CHANGES IN CARBOHYDRATE METABOLISM AND ADIPOCYTOKINES UNDER THE INFLUENCE OF TREATMENT OF PATIENTS WITH ALCOHOLIC CIRRHOSIS OF THE LIVER IN COMBINATION WITH OBESITY USING ADAMETHIONINUM AND ARGinine GLUTAMATE

Introduction. Alcohol-related liver cirrhosis is one of the leading causes of hospitalization and disability in patients with gastrointestinal disease.

The aim of the study – to learn the effectiveness of treatment of patients with alcoholic liver cirrhosis (ALC) combined with obesity depending on the stage of decompensation using ademethionine and arginine glutamate.

Research Methods. 215 patients, diagnosed with ALC, took part in the study, including 66 women and 149 men. 109 people had ALC with obesity and 106 people had ALC without obesity. Patients were divided into subgroups depending on the stage of decompensation according to Child-Pugh. Depending on the treatment protocol (b protocol – basic therapy, h protocol – basic therapy in combination with ademethionine and arginine glutamate), all patients were divided into subgroups.

Results and Discussion. In this study, the values of carbohydrate metabolism, adiponectin and leptin were used to evaluate the effectiveness of a three-month treatment regimen with ademethionine and arginine glutamate in patients with ALC in combination with obesity. Obese patients with ALC have a more severe course of the disease, accompanied by more pronounced clinical manifestations, impaired carbohydrate metabolism and imbalance of leptin and adiponectin levels. The inclusion of ademethionine and arginine glutamate in the treatment regimen for 3 months allowed to improve the general condition of patients, their clinical and laboratory parameters and reduce the rate of disease progression, which is reflected in improved carbohydrate metabolism, leptin and adiponectin and reduced indicators of the severity scale according to Child-Pugh and the 3-month MELD mortality score.

Conclusions. Inclusion of ademethionine and arginine glutamate in the complex treatment of patients with ALC in combination with obesity helps to reduce insulin resistance, improve leptin and adiponectin. In patients with ALC in combination with obesity, the inclusion in the complex treatment of ademethionine and arginine glutamate helps to improve the course of the disease according to the indicators of the severity scale using the Child-Pugh and MELD scales.

KEY WORDS: alcoholic liver disease; liver cirrhosis; obesity; adipocytokines; carbohydrate metabolism.

INTRODUCTION. The steady increase in metabolic disorders among the world’s population, associated with the development of obesity and its consequences, attracts special attention of scientists. The problem affects about a quarter of the adult population due to certain changes in society, including a high prevalence of sedentary lifestyles, low physical activity, excessive consumption of high-calorie foods with insufficient energy expenditure and unhealthy food, which leads to unbalanced diet [1–3]. The prevalence of metabolic disorders among the population of wealthy countries in people with normal weight is also high. In addition, there is currently no clear nomenclature of liver diseases caused by metabolic factors, and, as a result, there is no defined algorithm for the management of such patients [4–6]. With regard to liver damage, an international group of experts proposed to clarify the definitions and criteria for assessing metabolic dysfunction associated with fatty liver disease [7–9]. As the pathogenetic mechanisms underlying the development of this condition are associated with systemic metabolic dysfunction, metabolic dysfunction, which is associated with fatty liver disease, is often perceived as an independent disease with inherent criteria that do not depend on the amount of alcohol consumed and therefore can be used for patients under any clinical conditions [10–12]. Liver cirrhosis (LC) on the background of metabolic dysfunction, associated with fatty liver disease, includes the following criteria of confirmation of
metabolic risk factors: waist circumference ≥102/88 cm in men and women of Caucasian race; blood pressure ≥130/85 mm Hg or specific drug treatment; plasma triglycerides ≥150 mg/dl (≥1.70 mmol/l) or specific drug treatment; plasma HDL cholesterol <40 mg/dl (<1.0 mmol/l) for men and <50 mg/dl (<1.3 mmol/l) for women or specific drug treatment; signs of prediabetes; evaluation of the homeostasis model for the evaluation of insulin resistance ≥2.5; high-sensitivity plasma C-reactive protein level >2 mg/l), documented data on steatosis by imaging or previous liver biopsy (if performed) taking into account the possible dual etiology of the disease – a combination of alcohol abuse [13–15].

Alcohol-related LC is one of the leading causes of hospitalization and disability in patients with gastrointestinal disease. The mortality rate among such people is 15–25 % [16]. With combined pathology, the prognosis for patients deteriorates. Therefore, we are looking for ways to prevent the progression, complications of LC and improve the quality of life of such patients [17–19].

The aim of the study was to learn the effectiveness of treatment of patients with alcoholic liver cirrhosis (ALC) combined with obesity depending on the stage of decompensation using ademethionine and arginine glutamate.

RESEARCH METHODS. 215 patients, diagnosed with alcoholic liver cirrhosis (ALC), took part in the study, including 66 women and 149 men aged (48.1±9.7) years and a median disease duration (5.8±2.6) years. 109 people had ALC with obesity (group I) and 106 people had ALC without obesity (group II). Patients were divided into subgroups depending on the stage of decompensation according to Child-Pugh: class A – group IA (n=40), class B – group IB (n=39), class C – group IC (n=30) and IIA (n=39), IIB (n=36), IIC (n=31) groups, respectively; and also depending on the treatment protocol all patients were divided into subgroups (b protocol – basic therapy, h protocol – basic therapy in combination with intravenous administration of ademethionine and arginine glutamate): patients receiving basic therapy were included in IAb (n=19), IBb (n=20), ICb (n=15), IIAb (n=22), IIb (n=18), IICb (n=16) groups; patients who additionally received ademethionine and arginine glutamate were included in IAh (n=21), IBh (n=19), ICh (n=15) in IIAh (n=17), IIb (n=18), IICh (n=15) groups.

Groups I and IIaH, in addition to the basic treatment, received intravenously 500 mg of ademethionine per day during two weeks, followed by oral administration of 500 mg of ademethionine and 1500 mg of arginine glutamate per day for 12 weeks.

Groups IBh and IIbH, in addition to the basic treatment, received intravenously 1000 mg of ademethionine per day for two weeks, followed by oral administration of 1000 mg of ademethionine and 3000 mg of arginine glutamate for 12 weeks.

Groups ICh and IICh, in addition to their basic treatment, received intravenously 1000 mg of ademethionine per day for two weeks, followed by oral administration of 1500 mg of ademethionine and 4500 mg of arginine glutamate per day for 12 weeks.

Diagnosis was verified using clinical and laboratory-instrumental methods in accordance with the order of the Ministry of Health of Ukraine No. 826 dated November 6, 2014, adapted clinical guidelines “Non-Alcoholic Fatty Liver Disease”, 2014, adapted clinical guidelines “Alcoholic Liver Disease”, 2014, adapted clinical guidelines “ Liver Cirrhosis, 2017 (State Expert Centre of the Ministry of Health of Ukraine, Ukrainian Gastroenterology Association, Kyiv), recommendations of the European Association for the Study of Liver, Diabetes and Obesity (EASL-EASD-EASO, 2016).

Exclusion criteria were liver cirrhosis of the viral, toxic and autoimmune genesis, metabolic diseases of the liver, oncological diseases, and the lack of individual consent of the patient to conduct the study. All patients were matched according to age and sex. The research was carried out in accordance with the ethical principles of conducting scientific research and principles of the Helsinki Declaration.

The carbohydrate metabolism was evaluated for immunoreactive insulin (IRI) indicators, glycosylated haemoglobin (HbA1c), HOMA-IR indexes (calculated using the formula HOMA-IR = (glycemia in the fasted state, mmol/l* insulin in the fasted state (mcU/ml))/22.5) and QUICKI (www.ndapp.co/insulin-sensitivity-quicki-calculator-324). The level of leptin and adiponectin was determined by immunoassay using Human Leptin ELISA (Biovendor, Czech Republic) and Human Adiponectin ELISA kit (Biovendor, Czech Republic) respectively. The severity of the LC was assessed using the Child-Pugh score and the MELD score [20]. The control group consisted of 20 healthy individuals, who were age and gender matched. Assessment of patients was performed before and after 3 months from the beginning of treatment.

Statistical processing of the obtained results was carried out using the software package Statistica v. 12.0 (StatSoft, USA, trial) and Microsoft Excel. The average values are presented in the form (M±m), where “M” is the average value of the
was a significant increase in IRI in patients of group I consequences.

An inflammatory response, which ultimately leads to the blood flow, more severe systemic inflammation in patients with a combination of ALD and obesity accompanied by significant systemic changes in necrotic process and fibrosis in the liver and due to a more pronounced increase in inflammatory-

A correlation between leptin, adiponectin levels, carbohydrate metabolism and leptin levels in patients of group I were as follows: for adiponectin – 0.74, -0.62 and -0.64 for A, B and C classes, respectively; for IRI – 0.69, -0.53 and -0.49 for A, B and C classes, respectively; for HOMA-IR – -0.54, -0.41 and -0.33 for A, B and C classes, respectively; for QUICKI – 0.63, 0.69 and 0.74 for A, B and C classes, respectively; for HbAlc – -0.35, -0.33 and -0.31 for A, B and C classes, respectively. Correlations between adiponectin levels and carbohydrate metabolism in patients of group I were for IRI – 0.45, 0.38 and 0.31 for A, B and C classes, respectively; for HOMA-IR – 0.36, 0.31 and 0.27 for A, B and C classes, respectively; for QUICKI – -0.42, -0.38 and -0.37 for A, B and C classes, respectively; for HbAlc – -0.31, 0.27 and 0.25 for A, B and C classes, respectively. The relationship between adiponectin levels, disease severity and the MELD score was more pronounced in patients of group I.

Acute correlation between leptin, adiponectin levels, Child-Pugh and MELD scores showed a stronger relationship in patients of group I with increased decompensation. The correlation between leptin level, Child-Pugh and MELD scores was as follows: for Child-Pugh – r=0.72, r=0.58, r=0.44, and for MELD – r=0.66, r=0.61, r=0.68 for classes A, B, C, respectively. The relationship between the level of adiponectin and the severity of the disease and the MELD score was for the Child-Pugh index – r=0.69, r=0.49, r=0.67, and for the MELD score – r=0.73, r=0.52, r=0.34 for classes A, B, C, respectively.

Three months after the prescribed course of treatment, clinical and laboratory manifestations improved in most patients receiving the h protocol, whereas in patients with the b protocol, deterioration was observed, especially at the stages of subcompensation and decompensation. In patients re-
Table 1 – Dynamics of adipocytokines levels, carbohydrate metabolism, severity and prognostic MELD score in patients with alcoholic liver cirrhosis with stage A by Child-Pugh, depending on the combination with obesity, М±m

| Values                  | Control, n=20 | ALC with obesity | ALC with obesity |
|-------------------------|---------------|------------------|------------------|
|                         |               | Before treatment | After 3 month    |
|                         |               | treatment        | treatment        |
| Adiponectin, μg/ml      | 8.46±0.11     | 2.86±0.15*       | 2.67±0.18**      | 2.79±0.16** | 5.58±0.14* |
| Leptin, ng/ml           | 7.92±0.28     | 21.72±0.58*      | 22.52±0.66**     | 21.43±0.62* | 12.37±0.52* |
| IRI, mcod/l             | 5.89±0.32     | 23.5±1.46*       | 25.81±1.22**     | 24.65±1.34* | 6.36±0.38  |
| HOMA-IR                 | 1.05±0.06     | 5.39±0.15*       | 5.57±0.11**      | 5.43±0.12** | 1.11±0.09  |
| QUICKI                  | 0.68±0.004    | 0.37±0.007*      | 0.36±0.005**     | 0.372±0.006* | 0.664±0.006 |
| HbA1c, %                | 4.63±0.18     | 6.33±0.14*       | 6.69±0.16**      | 6.35±0.12** | 4.71±0.15  |
| Child-Pugh score        | -             | 5.76±0.11*       | 5.94±0.12**      | 5.81±0.09*  | 5.32±0.11  |
| MELD score              | -             | 13.64±0.92*      | 14.79±0.95**     | 13.47±0.84* | 8.17±0.75  |

Continuation of Table 1

| Values                  | Control, n=20 | ALC | ALC |
|-------------------------|---------------|-----|-----|
|                         |               | IIAb, n=22 | IIAb, n=22 |
|                         |               | Before treatment | After 3 month treatment |
| Adiponectin, μg/ml      | 8.46±0.11     | 3.29±0.06**   | 3.04±0.08**       | 3.24±0.09** | 5.34±0.06* |
| Leptin, ng/ml           | 7.92±0.28     | 15.47±0.82**  | 18.67±0.94**      | 15.52±0.76* | 10.21±0.88* |
| IRI, mcod/l             | 5.89±0.32     | 31.37±1.25**  | 34.11±1.18**      | 31.43±1.22* | 17.32±1.16* |
| HOMA-IR                 | 1.05±0.06     | 7.12±0.21**   | 7.54±0.17**       | 7.15±0.19** | 2.97±0.15*  |
| QUICKI                  | 0.68±0.004    | 0.348±0.004** | 0.339±0.003**     | 0.349±0.005** | 0.652±0.005* |
| HbA1c, %                | 4.63±0.18     | 6.38±0.14**   | 6.71±0.11**       | 6.42±0.17** | 4.97±0.15  |
| Child-Pugh score        | -             | 8.73±0.19**   | 9.17±0.15**       | 8.82±0.12** | 5.47±0.14  |
| MELD score              | -             | 19.74±0.72**  | 21.86±1.15**      | 19.95±1.12* | 8.65±0.43  |

Notes:
1. * – probability of difference of values between groups I and II (p<0.05).
2. ● – probability of differences of values before and after treatment (p<0.05).
3. # – probability of differences of values between groups A and Ah with treatment protocols (p<0.05).

Table 2 – Dynamics of adipocytokines levels, carbohydrate metabolism, severity and prognostic MELD score in patients with alcoholic liver cirrhosis with stage B by Child-Pugh, depending on the combination with obesity, М±m

| Values                  | Control, n=20 | ALC with obesity | ALC with obesity |
|-------------------------|---------------|------------------|------------------|
|                         |               | Before treatment | After 3 month    |
|                         |               | treatment        | treatment        |
| Adiponectin, μg/ml      | 8.46±0.11     | 2.86±0.15*       | 2.67±0.18**      | 2.79±0.16** | 5.58±0.14* |
| Leptin, ng/ml           | 7.92±0.28     | 21.72±0.58*      | 22.52±0.66**     | 21.43±0.62* | 12.37±0.52* |
| IRI, mcod/l             | 5.89±0.32     | 23.5±1.46*       | 25.81±1.22**     | 24.65±1.34* | 6.36±0.38  |
| HOMA-IR                 | 1.05±0.06     | 5.39±0.15*       | 5.57±0.11**      | 5.43±0.12** | 1.11±0.09  |
| QUICKI                  | 0.68±0.004    | 0.37±0.007*      | 0.36±0.005**     | 0.372±0.006* | 0.664±0.006 |
| HbA1c, %                | 4.63±0.18     | 6.33±0.14*       | 6.69±0.16**      | 6.35±0.12** | 4.71±0.15  |
| Child-Pugh score        | -             | 5.76±0.11*       | 5.94±0.12**      | 5.81±0.09*  | 5.32±0.11  |
| MELD score              | -             | 13.64±0.92*      | 14.79±0.95**     | 13.47±0.84* | 8.17±0.75  |

Continuation of Table 2

| Values                  | Control, n=20 | ALC | ALC |
|-------------------------|---------------|-----|-----|
|                         |               | IIAb, n=22 | IIAb, n=22 |
|                         |               | Before treatment | After 3 month treatment |
| Adiponectin, μg/ml      | 8.46±0.11     | 3.29±0.06**   | 3.04±0.08**       | 3.24±0.09** | 5.34±0.06* |
| Leptin, ng/ml           | 7.92±0.28     | 15.47±0.82**  | 18.67±0.94**      | 15.52±0.76* | 10.21±0.88* |
| IRI, mcod/l             | 5.89±0.32     | 31.37±1.25**  | 34.11±1.18**      | 31.43±1.22* | 17.32±1.16* |
| HOMA-IR                 | 1.05±0.06     | 7.12±0.21**   | 7.54±0.17**       | 7.15±0.19** | 2.97±0.15*  |
| QUICKI                  | 0.68±0.004    | 0.348±0.004** | 0.339±0.003**     | 0.349±0.005** | 0.652±0.005* |
| HbA1c, %                | 4.63±0.18     | 6.38±0.14**   | 6.71±0.11**       | 6.42±0.17** | 4.97±0.15  |
| Child-Pugh score        | -             | 8.73±0.19**   | 9.17±0.15**       | 8.82±0.12** | 5.47±0.14  |
| MELD score              | -             | 19.74±0.72**  | 21.86±1.15**      | 19.95±1.12* | 8.65±0.43  |

Receiving basic treatment, the Child-Pugh and MELD scores deteriorated, indicating further disease progression and, consequently, a worsening of the mortality prognosis. Within 3 months from the beginning of treatment, 3 people died in group ICB and 2 people – in group IICB due to deterioration of patients’ condition and the development of complications (in 2 patients of group IICB and 1 patient
### Table 3 – Dynamics of adipocytokines levels, carbohydrate metabolism, severity and prognostic Meld score in patients with alcoholic liver cirrhosis with stage C by Child-Pugh, depending on the combination with obesity, М±m

| Values                  | Control, n=20 | ALC with obesity | ALC with obesity |
|-------------------------|---------------|------------------|------------------|
|                         |               | IICb, n=16       | ICh, n=15        |
|                         | Before treatment, n=16 | After 3 month treatment, n=14 | Before treatment, n=15 | After 3 month treatment, n=15 |
| Adiponectin, μg/ml      | 8.46±0.11     | 15.78±0.98*     | 17.91±0.75*     | 16.12±0.79*     | 12.82±0.84*     |
| Leptin, ng/ml           | 7.92±0.28     | 6.73±0.37*      | 5.21±0.11*      | 6.81±0.13*      | 7.15±0.16       |
| IRI, mcod/l             | 5.89±0.32     | 26.53±1.87*     | 28.32±1.54**    | 26.89±1.75**    | 19.61±1.49*     |
| HOMA-IR                 | 1.05±0.06     | 7.08±0.16**     | 7.42±0.13**     | 7.11±0.17**     | 4.26±0.11*      |
| QUICKI                  | 0.681±0.004   | 0.348±0.003*    | 0.345±0.004**   | 0.349±0.003**   | 0.526±0.005*    |
| HbA1c, %                | 4.63±0.18     | 5.92±0.24       | 6.12±0.14*      | 5.89±0.26*      | 5.32±0.14       |
| Child-Pugh score        | -             | 13.98±0.61**    | 15.38±0.52**    | 14.21±0.64**    | 7.84±0.41       |
| MELD score              | -             | 27.43±1.19**    | 30.13±1.21**    | 28.13±1.23**    | 17.52±1.15      |

### Notes.
1. * – probability of difference of values between groups I and II (p<0.05).
2. ** – probability of differences of values before and after treatment (p<0.05).
3. *** – probability of differences of values between groups A and Ah with treatment protocols (p<0.05).
of group ICb liver failure was developed, 1 patient of group ICb group had mesenteric thrombosis, 1 patient of group ICb had bleeding from varicose veins).

In patients of groups I and II who received the h protocol, at the stage of compensation, sub-compensation and decompensation, the indicators of carbohydrate metabolism, adiponectin and leptin significantly improved (p<0.05). In patients of groups I and II who received basic treatment, at the stage of compensation such indicators worsened, but no significant difference was observed before and after the treatment (p>0.05). In patients of group I who received basic treatment, significantly worsened carbohydrate metabolism, adiponectin and leptin at the stage of subcompensation (p<0.05), and in patients of group II no significant difference before and after treatment was observed (p>0.05). In patients of group I receiving basic treatment, at the stage of decomposition the indicators of HOMA-IR, adiponectin and leptin significantly deteriorated (p<0.05), other indicators of carbohydrate metabolism deteriorated, but there was no significant difference before and after treatment (p>0.05). In patients of group II who received basic treatment, at the stage of decomposition carbohydrate metabolism, adiponectin and leptin values deteriorated, but no significant difference was observed before and after treatment (p>0.05).

Significant deterioration in indicators of carbohydrate metabolism, adiponectin and leptin in patients receiving basic treatment was accompanied by deterioration of their condition and increased the risk of 3-month mortality.

In this study, the values of carbohydrate metabolism, adiponectin and leptin were used to evaluate the effectiveness of a three-month treatment regimen with ademethionine and arginine glutamate in patients with ALC in combination with obesity. Obese patients with ALC have a more severe course of the disease, accompanied by more pronounced clinical manifestations, impaired carbohydrate metabolism and imbalance of leptin and adiponectin levels.

Changes in carbohydrate metabolism were characterized by a significant increase in IRI, HOMA-IR index and decrease in QUICKI index in patients of group I compared with patients of group II. The HbA1c index was significantly increased at the stage of compensation and subcompensation in patients of group I compared with patients of group II (p<0.05), but at the stage of decompensation no significant difference was observed (p>0.05). The detected changes in carbohydrate metabolism indicate that patients in group I have insulin resistance. Elevated levels of HbA1c and IRI were found in group II patients at the decompensation stage, which is probably due to the direct involvement of the liver in carbohydrate metabolism.

The peculiarity of adipocytokine parameters was that with the progression of LC the level of leptin decreased and the level of adiponectin increased. Resistance to leptin is associated with adipose tissue as an endocrine organ and is characteristic of overweight people, which is confirmed by higher levels of leptin in patients of group I. Higher leptin content in class A and B patients is accompanied not only by impaired liver function, but also increased its release from adipose tissue. In class C patients, fat depots are depleted, that's why leptin levels are reduced. Moreover, this decrease correlates with the severity of the disease and the prognostic MELD score. Adiponectin levels were decreased in class A patients and increased in patients with more severe course of the disease and correlated with disease severity and the MELD score. Due to the hepatoprotective effect of adiponectin, some scientists believe that its increased level reflects the anti-inflammatory response to liver damage, which depends on the severity of the disease.

An analysis of the relationship between leptin and adiponectin levels and carbohydrate metabolism showed that a stronger correlation was observed in patients with ALC combined with obesity. The identified correlations between leptin and adiponectin levels with the severity of LC and the MELD score allow us to consider their changes for assessment of the severity of LC and prediction of the course of the disease.

The inclusion of ademethionine and arginine glutamate in the treatment regimen for 3 months allowed to improve the general condition of patients, their clinical and laboratory parameters and reduce the rate of disease progression, which is reflected in improved carbohydrate metabolism, leptin and adiponectin and reduced indicators of the severity scale according to Child-Pugh and the 3-month MELD mortality score.

CONCLUSIONS. Progression of liver cirrhosis in patients with ALC in combination with obesity is accompanied by more severe clinical and laboratory manifestations. Changes in carbohydrate metabolism (IRI, HOMA-IR index, QUICKI index and HbAlc) in patients with ALC combined with obesity indicate the presence of insulin resistance. Leptin and adiponectin levels in patients with ALC in combination with obesity correlate with changes in carbohydrate metabolism, the severity of LC and the prognostic MELD score, which allows them to be used to assess the severity and prediction of ALD in combination with obesity. Inclusion of ademethionine and arginine glutamate in the complex
treatment of patients with ALC in combination with obesity helps to reduce insulin resistance, improve leptin and adiponectin. In patients with ALC in combination with obesity, the inclusion in the complex treatment of ademethionine and arginine glutamate helps to improve the course of the disease according to the indicators of the severity scale using the Child-Pugh and MELD scales.

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Н. Р. Матковська

**ІВАНО-ФРАНКІВСЬКІЙ НАЦІОНАЛЬНИЙ МЕДИЧНИЙ УНІВЕРСИТЕТ МОЗ УКРАЇНИ**

**ЗМИНИ ПОКАЗНИКІВ ВУГЛЕВОДНОГО ОБМІНУ ТА АДІПОЦИТОКІНІВ ПІД ВПЛИВОМ ЛІКУВАННЯ ХВОРИХ НА АЛКОГОЛІЙНИЙ ЦИРОЗ ПЕЧІНКИ В ІЗОГІРНЯННІ З ВИКОРИСТАННЯМ АДЕМЕТИОНІЮ І АРГІНІНУ Г ЛУТАМАТУ**

**Резюме**

**Вступ.** Цироз печінки, що зумовлений зволоженням алкоголю, є однією з провідних причин госпіталізації та амбулаторного обслуговування хворих на цироз печінки, особливо в молодих і середніх відділеннях лікарень та поликлиник. Він вчинені важливе значення для економіки України, оскільки він та його інсфекції є головною причиною смерті хворих з діагностічними установами системи охорони здоров'я. Цироз печінки, як звісно, є одним з лідируючих факторів ризику підтримки хворих і згідно з даними відомих авторів, включає до 1-ї-3-ї стадії. Мета становить вивчення ефективності лікування хворих на алкогольний цироз печінки (АЦП) у поєднанні з ожирінням (За іншими або москівським) та АЦП без ожиріння. Як необхідно, дана мета вивчена в хворих з АЦП без ожиріння.

**Мета дослідження** — вивчити ефективність лікування хворих на алкогольний цироз печінки (АЦП) у поєднанні з ожирінням без стадії декомпенсації з використанням ацетилметіоніну (AM) в ампули у комбінації з аргініново-лутаматовим протоколом. Даний варіант використовує спектральний аналіз та дослідження в рамках проведення терапії в поєднанні з ацетилметіоніном і аргініну глутаматом.

**Методи дослідження.** У дослідженні взяли участь 215 хворих з діагностічним алкогольним цирозом печінки, серед яких було 66 жінок та 149 чоловіків. У 109 осіб діагностовано АЦП з ожирінням, у 106 осіб — АЦП без ожиріння. Хворі підійшли на підготовлені в зв'язку з використанням протоколу лікування (в протокол — базова терапія, h протокол — базова терапія в поєднанні з ацетилметіоніном і аргініну глутаматом).
Результати й обговорення. У цій роботі для оцінки ефективності тримісячної схеми лікування з включенням адеметіоніну й аргініну глутамату у хворих на АЦП у поєднанні з ожирінням використано показники углеводного обміну, адипонектина та лептину. В пацієнтів з АЦП у поєднанні з ожирінням спостерігали тяжкій перебіг захворювання, що супроводжувалося більш вираженими клінічними проявами, порушенням углеводного обміну і дисбалансом інших показників. Включення в схему лікування адеметіоніну й аргініну глутамату дозволило покращити загальній стан пацієнтів, що відбувалося за рахунок зниження показників углеводного обміну і адипонектина, а також зменшення тяжкості захворювання за показниками шкали Чайльда – Пью та індексу MELD.

Висновки. Включення в комплексне лікування хворих на АЦП у поєднанні з ожирінням адеметіоніну й аргініну глутамату сприяло зменшенню інсулінорезистентності, покращенню показників углеводного обміну та адипонектина. Використання в комплексному лікуванні пацієнтів з АЦП в поєднанні з ожирінням адеметіоніну й аргініну глутамату сприяло зменшенню тяжкості захворювання за показниками шкали Чайльда – Пью та індексу MELD.

КЛЮЧОВІ СЛОВА: алкогольна хвороба печінки; цироз печінки; ожиріння; адипоцитокіни; углеводний обмін.

Н. Р. Матковская

ИВАНО-ФРАНКОВСКИй НАЦИОНАЛЬНЫй МЕДИЦИНСКИй УНИВЕРСИТЕТ МОЗ УКРАИНy

ИЗМЕНЕНИЯ ПОКАЗАТЕЛЕЙ УГЛЕВОДНОГО ОБМЕНА И АДИПОЦИТОКИНОВ ПОД ВЛИЯНИЕМ ЛЕЧЕНИЯ БОЛЬНЫХ АЛКОГОЛЬНЫМ ЦИРРОЗОМ ПЕЧЕНИ В СОЧЕТАНИИ С ОЖИРЕНИЕМ С ИСПОЛЬЗОВАНИЕМ АДЕМЕТИОНИНА И АРГИНИНА ГЛУТАМАТА

Резюме

Вступление. Цирроз печени, обусловленный злоупотреблением алкоголя, является одной из ведущих причин госпитализации и потери трудоспособности пациентами с заболеваниями органов желудочно-кишечного тракта.

Цель исследования – изучить эффективность лечения больных алкогольным циррозом печени (АЦП) в сочетании с ожирением в зависимости от стадии декомпенсации с использованием адеметионина и аргинина глутамата.

Методы исследования. В исследовании приняли участие 215 больных с диагностированым алкогольным циррозом печени, среди которых было 66 женщин и 149 мужчин. В 109 лиц диагностирован АЦП с ожирением, в 106 лиц – АЦП без ожирения. Пациентов разделили на подгруппы в зависимости от стадии декомпенсации по Чайльду – Пью, а также в зависимости от примененного протокола лечения (b протокол – базовая терапия, h протокол – базовая терапия в сочетании с адеметионином и аргинина глутаматом).

Результаты и обсуждение. В этой работе для оценки эффективности трехмесячной схемы лечения с включением адеметионина и аргинина глутамата у больных АЦП в сочетании с ожирением использовано показатели углеводного обмена, адипонектина и лептину. У пациентов с АЦП в сочетании с ожирением наблюдалось более тяжёлое течение заболевания, что сопровождалось более выраженным клиническими проявлениями, нарушением углеводного обмена и дисбалансом уровней лептину и адипонектина. Включение в схему лечения адеметионина и аргинина глутамата позволяло улучшить общее состояние пациентов, клинико-лабораторные показатели и уменьшить темпы прогрессирования заболевания, о чем свидетельствовали улучшение показателей углеводного обмена, лептину и адипонектина и снижение показателей шкалы тяжести Чайльда – Пью и индекса трехмесячной летальности MELD.

Выводы. Включение в комплексное лечение больных АЦП в сочетании с ожирением адеметионина и аргинина глутамата способствует уменьшению инсулинорезистентности, улучшению показателей углеводного обмена, лептину и адипонектина. Использование в комплексном лечении пациентов с АЦП в сочетании с ожирением адеметионина и аргинина глутамата способствует улучшению течения заболевания по показателям шкалы тяжести Чайльда – Пью и индекса MELD.

КЛЮЧЕВЫЕ СЛОВА: алкогольная болезнь печени; цироз печени; ожирение; адипоцитокины; углеводный обмен.

Address for correspondence: N. R. Matkovska, Ivanо-Frankivsk National Medical University, Ministry of Health of Ukraine, Kropyvnytskyi 16 St., Ivanо-Frankivsk, 76018, Ukraine, e-mail: nmail4you@gmail.com.

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