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Original article

POST-STROKE DEPRESSION AND ITS RISK FACTORS – CROSS-SECTIONAL STUDY

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ABSTRACT:

Introduction: Post-stroke depression (PSD) is one of the leading, although preventable complications, after ischemic stroke (IS). Our study aimed to examine PSD and the leading causes for its development

Contingent and methods: In this cross-sectional study, we examined 107 post-stroke survivors (66.67±9.03 years old, 65 males and 42 females) for PSD in a two-step model (at the acute stage and at the 3rd month after stroke) with 21 Hamilton Depression Rating Scale (HDRS).

Results: 33% of examined patients showed depression at the acute and 30% at the chronicIS stage. 1/4 to 1/3 of the others had subclinical depression. The severity of depression in most of the cases was mild. Very few of our patients (2 at the acute tage and 1 at the chronic stroke stage) had severe depression. The main risk factors for PSD were stroke severity, subcortical localization of stroke, leukoaraiosis, ageing, loneliness and some comorbidities (that lead to systemic inflammation, changes in neurotransmission and impaired brain plasticity).

Conclusion: PSD is one of the main complications of acute IS. It should be assessed, prevented and treated as soon as possible.

Keywords: post-stroke depression, risk factors,

INTRODUCTION

Post-stroke depression (PSD) is one of the leading, although preventable, complications after stroke. It is considered that 10-27% of post-stroke survivors develop major depressive disorder, and 15-40% of the others have symptomatic depression during the first three months after the incident [1].

There are two main hypotheses for the development of PSD – reactive depression (the depression is considered as psychogenic reaction to the disease) and organic (the depression is due to the brain's structural and functional changes and underlying inflammation) [2].

The aim of our study was to examine the PSD and the leading risk factors for its development.

Contingent and methods:

Contingent: In this cross-sectional study, we recruited 107 poststroke survivors (66.67±9.03 years old, 65 males and 42 females).

The inclusion criteria were: 1. Clinically verified ischemic stroke (IS); 2. Stroke severity 1 to 15 points on the National Institutes of Health Stroke Scale (NIHSS); 3. Luck of other severe brain diseases (tumors, degenerative brain diseases, epilepsy), except leucoaraiosis; 4. Luck of family history or previous history of depression or severe psychiatric disorders; 5. Luck of preceding treatment with antipsychotics, antidepressants, or opioid drugs; 6. Ability to fulfill the neuropsychological battery.

METHODS

The IS were diagnosed with clinical examination and imaging technique (computer tomography – CT).

The patients were examined twice – firstly, during the first three days after IS and secondly - at the third month after IS. We used 21 - Hamilton Depression Rating Scale (HDRS) for assessment of PSD (0-7p. not depressed; 8-13p. borderline (subthreshold), 14-18 p. - Mild depression; 19-22 p. – Moderate depression; >23p. Severe depression).

Statistical analysis was done via SPSS 20.0. The results were interpreted at a 95% confidential level.

RESULTS

1. PSD frequency and severity.

The average HDRS score during the first three days was 12.52±5.69p, and in the third month was 9.61±4.29p.

Around 33% of our patients were with clinical depression during the first 3 days after IS, and 30% of them had depression in the 3rd month (see Fig. 1 and 2). Nearly 1/3 of our patients (in the first three days) and over 1/4 of them (in the third month) showed additional subclinical depression. Most of the depressed patients had mild depression, and only 2 of them at acute stroke stage and 1 of them at 3rd month developed severe depression.

Fig. 1. Depressive signs during the first 3 days after ischemic stroke

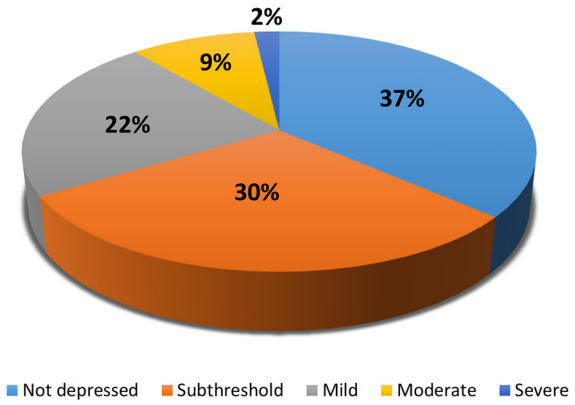
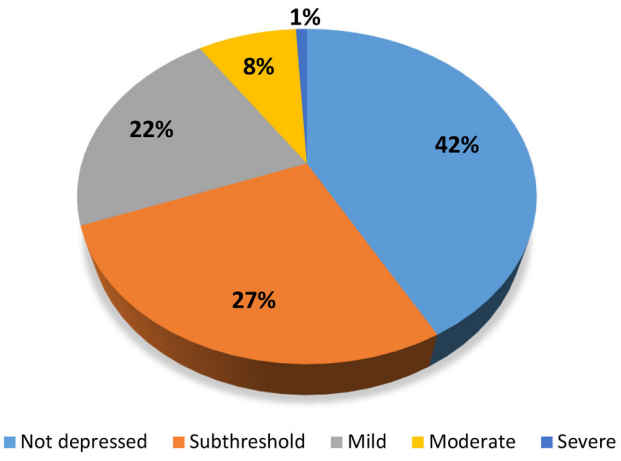


Fig. 2. Depressive signs in the 3rd month after ischemic stroke



2. Risk factors for Post-stroke depression

2.1. The impact of stroke severity and localization and Leukoaraiosis on PSD.

The stroke severity (measured by NIHSS) strongly correlated with the HDRS score in the third month (rr=0.43; p=0.0001) but not with HDRS at the acute IS stage (p>0.05). The patients with subcortical strokes had more severe depressive signs than those with cortical strokes both at the acute stage (HDRS 13.46±4.27p. vs 10.94±4.29p., p=0.0448) and at the 3rd month (HDRS 10.37±4.05p. vs. 8.24±3.93p., p=0.0194) after IS. The underlying leukoaraiosis caused more severe depression in the 3rd month (HDRS 10.67±0.83 vs 7.97±0.62p., p=0.0042), but not at acute stroke stage (p>0.05). Patients with left and right hemispheric strokes and those with brainstem strokes didn't show any differences in the severity of depression (p>0.05).

2.2. The influence of ageing, sex and loneliness on PSD

Ageing is associated with more severe depression at the 3rd month after IS (rr=0.32; p=0.0006), but not at the acute stroke stage (p>0.05). Females had more severe depression than males only during the acute stroke stage (HDRS 14.58±6.49p. vs. 11.47±4.81p., p=0.0045). We didn't find sex-related difference in PSD severity at 3rd month after the incident (p>0.05). Loneliness was associated with high HDRS scoring at acute (15.69 vs 19.8p., p=0.0121) but not at chronic IS phase (p>0.05). A high Barthel index was associated with more severe depression at the chronic stroke phase (rr=0.33; P=0.0001).

2.3. Comorbidity and PSD

We examined the influence of arterial hypertension, chronic ischemic heart disease, diabetes mellitus, and dyslipidemia on PSD. Arterial hypertension was not associated with depression (p>0.05). Patients with chronic ischemic heart disease showed more severe depression at the acute p=0.0217) and chronic (p=0.0052) stroke stages. Diabetes mellitus (type 2) correlated with the PSD severity at 3rd month (p=0.0021). The serum cholesterol level (total cholesterol at mmol/l) also correlated with severity of depression at chronic stroke stage (rr=0.27; p=0.0105). Total triglyceride level was not associated with PSD. The initial fibrinogen level was associated with more severe depression at acute (rr=20; p=0.0361) and chronic stroke stage (rr=18, p=0.0421).

DISCUSSION

PSD is one of the leading and treatable complications after acute stroke. More than 1/3 of patients develop PSD during the first three months after IS (Wang Z, et al. 2019 [3]). It severely impacts the quality of life and functional outcome of post-stroke survivors [2, 3, 4]. Although a treatable and important condition, PSD is underestimated and even ignored in everyday practice due to different subjective and objective factors. Some of its signs and symptoms (sleep disorders, pain, energy decline, losing interests, hobbies, pleasure, memory decline, etc.) are often attributed to the stroke itself [3]. Moreover, there is an enormous group of patients with borderline depression (according to our study, 1/4 to nearly 1/3 of all post-stroke survivors develop subclinical depression – HDRS 7-14p.), which also should be assessed, followed up, prevented and even treated. However, PSD in our patients was most often mild in severity, with very few patients with severe depressive signs. The majority of depressive subjects in our study complain of somatic signs (pain, cognitive decline, sleep disorders, etc.) with or without psychological ones (e.g. loss of energy, losing interest in life, etc.).

There are many factors that have effects on PSD [5]. They can be divided into stroke-related factors, comorbidity and inflammation–associated factors and factors associated with the personality of the patient (sex, age, lone-

liness, family history for depression, history for depression or other psychiatric disorders, etc.) [3, 4, 5, 6].

According to our study, the leading PSD risk factors are stroke severity, subcortical distribution of IS, co-existence of leukoaraiosis, ageing, loneliness and some comorbidities associated with increasing of system inflammation and changes in brain neurotransmission and plasticity (diabetes mellitus, chronic ischemic heart disease, dyslipidemia and high initial fibrinogen level). So PSD is not only psychogenic phenomenon (so called reactive depression) but it should be considered as co-existing brain disease with its own specific organic causes and risk factors.

The recent treatment strategy for PSD includes a combination of pharmacological, psychosocial and stroke-focused interventions [4].

CONCLUSION

PSD is one of the main complications of acute IS and should be taught as an independent brain disease due to organic and psychological factors. It should be assessed, prevented and treated as soon as possible.

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