ფუნქციურ-მორფოლოგიური მდგომარეობის, ჰიპოთირეოზის სიხშირის, ფორმების და მათ კლინიკურ გამოხატულებაზე ზემოქმედების, მეტაბოლური დარღვევების,ოსტეოპოროზის და მისი კომორბიდური პროცესების შესწავლა.

კომპლექსურად შესწავლილია 312 ოსტეოპოროზით ავადმყოფი 37-76 წ.წ. ასაკში. გამოყენებულია ანტროპომეტრული,კლინიკური,ულტრასონოგრაფიული, ბიოქიმიური, რადიოიმუნოლოგიური (თირეოთროპული ჰორმონის დონე, თავისუფალი ტიროქსინი, ანტისხეულები თირეოიდული პეროქსიდაზასადმი) მეთოდები.

გამოვლენილია, რომ ავადმყოფების ასაკის მატებასთან ერთად პროგრესირებს ოსტეოართროზის მოვლენები, აღინიშნება კომორბიდულ დაავადებათა რაოდენობრივი მატება, განსაკუთრებით ათეროსკლეროზული გენეზის გულსისხლძარღვთა დაავადებები და მათი სიმძიმე. ამ ფონზე 4,44% გამოვლინდა ჰიპერთირეოზის მანიფესტური ფორა, ხოლო 13,78% - სუბკლინიკური. ჰიპერთირეოზის მანიფესტური ფორმის დაშრევება იწვევს დაავადების მიმდინარეობის და მკურნალობის შედეგების გაუარესებას. ჰიპერთირეოზის ორივე ფორმა აძლიერებდა სისხლში მეტაბოლიურ ცვლილებების ხარისხს, ამცირებდა თირქმლის ბოჭკოვან ფილტრაციას, განსაკუთრებით მანიფესტური ფორმა. ჰიპერთირეოზის გამოხატულება განსაკუთრებით აღენიშნებოდა ავადმყოფებს ოსტეოართრიტით მისი მნიშვნელოვანი სისტემური გამოხატულებით, კომორბიდობის მაღალი ფორმით, 50 წ., განსაკუთრებით კი 60 წლის ასაკში უმეტესად ქალებში (83,72%). 50 წლის ასაკის ავადმყოფებს ოსტეოართროზით და კომორბიდობის მაღალი ფონით მიზანშეწონილია ჩაუტარდეს ფარისებრი ჯირკვლის ულტრაბგერითი გამოკვლევა, ასევე თირეოტროპული ჰორმონის დონეების და თავისუფალი თიროქსინის განსაზღვრა ჰიპერთირეოზის დროული დიაგნოსტიკის და მკურნალობის ტაქტიკის გაუმჯობესების მიზნით.

FACTORS ASSOCIATED WITH POST-STROKE FATIGUE DURING THE SECOND HALF YEAR AFTER STROKE

Delva M., Lytvynenko N., Delva I.

Ukrainian Medical Stomatological Academy, Poltava, Ukraine

At our time, as consequence of urbanization and significant lifestyle changes there is an accelerating growth of modern society diseases and their vascular complication, in particular stroke [20]. Post-stroke fatigue (PSF) is a common and often debilitating sequel of both ischemic and hemorrhagic strokes which predicts patients' death, disability and quality of post-stroke life [3]. As known, PSF is multi-domain entity with complex multi-factorial etiopathogenesis [7]. A myriad of biological, psychological, behavioral and social factors might be associated with PSF and (or) predict PSF [18]. Given the concepts of PSF as evolving process it is plausible that different factors contribute to PSF experienced at different post-stroke periods [18]. So, for rational prevention of PSF it's important to clarify the temporal relationships between PSF and definite factors. In particular, in previous work we revealed some personal and psychological factors that may be contributing to PSF development within the first three months after stroke occurrence [1]. However, up to now almost nothing is known about risk factors for global PSF as well as about risk factors for PSF different domains over subsequent periods after stroke.

The objectives of this study were to identify sociodemographic, personal and psychological factors associated with global PSF and with certain PSF domains over the second half year after stroke occurrence.

Material and methods. Patients were included in the study if they had an acute stroke (ischemic or hemorrhagic), agreed to participate in the study and were able to provide informed consent. Exclusion criteria were major medical illnesses that could cause secondary fatigue (oncological, hematological diseases, cardiac, liver, kidney and respiratory insufficiency, progressive angina pectoris, acute myocardial infarction), alcohol abuse, consciousness impairments, insufficient cognitive ability (Mini-Mental State Examination scores less than 24) [2], depressive and anxious disorders (Hospital Anxiety and Depression Scale scores more than 10 for both pathologies) [19], impaired speech function to participate (severe dysphasia or dysarthria), impaired language or written ability to complete the study questionnaires, severe functional disabilities (modified Rankin scale scores ≥4).

Patients' characteristics were evaluated in definite time points: at 6 months (156 patients), at 9 months (139 patients) and at 12 months (128 patients) after stroke occurrence.

PSF was measured by self-report multidimensional fatigue inventory-20 (MFI-20) questionnaire which covers

global, physical, mental, activity-related and motivational fatigue dimensions. A cut-off of 12 out of 20 for every sub-scale has been suggested for use with people with stroke [12].

Socio-demographic factors such as age, gender, marital status (married/single), formal education level (higher/non-higher) were recorded.

Signs of anxiety and depression were assessed by the Hospital Anxiety and Depression Scale (anxiety and depression sub-scales using a cut-off of 4, which has been recommended for persons who have had a stroke) [11]. Apathy symptoms were assessed by the Starkstein apathy scale (a cut-off point 14 or more from the total score of the scale was used to dichotomize the patients into apathetic and non-apathetic) [14]. Cognitive impairments were evaluated by the Montreal cognitive assessment (cut-off scores less than 26) [8]. Sleepiness was measured using Epworth scale (scores 10 or more indicate excessive daytime sleepiness) [5]. For abdominal obesity was measured waist circumference (cut-off 102 cm for males and 88 cm for females). The co-morbidities included arterial hypertension, ischemic heart disease, atrial fibrillation and diabetes mellitus.

Continuous variables were represented as mean±standard deviation (M±SD) and categorical data were represented by number (n) and percentage. Univariate logistic regression analysis was performed to analyze the odds ratio (OR) with 95% confidence intervals (CI) of factors associated with PSF domains. Variables having a p value less than 0,05 in the univariate analysis were selected and evaluated by multivariate logistic regression models. P values less than 0,05 were considered significant. Statistical analyses were performed using SPSS 14.0 statistics software.

Results and thir discussion. As article is limited we present only significant results. First of all, in univariate logistic regression analysis most of the studied variables (gender, marital status, education level, apathetic impairments, excessive daytime sleepiness, abdominal obesity, arterial hypertension, ischemic heart disease, atrial fibrillation, diabetes mellitus) were not significantly associated with global PSF as well as with any PSF domain at 6, 9 and 12 months after stroke occurrence.

As can be seen from Table 2, at 6 months after stroke occurrence, depressive signs were associated with increased risk of global PSF and with increased risk of all PSF domains (in three-four times); anxious symptoms were associated with higher risk of global PSF and with higher risk of PSF motivational domain

Table 1. Characteristics of the baseline study sample

Characteristics		**Patients**
age (years), M±SD		63,6±8,3
males, n (%)		73 (46,8%)
married, n (%)		101 (64,7%)
higher education, n (%)		53 (34,0%)
anxious signs, n (%)		33 (21,2%)
depressive signs, n (%)		31 (19,9%)
apathy symptoms, n (%)		38 (24,4%)
cognitive impairments, n (%)		64 (41,0%)
excessive daytime sleepiness, n (%)		59 (37,8%)
abdominal obesity, n (%)		51 (32,7%)
co-morbidities	arterial hypertension, n (%)	136 (87,2%)
	ischemic heart disease, n (%)	110 (70,5%)
	atrial fibrillation, n (%)	35 (22,4%)
	diabetes mellitus, n (%)	46 (29,5%)

Table 2. Factors significantly associated with different PSF domains from univariate logistic regression models at 6 months after stroke occurrence

PSF domains	Factors		
	cognitive impairments	anxious signs	depressive signs
global	-	2,57 (CI, 1,17-5,63)	3,27 (CI, 1,44-7,42)
physical	-	-	2,71 (CI, 1,20-6,11)
mental	3,23 (CI, 1,54-6,75)	-	2,84 (CI, 1,27-6,35)
motivational	-	3,57 (CI, 1,55-8,22)	4,32 (CI, 1,84-10,15)
activity-related	-	-	2,99 (CI, 1,31-6,83)

Table 3. Factors significantly associated with different PSF domains from univariate logistic regression models at 9 months after stroke occurrence

PSF domains 3,44 (CI, 1,48-8,01)3,78 (CI, 1,62-8,77)	Factors		
	cognitive impairments	anxious signs	depressive signs
global	-	3,44 (CI, 1,48-8,01)	3,78 (CI, 1,62-8,77)
physical	-	3,60 (CI, 1,54-8,41)	-
mental	3,94 (CI, 1,86-8,34)	3,28 (CI, 1,41-7,63)	3,43 (CI, 1,86-10,13)
motivational	-	-	3,37 (CI, 1,39-8,19)
activity-related	-	-	3,02 (CI, 1,28-7,15)

Table 4. Factors significantly associated with different PSF domains from univariate logistic regression models at 12 months after stroke occurrence

PSF domains -4,34 (CI, 1,32-14,26)	Factors		
	cognitive impairments	anxious signs	depressive signs
global	-	-	4,34 (CI, 1,32-14,26)
mental	5,25 (CI, 2,27-12,13)	3,34 (CI, 1,30-8,59)	6,10 (CI, 1,75-21,23)
motivational	-	-	5,89 (CI, 1,78-19,50)

(in two-three times), whereas cognitive impairments were only associated factor of mental PSF. However, in multivariate logistic regression analysis with factors proven to be significant in univariate analysis (cognitive impairments, anxious signs and depressive signs), only depressive signs were as independent predictor of mental PSF (OR, 2,55; CI, 1,12-5,80; p=0,03).

Table 3 demonstrates that at 9 months after stroke occurrence depressive signs were associated with more than threefold increase of global PSF and of all PSF domains (except physical PSF). Moreover, patients with anxious symptoms experienced threefold higher risk of global PSF, of physical and mental PSF domains, whereas cognitive impairments increased the risk of mental PSF development almost in four times. But multivariate logistic regression analysis, after adjusting for reliable variables, from the univariate analyses, demonstrated significant associations only between cognitive impairments and risk of mental PSF (OR, 2,77; CI, 1,12-6,88; p=0,03).

Table 4 shows that at 12 months after stroke occurrence depressive signs were associated with quite high probability of global PSF, of PSF mental and motivational components. Anxious signs as well as cognitive impairments were associated with higher risk of mental PSF. Multivariate logistic regression analysis revealed significant predictors only for mental PSF – depressive signs (OR, 9,33; CI, 2,27-38,45; p=0,002) and cognitive impairments (OR, 5,95; CI, 2,18-16,28; p=0,005).

So, in univariate logistic regression analysis it has been revealed that among diversity of the studied factors only depressive signs and anxious signs may contribute to global PSF and to all PSF domains during the second half year after stroke occurrence. The only exception from above mentioned was contribution of cognitive impairments to risk of PSF mental component. Further, multivariate logistic regression analysis found that among all PSF domains only mental PSF had independent predictors (depressive signs and cognitive impairments) over the studied period.

Wu S. and co-authors proposed PSF as part of an evolving process (they conditionally divided PSF on «early» and «late») i.e. there may be specific factors associated with PSF at different post-stroke periods [18]. Up to now, there are a few studies investigated associations of PSF at different time points after stroke. In one study, baseline depression and anxiety were associated with PSF at 2 and 18 months after stroke [13]. In another study, the 3 strongest correlates of PSF at 6 months were initial stroke severity, disability, and depression, whereas at 1 year, the strongest correlates were depression, anxiety, and language impairments [10]. We previously revealed that employing status before stroke, pre-stroke fatigue, anxious signs, excessive daytime sleepiness and chronic pain may be contributing factors to risk of global PSF and risk of all PSF domains within the first three months after stroke occurrence; moreover, cognitive impairments were risk factor of mental PSF and depressive symptoms – risk factor of motivational PSF [1].

Generally, revealed connections between anxious signs as well as depressive signs and PSF are in accordance with literature data. In systematic review Ponchel A. with co-authors demonstrated that anxiety and depression are reliable predictors of PSF and they played important role in triggering PSF [9]. This is especially true for later periods

after stroke: according to Wu S. and co-authors «late» PSF may be more attributable to psychological and behavioral factors [18], depression seems to play a more important role in the long-term stages of stroke [16]. However, the strength of our study is that PSF were associated with anxious and depressive symptoms, even in patients not meeting clinical criteria for depression and anxiety.

Interesting is the fact that in multivariate logistic regression analysis only depressive signs (but not anxious signs) were independent predictors of mental PSF. On the one hand, the relationship between PSF and depression is difficult to evaluate because many of the tools for assessing depression contain items about fatigue [3]. On the other hand, according to meta-analysis, studies that find an association between anxiety and PSF, the association weakens after controlling for depression (a statistically significant association between PSF and depressive symptoms and a trend toward an association between PSF and anxious symptoms) [17]. To better clarify whether depressive symptoms are confounders, the association between PSF and anxiety needs to be compared between patients with stroke with and without depressive symptoms [17].

In our study mild cognitive impairments within the second half year after stroke occurrence were associated only with mental PSF in univariate, as well as in multivariate logistic regression analyses. Until now literature data about cognitive characteristics and global PSF are quite contradictory [6]. Mental fatigue, according to corresponding MFI-20 sub-scale, is mainly described as "loss of concentration". Evidence suggests that cognitive impairment, mental slowing, and difficulty in concentration may contribute to the decreased mental energy aspect of PSF [3]. As rule, persons with cognitive impairments try to compensate the cognitive deficits by making extra effort (so called «coping theory») [15] with a subsequent faster mental exhaustion and longer time to regain cognitive ability after being exhausted [4].

Summing up we can say that this is the first study to examine factors that could be related to certain PSF domains using MFI-20 sub-scales within the second half year after stroke occurrence. The clinical implications of our study are that clinicians in routine practice should be aware of depressive and anxious symptoms (even without clinical depression and anxiety) as well as be aware of cognitive impairments in patients with stroke and screen for them even in those without clinical depression and anxiety disorder. Subclinical depressive and anxious signs, mild cognitive impairments might be potential targets for prevention and treatment of PSF.

Conclusions. 1. Anxious and depressive signs may be contributing factors to global PSF and to all PSF domains (physical, mental, motivational, activity-related) over the second half year after stroke occurrence. 2. Cognitive impairments may be contributing factors to mental PSF over the second half year after stroke occurrence.

REFERENCES

1. Delva I, Lytvynenko N, Delva M. Factors associated with post-stroke fatigue within the first 3 month after stroke. Georgian Med News. 2017;6(267):38-42.
2. Folstein M, Folstein S, McHugh P. Mini-mental state: A practical method for grading the cognitive state of patients for the clinician. J Psychiatr Res. 1975;12:189-98.
3. Hinkle J, Becker K, Kim J, Choi-Kwon S, Saban K, McNair N, et al. Poststroke fatigue: emerging evidence and approaches to management: a scientific statement for healthcare professionals from the American Heart Association. Stroke 2017;48:e000-e000.
4. Johansson B, Ronnback L. Mental fatigue and cognitive impairment after an almost neurological recovered stroke. ISRN Psychiatry 2012;686425.
5. Johns M. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. Sleep. 1991;14(6):540-5.
6. Lagogianni C, Shirley T, Nadina L. Examining the relationship between fatigue and cognition after stroke: A systematic review. Neuropsychol Rehabil. 2016; 1-60.
7. Nadarajah M, Goh T. Post-stroke fatigue: a review on prevalence, correlates, measurement, and management. Top Stroke Rehabil. 2015;22:208-20.
8. Nasreddine S, Phillips N, Bedirian V, Charbonneau S, Whitehead V, Collin I, et al. The Montreal cognitive assessment, MoCA: a brief screening tool for mild cognitive impairment. J Am Geriatr Soc. 2005;53:695–9.
9. Ponchel A, Bombois S, Bordet R, Henon H. Factors associated with post-stroke fatigue: a systematic review. Stroke Res Treat. 2015.Http://dx.doi.org/10.1155/2015/347920.
10. Radman N, Staub F, Aboulafia-Brakha T, Berney A, Bogousslavsky J, Annoni J. Poststroke fatigue following minor infarcts: a prospective study. Neurology. 2012;79:1422–1427.
11. Sagen U, Vik T, Moum T, Murland T, Finset A, Dammen T. Screening for anxiety and depression after stroke: comparison of the hospital anxiety and depression scale and the Montgomery and Asberg depression rating scale. J. Psychosom. Res. 2009;67(4):325–32.
12. Smets E, Garssen B, Bonke B, Dehaes J. The multidimensional fatigue inventory (MFI) psychometric qualities of an instrument to assess fatigue. J Psychosom Res. 1995;39: 315–25.
13. Snaphaan L, van der Werf S, de Leeuw FE. Time course and risk factors of post-stroke fatigue: a prospective cohort study. Eur J Neurol. 2011;18:611–617.
14. Starkstein S, Migliorelli R, Manes F, Teson A, Petracca G, Chemerinski E, et al. The prevalence and clinical correlates of apathy and irritability in Alzheimer's disease. Eur J Neurol. 1995;2:540-6.
15. Staub F, Bogousslavsky J. Fatigue after stroke: a major but neglected issue. Cerebrovasc Dis. 2001;12(2):75–81.
16. Tseng BY, Billinger SA, Gajewski BJ, Kluding PM. Exertion fatigue and chronic fatigue are two distinct constructs in people post-stroke. Stroke. 2010;41:2908–12.
17. Wu S, Barugh A, Mcleaod M, Mead G. Psychological associations of post-stroke fatigue: a systematic review and meta-analysis. Stroke. 2014;45:893-8.
18. Wu S, Mead G, Macleod M, Chalder T. Model of understanding fatigue after stroke. Stroke. 2015;46 (3):893–8..
19. Zigmond A, Snaith R. The hospital anxiety and depression scale. Acta Psychiatr Scand. 1983;67(6):361-70.
20. Кайдашев ИП. Изменение образа жизни, нарушение энергетического метаболизма и системное воспаление как факторы развития болезней цивилизации. Укр Мед Часопис. 2013;5(97).103-8.

SUMMARY

FACTORS ASSOCIATED WITH POST-STROKE FATIGUE DURING THE SECOND HALF YEAR AFTER STROKE

Delva M., Lytvynenko N., Delva I.

Ukrainian medical stomatological academy, Poltava, Ukraine

Post-stroke fatigue (PSF) is a common and often debilitating sequel of both ischemic and hemorrhagic strokes

Aim - identify socio-demographic, personal and psychological factors associated with different PSF domains over the second half year after stroke occurrence.

There were examined patients consequently in definite time points: at 6 months (156 patients), at 9 months (139 patients) and at 12 months (128 patients) after ischemic or hemorrhagic strokes. Global PSF and certain PSF domains (physical, mental, motivational, activity-related) were measured by multidimensional fatigue inventory-20 scale.

In univariate logistic regression analysis most of the studied variables (gender, marital status, education level, apathetic impairments, excessive daytime sleepiness, waist circumference, arterial hypertension, ischemic heart disease, atrial fibrillation, diabetes mellitus) were not significantly associated with global PSF as well as with any PSF aspect at 6, 9 and 12 months after stroke occurrence. On the other hand, univariate logistic regression analysis showed reliable associations between risk of global PSF, risk of all PSF domains and anxious as well as depressive signs, reliable associations between risk of mental PSF and cognitive impairments over the second half year after stroke. Multivariate logistic regression analysis revealed that depressive signs and cognitive impairments were independent predictors of mental PSF over studied post-stroke period.

Screening and early management of depressive signs, anxious signs and cognitive impairments probably may be helpful for PSF prevention over the second half year after stroke occurrence.

Keywords: stroke, fatigue, risk factors.

РЕЗЮМЕ

ФАКТОРЫ, АССОЦИИРОВАННЫЕ С ПОСТИНСУЛЬТНОЙ УСТАЛОСТЬЮ В ТЕЧЕНИЕ ВТОРОГО ПОЛУГОДИЯ ПОСЛЕ ИНСУЛЬТА

Дельва М.Ю., Литвиненко Н.В., Дельва И.И.

Украинская медицинская стоматологическая академия, Полтава, Украина

Постинсультная усталость (ПИУ) является весьма распространенным феноменом и сопровождается разнообразными негативными последствиями.

Цель исследования - идентифицировать социально-демографические и психологические характеристики пациентов, ассоциированные с определенными компонентами постинсультной усталости в течение второго полугодия после развития инсульта.

Проведено последовательное обследование пациентов в определенных временных точках: спустя 6 месяцев (156 пациентов), 9 месяцев (139 пациентов) и 12 месяцев (128 пациентов) после развития ишемических или геморрагических инсультов. Наличие общей ПИУ, как и определенных ее компонентов (физический, психический, мотивационный, связанный с активностью) определяли с помощью многомерной шкалы усталости MFI-20.

В результате проведенного одновариантного логистического регрессионного анализа каких-либо достоверных ассоциаций между большинством изучаемых факторов (пол, семейное положение, уровень образования, апатические нарушения, повышенная дневная сонливость, абдоминальное ожирение, артериальная гипертензия, ишемическая болезнь сердца, фибрилляция предсердий, сахарный диабет) и риском наличия как общей ПИУ, так и отдельных ее компонентов на протяжении второго постинсультного полугодия не выявлено; выявлены статистически достоверные ассоциации между риском общей ПИУ и всех ее компонентов и наличием тревожных и депрессивных симптомов. Определены статистически достоверные ассоциации между риском психической ПИУ и когнитивными нарушениями. Мультивариантный логистический регрессионный анализ показал, что депрессивные проявления и когнитивные нарушения являются независимыми предикторами риска развития психического компонента ПИУ за изучаемый постинсультный период.

Авторами делается вывод, что раннее выявление и эффективная коррекция депрессивных и тревожных проявлений, а также когнитивных нарушений позволит проводить эффективную профилактику ПИУ в течение второго полугодия после возникновения инсульта.

რეზიუმე

პოსტინსულტურ დაღლილობასთან ასოცირებული ფაქტორები ინსულტისშემდგომი მეორე ექვსი თვის განმავლობაში

მ. დელვა, ნ. ლიტვინენკო, ი. დელვა

უკრაინის სამედიცინო სტომატოლოგიური აკადემია, პოლტავა, უკრაინა

პოსტინსულტური დაღლილობა (პიდ) წარმოადგენს საკმაოდ გავრცელებულ ფენომენს, მრავალმხრივი თანმხლები ნეგატიური შედეგებით. კვლევის მიზანს შეადგენდა პაციენტებში ინსულ-

ტისშემდგომი მეორე ექვსი თვის განმავლობაში პიდ-ის გარკვეულ კომპონენტებთან ასოცირებული სოციალ-დემოგრაფიული და ფსიქოლოგიური მახასითებლების იდენტიფიცირება. ჩატარდა იშემიური და ჰემორაგიული ინსულტის შემდეგ პაციენტების თანმიმდევრული კვლევა დროის სხვადასხვა მონაკვეთებში: 6 თვის (156 პაციენტი), 9 თვის (139 პაციენტი) და 12 თვის შემდეგ (128 პაციენტი). პიდ-ის საერთო ასევე, მისი ფიზიკურ, ფსიქიკურ,მოტივაციურ,აქტივობასთან დაკავშირებული კომპონენტების არსებობა განისაზღვრა დაღლილობის მრავალგანზომილებიანი სკალით - MFI-20. ერთვარიანტული რეგრესიული ანალიზით რაიმე სარწმუნო ასოციაცია შესწავლილ ფაქტორებს (სქესი, ოჯახური მდგომარეობა, განათლების დონე, აპათიური დარღვევები, მომატებული დღის ძილიანობა, აბდომინური სიმსუქნე, არტერიული ჰიპერტენზია, გულის იშემიური დაავადება, წინაგულების ფიბრილაცია, შაქრიანი დიაბეტი) და საერთო პიდ-სა და მის ცალკეულ კომპონენტს შორის ინსულტისშემდგომი მეორე ექვსი თვის განმავლობაში არ გამოვლინდა. ერთვარიანტული რეგრესიული ანალიზით მეშვეობით აღმოჩნდა საერთო პიდ-ის და მისი ცალკეული კომპონენტის სტატისტიკურად სარწმუნო ასოციაცია შფოთვით და დეპრესიულ სიმპტომებთან, ასევე, ფსიქიკური პიდ-ის რისკის - კოგნიტიურ დარღვევებთან. მულტივარიანტულმა რეგრესიულმა ანალიზმა აჩვენა, რომ პოსტინსულტურ პერიოდში დეპრესიული გამოვლინებები და კოგნიტიური დარღვევები წარმოადგენენ პიდ-ის ფსიქიკური კომპონენტის განვითარების დამოუკიდებელ პრედიქტორებს. სავარაუდოა, რომ დეპრესიული და შფოთვითი გამოვლინებების, კოგნიტიური დარღვევების ადრეული გამოვლენა და ეფექტური კორექცია უზრუნველყოფს პიდ-ის პროფილაქტიკას.

CLINICAL FEATURES OF THERAPY OF ANEMIA AND THE SIGNIFICANCE OF ITS FLUCTUATION IN THE DEVELOPMENT OF ANEMIA IN DIALYSIS PATIENTS

Lapina E., Batiushin M., Terentiev V., Gasanov M., Gasanova A.

Rostov State Medical University, Rostov-on-Don, Russian Federation

The results of numerous epidemiological studies have shown a high prevalence of chronic kidney disease (CKD) in the population, including its terminal stage [1,6]. Anemia is one of the most common complications of CKD, is observed in every second patient with CKD (47%), while its frequency is back correlated with the degree of decrease in glomerular filtration rate. Anemia occurs in 5% of patients with CKD 1-2nd stages, 15-20% - with the 3rd stage, in 50-55% of patients - with the 4th stage and in 80% - with the 5th stage [3,5,7].

Anemia worsens the quality of life of patients with CKD, causing the decrease in their working capacity, exercise tolerance, impairment of cognitive and sexual functions [10,15]. In addition, anemia acts as an independent risk factor of cardiovascular complications [11,16,18], contributes to the progression of CKD [2,9,18].

The development of anemia in CKD is a multifactorial process, in which the leading role is played by the deficiency in the formation of the erythrocyte glycoprotein growth factor - erythropoietin and the depletion of available iron pool for erythropoiesis [4]. In accordance with this, the treatment of anemia in CKD is based on the using of recombinant erythropoietin and iron drugs [3].

The treatment of anemia is aimed at achieving the target hemoglobin level, the excess of which is associated with an increased risk of cardiovascular complications (myocardial infarction, stroke, congestive heart failure etc.) and death, which was demonstrated in large randomized trials: CHOIR, CREATE, TREAT [8,11,17]. In addition, the great importance in the treatment of patients with erythropoiesis-stimulating agents has not only the achievement of hemoglobin targets, but also their maintain at a constant level. The fluctuation in hemoglobin level, constituting 1,5 g/dl in amplitude up or down from some point of equilibration, with a cycle time at least 8 weeks, is a reliable predictor of high mortality among patients with CKD [12]. Thus, despite the progress in the treatment of anemia in CKD, a number of questions of therapy of anemia are unresolved: the timing of initiation of erythropoiesis-stimulating therapy, the target values of hemoglobin level, the fight against resistance to erythropoiesis-stimulating agents, the exclusion of their fluctuations etc.

The purpose of our study was to research the features of anemia's treatment and the influence of fluctuation in dose of antianemic drugs on hemoglobin level in patients with CKD stage 5D receiving hemodialysis (HD).

Material and methods. The study included 100 patients with stage 5 CKD (51 women and 49 men, mean age was 53,4±15,8 years). The study was conducted in accordance with ethical principles of the Declaration of Helsinki, its design approved by the local ethics committee of Rostov State Medical University. Written informed