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# NEUROLOGICAL AND NEUROIMAGING FACTORS ASSOCIATED WITH POST-STROKE FATIGUE OVER THE SECOND HALF YEAR AFTER ACUTE CEREBROVASCULAR EVENTS

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Post-stroke fatigue (PSF) is a common syndrome for stroke survivors. Aim. Identify clinical and neuroimaging factors associated with different PSF domains over the second half year after acute cerebrovascular events (ACE). Material and methods. There were examined 194 patients at 6, 9 and 12 months after ACE. Global PSF and PSF domains were measured by multidimensional fatigue inventory-20 scale. Results. It had been identified the significant prevalence of global PSF and physical PSF in stroke patients in comparison with TIA. In univariate logistic regression analysis, in stroke patients it had been found reliable associations between MRS score and risk of global PSF as well as risk of PSF domains related to physical activity. Infratentorial infarcts were significantly associated with increased risk of global PSF and white matter lesion extension, according to Fazekas scale score, was directly associated with significant higher risk of mental and motivational PSF. Conclusion. Decreased post-stroke functional ability, infratentorial ischemic stroke location and leukoaraiosis grade could be prognostic factors for certain PSF domains over the second half year after ACE.

**Key words:** postnatal fatigue, cerebral blood flow, infact, stroke.

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Post-stroke fatigue (PSF) is a common syndrome for stroke survivors [3, 4]. PSF exerts a negative impact on participation in physical activities and rehabilitation [2]. Patients with PSF have difficulty in resuming social, familial, and professional activities [19]. Evidences indicate that PSF is evolving multi-domain entity [3]. PSF has multidimensional causes varying depending on timing after stroke occurrence [24]. Understanding triggering factors for PSF development over different post-stroke periods has crucial importance for early prevention and effective PSF management. In our previous works we revealed that some neurological and neuroimaging factors have significant associations with certain PSF components within the first 3 months after stroke occurrence [3, 5]. Moreover, to the best of our knowledge, up to now there has been no research on the clinical and neuroimaging determinants of certain PSF domains over the later periods after acute cerebrovascular events (ACE).

**The purpouse** of the study were to identify clinical and neuroimaging factors associated with different PSF domains over the second half year after ACE occurrence.

**Material and methods.** Initially we enrolled in the study 194 patients: 137 with ischemic strokes, 19 with hemorrhagic strokes and 38 with TIA. Patients were included in the study if they agreed to participate and were able to provide informed consent. Exclusion criteria were major medical illness 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) [6], depressive and anxious disorders (Hospital Anxiety and Depression Scale scores more than 10 for both pathologies) [25], impaired speech function to participate (severe dysphasia or dysarthria), impaired language or written ability to complete the study questionnaire, severe functional disabilities (modified Rankin scale (MRS) scores ≥4).

Patients' characteristics had been evaluated consequently in definite time points: at 6, 9 and 12 months after ACE occurrence. During the third quarter of post-stroke year 21 patients (12 with ischemic stroke, 5 with hemorrhagic stroke, and 4 with TIA) and during the last quarter of post-stroke year 14 more patients (9 with ischemic stroke, 2 with hemorrhagic stroke and 3 with TIA) were dropped out due to different reasons. So, at 9 and 12 months after ACE we have examined 173 and 159 patients, respectively. 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 [15]. For all stroke patients we recorded such clinical variables as affected cerebral hemisphere (right – left) and post-stroke functional disability (according to MRS score). Additionally, for ischemic stroke patients we recorded some specific variables – stroke subtype (non-lacunar – lacunar, according to TOAST criteria [1]) and affected cerebral arterial region (carotid – vertebrobasilar). Among enrolled patients 141 subjects underwent head magnetic resonance imaging (MRI) – 107 with ischemic strokes, 5 with hemorrhagic strokes and 29 with TIA. MRI studies were performed with a 1,5-T system (Siemens MAGNETOM Avanto 1.5T) and 0,2-T system (Signa Profile HD GE 0.2T).

For measurement of brain atrophy we used planimetrical indexes: bifrontal index (BFI), bicaudate index (BCI), maximum diameter of the third ventricle and cortical atrophy index (CAI) on T1 MRI sequence [9]. BFI – maximum width of the anterior horns of the lateral ventricles in relation to the inner skull width at the same level. BCI – minimum width of the lateral ventricles in relation to the inner skull at the same level. CAI – sum of the width of the four widest sulci at the two highest scanning levels divided into maximum inner scull diameter. White matter lesions derived from fluid-attenuated inversion recovery (FLAIR) imaging was graded from 0 to 3 on Fazekas scale on the basis of visual assessment both periventricular (0=absent, 1=caps or pencil lining, 2=smooth halo, and 3=irregular periventricular hyperintensities extending into deep white matter) and subcortical areas (0=absent, 1=punctuate foci, 2=beginning confluence of foci, and 3=large confluent areas) [6]. The total Fazekas scale score was calculated by adding the periventricular and subcortical scores [22]. Leukoaraiosis severity was graded according to the Fazekas scale as mild (1–2), moderate (3–4), and severe (5–6). Continuous variables with parametric distribution (according to Shapiro-Wilk test) were represented as mean±standard deviation. Categorical data were represented by number (n) and percentage. Differences in categorical variables were compared using chi-square test. Univariate logistic regression analysis was performed to analyze the odds ratio (OR) with 95% confidence intervals (CI) of clinical and neuroimaging variables associated with PSF domains. P-value less 0,05 was taken to indicate statistical significance. Statistical analyses were performed using SPSS 14.0 statistics software.

**Results and its disscusion.** Patients' age ranged from 41 to 79 years (63,1±8,7 years). There were 93 (47,9%) males and 101 (52,1%) females. As can be seen from table 1, over the second half year after ACE occurrence it had been observed a significant increase of the risk of global PSF (probably, due to reliable increasing of the risk of physical PSF domain) in stroke patients in comparison with TIA patients. So, patients with strokes, in relation to TIA, had more common global PSF and physical PSF in all time points within the observation period: at 6 months - OR 4,37 (95% CI, 1,47-12,98; p=0,01) and OR, 3,91 (95% CI, 1,44-10,57; p=0,01), at 9 months - OR 3,59 (95% CI, 1,19-10,81; p=0,02) and OR 3,47 (95% CI, 1,15-10,46; p=0,03), at 12 months – OR, 4,08 (95% CI, 1,17-14,26; p=0,03) and OR 4,24 (95% CI, 1,22-14,78; p=0,02), respectively. Moreover, at 9 months after stroke mental PSF component was significant more frequently than after TIA (OR, 2,87; 95% CI, 1,04-7,90; p=0,04).

Table 1

**Frequencies of certain PSF domains over the second half year after ACE occurrence**

| PSF domain | Time point after ACE occurrence | | |
|---|---|---|---|
| | 6 months | 9 months | 12 months |
| global | | | |
| TIA, n (%) | 4 (10,5%)* | 4 (11,8%)* | 3 (9,7%)* |
| stroke, n (%) | 53 (34,0%) | 45 (32,4%) | 39 (30,5%) |
| physical | | | |
| TIA, n (%) | 5 (13,2%)* | 4 (11,8%)* | 3 (9,7%)* |
| stroke, n (%) | 58 (37,2%) | 44 (31,7%) | 40 (31,3%) |
| mental | | | |
| TIA, n (%) | 6 (15,8%) | 5 (14,7%)* | 5 (16,1%) |
| stroke, n (%) | 49 (31,4%) | 46 (33,1%) | 40 (31,3%) |
| motivational | | | |
| TIA, n (%) | 5 (13,2%) | 4 (11,8%) | 4 (12,9%) |
| stroke, n (%) | 34 (21,8%) | 30 (21,6%) | 26 (20,3%) |
| activity-related | | | |
| TIA, n (%) | 5 (13,2%) | 5 (14,7%) | 3 (9,7%) |
| stroke, n (%) | 41 (26,3%) | 35 (25,2%) | 29 (22,7%) |

* - significant difference (p<0,05) by chi-square test in comparison with stroke patients.

Table 2 shows patients' distribution according to their clinical characteristics. In patients with stroke univariate logistic regression analysis did not reveal any association between any PSF domain in all time points within the second half year after disease onset and all analyzed stroke characteristics (affected cerebral hemisphere, ischemic stroke subtype and affected cerebral arterial region for ischemic stroke). Table 3 demonstrates patients' distribution on the basis of functional limitation degree. Over the second half year after stroke occurrence there were some reliable associations between MRS score values and risk of global PSF as

well as risk of such PSF domains that are just related to physical activity (physical PSF and activity-related PSF). Univariate logistic regression analysis showed that at 6 months after stroke the MRS score increment of 1 point was significantly associated with higher risk of global PSF (OR, 1,81; CI, 1,27-2,59; p=0,001), physical PSF (OR, 1,83; CI, 1,29-2,60; p=0,001) and activity-related PSF (OR, 1,53; CI, 1,06-2,22; p=0,02). In the same way, at 9 and 12 months after stroke occurrence identical values were – OR 1,91 (CI, 1,30-2,80; p=0,001), OR 1,95 (CI, 1,33-2,88; p=0,001), OR 1,57 (CI, 1,06-2,32; p=0,03) and – OR 1,65 (CI, 1,11-2,47; p=0,01), OR 1,77 (CI, 1,18-2,65; p=0,01), OR 1,63 (CI, 1,06-2,51; p=0,03), respectively.

Table 2

**Clinical characteristics of stroke patients at the baseline**

| | | | |
|---|---|---|---|
| Affected cerebral hemisphere | | right | 84 (53,8%) |
| | | left | 72 (46,2%) |
| Ischemic stroke characteristics | subtype | non-lacunar | 98 (71,5%) |
| | | lacunar | 39 (28,5%) |
| | affected cerebral arterial region | carotid | 90 (65,7%) |
| | | vertebrobasilar | 47 (34,3%) |

Table 3

**Rates of MRS scores over the second half year after stroke occurrence**

| MRS score | Time point after stroke occurrence | | |
|---|---|---|---|
| | 6 months | 9 months | 12 months |
| 0 | 24 (15,4%) | 31 (22,2%) | 31 (24,2%) |
| 1 | 54 (34,6%) | 52 (37,4%) | 45 (35,2%) |
| 2 | 43 (27,6%) | 32 (23,8%) | 39 (30,5%) |
| 3 | 35 (22,4%) | 24 (16,6%) | 13 (10,1%) |

First of all, univariate logistic regression analysis did not reveal any reliable statistical regularities between frequencies of any PSF domain and any brain atrophy indexes (BFI, BCI, third ventricle diameter, CAI) over the second half year after ACE occurrence. On the other hand, it had been shown that cerebral infarcts of infratentorial locations were significantly associated with increased risk of global PSF at 6 months (OR, 3,19; CI, 1,34-7,58; p=0,01) and at 12 months (OR, 3,10; CI, 1,25-7,65; p=0,01) after disease occurrence. Moreover, univariate logistic regression analysis showed that the Fazekas scale score increment of 1 point was significantly associated with higher risk of mental PSF (OR, 1,49; CI, 1,11-2,00; p=0,01) at 6 months after ACE occurrence as well as with higher risk of mental PSF and motivational PSF in later observation time points – OR 1,56 (CI, 1,13-2,16; p=0,01) and OR 1,61 (CI, 1,11-2,34; p=0,01) at 9 months, OR 1,78 (CI, 1,24-2,55; p=0,002) and OR 1,64 (CI, 1,10-2,46; p=0,02) at 12 months, respectively.

The first finding is that in comparison with TIA, we can see the significant prevalence of global and physical PSF over the second half year after ACE occurrence. The same peculiarity we previously revealed within the first 3 months after ACE occurrence [3].

Table 4

**Neuroimaging characteristics of stroke patients at the baseline**

| | |
|---|---|
| Ischemic lesion location, n (%)<br>cortical-subcortical<br>subcortical infratentorial | 33 (30,8%)<br>38 (35,5%)<br>36 (33,7%) |
| Brain atrophy indexes<br>BFI<br>BCI<br>third ventricle diameter, mm<br>CAI | <br>0,33±0,04<br>0,23±0,06<br>8,3±2,0<br>0,04±0,02 |
| Fazekas scale score, n (%)<br>1<br>2<br>3<br>4<br>5 | <br>18 (12,8%)<br>38 (26,9%)<br>35 (24,8%)<br>33 (23,4%)<br>17 (12,1%) |

Possibly this phenomenon may be determined in some degree by post-stroke functional limitations (whereas TIA don't have any functional deficit) and by extended and permanent ischemic brain lesion due to stroke (PSF, at least partially, may be of central origin [23]). In our study all neurological characteristics of stroke were not predictive of any PSF domain in any time point within the second half year after disease onset.

In literature there are controversial data regarding connections between stroke features and PSF as whole entity. According to systematic review by Ponchel A. et al. in 11 studies it had not been discovered any connection between stroke site and PSF [14]. On the other hand, it should be noted studies that had shown relationships between PSF and vertebrobasilar stroke [11, 18] and this phenomenon was explained by the lesion of ascending activating reticular formation in brainstem [17]. The second finding is the direct significant associations between decreased functional ability according to MRS score and risk of global PSF as well as risk of PSF domains that are related to physical activity (physical PSF and activity-related PSF). Whereas, to the above mentioned associations, our previous study showed also significant negative connections between MRS score and risk of mental PSF at 1 month as well as at 3 months after stroke [3]. Generally, literature data about post-stroke functional status and PSF are quite contradictory – systematic review of factors that contribute to PSF identified 25 studies in which PSF was associated with greater disability and dependency, though this association was not detected in other 15 studies [14]. In any case, PSF is an independent contributor to post-stroke disability (maybe through the various behavioral impacts), so the effective early management of PSF might be an important step in post-stroke rehabilitation [12]. The third finding is some significant regularities between neuroimaging characteristics (ischemic stroke location, extent of white matter lesion) and increased risk of definite PSF domains over the second half year after ACE occurrence. Infratentorial infarcts were associated with increased risk of global PSF. In the same way, cerebral infarcts of infratentorial locations were significantly associated with increased risk of global PSF at 3 months after stroke [5]. Our results support others who found an association between PSF and brainstem strokes and this phenomenon may be explained, according to authors, by the interruption of brainstem reticular activating system which is involved in attention [16, 19]. Additionally, leukoaraiosis extension was directly associated with risk of mental PSF and motivational PSF over the second half year after ACE. Our results are supported by other work where the presence of leucoareosis on CT was independently associated with PSF at 6 months and later after ischemic and hemorrhagic strokes occurrence [13]. Generally, it's known the importance of white matter lesions in the pathophysiology of fatigue at all, for example in chronic fatigue syndrome was observed reduced white matter volume [8]. It's well known that white matter lesions are directly connected with cognitive decline [20]. Associations between leukoaraiosis severity and risk of mental PSF can be explained, at least partially, by the fact that persons with cognitive impairments try to compensate the cognitive deficits by making extra effort with subsequent faster tiredness [19]. According to MFI-20, the essence of fatigue motivational component has close overlap with depressive signs [15]. As known, white matter hyperintensities may contribute to the development of post-stroke depressive signs (and thus to risk of motivational PSF) as directly as well as indirectly through different mechanisms [10, 21].

## Conclusions

1. Over the second half year after strokes, in comparison with TIA, it had been identified significant higher rates of global and physical PSF domains.
2. The MRS score was directly associated with significant higher risk of global PSF and of PSF domains related to physical activity (physical and activity-related PSF).
3. Infratentorial infarcts were associated with significantly increased risk of global PSF. 4. White matter lesion extension according to Fazekas scale score was directly associated with significant higher risk of mental and motivational PSF.

***Future investigations in this field*** *should be directed toward identification of clinical and neurological factors associated with general PSF as well as with certain PSF domains during later post-stroke periods.*

## Referances

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## Реферати

### КЛІНІКО-НЕВРОЛОГІЧНІ ТА НЕЙРОВІЗУАЛІЗАЦІЙНІ ФАКТОРИ, АСОЦІЙОВАНІ З ПОСТІНСУЛЬТНОЮ ВТОМОЮ НА ПРОТЯЗІ ДРУГОГО ПІВРІЧЧЯ ПІСЛЯ РОЗВИТКУ ГОСТРИХ ПОРУШЕНЬ МОЗКОВОГО КРОВООБІГУ

**Дельва І. І.**

Постінсультна втома (ПІВ) – розповсюджене ускладнення гострих порушень мозкового кровообігу (ГПМК). Мета. Ідентифікувати клінічні та нейровізуалізаційні фактори, асоційовані з різними компонентами ПІВ на протязі другого півріччя після розвитку ГПМК. Матеріал та методи. Обстежено 194 пацієнти через 6, 9 та 12 місяців після ГПМК. Глобальну ПІВ та окремі її компоненти вимірювали за допомогою багатомірної шкали оцінки втоми (MIF-20). Результати. Виявлена достовірно вища розповсюдженість глобальної та фізичної ПІВ при інсультах, порівняно з транзиторними ішемічними атаками. В одновимірному логістичному регресійному аналізі знайдено достовірні асоціації між ступенем функціональної неспроможності, згідно модифікованої шкали Ренкіна, та ризиком наявності глобальної і фізичної ПІВ. Наявність інфартенторіальних інфарктів асоціювалася з підвищеним ризиком глобальної ПІВ, а ступінь лейкоареозу, згідно шкали Фазекас, прямо асоціювалася з підвищеним ризиком психічної та мотиваційної ПІВ. Висновки. Рівень функціональної неспроможності, інфратенторіальна локалізація інфарктів та ступінь лейкоареозу можуть розглядатися як прогностичні фактори наявності ПІВ протягом другого півріччя після розвитку ГПМК.

**Ключові слова:** постінсультна втома, мозковий кровообі, інфакт, інсульт.

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### КЛИНИКО-НЕВРОЛОГИЧЕСКИЕ И НЕЙРОВИЗУАЛИЗАЦИОННЫЕ ФАКТОРЫ, АССОЦИИРОВАННЫЕ С ПОСТИНСУЛЬТНОЙ УСТАЛОСТЬЮ НА ПРОТЯЖЕНИИ ВТОРОГО ПОЛУГОДИЯ ПОСЛЕ РАЗВИТИЯ ОСТРЫХ НАРУШЕНИЙ МОЗГОВОГО КРОВООБРАЩЕНИЯ

**Дельва И. И.**

Постинсультная усталость (ПИУ) – распространенное осложнение острых нарушений мозгового кровообращения (ОНМК). Цель. Идентифицировать клинические и нейровизуализационные факторы, ассоциированные с разными компонентами ПИУ на протяжении второго полугодия после развития ОНМК. Материал и методы. Обследовано 194 пациента через 6, 9 и 12 месяцев после ОНМК. Глобальную ПИУ и отдельные ее компоненты измеряли с помощью многомерной шкалы оценки усталости (MIF-20). Результаты. Выявлена достоверно большая распространенность глобальной и физической ПИУ при инсультах, в сравнении с транзиторными ишемическими атаками. В одномерном логистическом регрессионном анализе найдены достоверные ассоциации между степенью функциональной несостоятельности, согласно модифицированной шкале Рэнкина, и риском глобальной и физической ПИУ. Инфартенториальные инфаркты прямо ассоциировались с повышенным риском глобальной ПИУ, а степень лейкоареоза, по шкале Фазекас, - с повышенным риском психической и мотивационной ПИУ. Выводы. Уровень функциональной несостоятельности, инфратенториальная локализация инфарктов и степень лейкоареоза могут рассматриваться в качестве прогностических факторов наличия ПИУ на протяжении второго полугодия после развития ОНМК.

**Ключевые слова:** постинсультная усталость, мозговое кровообращение, инфакт, инсульт.

Рецензент Литвиненко Н.В.