DOI: 10.1590/2317-1782/20152015005

Original Article

Artigo Original

Depressive signs and cognitive performance in patients with a right hemisphere stroke

Sinais depressivos e desempenho cognitivo em pacientes com lesão de hemisfério direito

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Keywords

Stroke
Right-hemisphere
Depression
Cognition
Neuropsychological Tests

Descritores

Acidente Vascular Cerebral
Hemisfério Direito
Depressão
Cognição
Testes Neuropsicológicos

ABSTRACT

Purpose: This study investigated the influence of suggestive signs of depression (SSD) in right-hemisphere brain-damaged (RHD) patients following a stroke on their cognitive performance measured by a brief neuropsychological assessment battery. **Methods:** Forty-two adults with RHD after a single episode of stroke and 84 matched controls participated in this study. They were assessed by means of the Geriatric Depression Scale and by Brief Neuropsychological Assessment Battery NEUPSILIN. **Results:** Almost half of the patients showed SSD. The RHD group with SSD (RHD+) showed poorer performance in at least one task among all evaluated cognitive domains (concentrated attention, visual perception, working memory, episodic verbal memory and semantic memory, auditory and written language, constructional praxia and verbal fluency). **Conclusion:** The association of depression and RHD seems to enhance the occurrence and the severity of cognitive déficits. A brief neuropsychological assessment can be useful to identify cognitive impairment caused by this neuropsychiatric disorder.

RESUMO

Objetivo: Investigar a influência de sinais sugestivos de depressão (SSD) em pacientes com acidente vascular cerebral (AVC) de hemisfério direito (HD) no desempenho em um instrumento de avaliação neuropsicológica breve. **Métodos:** Participaram 42 adultos pós-episódio único de lesão cerebrovascular de HD (LHD) e 84 controles emparelhados. Administraram-se a Escala de Depressão Geriátrica GDS-15 e o Instrumento de Avaliação Neuropsicológica Breve NEUPSILIN. **Resultados:** Aproximadamente metade da amostra de pacientes apresentou SSD. O grupo com LHD com SSD (LHD+) obteve desempenho inferior em no mínimo uma tarefa dos domínios cognitivos avaliados (atenção concentrada, percepção visual, memória de trabalho, memória verbal episódica e memória semântica; linguagem oral e escrita; praxia construtiva e fluência verbal). **Conclusão**: A associação de depressão e LHD parece aumentar a extensão dos déficits cognitivos, sendo que uma avaliação neuropsicológica breve pode ser útil na identificação de alterações cognitivas neste quadro neuropsiquiátrico.

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Received: 01/19/2015

Accepted: 03/31/2015

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Conflict of interests: nothing to declare.

INTRODUCTION

Depression is considered to be one of the most common neuropsychiatric consequences following a stroke, reaching approximately a third of the patients. Besides generating a negative impact on functional rehabilitation, it represents one of the most prominent causes to increase mortality in this population[1]. Depression has been detected in between 20 and 60% of stroke patients, this rate ranging in accordance with the evaluation criteria established (by means of scales or semi-structured interviews, for instance), as well as to the population investigated (such as ischemic and/or hemorrhagic, chronic and/or acute stroke)[2] and lesion location. Patients with lesions involving left-hemisphere prefrontal or basal ganglia structures had a higher frequency of depressive disorder (75%) than other left-hemisphere lesions (8%) or those with right-hemisphere lesions (29%)[3]. However, studies investigating the relationship between injury localization and depression are contradictory[4,5]. Therefore, further investigations are necessary, specially considering the cognitive issues in right-hemisphere.

Despite the high incidence of depressive symptoms associated with the stroke framework, it seems that literature does not have enough studies with converging evidences concerning the frequency and the relationship between this psychiatric disease and stroke aspects, such as the lesion side and associated cognitive impairment[6]. More specifically, when comparing what has been found about cognitive impairment following a stroke in isolation[7], as well as cognitive changes following depression cases[8], little is known about the relationship between depression, unilateral right-hemisphere stroke and cognitive deficits derived by the association of these aspects. Thus, there is an important lack of knowledge on the comprehension of cognitive impairment associated with stroke and depression versus hemispheric specializations.

Although the left-hemisphere has been related to higher incidence of depression, the right-hemisphere seems to be more related to emotions in general, being pointed in the literature as the main neurobiological correlate to facial emotions and prosodic processing[9]. Therefore, vascular lesions in this hemisphere may impair the patient's mental and emotional state, as well as negatively affecting motivation, comprehension and prosodic emotional, metaphoric and humor expression[10].

Some few existing studies[6,11-13] propose to discuss the relationship between cognitive dysfunctions and post-stroke depression, but not followed by the association's investigation of the neurocognitive deficits of this comorbidity with the affected hemisphere. Among those studies, the research conducted by Alexopoulos[11], for instance, emphasizes that lesions in the frontolimbic and frontoestriatic networks are associated withwith executive functions deficits; however, the contributions of each hemisphere are not considered in this process. According to Sneed and Culang-Reinlieb[12], post-stroke depression is also associated withwith executive dysfunction, although independently of lesion type. Brodaty et al.[13] identified that apathy following stroke is linked to low performance in tasks measuring concentrated attention, working memory and processing speed. Marazziti et al.[6], on their hand, observed the incidence of attention, working memory and executive functions impairment, including cognitive inhibition, planning and problem solution, suggesting a strong relationship between low performance in cognitive tasks and positive signs of depression associated with stroke, not necessarily unilateral, though.

As for the characterization of cognitive processing in this neuropsychiatric framework of post-stroke depression, besides the lack of studies about several cognitive functions, some limitations may be pointed out regarding assessment methods. The researches had, in general, administrated screenings such as the Mini Mental State Exam – MMSE[14], some tasks of expanded batteries[15] or entire expanded batteries[13]. Therefore, seems to be an even larger gap in the literature on the relationship between stroke, suggestive signs of depression (SSD) and hemispheric specialization with the processing of several cognitive functions measured by brief neuropsychological batteries.

Taking into account the importance of brief batteries to the neuropsychological examination in contexts where initial diagnosis is necessary in a shorter time[16], associated with the scarceness of empirical evidence relating unilateral stroke, post-stroke depression and cognitive impairment following this comorbidity, the present article aims to investigate the incidence of SSD in a sample of right-hemisphere brain-damaged (RHD) adults and verify the influence of these signs and symptoms in the processing of several cognitive functions — temporo-spacial orientation, attention, perception, memory, language, arithmetical skills, praxias and executive functions.

METHOD

Participants

In this study a total sample of 126 adults participated, divided in two groups: clinical (with two subgroups) and a control group.

RHD clinical group

A total of 56 participants with a single incident of unilateral right-hemisphere stroke, with an ischemic or hemorrhagic etiology, were recruited. They signed an informed consent and underwent a brief neuropsychological assessment. Participants should be native Brazilians with no history of abuse of psychoactive substances, neither have presented neuropsychological or psychiatric self-reported disorders or diagnosed depression. Moreover, they could not have received previous neuropsychological and/or speech rehabilitation treatment nor psychiatric of psychotherapeutic treatment (due to previous psychiatric diagnosis). From the sample 14 individuals were excluded due to ethylic history (n=3), previous stroke(s) (n=2), left handedness dominance (n=2), illiteracy (n=3) and not conclusion of evaluation (n=4). Thus, the final sample of the study was composed by 42 RHD adults. According to the presence or not of SSD, the patients were subdivided in two clinical groups:

RHD patients with SSD (RHD+), between 5 and 15 points in 15 Item Geriatric Depression Scale (GDS-15), and with no SSD (RHD-), between 0 and 4 points.

Control Group

This group was composed by 84 neurologically preserved participants, paired with brain-damaged patients according to age criteria, years of formal education, reading and writing habits and gender. The control group formation should observe the same inclusion criteria of brain-damaged patients, including absence of the diagnosis of a cerebrovascular disease, no signs of cognitive impairment measurement by the MMSE (adapted by Chaves and Izquierdo[17]) and non-occurrence of SSD.

Procedures and instruments

It is important to state that all ethical procedures have been respected, with the guarantee of voluntary participation in the study, and under the approval of a Research Committee on Ethics of a higher education institution (protocol number 10/05134). The evaluations were developed in individual sessions with approximated duration of one hour and a half. Participants answered to sociodemographic characterization and general health conditions questionnaire.

In order to verify the presence of SSD, participants were administered GDS-15 (adapted by Almeida and Almeida[18], in order to obtain same measure in adult and elderly. The validity of the GDS-15 for other age groups, like younger and adults, has already been reported in the literature[19]. The cognitive abilities were assessed by the Instrument of Brief Neuropsychological Assessment (NEUPSLIN)[20], which is an instrument with a brief administration time aiming at verifying preserved and impaired abilities in components of eight different cognitive functions, giving support to a neuropsychological diagnosis. It includes the following tasks, all of them administered in this study: Concentrated attention (inverted counting and digit repetition); Visual perception (verification of similarities and differences of lines, hemineglect, faces perception and recognition); Working memory (ascendant ordering of digits and auditory span of words in sentences); Episodic-semantic verbal memory (delayed and immediate recall and word list recognition); Long-term semantic memory; Short-term visual memory (figures); Prospective memory; Arithmetic abilities (simple addition, subtraction, multiplication and division calculation); Oral language (objects and figures' naming, words and non-words' repetition, automatic language, inference comprehension and processing); Written language (reading aloud of words and non-words, written comprehension, spontaneous writing, copying, words and non-words dictation); Apraxia (ideomotor, constructive and reflexive), and Executive functions (simple problems' resolution and orthographic verbal fluency – letter F).

Data analysis

Initially an analysis of frequency was performed to verify the incidence of SSD and its intensity in RHD patients as well as to subdivide the RHD+ and RHD- clinical groups. The dependent variables presented a normal distribution in the Kolmogorov-Smirnov Test (p>0.05). In order to compare the performance of RHD+, RHD- and the Clinical Group, a One-way ANOVA analysis was used, with a Bonferroni *post hoc* test. The incidence of hemineglect between RHD+ and RHD- groups was compared through a χ^2 analysis.

RESULTS

The groups' sociodemographic and clinical characteristics are depicted in Table 1. Regarding the characterization variables, the groups only present significant differences in the GDS-15 score (RHD+ > RHD- > Controls, p≤0.001, indicating that pairing was correctly done. Moreover, a predominance of cortical lesion was observed in the sample.

The incidence of SSD in RHD patients was of 47.62%. As for the symptoms intensity in the RHD+ group through the GDS-15 scores, 40% of the cases presented a mild SSD level, 35% moderate and 25% severe cases. Means and standard deviations of NEUPSILIN tasks are described in Table 2.

The *post hoc* analysis identified a significant lower performance of the RHD+ group as compared to the Control Group's performance in at least one of the subtests of all cognitive domains assessed. Regarding the tasks of Concentrated attention, Visual perception, Working memory, Auditory and Written language, in which differences were observed between RHD+ and RHD- groups, the RHD+ also presented lower scores.

Table 1. Sociodemographic and Clinical Characteristics

	RHD+	RHD-	Controls	F value	p-value
	Mean (SD)	Mean (SD)	Mean (SD)		
Gender (Male/Female)	8 M / 12 F	12 M / 10 F	21 M / 63 F	-	0.347
Age (years)	57.35 (11.11)	59.14 (12.59)	58.35 (12.08)	0.12	0.891
Education (years)	7.45 (4.25)	9.36 (5.65)	9.02 (5.69)	0.79	0.456
Frequency reading and writing habits	9.80 (6.46)	11.05 (5.51)	10.90 (5.03)	0.38	0.683
GDS-15	8.10 (2.59)	3.00 (1.57)	1.88 (1.26)	123.73	<0.001
Post-lesion time (months)	22.06 (24.88)	17.68 (24.46)	–	1.03	0.569
General locus of lesion (Cortical/Subcortical/Mixed/NR)	9 / 2 / 8 / 1	12 / 9 / 1 / 0	–	–	–
Hemineglect (Yes/No)	5 Y / 15 N	4 Y / 18 N	–	–	0.714

df (degrees of freedom) = 2

Caption: SD = standard deviation; GDS-15 = Geriatric Depression Scale of 15 points; NR = Not reported

Table 2. Groups' Performances in NEUPSILIN Tasks

Variables	RHD+	RHD-	Controls	F value	p-value	Post hoc
	Mean (SD)	Mean (SD)	Mean (SD)			
Attention						
Inverted counting	16.7 (6.39)	18.86 (4.25)	17.87 (5.22)	0.88	0.416	
Inverted counting (time)	42.68 (26.92)	28.53 (15.06)	26.11 (14.43)	7.61	0.001	RHD+ > RHD-* RHD+ > Controls***
Digit repetition	2.6 (1.57)	2.45 (1.82)	3.17 (1.93)	1.71	0.185	
Perception						
Line verification	4.65 (1.53)	5.24 (1.04)	5.26 (1.04)	2.42	0.094	
Visual neglect	0.85 (0.37)	0.86 (0.35)	1 (0)	6.86	0.001	RHD+ < RHD-** RHD- < Controls*
Face perception	2.1 (0.64)	2.05 (0.72)	2.39 (0.64)	3.41	0.036	
Face recognition	1.55 (0.51)	1.82 (0.39)	1.88 (0.33)	6.39	0.002	RHD+ < Controls**
Memory						
Working memory (ADO)	3.95 (2.7)	6.27 (2.47)	6.38 (2.37)	8.21	0	RHD+ < RHD-** RHD+ < Controls***
Working memory (ASWS)	9.55 (5.65)	11.91 (4.94)	13.5 (6.13)	3.84	0.024	RHBD+ < Controls*
Verbal memory (immediate recall)	3.65 (1.5)	3.91 (1.27)	4.64 (1.37)	5.65	0.005	RHD+ < Controls**
Verbal memory (delayed speech)	0.8 (1.32)	1.14 (1.39)	1.99 (1.89)	4.98	0.008	RHD+ < Controls*
Verbal memory (recognition)	10.35 (1.5)	11.45 (1.57)	12.45 (2.39)	8.44	0	RHD+ < Controls***
Long-termsemanticmemory	4.1 (0.91)	4.55 (0.91)	4.67 (0.63)	4.84	0.009	RHD+ < Controls**
Short-term visual memory	2.35 (0.93)	2.55 (0.74)	2.64 (0.61)	1.47	0.234	
Prospective memory	1.25 (0.72)	1.05 (0.84)	1.39 (0.78)	1.8	0.17	
Arithmetic abilities	4.8 (2.93)	6 (2.54)	6.79 (2.04)	6.37	0.002	RHD+ < Controls**
Language						
Oral naming	3.9 (0.45)	4 (0)	3.99 (0.11)	1.8	0.169	
Repetition	9.55 (0.69)	9.59 (0.67)	9.7 (0.72)	0.5	0.611	
Automatic language	1.8 (0.41)	2 (0)	1.94 (0.24)	3.54	0.032	RHD+ < RHD-*
Oral comprehension	2.7 (0.57)	2.73 (0.55)	2.8 (0.51)	0.36	0.695	
Inference processing	2.3 (0.73)	2.5 (0.6)	2.36 (0.77)	0.44	0.643	
Reading aloud	9.75 (3.26)	11.55 (0.67)	11.46 (0.91)	11.1	0	RHD+ < RHD-*** RHD+ < Controls***
Written comprehension	2.7 (0.57)	2.5 (0.67)	2.82 (0.42)	3.79	0.025	RHD- < Controls*
Spontaneous writing	1.05 (0.76)	1.41 (0.73)	1.56 (0.72)	4.02	0.02	RHD+ < Controls*
Copied writing	1.5 (0.69)	1.68 (0.57)	1.82 (0.42)	3.64	0.029	RHD+ < Controls*
Dictated writing	8.65 (3.08)	10.23 (1.34)	10.04 (2.27)	3.32	0.039	RHD+ < Controls*
Praxias						
Ideomotor	2.95 (0.22)	3 (0)	2.95 (0.21)	0.54	0.582	
Constructive	8.65 (3.7)	10.59 (3.19)	11.2 (2.99)	5.35	0.006	RHD+ < Controls**
Reflexive	1.8 (1.15)	2.09 (1.11)	2.15 (0.92)	1.03	0.362	
Executive functions						
Problem resolution	1.5 (0.61)	1.64 (0.49)	1.64 (0.53)	0.59	0.558	
Verbal fluency (letter F)	8.65 (4.07)	11.36 (4.89)	12.01 (5.01)	3.87	0.023	RHD+ < Controls*

*p≤0.05; **p≤0.01; ***p≤0.001; df (degrees of freedom) = 2

Caption: SD = standard deviation; ADO = Ascendant Digit Ordering; ASWS = Auditory *Span* of Words in Sentences

DISCUSSION

The SSD incidence in RHD patients in this study sample (47.62%) was compatible with indexes levels of depressive symptoms presented in the literature, suggesting that it is definitively a framework commonly found following a stroke. According to the study developed by Kouwenhoven et al.[21], for instance, the index of depressive signs varied from 5 to 54% of the sample. Haq et al.[22] observed depression signs in 28% of their patients. In another study, a varying level of 23 to 60% of patients' post-stroke depression was observed[2]. This variability may be related to the variety of instruments utilized to assess depressive symptoms as well as the inclusion of patients with language difficulties, which limits the comprehension of depression scales and inventories (in rehabilitation, ambulatories or hospitals). Although some studies suggested increased frequency of depression related to the left-hemisphere, mainly associated with left anterior lesions, little focus has been given to the cognitive consequences of SSD in patients with right-hemisphere lesion[5,8]. Thus, there is still a prominent lack of epidemiologic data on depression unilateral following post-stroke.Considering the aim to verify whether there is an influence of post-RHD depression in eight cognitive functions, in a general perspective RHD+ adults presented cognitive changes in at least one subtest in all neuropsychological domains assessed. These findings corroborate the literature which investigates the relationship between post-stroke depression and cognitive performance, even when they do not consider the isolated contribution of each cerebral hemisphere in the execution of the tasks adopted[12]. Despite of the fact that this has not been the main aim of Barker-Collo's[23] study, for instance, RHD patients with a diagnosis of depression presented lower performance in late recall in semantic-verbal memory.

In adults with post-stroke depression deficits are observed in the domains of memory, visual-perception, language, executive functions and attention[23]. In a study conducted by Nys et al.[24], in which patients were evaluated six months after the stroke, changes were observed in abstract reasoning and in verbal memory. Verhoeven et al.[25] assessed patients with depression following both hemorrhagic or ischemic CVA with a battery of neuropsychological tests which included the Token Test, Boston Naming Test, Trail Making Test, Rey Auditory Verbal Learning Test, Doors Test, Benton Facial Recognition Test, Judgment of Line Orientation Test, and Letter Cancelation Task. The authors found changes in linguistic and visuo-perceptive abilities.

Specific late-onset depression is known for generating cognitive impairment which in general encompasses changes in attention, episodic, working and prospective memory, besides deficits in executive components, such as processing speed[26]. Performance in RHD+ patients was lower as compared to other groups in attention and working memory tasks, confirming this profile associated with depression. However, the battery administered in this study contains few executive functions tasks and presents reduced punctuation variability, which could have led it to not to be sufficient to discriminate RHD+ and RHD- patients[27]. It is possible that tasks of problem resolution may have been too simple and that other paradigms of verbal fluency could have been more sensitive to discriminate it, such as free and semantic verbal fluency, as well as fluency tasks with higher duration time, which demand more searching cognitive strategies[28]. Regarding the differences found in written and auditory language, initially unexpected for being more associated with LH specialization, a plausible hypothesis is that they occurred due to comorbidity with an attentional deficit framework. This is the case because tasks used present a high accuracy level in the normative sample, being few errors enough to deficit occurrence.

An important finding in the literature refers to sensory hemineglect as a determining factor to depressive humor in stroke patients[25]. In the sample of the present study, although both groups include patients with hemiplegia, the distribution of the occurrence of this syndrome did not differentiate between them, which seems not to directly justify the differences in cognitive performance found between RHD+ and RHD- groups.

Although the performance in the brief neuropsychological battery has discriminated two groups (RHD+ and RHD-) in different cognitive functions, it is important to highlight that the current study presents some limitations, such as assessing SSD with a single scale. Such limitation is justifiable since that GDS-15 presents good sensitivity and specificity when compared to clinical interviews based on the DSM-IV criteria[29]. Moreover, the cognitive performance was measured with a brief neuropsychological exam which did not encompass all cognitive functions, such as cognitive flexibility. However, it is noticeable that the majority of the studies which consider the post-stroke depressive signs adopt evaluations with the MMSE and the Barthel Index to assess altered cognitive functions[30]. However, the use of screenings instruments seems to be insufficient to assess the cognitive functioning of these patients in a broader way. Such deficiencies are frequent and may cause a significant impact both to the patients and to their relatives. Furthermore, participants with left-hemisphere damage were not included in this study in order to control the effect of the local brain injury as well as other variables have not been investigated that could influence cognitive performance in addition to the occurrence of SSD (specific injury localization and socioeconomic status, for example). Age and education are related to cognitive performance, and may have an interaction effect with depressive symptoms. However, the groups of this study had no significant differences in these variables.

In this way, as a follow up study, we suggest a complementary neuropsychological battery with specific tasks, validated in the literature to examine executive functions, both with formal and ecological tasks. It is important to state that, despite the limitations cited above, the brief neuropsychological battery adopted seems to be relevant applicability to differentiate RHD+ and RHD- patients, demonstrating its contribution to evaluations with a limited time in clinical routines of cerebrovascular diseases and neuropsychiatric frameworks services. Moreover, the inclusion and comparison to other clinical samples in future studies, such as LHD patients, may promote a better understanding of the impact of SSD in the cognitive performance in relation to hemispheric specializations. Other aspects that should be investigated are the relationship between functional capacity and social support received and the impact on the severity of depression and cognitive functioning in post-stroke patients.

CRO, KCP, LFC and GG contributed to data collection, data analysis and writing; IILA contributed to writing; RPF contributed to data analysis and writing.

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