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# Subjective Cognitive Complaints in Distant Phase After Stroke – Preliminary Study

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## Abstract

**Introduction.** Subjective cognitive decline (SCD) is often reported by healthy individuals and by different clinical groups. Current data do not clearly show the relationship between SCD and cognitive functioning, but the predictors of SCD are: age, depression and sociodemographic factors. Inconclusive data also applies to people suffering from stroke in the distal post-stroke phase. Correct identification of the causes of SCD will help to take adequate forms of psychological therapy (neuropsychological rehabilitation and/or psychotherapy). **Methodology.** The aims of research was to compare the intensity and structure of SCD as well as their determinants in healthy persons and those after stroke. In our study 193 adults participated: 118 osób without brain pathology and 75 patients who suffered stroke 2–3 years earlier. DEX-S and ProCog as methods of evaluation of SCD and cognitive assessment techniques: the Montreal Cognitive Assessment Scale (MoCA), WAIS-R subtests and intensity of depressive mood (Geriatric Depression Scale – 15) were used in the study. **Results.** Patients after stroke were characterized by more severe SCD than healthy people. People in both groups likewise (low) rated one's own long-term memory, general cognitive and executive function. It has also been shown that people after stroke have significantly lower cognitive competencies compared to healthy individuals, but similar (low) level of depressive mood. The later part of the analysis showed that some complaints may be determined by depressive mood, others are due to the interaction of cognitive deficits and depressive mood. **Conclusion.** Our results confirm the need for proper qualifications complaints in patients after stroke (in distant post-stroke phase).

**Keywords:** subjective cognitive decline, stroke, cognitive functioning, depression

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## 1. Introduction

Subjective cognitive decline (SCD) is the belief about weakening/deterioration of own cognitive abilities compared to previous possibilities and to peers [1, 2]. SCD may be of general character or affect some areas of functioning, for example different memory aspects (SMD – *subjective memory decline)* [3], language (SLD – *subjective language deficits*) [4], executive functions (SEFD – *subjective executive function deficits*) [5, 6], and others. Current reports in this field of studies focus on issues of SCD conditions [7] and their predictive value (e.g., in MCI – *mild cognitive impairment* development or dementia) [8–14]. The SCD classification is i.a. difficult for the reason that they are frequent both in healthy people and people with brain dysfunctions. The data concerning the relation between real cognitive functioning and SCD are not conclusive, therefore other individual and social factors are included. A significant determinants are: age [2], depressive mood [15], higher level of anxiety or neuroticism [16, 17], sex (female), low education level [13], economic and environmental factors [18]. A significant factors contributing to reporting different SCD are brain diseases of different aetiologies. In comparison to healthy people, an increased SCD is the feature of people with Parkinson disease [19], after head injuries [20, 21], with cardiovascular diseases [22], after strokes [23, 24], in multiple sclerosis [25, 26] and in early stages of Alzheimer disease [27]. Similarly to healthy, in clinic groups the connection between increased SCD and depression, anxiety, feeling of stress was noted [28]. Few analyses concern people after stroke, including the connection between lateralization of pathology and the profile/intensity SCD. Based on the research overview, van Rijsbergen et al. [29] indicates that patients with different localization/lateralization of stroke did not differ in various type SCD, but they differed from the healthy persons. In the acute stroke stage, the intensification and scope of complaints show some dynamics. Initially, depending on the insight level, a patient focuses attention on physical disability, however within 3–9 months after the stroke there is an increased feeling of deteriorated psychomotor speed, attention, increased fatigue, depression, anxiety [30], and language difficulties [31]. Interestingly, no clear connection was established between intensity of SCD and cognitive ability. Van Rijsbergen et al. [32] indicates the correlation between a higher scope of cognitive deficits (including many domains) and the intensity of SCD, while other authors of the reports emphasize the significance of depression [31] and low feeling of social support [33]. It was also established that these correlations may be revealed only in further stage after the stroke (2–8 years), when an patients encounters difficulties in his everyday life, and these difficulties could not have been suffered during the

treatment and rehabilitations stage [22]. In clinical practice, it is the responsibility of the neuropsychologist to explain what functional aspects the SCD refer to, what is their source and mechanism [34]. The main basis of the diagnostic reasoning is the analysis of the documents, the interview with a patient, his family and psychological diagnosis including neuropsychological assessment [35]. The incorrect classification of SCD exposes the patient to wrong diagnosis and inefficient therapeutic support [36]. On the other hand, lack of SCD in the presence of cognitive deficits proves their unawareness [37]. The current stage of research indicates that the SCD has double character: some of the complaints are mainly determined by emotional factors (SCD *worry*) while the others have relation with real cognitive difficulties (SCD *content*), particularly the subjective executive difficulties, deficits of prospective and recent memory [38]. In relation to commonly found SCD in the healthy people and clinic groups and their complex determinants, the research was undertaken with the aim to compare the intensity and structure of SCD as well as their determinants in healthy persons and those after strokes. The permission of local Bioethics Committee was obtained.

## 2. Methodology

The results of 193 people were included in the analysis. The control group (C; $N$ = 118; 63 women, 55 men) was formed by volunteers, without somatic, psychiatric and neurological diseases. The post-stroke group (PS) included 75 persons (33 women, 42 men), who documented (CT, MRI) the ischemic stroke of the right hemisphere ($N$ = 18), left hemisphere ($N$ = 10) or in both hemispheres ($N$ = 47) from 2 to 3 years before. Since suffering the stroke, these persons have been under the control of the neurologic and neuropsychological clinic, they are independent in their everyday functioning, do not show aphasia, agnosia or behavior disorders. Based on the neuropsychological assessment and the interviews, people with deficits in these areas were excluded [35, 39]. The persons from the C group were (not statistically significant) slightly younger (63.62 ± 12.32) than the persons from PS group (64.75 ± 12.0, $t$ = 1.32 $p$ = 0.09). People with high school education were dominant in each group (C, $N$ = 64, PS, $N$ = 44), while in C group other persons had higher ($N$ = 45) and primary education ($N$ = 9). Respectively, there were patients with primary education ($N$ = 17) and lower number ($N$ = 14) with higher education.

We used the following methods:

A. ProCog questionnaire (*Patient Reported Outcomes in Cognitive Impairment*) [40]. Due to the lack of Polish adaptation of the tools in our study we used the

procedure *backtranslation*. This is 55 items grouped in several scales (combined achieved an overall score – the sum). The questions refer to self-assessment of general cognitive functioning, including attention and thinking (Cognitive Functioning), the long-term memory (Long-term memory), episodic memory (Memory for Events), semantic memory (Semantic Memory), emotional reactions to perceived cognitive difficulties (Affect), a feeling of loss of acquired skills (Skill Lost) and restrictions in social relations associated with cognitive deficits (Social Impact). The test is to choose a response to a Likert scale; standardized scores so that the lower assessment meant no feelings of difficulty, the higher – of greater intensity SCD. The sum of the results is within the range of 0 points (no difficulty) and 220 points (very high sense of cognitive disorders). Due to unequal points in subscales, the raw results and percentages in each scale for each test person were calculated.

B. MoCA (*Montreal Cognitive Assessment Scale*) [41]. The tool is used to evaluate cognitive functions such as short-term memory, visual-spatial, executive functions, language, verbal fluency, attention, naming, abstraction and allopsychic orientation.

C. DEX-S (*Dysexecutive Questionnaire*/Self) for self-description of the severity of the executive difficulties in everyday life. DEX-S consists of 20 questions relating to behavior involving executive functions. The tested shall respond to them by selecting a point on the Likert scale from 0 (never) to 4 (very often). The maximum score (range 0–80) points to ones perception of deficits in the realization of tasks involving executive function [42].

D. Digits forward and backward – subtests from Polish version WAIS (WAIS-PL), indicating the level of direct and working memory, and the subtest Vocabulary indicating the level of abstract thinking, linguistic competences and semantic memory [43].

e. GDS – 15 (*Geriatric Depression Scale – short form*) [44].

## 3. Results

The quantitative analysis was performed with IBM SPSS Statistics version 22 software. The parametric tests were used due to a normal distribution of variables (Shapiro–Wilk test). The intensity of SCD in C and PS groups was compared. The intergroup differences concern the results in all subscales except from the self-assessment of long-term

Table 1: C group and PS group: subjective cognitive complaints (ProCog i DEX-S).

| Scales (ProCog and DEX-S) | PS group M ± SD | Control group M ± SD | t-Student test (p) | Effect size (d'Cohen) |
|---|---|---|---|---|
| ProCog – sum$^{p}$ | 25.2 ± 19.9 | 16.03 ± 10.3 | 4.04 (0.001)*** | 0.580 |
| Cognitive functioning | 30.8 ± 17.4 | 26.8 ± 13.2 | 1.69 (0.07) | – |
| Affect | 31.2 ± 24.1 | 17.5 ± 17.9 | 4.23 (0.001)*** | 0.645 |
| Skill lost | 19.1 ± 20.5 | 6.1 ± 7.7 | 5.53 (0.001)*** | 0.837 |
| Semantic memory | 33.9 ± 21.7 | 23.4 ± 15.9 | 3.59 (0.001)*** | 0.548 |
| Memory for events | 21.9 ± 19.9 | 14.02 ± 12.06 | 3.08 (0.003)** | 0.478 |
| Social impact | 16.8 ± 18.05 | 8.6 ± 8.2 | 3.75 (0.001)*** | 0.587 |
| Long memory | 29.7 ± 30.7 | 34.3 ± 27.3 | –1.07 (0.28) | – |
| DEX-S$^{s}$ | 23.2 ± 16.1 | 19.4 ± 11.5 | 1.76 (0.08) | – |

Note: mean – M, standard deviation – SD, between groups comparisons – *t*-Student test, effect size - d-Cohen test, p – percentage indicators; r – raw results, ***$p \leq 0.001$; **$p \leq 0.01$.

memory and cognitive functioning (subtests of ProCog) and executive functions (DEX-S) (Table 1).

The results indicate a higher intensity of SCD in PS group (effect size: average and high). PS group assess poorly their semantic memory and indicate the anxiety related with experiencing difficulty. In turn, the C group also indicates the feeling of difficulty in semantic memory, but the level of anxiety related to own cognitive ability is low. Table 2 includes the results of cognitive tests and GDS-15. PS group obtained lowered results than the C group in: semantic memory (Vocabulary), direct memory (digits forward), working memory and attention (digits backward) subtests of WAIS, and in MoCA sum and subtests (effect size – medium and high) except for in the naming (MoCA), and GDS-15.

The results were analyzed with taking into consideration the lateralization of stroke (right hemisphere, left hemisphere, pathology of both hemispheres). No differences were noted between the groups in ProCog and DEX-S results, however higher SCD were consequently noted (insignificant) among persons with pathology of the right hemisphere.

In order to define the determinants of SCD intensity, the linear regression analysis of multivariate stepwise was applied. The factor analysis was used at the beginning in order to reduce the predictors, it allowed to distinguish 3 factors: cognitive (combined factor from the MoCA subscales: visual-spatial, attention, naming, language, abstracting and delayed recall), emotional (result in GDS) and age. These factors were included as predictors in regression analysis. Table 3 shows the data.

Table 2: C group and PS group – cognitive functioning and depression.

| Cognitive functioning and depression | PS group M ± SD | Control group M ± SD | t-Student test (p) | Effect size (d'Cohen) |
|---|---|---|---|---|
| Vocabulary | 29.6 ± 15.4 | 47.6 ± 12.3 | −8.60 (0.001)*** | 1.293 |
| Digits forward | 5.08 ± 1.8 | 6.9 ± 1.9 | −6.43 (0.001)*** | 0.964 |
| Digits backward | 3.8 ± 1.8 | 6.5 ± 2.2 | −9.25 (0.001)*** | 1.339 |
| MoCA sum | 22.5 ± 4.4 | 27.6 ± 2.4 | −9.18 (0.001)*** | 1.431 |
| MoCA visual-spatial | 4.03 ± 1.2 | 4.7 ± 0.5 | −4.80 (0.001)*** | 0.760 |
| MoCA naming | 2.9 ± 0.4 | 2.9 ± 0.2 | −1.75 (0.08) | – |
| MoCA attention | 4.4 ± 1.4 | 5.34 ± 1.13 | −4.60 (0.001)*** | 0.715 |
| MoCA language | 2.2 ± 0.8 | 2.6 ± 0.6 | −3.41 (0.001)*** | 0.490 |
| MoCA abstract thinking | 1.2 ± 0.8 | 1.9 ± 0.4 | −6.74 (0.001)*** | 1.073 |
| MoCA delayed recall | 1.7 ± 1.7 | 3.8 ± 1.5 | −8.62 (0.001)*** | 1.297 |
| MoCA orientation | 5.8 ± 0.6 | 5.99 ± 0.09 | −1.19 (0.05)* | 0.355 |
| GDS-15 | 4.6 ± 3.9 | 4.2 ± 2.7 | 0.79 (0.43) | – |

Note: mean – M, standard deviation – SD, comparison of the results (*t*-Student test), effect size d-Cohen test, ***$p \leq 0.001$, **$p \leq 0.01$, *$p \leq 0.05$.

The intensity of SCD is mostly determined by the level of the depressive mood and/or cognitive functioning. These variables, in isolation or in interaction, explain from 11% to 25% variations of the results in ProCog subtests. A higher level of depressive mood favors the subjective general cognitive decline and executive dysfunctions (DEX-S). The higher level of depressive mood along with diminished cognitive abilities explained the increased feeling of: anxiety, semantic memory disorders, negative impact of the cognitive deficits on social functioning and loss of previous skills. The variables included in regression do not explain the feeling of long-term memory disorders.

## 4. Conclusion

The tendency to negative self-assessment of own cognitive functioning is more characteristic for persons after strokes rather than for healthy persons [45]. In patients' SCD profile there is a dominant feeling of difficulty: in coping with the situations involving cognitive functions, semantic memory, reminding old facts and knowledge and anxiety related with conviction of cognitive difficulties. In turn, the healthy persons complain about difficulties in reminding old facts and they have feeling of general weakening of cognitive competences, without feeling of anxiety. The subjective long-term memory

Table 3: Determinants of SCD: linear regression analysis of multivariate stepwise.

| SCD: scales ProCog and DEX-S | | age | Cognitive factor | Emotional factor (depressive mood) | Adjusted $R^2$ | F (p) |
|---|---|---|---|---|---|---|
| Cognitive functioning | | −0.09 | −0.026 | 0.34 (0.001)*** | 0.11 | 24.7 (0.001)*** |
| Affect | 1 step | 0.10 | −0.21 | 0.35 (0.001)*** | 0.12 | 26.7 (0.001)*** |
| | 2 step | 0.02 | −0.22 (0.001)*** | 0.32 (0.001)*** | 0.17 | 19.24 (0.001)*** |
| Skill lost | 1 step | 0.07 | −0.39 (0.001)*** | 0.29 | 0.15 | 36.12 (0.001)*** |
| | 2 step | 0.08 | 0.29 (0.001)*** | 0.27 (0.001)*** | 0.25 | 30.82 (0.001)*** |
| Semantic memory | 1 step | 0.17 | −0.17 | 0.35 (0.001)*** | 0.12 | 26.38 (0.001)*** |
| | 2 step | 0.11 | −0.17 (0.01)** | 0.32 (0.001)*** | 0.15 | 16.91 (0.001)*** |
| Memory for events | 1 step | 0.05 | −0.13 | 0.36 (0.001)*** | 0.13 | 27.54 (0.001)*** |
| | 2 step | −0.03 | −0.14 (0.04)* | 0.34 (0.001)*** | 0.14 | 16.0 (0.001)*** |
| Social impact | 1 step | 0.09 | −0.18 | 0.34 (0.001)*** | 0.12 | 25.48 (0.001)*** |
| | 2 step | −0.01 | −0.18 (0.008)** | 0.32 (0.001)*** | 0.14 | 16.75 (0.001)*** |
| Long-term memory | | −0.05 | 0.009 | −0.002 | −0.001 | 0.91 (0.44) |
| DEX-S | | 0.009 | −0.09 | 0.38 (0.001)*** | 0.14 | 33.07 (0.001)*** |

Note: ***$p \leq 0.001$; **$p \leq 0.01$, *$p \leq 0.05$.

and general cognitive functioning deficits seems to be independent of the group the persons belong to. The data are similar with other data which indicate that the beliefs about general cognitive deficits, memory, executive functions and attention deficits are most often found in the complaint structure of healthy and persons after brain pathology. In the patient group, there is a clear anxiety connected with feeling of having difficulties. A factor explaining a slight increase of the complaints (e.g., to 33% of the possible result in semantic memory scale in ProCog) may be the time, which has passed since brain damage (in this case 2–3 years) and general good cognitive functioning of the patients. Some data indicate, that the SCD intensifying with time may be the symptom of developing vascular dementia [22]. The real cognitive abilities are significantly lower in the PS group. It is in line with many other reports indicating long-term neuropsychological effects of strokes. It is also known that there are connections between depression and cognitive functioning and between the depression and SCD

[46], but most of the data (non-conclusive) stems from the research of people with depression or seniors with/without dementia [47]. The newest data [32] confirm that within 3 months after the stroke, SCD are correlated to a higher extent with cognitive deficits than at an early stage. Directly after a brain injury, depression may hide the cognitive dysfunctions and explain SCD, however in the long-term stage there is a clear connection of emotional and/or cognitive factors and SCD [48], which is also shown in own research. Therefore, the SCD, depending on their type, may have different determinants. The results should be considered very interesting. For example, the subjective executive deficits and subjective cognitive weakening would rather have the connection with negative mood, however, the complaints concerning some areas (e.g., episodic memory) are connected with the decline of cognitive functions and higher severity of the depressive mood. This observation is according to the opinions of other authors, who underline the necessity of extending the self-report questionnaires with the positions referring to the narrow cognitive aspects (reading, counting, writing etc.), because the SCD in this areas may be connected with the real neuropsychological problems [49]. Summing up, it was indicated that in distant stage after the stroke, the patients report the SCD despite the fact that they are independent persons in their everyday functioning. Due to different conditioning of the complaints, the appropriate forms of neuropsychological diagnosis and rehabilitation should be chosen (neuropsychological rehabilitation and/or psychotherapy).

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