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Cognitive deficit and aphasia – a challenging diagnosis

Déficit cognitivo e afasia – um diagnóstico desafiador

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One of the greatest challenges in cognitive neurology is to determine severity of cognitive impairment in patients with aphasia. Most of the cognitive evaluation is performed through language assessment; lesions causing aphasia can be spread in some different brain areas by committing different networks and, consequently, different cognitive domains. Otherwise, verbal based evaluations could show a false positive impairment.

One of the vascular cognitive impairment (VCI) criteria classified it into vascular mild cognitive impairment and vascular dementia, based on functional impairment and number of evaluated cognitive domains. The neuropsychological evaluation must include memory, visuospatial, language, and executive domains[1]. The most recent diagnostic criteria divides VCI into mild cognitive disorder and dementia or major cognitive disorder; there is a recommendation to evaluate praxis-gnosis-body schema, and social cognition, besides previous cognitive domains[2]. In both of them there are no concerns about cognitive evaluation in aphasic patients, which could be unsuitable in most studies.

Studies in tertiary outpatient clinics report a prevalence of vascular dementia between 24.9 and 32.25%; and among presenile dementia there was a prevalence of 36.9%[3,4,5]. There are a few considerations regarding aphasia in these studies. In a prospective study, that have also included aphasics, frequency of VCI was 16.8% in 12-month follow-up[6].

In the majority of the studies, aphasic patients have shown poor performance in attention, executive functions, working memory, and short-term memory[7,8,9].

The study published in this number of Arq Neuropsiquiatr tries to fulfill a gap in this issue in Brazil, characterizing cognitive deficit in a sample of first-stroke patients. Bonini and Radanovic have evaluated 47 stroke patients without depression (non-aphasics: left hemisphere lesion: 17, right hemisphere lesion: 9; and 21 aphasics) with a comprehensive neuropsychological battery; functional activities and quality of life were measured, as well. Aphasics presented a poorer performance on digit span, verbal and visual memory, constructional praxis recall, clock design test, and phonemic verbal fluency. Quality of life was better in right hemisphere lesion and non-aphasic patients[10].

Aphasia severity correlated with scores in the Trail Making Test (TMT) part B, Digit Span forwards and backwards, and Gesture Praxis in this study. In another report, only attention was correlated with aphasia severity[9].

Although this study has limitations as a small number of patients with heterogeneous vascular lesions, it is needed to highlight the importance of the development of specific batteries for evaluating cognition in aphasic patients.

Difficulties on interpretation of cognitive deficit in aphasics are relatively common, according to different lesioned topographies, involvement of cortical and subcortical areas, and white matter tracts, near common language areas[11].

More studies must be performed in Brazil to evaluate cognition in aphasics, using more suitable tests for this type of patients.

References

1. Gorelick PB, Scuteri A, Black SE, Decarli C, Greenberg SM, Iadecola C et al. Vascular contributions to cognitive impairment and dementia: a statement for healthcare professionals from the American Heart Association/American Stroke Association. Stroke. 2011;42(9):2672-713. doi:10.1161/STR.0b013e3182299496
2. Sachdev P, Kalaria R, O'Brien J, Skoog I, Alladi S. Black SE et al. Diagnostic criteria for vascular cognitive disorders: a VASCOG statement. Alzheimer Dis Assoc Disord. 2014;28(3):208-18. doi:10.1097/WAD.0000000000000034
3. Silva DW, Damasceno BP. [Dementia in patients of UNICAMP University Hospital]. Arq Neuropsiquiatr. 2002;60(4):996-9. Portuguese. doi:10.1590/S0004-282X2002000600020
4. Vale FA, Miranda SJ. Clinical and demographic features of patients with dementia attended in a tertiary outpatient clinic. Arq Neuropsiquiatr. 2002;60(3A):548-52. doi:10.1590/S0004-282X2002000400006
5. Fujihara S. Brucki SMD, Rocha MSG, Carvalho AA, Piccolo AC. Prevalence of presenile dementia in a tertiary outpatient clinic. Arq Neuropsiquiatr. 2004;62(3A):592-5. doi:10.1590/S0004-282X2004000400005
6. Brucki SMD, Machado MF, Rocha MSG. Vascular Cognitive Impairment (VCI) after non-embolic ischemic stroke during a 12-month follow-up in Brazil. Dement Nuropsychol. 2012;6(3):164-9.
7. Helm-Estabrooks N. Cognition and aphasia: a discussion and a study. J Commun Disord. 2002;35:171-86.
8. Majerus S, Attout L, Artielle MA, Van der Kaa MA. The heterogeneity of verbal short-term memory impairment in aphasia. Neuropsychologia; 2015;11:NSYD1500380. doi:10.1016/j.neuropsychologia.2015.08.010
9. Lee B, Pyun SB. Characteristics of cognitive impairment in patients with post-stroke aphasia. Ann Rehabil Med. 2014;38(6):759-65. doi:10.5535/arm.2014.38.6.759
10. Bonini MV, Radanovic M. Cognitive deficits in post-stroke aphasia. Arq Neuropsiquiatr, 2015;73(10): 840-47 doi:10.1590/0004-282X20150133
11. Catani M, Dell'Acqua F, Bizzi A, Forkel SJ, Wiliams SC, Simmons A et al. Beyond cortical localization in clinic-anatomical correlation. Cortex. 2012;48(10):1262-87. doi:10.1016/j.cortex.2012.07.001