioning (RP) | 48,0±3,91 | 69,2±3,3 | p<0,05 (p=5,61E-05) |
| | Bodily Pain (BP) | 63,6±2,93 | 72,17±2,9 | p<0,05 (p=3,99E-02) |
| | General health (GH) | 47,9±2,52 | 67,05±2,5 | p<0,05 (p=2,28E-07) |
| SF-36 – mental component of health, points | Vitality (VT) | 53,0±1,79 | 60,87±2,6 | p<0,05 (p=7,19E-03) |
| | Social Functioning (SF) | 55,4±2,68 | 63,43±2,6 | p<0,05 (p=3,30E-02) |
| | Role Emotional (RE) | 47,5±4,53 | 46,20±4,1 | p>0,05 (p=0,827) |
| | Mental Health (MH) | 52,4±3,01 | 65,28±2,8 | p<0,05 (p=2,23E-03) |

(WHR ($p(w/m)=2.91E-32$); ConI ($p(w/m)=1.56E-31$)). These results show the low sensitivity of the WHR and ConI indices to the description of body shape and determination of abdominal obesity in the gender aspect, the insensitivity of these indices to the features of the physique of female and male organisms.

On the contrary, the ABSI and AVI indices (Table 1) turned out to be more sensitive to the features of body structure depending on gender – significant differences were determined between subgroups of men (ABSI ($p(m/m)=1.32E-04$); AVI ($p(m/m)=1.92E-07$)) and women (ABSI ($p(w/w)=2.37E-21$); AVI ($p(w/w)=5.17E-23$)) of study and control groups, but when comparing these indices in men and women of the study group, no significant difference was found in contrast to the control group (ABSI ($p(w/m)=0.70$); AVI ($p(w/m)=0.46$)), where the difference was significant (ABSI ($p(w/m)=3.75E-30$); AVI ($p(w/m)=7.64E-32$)). This indicates the greatest informativeness of this index for determining abdominal obesity in the gender aspect.

Therefore, in the patients of the research group, the presence of abdominal obesity was determined in 100% of patients, both in men and in women, which is one of the risk factors for non-infectious diseases and their complications. Although the «gold standard» for diagnosing visceral obesity is MRI diagnosis [33, 34], this method is expensive, not always available and safe for the patient, so such alternative simpler, sensitive and easy methods as ABSI are more appropriate for primary medical care.

Obesity, especially of the abdominal type, is usually caused by long-term psycho-emotional experiences, which affects the eating behavior of patients, sleep disturbances, often associated with limiting the active functioning of the body, violations of the respiratory and musculoskeletal system, and as a result deepens changes in mental health and causes deterioration of the quality of life [35, 36].

The examination of the mental health of the patients determined that the patients of the study group had mental disorders, such as anxiety or depression. Thus, the presence of depression in the study group was indicated by the average scores on the HADS scale= 12.04 ± 0.57 points, on the Beck scale= 13.12 ± 0.80 points, which corresponds to clinically expressed depression. The presence of anxiety was confirmed by the average score on the HADS scale= 10.15 ± 0.54 points and on the Hamilton scale= 11.76 ± 0.58 points, which corresponds to clinically expressed anxiety. While in the control group the mental disorders were not determined, which was reliably significant in comparison with the study group ($p<0.05$).

The presence of mental disorders influenced the appearance of changes in eating behavior and sleep disorders. According to the Dutch Eating Behavior Questionnaire (DEBQ), the scale of the Emotional Type, the study group had a significantly higher tendency to “eat emotions” than the control group ($p<0.05$, $p=1.19E-04$), according to the External type scale of this questionnaire, the tendency of obese patients to overeat when food is available, in contrast to the control group, was revealed ($p<0.05$, $p=4.22E-11$) and according to the Restrictive type scale, the study group had a significantly more pronounced habit of eating without restrictions than the control group ($p<0.05$, $p=6.43E-04$).

When studying sleep disorders and sleepiness using the Epworth and PSQI Global score questionnaires, it was determined that the patients of the study group had excessive daytime sleepiness of a moderate degree, in contrast to the control group, and low quality of sleep, which was reliably significant (Table 1).

Obesity, as well as psychoemotional disorders, are risk factors for many non-infectious diseases and are often associated with arterial hypertension, carbohydrate and lipid metabolism disorders, hypodynamia, etc [37]. The results of the clinical and laboratory examination of the patients of the study group revealed normal high levels of blood pressure, both systolic (BP= 135.87 ± 1.75 mmHg) and diastolic (BP= 86.67 ± 1.19 mmHg.st.), although with a significant difference in contrast to healthy people (BP= 119.12 ± 1.5 mmHg, BP= 69.35 ± 1.3 mmHg, $p<0.05$).

The patients of the study group had impaired tolerance to carbohydrates, insulin resistance and significantly higher levels of insulin in the blood, in contrast to the control group. In addition, in the experimental group there was a violation of lipid metabolism – total cholesterol values were significantly higher ($p<0.05$, $p=2.8E-14$) and significantly exceeded the target levels recommended for patients with increased cardiovascular risk. Other indicators of the lipid profile LDL, VLDL, and atherogenicity index in the study group were within the normal range, but also significantly exceeded the levels of indicators in the control group.

Identified metabolic disorders in the obese patients were accompanied by hormonal disorders. Thus, the average level of serotonin in the blood of patients of the study group was 154.91 ± 2.49 $\mu\text{g/l}$, which was within the normal range, but significantly lower than the level in the control group (175.73 ± 5.80 $\mu\text{g/l}$, $p<0.05$), which confirms the other authors' data on the serotonin-dependent mechanism of obesity development [12, 38–40]. The average levels of leptin in the study group exceeded normal values and were significantly higher than those of the control group ($p<0.05$), the average level of leptin in women was 11.64 ± 1.34 ng/ml and was slightly higher than the norm (for women is 3.7–11.1 ng/ml); and in men it was 11.80 ± 1.36 ng/ml and was twice higher (the norm for men is 2.0–5.6 ng/ml), which indicates an excessive amount of hormonally active visceral fat to a greater extent in men and causes higher cardiovascular risks. There was no significant difference between leptin levels in women and men ($p>0.05$).

Obesity, metabolic changes, and mental state changes were reflected in the quality of life and social activity of patients in the study group (Table 1). Thus, in contrast to the control group, the obese patients had significantly lower indicators of the components of the SF36 questionnaire, which describe the state of the physical component of health (PF, RP, BP, GH), and indicators of the mental component of health (VT, SF, MH), however, role emotional functioning (RE) did not have a significant difference $p=0.827$. It was established that physical functioning (Physical Functioning – PF) was at a low level. Role physical functioning (Role Physical – RP) and general health (General Health -

Table 2

Correlation of serotonin with the studied indicators of the study group

| Indicator | r | p | |
|--|------------------|---------|---------|
| BMI, kg/m ² | -0,55 | p<0,001 | |
| Wt, m | -0,64 | p<0,001 | |
| H, m | -0,60 | p<0,001 | |
| BSA, м ² | -0,54 | p<0,001 | |
| WHR | 0,06 | p<0,1 | |
| ConI, м ^{3/2} /кг ^{-1/2} | -0,45 | p<0,001 | |
| ABSI, м ^{5/3} ·кг ^{-2/3} | 0,71 | p<0,001 | |
| BPs, mmHg | -0,48 | p<0,001 | |
| BPd, mmHg | -0,40 | p<0,001 | |
| Glucose, mmol/l | -0,59 | p<0,001 | |
| Insulin, μIU/ml | -0,30 | p<0,01 | |
| HOMA index, μmol·μl·ml ² | -0,45 | p<0,001 | |
| Total cholesterol, mmol/l | -0,35 | p<0,01 | |
| HDL, mmol/l | 0,56 | p<0,001 | |
| LDL, mmol/l | -0,46 | p<0,001 | |
| VLDL, mmol/l | -0,53 | p<0,001 | |
| Atherogenic index | -0,49 | p<0,001 | |
| Leptin, ng/ml | -0,61 | p<0,001 | |
| Hamilton anxiety scale, points | -0,57 | p<0,001 | |
| HADS, points | anxiety | -0,54 | p<0,001 |
| | depression | -0,56 | p<0,001 |
| Beck's (depression scale), points | -0,63 | p<0,001 | |
| DEBQ, points | Emotional type | -0,57 | p<0,001 |
| | External type | -0,02 | p<0,1 |
| | Restrictive type | -0,02 | p<0,1 |
| Sleep quality (PSQI Global score), points | -0,66 | p<0,001 | |
| Drowsiness (ESS), points | -0,66 | p<0,001 | |

GH) in patients with obesity are significantly lower, that indicates a worse physical condition in contrast to the control group, and does not allow to ensure full performance of daily activities (p<0.05). Limitation of daily activities was associated with pain syndromes, which is confirmed by a significantly lower index of pain intensity (Bodily Pain – BP) of patients in the experimental group.

Social and vital activity of obese patients (Social Functioning – SF and Vitality – VT) were significantly lower than those of control patients (p<0.05), which may be due to certain complexes or psychoemotional disorders, which is confirmed by a significantly lower total score assessment of mental health (Mental Health – MH), although role activity due to emotional state (Role emotional – RE) remained at the same level as in the control group (p>0.05). The obtained results indicate a lower level of quality of life in obese patients, which is reflected in social and physical activity.

Correlation analysis showed a strong or moderate positive correlation of BMI with WC (r=0.78, p<0.001), HC (r=0.80, p<0.001), ConI index (r=0.58, p<0.001), systolic blood pressure (r=0.71, p<0.001), diastolic blood pressure (r=0.66, p<0.001), glucose level (r=0.75, p<0.001), cholesterol (r=0.65, p<0.001), LDL (r=0.66, p<0.001), VLDL (r=0.64, p<0.001), leptin (r=0.73, p<0.001), atherogenic index (r=0.83, p<0.001), the HOMA index (r=0.63, p<0.001), depression scale scores (r=0.61, p<0.001), and HADS anxiety (r=0.51, p<0.001), Beck scale score (r=0.61, p<0.001) and Hamilton scale score (r=0.45, p<0.05), sleep scale scores (Epworth (r=0.64); PSQI (r =0.59, p<0.001), as well as a strong negative correlation with the ABSI index (r=-0.79, p<0.001), HDL (r=-0.78, p<0.001), serotonin (r=0.55, p<0.001); low correlations of the WHR index (r=-0.16, p<0.1) and eating behavior scores (rI=0.35, p<0.01; rII=-0.04, rIII=-0,01, p<0.1). In addition, a positive correlation was observed between the level of leptin and the scores of the depression (r=0.68, p<0.001) and anxiety scales of the HADS (r=0.60, p<0.001), Beck (r=0.72 , p<0.001) and Hamilton (r=0.58, p<0.001), eating behavior scores (rI=0.53, p<0.001; rII=0.08, rIII=-1.25E-04, p<0.1), sleepiness (Epworth (r=0.65); PSQI (r=0.60), p<0.001)) and the negative correlation of these indicators with the level of serotonin (Table 2).

The obtained data indicate a close relationship between obesity and psycho-emotional disorders, sleep disorders, eating behavior and leptin and serotonin levels, metabolic disorders. SF-36 scores were not significantly correlated with obesity scores, suggesting that patients' poor quality of life was related to other factors, which requires further study.

CONCLUSIONS

Comparative characteristics of obese patients and people with normal body weight showed that 100% of patients in the study group had abdominal obesity. The ABSI and AVI indices turned out to be the most sensitive calculated indicators that took into account the features of the body structure in obesity, related to sex and allowed

to determine abdominal obesity and can be an alternative to MRI for the diagnosis of visceral obesity in primary care.

Obesity in the patients of the study group was accompanied by clinically expressed anxiety and depression, disordered eating behavior in the form of a tendency to “stress eating”, overeating when food is available, and eating without restrictions; sleep disorders – excessive daytime sleepiness, poor sleep quality, and metabolic and hormonal disorders – impaired glucose tolerance, insulin resistance, increased cholesterol, leptin levels, low serotonin levels, and reduced quality of life. Correlation analysis confirmed the interrelations between obesity, metabolic disorders, and psychoemotional disorders, sleep disorders, leptin and serotonin levels. The obtained results should be taken into account in patient-centred treatment in order to improve the quality of the management of obese patients.

Information about the authors

Tkachenko Victoria I. – MD, PhD, DSc, Professor, Department of Family Medicine, Shupyk National Healthcare University of Ukraine, Kyiv. *E-mail: wtk@ukr.net*
 ORCID: 0000-0002-0789-5340
Bagro Taisia O. – MD, PhD-student, Department of Family Medicine, Shupyk National Healthcare University of Ukraine, Kyiv. *E-mail: taisia80@gmail.com*
 ORCID: 0000-0001-6881-8229

Відомості про авторів

Ткаченко Вікторія Іванівна – д-р мед. наук, проф., кафедра сімейної медицини, Національний університет охорони здоров'я України імені П.Л. Шупика, м. Київ. *E-mail: wtk@ukr.net*
 ORCID: 0000-0002-0789-5340
Багро Таїсія Олександрівна – лікар-терапевт, лікар загальної практики–сімейної медицини, аспірант, кафедра сімейної медицини, Національний університет охорони здоров'я України імені П.Л. Шупика, м. Київ. *E-mail: taisia80@gmail.com*
 ORCID: 0000-0001-6881-8229

REFERENCES

- Melnik OB, Fediv OI. Quality of life of patients with bronchial asthma combined with obesity, depending on the identification of FTO RS9939609 and RS324011 STAT6 gene polymorphism. *Inter J Endocrinol.* 2017;13(6):424-8. doi: 10.22141/2224-0721.13.6.2017.112884.
- Bray GA, Kim KK, Wilding JPH; World Obesity Federation. Obesity: a chronic relapsing progressive disease process. A position statement of the World Obesity Federation. *Obes Rev.* 2017;18(7):715-23. doi: 10.1111/obr.12551.
- Wharton S, Lau DCW, Vallis M, Sharma AM, Biertho L, Campbell-Scherer D, et al. Obesity in adults: a clinical practice guideline. *CMAJ.* 2020;192(31):875-91. doi: 10.1503/cmaj.191707.
- van Galen KA, Ter Horst KW, Serlie MJ. Serotonin, food intake, and obesity. *Obes Rev.* 2021;22(7):e13210. doi: 10.1111/obr.13210.
- Patriquin MA, Mathew SJ. The Neurobiological Mechanisms of Generalized Anxiety Disorder and Chronic Stress. *Chronic Stress (Thousand Oaks).* 2017;1:2470547017703993. doi: 10.1177/2470547017703993.
- Zagayko A, Shkapo A, Bryukhanova T. Study of mechanisms of influence of hydroxytryptophan on the content of serotonin in the brain under conditions of a high-calorie diet in rats. In: *Material VII Nat. congress of pathophysiology of Ukraine with international with the participation of Pathophysiology and pharmacy: ways of integration;* 2016 October 5-7; Kharkiv. Kharkiv: NFAU Publishing House; 2016. p. 88.
- Froy O. Circadian rhythms and obesity in mammals. *ISRN Obes.* 2012; 2012:437198. doi: 10.5402/2012/437198.
- Sridhar GR, Lakshmi G. Sleep, obesity and diabetes: the circadian rhythm. *Advances in diabetes: newer insights.* In: Sridhar GR (Ed), editor. New Delhi: The Health Services Publisher; 2016. p. 196-207.
- Sridhar GR, Sanjana NS. Sleep, circadian dysrhythmia, obesity and diabetes. *World J Diabetes.* 2016;7(19):515-22. doi: 10.4239/wjcd.v7.i19.515.
- Namkung J, Kim H, Park S. Peripheral Serotonin: a New Player in Systemic Energy Homeostasis. *Mol Cells.* 2015;38(12):1023-8. doi: 10.14348/molcells.2015.0258.
- Voigt JP, Fink H. Serotonin controlling feeding and satiety. *Behav Brain Res.* 2015;277:14-31. doi: 10.1016/j.bbr.2014.08.065.
- Flores RA, da Silva ES, Ribas AS, Tachetto APD, Zampieri TT, Donato J Jr, et al. Evaluation of food intake and Fos expression in serotonergic neurons of raphe nuclei after intracerebroventricular injection of adrenaline in free-feeding rats. *Brain Res.* 2018;1678:153-63. doi: 10.1016/j.brainres.2017.10.021.
- Anderberg RH, Richard JE, Eerola K, Lopez-Ferreras L, Banke E, Hansson C, et al. Glucagon-Like Peptide 1 and Its Analogs Act in the Dorsal Raphe and Modulate Central Serotonin to Reduce Appetite and Body Weight. *Diabetes.* 2017 Apr;66(4):1062-1073. doi: 10.2337/db16-0755.
- Versteeg RI, Koopman KE, Booij J, Ackermans MT, Umhedeo UA, Fliers E, et al. Serotonin Transporter Binding in the Diencephalon Is Reduced in Insulin-Resistant Obese Humans. *Neuroendocrinology.* 2017;105(2):141-9. doi: 10.1159/000450549.
- D'Agostino G, Lyons D, Cristiano C, Lettieri M, Olarte-Sanchez C, Burke LK, et al. Nucleus of the Solitary Tract Serotonin 5-HT2C Receptors Modulate Food Intake. *Cell Metab.* 2018;28(4):619-30.e5. doi: 10.1016/j.cmet.2018.07.017.
- Zhan C, Zhou J, Feng Q, Zhang JE, Lin S, Bao J, et al. Acute and long-term suppression of feeding behavior by POMC neurons in the brainstem and hypothalamus, respectively. *J Neurosci.* 2013;33(8):3624-32. doi: 10.1523/JNEUROSCI.2742-12.2013.
- Nonogaki K. Serotonin conflict in sleep-feeding. *Vitam Horm.* 2012;89:223-39. doi: 10.1016/B978-0-12-394623-2.00012-3.
- Hodge S, Bunting BP, Carr E, Strain JJ, Stewart-Knox BJ. Obesity, whole blood serotonin and sex differences in healthy volunteers. *Obes Facts.* 2012;5(3):399-407. doi: 10.1159/000339981.
- Tavares GA, Torres A, de Souza JA. Early Life Stress and the Onset of Obesity: Proof of MicroRNAs' Involvement Through Modulation of Serotonin and Dopamine Systems' Homeostasis. *Front Physiol.* 2020;11:925. doi: 10.3389/fphys.2020.00925.
- Kullmann S, Veit R, Crabtree DR. The effect of hunger state on hypothalamic functional connectivity in response to food cues. *Hum Brain Mapp [Internet].* 2022;44(2):418-28. Available from: doi: 10.1002/hbm.26059.
- Velichko VI. Complex staged system of supervision, prevention, differentiated therapy of children with excess body weight and obesity [thesis]. Odesa: Odesa. national honey. university; 2012. 353 p.
- Choi YH, Fujikawa T, Lee J, Reuter A, Kim KW. Revisiting the Ventral Medial Nucleus of the Hypothalamus: The Roles of SF-1 Neurons in Energy Homeostasis. *Front Neurosci.* 2013;7:71. doi: 10.3389/fnins.2013.00071.
- van der Valk ES, Savas M, van Rossum EFC. Stress and Obesity: Are There More Susceptible Individuals? *Curr Obes Rep.* 2018;7(2):193-203. Available from: doi: 10.1007/s13679-018-0306-y.
- Bassols J, Prats-Puig A, Vázquez-Ruiz M, García-González MM, Martínez-Pascual M, Avellí P, et al. Placental FTO expression relates to fetal growth. *Int J Obes (Lond).* 2010;34(9):1365-70. doi: 10.1038/ijo.2010.62.
- Obradovic M, Sudar-Milovanovic E, Soskic S, Essack M, Arya S, Stewart AJ, et al. Leptin and Obesity: Role and Clinical Implication. *Front Endocrinol (Lausanne).* 2021;12:585887. doi: 10.3389/fendo.2021.585887.
- Izquierdo AG, Crujeiras AB, Casanueva FF, Carreira MC. Leptin, Obesity, and Leptin Resistance: Where Are We 25 Years Later? *Nutrients.* 2019;11(11):2704. doi: 10.3390/nu11112704.
- Schepers J, Gebhardt C, Bracke A, Eifler I, von Bohlen Und Halbach O. Structural and functional consequences in the amygdala of leptin-deficient mice. *Cell Tissue Res.* 2020;382(2):421-6. Available from: doi: 10.1007/s00441-020-03266-x.
- Guo M, Lu Y, Garza JC, Li Y, Chua SC, Zhang W, Lu B, Lu XY. Forebrain glutamatergic neurons mediate leptin action on depression-like behaviors and synaptic depression. *Transl Psychiatry.* 2012;2(2):83. doi: 10.1038/tp.2012.9.
- Lee JS, Lee EY, Lee HS. Hypothalamic, feeding/arousal-related peptidergic projections to the paraventricular thalamic nucleus in the rat. *Brain Res.* 2015;1598:97-113. doi: 10.1016/j.brainres.2014.12.029.
- Yang LZ, Solivan-Rivera J, Corvera S. Adipocyte Heterogeneity Underlying Adipose Tissue Functions. *Endocrinol.* 2022;163(1):bqab138. doi: 10.1210/en-docr/bqab138.
- Castanon N, Luheshi G, Layé S. Role of neuroinflammation in the emotional and cognitive alterations displayed by animal models of obesity. *Front Neurosci.* 2015;9:229. doi: 10.3389/fnins.2015.00229.
- Farooqui AA, Farooqui T, Panza F, Frisardi V. Metabolic syndrome as a risk factor for neurological disorders. *Cell Mol Life Sci.* 2012;69(5):741-62. doi: 10.1007/s00018-011-0840-1.
- Nagayama D, Fujishiro K, Watanabe Y, Yamaguchi T, Suzuki K, Saiki A, et al. A Body Shape Index (ABSI) as a Variant of Conicity Index Not Affected by the Obesity Paradox: A Cross-Sectional Study Using Arterial Stiffness Parameter. *J Pers Med.* 2022;12(12):2014. doi: 10.3390/jpm12122014.
- Thomas EL, Frost G, Taylor-Robinson SD, Bell JD. Excess body fat in obese and normal-weight subjects. *Nutr Res Rev.* 2012;25(1):150-61. doi: 10.1017/S0954422412000054.
- Cooper CB, Neufeld EV, Dolezal BA, Martin JL. Sleep deprivation and obesity in adults: a brief narrative review. *BMJ Open Sport Exerc Med.* 2018;4(1):000392. doi: 10.1136/bmjsem-2018-000392.
- Pimenta FB, Bertrand E, Mograbi DC, Shinohara H, Landeira-Fernandez J. The relationship between obesity and quality of life in Brazilian adults. *Front Psychol.* 2015;6:966. doi: 10.3389/fpsyg.2015.00966.
- Mach F, Baigent C, Catapano AL, Koskinas KC, Casula M, Badimon L, et al. 2019 ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk. *Eur Heart J.* 2020;41(1):111-88. doi: 10.1093/eurheartj/ehz455.
- Voigt JP, Fink H. Serotonin controlling feeding and satiety. *Behav Brain Res.* 2015;277:14-31. doi: 10.1016/j.bbr.2014.08.065.
- Anderberg RH, Richard JE, Eerola K, Lopez-Ferreras L, Banke E, Hansson C, et al. Glucagon-Like Peptide 1 and Its Analogs Act in the Dorsal Raphe and Modulate Central Serotonin to Reduce Appetite and Body Weight. *Diabetes.* 2017;66(4):1062-73. doi: 10.2337/db16-0755.
- Versteeg RI, Koopman KE, Booij J, Ackermans MT, Umhedeo UA, Fliers E, et al. Serotonin Transporter Binding in the Diencephalon Is Reduced in Insulin-Resistant Obese Humans. *Neuroendocrinol.* 2017;105(2):141-9. doi: 10.1159/000450549.

Стаття надійшла до редакції 16.11.2022. – Дата першого рішення 24.11.2022. – Стаття подана до друку 21.12.2022