3. Shabanov P.D., Lebedev A.A., Morozov V.I. Rol grelina v kontrole emocionalnogo, issledovatelskogo i dvigatelnogo povedeniya pri eksperimentalnom posttravmaticheskom stressovom rasstroystve. Mediko-biologicheskie i socialno-psikhologicheskie problemy bezopasnosti v chrezvychajnykh situatsiyakh. 2018; 1: 65–74 [in Russian].
4. Bachmann C., Trenkwalder C. Body weight in patients with Parkinson's disease. Mov. Disord. 2006; 21:1824-1830.
5. Cabral J, Kringelbach ML, Deco G. Functional connectivity dynamically evolves on multiple time-scales over a static structural connectome: Models and mechanisms. Neuroimage. 2017; 160: 84-96.
6. Gong B, Jiao L, Du X, Li Y, Bi M, Jiao Q, Jiang H. Ghrelin promotes midbrain neural stem cells differentiation to dopaminergic neurons through Wnt/β-catenin pathway. J Cell Physiol. 2020; 235(11): 8558-8570.
7. Mao Y, Tokudome T, Kishimoto I. Ghrelin and blood pressure regulation. Curr Hypertens Rep. 2016; 18:15.
8. Mosińska P., Zatorski H., Storr M., Fichna J. Future treatment of constipation-associated disorders: role of relamorelin and other ghrelin receptor agonists. J. Neurogastroenterol. Motil. 2017; 23: 171–179.
9. Palacios N, Gao X, McCullough M, et al. Obesity, diabetes, and risk of Parkinson's disease. Mov Disord. 2011; 26(12): 2253-2259.
10. Scheer F, Morris C, Shea S. The internal circadian clock increases hunger and appetite in the evening independent of food intake and other behaviors. Obesity. 2013; 21: 421–423.
11. Song N, Wang W, Jia F, et al. Assessments of plasma ghrelin levels in the early stages of parkinson's disease. Mov. Disord. 2017; 32(10): 1487-1491.
12. Taheri S, Lin L, Austin D, Young T, Mignot E. Short sleep duration is associated with reduced leptin, elevated ghrelin, and increased body mass index. PLoS Med. 2004; 1(3):62.
13. Van der Mack MA, Dicke HC, Uc EY, et al. Body mass index in Parkinson's disease: a meta-analysis. Parkinsonism Relat Disord. 2012; 18: 263-267.
14. Wang L, Murphy NP, Stengel A, et al. Ghrelin prevents levodopa-induced inhibition of gastric emptying and increases circulating levodopa in fasted rats. Neurogastroenterol Motil. 2012; 24(5): 235-245.
15. Zhang P, Tian B. Metabolic Syndrome: An Important Risk Factor for Parkinson's Disease. Oxidative Medicine and Cellular Longevity. 2014; 1-7.

Стаття надійшла 29.11.2019 р.

DOI 10.26724/2079-8334-2020-4-74-149-153
UDC 616.89-008.46/.47-057.36-056.83-039.51:616.89-008.19]-07

O.S. Fitkalo, O.L. Lyzak, A.B. Neurova[1]
Danylo Halytsky National Medical University, Lviv
[1]Hetman Petro Sahaidachnyi National Army Academy, Lviv

ASSESSMENT OF COGNITIVE DYSFUNCTION IN THE MILITARY WITH ALCOHOL-INDUCED MENTAL AND BEHAVIORAL DISORDERS WITH DEPRESSION COMORBIDITY

e-mail: fitkalo@gmail.com

The article presents the results of a psychodiagnostic study aimed to identify cognitive impairment and comorbid depressive states in the military with alcohol-induced mental and behavioral disorders. Up to 50 % of borderline cognitive disorders were found in patients of both groups (N = 85). Depressive symptoms were found in 100 % of p□t□□ts □ the ex□er□me□t□l grou□; 95.21 % demonstrated anxiety symptoms. Depression has been shown to correlate with cognitive impairment in patients with alcohol-induced mental and behavioral disorders. To reduce cognitive impairment, the patients of both groups were administered Cytoflavin metabolic drug in combination with traditional treatment. Following the course of treatment with addition of Cytoflavin, in 68 (80 %) patients of both groups there was a decrease in the cognitive dysfunction symptoms, normalization of sleep, and improvement of mood, which affected the quality of life in patients of both groups.

Key words: cognitive impairment, alcoholism, depression, Cytoflavin.

О.С. Фітькало, О.Л. Лизак, А.Б. Неурова

ОЦІНКА КОГНІТИВНОЇ ДИСФУНКЦІЇ У ВІЙСЬКОВИХ З РОЗЛАДАМИ ПСИХІКИ ТА ПОВЕДІНКИ ВНАСЛІДОК ВЖИВАННЯ АЛКОГОЛЮ КОМОРБІДНОГО З ДЕПРЕСІЄЮ

У статті викладено результати психодіагностичного дослідження з метою виявлення когнітивних порушень та коморбідних депресивних станів у військових з розладами психіки та поведінки внаслідок вживання алкоголю. У пацієнтів обох груп (N=85) виявлено до 50% пограничних когнітивних відхилень. У 100% пацієнтів дослідної групи виявлено депресивні симптоми; у 95,21% - симптоми тривоги. Доведено, що депресія корелює з проявами когнітивних порушень у пацієнтів з розладами психіки та поведінки внаслідок вживання алкоголю. Для усунення когнітивних порушень пацієнтам обох груп було призначено в комплексі до традиційного лікування метаболічний препарат цитофлавін. У 68 (80%) пацієнтів обох груп після курсу лікування з додатковим призначенням цитофлавіну відзначалися зменшення проявів когнітивної дисфункції, нормалізація сну, покращення настрою, що впливало на якість життя пацієнтів обох груп.

Ключові слова: когнітивні порушення, алкоголізм, депресія, цитофлавін.

The work is a fragment of the research project "Features of comorbid states' clinical polymorphism in psychiatry and narcology", state registration No. 0119U100172.

Alcohol abuse and alcohol-induced disorders remain a pressing problem of modern healthcare and of society as a whole. As indicated by recent literature data, the growth of alcohol consumption has led to a

substantial increase in the incidence of alcohol-related dementia and other neurocognitive disorders [14]. Cognitive impairment (CI) in alcohol abusers is manifested in 50–70 % of cases; in 80 % of patients depression symptoms are revealed; alcohol-induced dementia constitutes 5 to 10 % of dementia incidence [1, 2, 4, 6, 10-15]. Due to the significant impact of mild cognitive impairment on health (varying in duration from several months to several decades and not yet reaching the level of dementia, but going beyond the age range), it was included in ICD–10 as an independent diagnostic unit – "moderate cognitive impairment".

Diagnosing alcohol-related neurocognitive impairment can be complicated due to varied types of manifestation and no specific brain pathology evident in other disorders with similar symptoms [14]. The patients' history frequently provides no explicit information on excessive alcohol consumption due to the lack of the patient's complaints or to cessation of one's drinking habits. However, detecting the correlation of neurocognitive impairment and alcohol abuse is essential, since the improvement of the patients' condition and reversing the harmful effects of alcohol intoxication is gradual and can only occur during prolonged abstinence [14]. Identifying mild and moderate cognitive impairment during a standard psychiatric assessment may be difficult as well, particularly with depression comorbidity. Due to their simplicity, non–invasiveness and cost–efficiency, neuropsychological tests remain an important tool to assess basic cognitive functions, leading to a more accurate diagnosis, and consequently to more adequate treatment strategies.

The purpose of the work was to study dynamics, validity and efficacy of the methods for assessing the cognitive status in patients with alcohol-induced mental and behavioral disorders associated with depression, and to correct cognitive dysfunction by additionally prescribing Cytoflavin.

Materials and methods. Eighty-five patients, aged from 21 to 59, with alcohol-induced mental and behavioral disorders, who were inpatients at Lviv Military Medical Center, Branch 16, were included in the study upon their informed consent.

Short-term and long-term memory, time and space orientation were assessed using the Mini Mental State Examination (MMSE) scale [11]. The scale was used both as a screening technique for assessing the degree of cognitive impairment and for monitoring the dynamics of improvement or stabilization due to treatment. The scale consists of 9 entries with a maximum total score of 30 points. A lower total score indicated more severe cognitive impairment. The time of completion is conveniently short, usually taking only 10 minutes. The methodology is standardized, easy to apply and to process the results; characterized by good predictive reliability; and informative in the assessment of intellectual defect, as it includes all major cognitive components. The next method applied was the clock–drawing test. Drawing a picture of a clock and placing the arrows correctly on the dial, serves to determine the patient's spatial, visual and, regulatory cognitive impairment. In the study of auditory and language functions we used the 'learning of 10 words' technique proposed in 1962 by A.R. Luria, and based on the hypothesis of "predominant damage to the anterior brain" (signs of damage to the anterior brain that are dominant in alcoholism), which explains the neuropsychological defect in the disease [2, 5]. The patient was asked to memorize 10 simple different words without any meaningful relationship between them. The number of words correctly reproduced and the number of repetitions were then calculated as points. Four to five words during the first time of reproduction was considered normal, as well as all 10 words after 3 to 5 repetitions. Assessment of depression level was performed using the Zung scale for pre-hospital diagnosis of depression, which included 20 variants of responses for self-assessment by the patients. Each question had four answers: 'never', 'some of the time', 'good part of the time', 'most of the time' to assess the patients' level of depression and to determine the degree of depressive disorder. The scale is characterized by high sensitivity and specificity, and permits avoiding additional economic and time expenses. For determining reactive and personality anxiety, the Spielberger-Khanin questionnaire consisting of two scales was used. Each of the questionnaire parts included 20 statements. A unique feature of this questionnaire is simultaneous identification of anxiety as a persisting individual characteristic, as well as a state describing subjectively experienced emotions. It is worth noting that in our study low anxiety levels on the Spielberger-Khanin scale correlated with depression symptoms. Low levels of anxiety were indicative of depressive state with low level of motivation [7]. The patients' condition was assessed twice (before and after alleviation of the symptoms), which permitted increasing the methods' sensitivity in the presented studies. The obtained results were processed using the statistics software Microsoft Excel and Statistica 6.0, together with Student's t–criterion calculations.

Results of the study and their discussion. In compliance with the biomedical ethics principles, 43 patients aged 22 to 59, with the mean age of 38.79±5.2 in the main group were examined. The control group consisted of 42 patients aged 21 to 59 (mean age 40.92±7.68) who showed no signs of depression.

Clinical and psychopathological presentation of adjustment disturbances at the beginning of treatment was characterized by depressed mood in 100 % of patients. In 95.21 % of patients psychopathological symptoms were accompanied by anxiety and were of generally neurotic nature: irritability – 87.12 %, tearfulness – 67.54 %, emotional reactivity – 71.12 %, attention-seeking – 37.6 %, and somatic complaints: headache – 41.13 %, shortness of breath – 18.5 %, blood pressure fluctuations – 64.2 %.

The findings of the clinical and psychopathological study were supported by the results obtained during the psychopathology dynamics scale application, as shown in table 1. The scale of psychopathology assessment included sleep disturbance, mood changes, anxiety, fear, irritability, and psychomotor agitation (table 1).

An important task of the study was to explore the mental state of patients with alcohol-induced mental and behavioral disorders considering the impact of depressive symptoms on their cognitive functioning. Cognitive abilities are a reliable predictor of crucial aspects in the patients' functional status. In the course of the study, which included before- and after-treatment examination, moderate cognitive impairment was found (table 1).

Table 1

Dynamics of psychopathological symptoms in patients with alcohol abuse

No	Symptoms	Main Group: patients with alcohol-induced mental and behavioral disorders with comorbid depression, n=43 (Points)		Control Group: patients with alcohol-induced mental and behavioral disorders without comorbid depression, n=42 (Points)	
		Day 0	Day 30	Day 0	Day 30
1	Sleep disturbance	2.5±0.09*	1.5±0.07*	2.5±0.08*	1.7±0.06*
2	Low mood	2.9±0.07*	1.6±0.06*	2.8±0.09*	1.8±0.08*
3	Anxiety	2.9±0.06*	1.1±0.02*	2.5±0.07*	1.6±0.08*
4	Dysphoria	2.8±0.08*	1.3±0.06*	2.7±0.04*	1.5±0.05*
5	Emotional reactivity	2.6±0.05*	1.0±0.04*	2.6±0.06*	1.9±0.01*
7	Behavior disturbance	2.5±0.05*	1.0±0.03*	2.5±0.14*	1.1±0.14*

Note: * - difference with Group I and Group II indices is reliable ($p<0.05$).

By the end of the study, the patients of the main group demonstrated improvement in mood, where the indices decreased from 2.9±0.07 to 1.6±0.06 points ($p<0.05$); their sleep improved in terms of less frequent awakenings during sleep – the index equaled 2.5±0.09 points at the beginning of the study, dropping to 1.5±0.07 points by its end ($p<0.05$); the anxiety levels decreased from 2.90±0.06 points to 1.1±0.02 points; and emotional reactivity decreased significantly from 2.6±0.05 to 1.0±0.04 points ($p<0.05$). Exacerbation of mental state was manifested as mild and moderate depressive disorders.

Table 2

Results of the scale scores in the study groups

No	Scales	Main Group: patients with alcohol-induced mental and behavioral disorders with comorbid depression, n=43 (Points)		Control Group: patients with alcohol-induced mental and behavioral disorders without comorbid depression, n=42 (Points)	
		Day 0	Day 30	Day 0	Day 30
1	MMSE	27.0±6.85	28.19±7.03	27.31±6.96	28.72±7.06
2	Luria method	3.95±3.00	5.93±3.64	4.83±3.35	5.05±3.42
3	Spielberger-Khanin Personality	27.29±6.87	33.93±7.30	31.05±7.22	45.50±7.78
4	Spielberger-Khanin Reactive	30.16±7.08	31.22±7.15	41.70±7.70	45.10±7.77
5	Zung	60.68±7.54	49.04±7.71	47.35±7.79	39.23±7.62

During the study of the MMSE results dynamics (mean scores) it was revealed that the scores before treatment were 27.0±6.85 in the main group (N=43) and 27.31±6.96 in the control group (N=42); after treatment, the scores equaled 28.19±7.03 and 28.72±7.06, respectively. According to ICD-10, the MMSE results indicated less severe cognitive disturbances (27.0±6.85 and 27.31±6.96) and were diagnosed as "moderate cognitive impairment". No statistically significant difference between groups was found ($p=0.05$). However, in almost all patients abnormalities were observed, indicating mild cognitive dysfunction. The most frequently observed disturbances were of alternative relationship, visual and spatial praxis, attention, memory (problems with memorizing and storing information). In addition, the patients complained of sleep disturbances, memory impairment and failure to concentrate. The majority of patients demonstrated a combined impairment of attention, memory, and information processing speed, which may indicate the diffuse nature of cognitive impairment.

After the applied treatment which included Cytoflavin medication, in 28 patients (65.11 %) of the main group and in 32 (76.19 %) patients of the control group (68 patients in total) improvement of memory and sleep and decreased anxiety was reported.

Analysis of the depression level indices determined by the above methods revealed the following: the mean score by the Zung scale in both groups of comparison was 60.68±7.54 and 47.35±7.79, respectively. Mild depressive symptoms were observed in 24 patients of the main group, while in 19 patients moderate depressive symptoms were found. Anxiety was observed in 38 (88.4 %) of patients of the main group and 37 (88.1 %) patients of the control group ($p\leq0.05$), indicating tension in both groups. Mental anxiety and affective

tension in patients of both groups at the beginning of the study manifested sudden anger and ended in physical weakness. In both groups, there were complaints of frequent headaches (3 points) at the beginning of the study, indicating an increase in withdrawal symptoms. Depressive disorders in the main group showed polymorphism of clinical symptoms, and depressive symptoms comorbidity with other disorders was found. In patients of the main group with a history of depressive disorders, autonomic crises revealed sympathetic-adrenal symptoms (characteristic of patients with substance dependence), starting with a fever, accompanied by palpitations, often with a feeling of numbness or decrease in extremity temperature.

Results on the Spielberger-Khanin scale revealed that in the course of treatment, anxiety indices decreased in the control group: personality anxiety after treatment was 31.05±7.22 points vs 45.50±7.78 points; reactive anxiety was 10.63±0.96 points vs 26.54±1.47 points. During the time of observation, the anxiety indices decreased by 1.6 times in the main group, while in the control group there was a decrease by 2.5 times. The control group had no history of comorbid depressive disorders. The character of depression was largely influenced by the dynamics of anxiety levels, which showed no changes for long periods of time during the study; this was explained by severe withdrawal syndrome.

In the course of the study, we found a functional correlation between cognitive impairment and depression indices, and the pharmacological safety of the Cytoflavin phyto-metabolic drug was proven. Cytoflavin can be regarded as a metabolic drug inducing the major metabolic pathways in cells, promoting utilization of free oxygen and preventing ischemic changes in organs and tissues through reducing the level of peroxide oxidation [8]. Patients with alcohol-induced mental and behavioral disorders with depression comorbidity were prescribed the Cytoflavin metabolic drug (2 tablets twice a day to be taken 30 min before meals) for 30 days in addition to the standard treatment protocol. Due to the significant reduction in symptoms of cognitive dysfunction and depression symptoms, the prescribed treatment was regarded as effective.

To achieve valid results while using subjective scales, we ensured self-completion of the scales by the patients (eliminating discussing the answer options with relatives, acquaintances, or fellow patients). To ensure spontaneity of the responses, a clear time frame was set for filling the scale (about 20-30 minutes). Within this time, the scale had to be completely filled. If the patients missed certain entries or had to interrupt scoring for a long period of time (several hours), retesting was performed on a blank form. In our study, both alcohol intoxication and depression were regarded as factors contributing to the development of cognitive deficit in the patients. We believe that the unity of pathogenetic mechanisms in cognitive and affective symptoms development lies in the established functional correlation between cognitive disorders and signs of depression. In both comparison groups, mental and behavioral disorders (F60-F69) were directly and reliably associated with neurotic and cognitive disorders as well as stress. On the other hand, there is evidence that certain cognitive disorders may be a stage in the psychosis development. This hypothesis is supported by the research results [9] on the detection of cognitive dysfunction both during psychotic relapse and in remission.

Conclusion

The key to effective therapy of patients with neurocognitive impairment is early diagnosis with the use of valid neuropsychological tests and a right choice of therapeutic strategy aimed at restoring cognitive functions and returning to the previous level of personality functioning and quality of life. Administration of Cytoflavin drug in such patients contributes to improvement of sleep and mood (in 80 % of patients according to our study results) and prevention of severe cognitive dysfunction manifestations.

Prospects for further research. Due to the fact that "cognitive complaints" do not always indicate cognitive impairment, comprehensive diagnostics of alcohol-induced CI including additional laboratory tests and diagnostic procedures should be developed and further implemented in clinical practice. The obtained results can serve as a basis for development of effective physical and psychological rehabilitation methods for veterans aimed at enhancing their adaptive capacities.

References

1. Ahaiev NA, Kokun OM, Pishko IO, Lozinska NS. Zbirnyk metodyk dlia diahnostyky nehatyvnykh psykhichnykh staniv viyskovosluzhbovtsiv: metodychnyi posibnyk. Kyiv: NDTsHPZSU; 2016. 234s. [in Ukrainian]
2. Voytsekhovskiy DV, Emelin AYu, Svistov DV. Skriningovaya otsenka vysshikh psikhicheskikh funktsiy s ispolzovaniem standartnykh neyropsikhologicheskikh shkal. Vestnik rossiyskoy voenno-meditsinskoy akademii. 2016; 53(1): 37-42. [in Russian]
3. Hrubliak VV, Hrubliak VT. Praktychne zastosuvannia metodyk otsinky kohnityvnoyi nedostatnosti ta psykhichnoho statusu. Problemy suchasnoyi psykholohiyi. 2011; 12:302-315. [in Ukrainian]
4. Zakharov VV, Stepkina DA. Lechenie kognitivnykh narusheniy pri distsirkulyatornoy entsefalopatii. Rossiyskiy meditsinskiy zhurnal. 2015;1:953-956. [in Russian]
5. Kokun OM, Ahaiev NA, Pishko IO, Lozinska NS Psykholohichna robota z viyskovosluzhbovtsiamy-uchasnykamy ATO na etapi vidnovlennia: metodychnyi posibnyk. K.: NDTs HP ZSU, 2017. 282 s. [in Ukrainian]
6. Levshenkova AA. Diagnostika sostoyaniya kognitivnykh funktsiy u patsientov s tranzitornoy ishemicheskoy atakoy. Voennaya meditsina. 2014. 4. S. 58–59. [in Russian]
7. Linska KI. Khronometrychnyi profil khvorykh na depresiyu pry vykorystanni testu Spilberhera-Khanina. Ukrayinskyi visnyk psykhonevrolohiyi. 2019; 27(1): 46 [in Ukrainian]
8. Minko AI, Linskiy IV, Kuzminov VN, Samoylova ES. Citoflavin v detoksikatsii bolnykh, zavisimykh ot alkogolya. Novosti meditsiny i farmatsii. 2010; 5: 10-12. [in Russian]

9. Molchanova OO, Dzerzhynska NO. Neirobiolohiya kohnityvnykh porushen u khvorykh na shyzofreniyu. Arkhiv psykhiatriyi. 2016; 22(2): 60-64. [in Ukrainian]
10. Odinak MM. Kriterii diagnostiki i klassifikatsiya posttravmaticheskikh kognitivnykh narusheniy. Vestn. Ross. voen.-med. akad. 2014; 48 (4): 12–17. [in Russian]
11. Folstein MF, Folstein SE, McHugh PR. "Mini-mental state": a practical method for grading the cognitive state of patients for the clinician. J. Psychiatr. Res. 1975; 12 (3): 189-198.
12. Hermens DF, Lagopoulos J, Tobias-Webb J et al. Pathways to alcohol-induced brain impairment in young people: A review. Cortex 2013; 49(1): 3-17.
13. Jellinger KA. Pathogenesis and treatment of vascular cognitive impairment. Neurodegenerative Disease Management. 2014; 4(6):471-490.
14. Sachdeva A, Chandra M, Choudhary M et al. Alcohol-Related Dementia and Neurocognitive Impairment: A Review Study. 2016; 5(3): e27976.
15. Vahnina N, Gromova D. Mild Cognitive Impairments: Diagnostics and Treatment. Effect Psychother Neurol Psychiatry. 2016; 25(3):18-24.

Стаття надійшла 22.12.2019 р.

DOI 10.26724/2079-8334-2020-4-74-153-159
UDC 616.718.51-001.515-089

Ye.E. Chip, A.V. Kalashnikov[1], O.V. Kalashnikov[1]
KNMP "Globyne Central District Hospital", Globyne, Poltava region.
[1]SI "Institute of Traumatology and Orthopedics of the NAMS of Ukraine", Kyiv

ALGORITHM FOR TACTICS OF SURGICAL TREATMENT IN PATIENTS WITH PROXIMAL TIBIA FRACTURES

e-mail: yevheniychip@rambler.ru

The purpose of the study performed was: to develop an algorithm for tactics for surgical treatment of patients with fractures of the proximal tibia. The algorithm was based on the analysis of domestic and foreign literature sources, the results of own clinical studies on the treatment efficacy in 125 patients with proximal tibia fractures, who were treated at the clinic of the SI "ITO NAMS of Ukraine" and the Traumatology Department of KNMP "Globyne Central District Hospital" in 2008-2017, and biomechanical studies to determine the strength of fixation using different methods of osteosynthesis in proximal tibia fractures. Based on our own clinical and biomechanical studies, analysis of literature sources, an algorithm for surgical treatment tactics in patients with proximal tibia fractures has been developed, which will permit effective treatment of this severe orthopedic pathology at all stages of surgery. The developed algorithm permits a differentiated selection of surgical accesses, repositions and methods of bone fragments fixation, improves the efficacy of surgical treatment in patients with proximal tibia fractures and can be recommended for implementation in our country.

Key words: proximal tibia fractures, surgical treatment, algorithm.

Є.Е. Чіп, А.В. Калашніков, О.В. Калашніков

АЛГОРИТМ ТАКТИКИ ОПЕРАТИВНОГО ЛІКУВАННЯ ХВОРИХ З ПЕРЕЛОМАМИ ПРОКСИМАЛЬНОГО ВІДДІЛУ ВЕЛИКОГОМІЛКОВОЇ КІСТКИ

Метою проведеного дослідження було: розробка алгоритму тактики оперативного лікування хворих з переломами проксимального відділу великогомілкової кістки. Базисом для створення алгоритму став аналіз літературних джерел вітчизняної та зарубіжної літератури, результати власних клінічних досліджень з ефективності лікування 125 хворих з переломами проксимального відділу великогомілкової кістки які проходили лікування в клініці ДУ «ІТО НАМН України» та відділенні травматології КНМП «Глобинська ЦРЛ» в період 2008-2017 рр. та біомеханічні дослідження з визначення міцності фіксації при використанні різних методів остеосинтезу за переломів проксимального відділу великогомілкової кістки. На основі власних проведених клініко-біомеханічних досліджень, аналізу літературних джерел розроблений алгоритм тактики оперативного лікування хворих з переломами проксимального відділу великогомілкової кістки, що дозволить проводити ефективне лікування даної тяжкої ортопедичної патології на всіх етапах проведення оперативного втручання. Розроблений алгоритм дозволяє проводити диференційований підбір операційних доступів, репозиції та методик фіксації кісткових уламків, збільшує ефективність оперативного лікування пацієнтів з переломами проксимального відділу великогомілкової кістки і може бути рекомендований для впровадження на теренах нашої держави.

Ключові слова: переломи проксимального відділу великогомілкової кістки, оперативне лікування, алгоритм

The work is a fragment of the research project "To develop tactics of surgical treatment for patients with post-traumatic tibia osteomyelitis depending on the trophic severity disorders", state registration No. 0117U003012.

Fractures of the proximal tibia (PTF) are severe injuries of the lower extremities and range from 2 to 5% among all skeletal bone fractures [2]. This type of injury is frequently accompanied by the impression of bone tissue near the joint surface and damage to important soft tissue structures, resulting from the complexity of the kinematics and features of the knee joint structure (close location of the major vessels, nerves, lack of significant muscle mass) [12]. The urgency of treating patients with proximal tibia fractures is determined not only by the high prevalence of this localization fractures, but also by the high frequency of unsatisfactory