

NEUROPSYCHOLOGY OF ACUTE STROKE

Osman Sinanović

Department of Neurology, University Clinical Center Tuzla, Medical Faculty University of Tuzla, Bosnia and Herzegovina

SUMMARY

Neuropsychology includes both the psychiatric manifestations of neurological illness (primary brain-based disorders) and neurobiology of «idiopathic» psychiatric disorders. Neurological primary brain disorders provoke broad spectrum of brain pathophysiology that cause deficit in human behaviour, and the magnitude of neurobehavioral-related problems is a world wide health concern. Speech disorders of aphasic type, unilateral neglect, anosognosia (deficit disorders), delirium and mood disorders (productive disorders) in urgent neurology, first of all in acute phase of stroke are more frequent disorders than it verified in routine exam, not only in the developed and large neurological departments. Aphasia is common consequence of left hemispheric lesion and most common neuropsychological consequence of stroke, with prevalence of one third of all stroke patients in acute phase although exist reports on greater frequency. Unilateral neglect is a disorder that mostly effects the patient after the lesion of the right hemisphere, mostly caused by a cerebrovascular insult (infarct or haemorrhage affecting a large area – up to two thirds of the right hemisphere), and in general the left-side neglect is the most widespread neuropsychological deficit after the lesion of the right cerebral hemisphere. Reports on the incidence of visual neglect vary and they range from 13 to 85%. Anosognosia is on the second place as neuropsychological syndrome of stroke in right hemisphere, characterized by the denial of the motor, visual or cognitive deficit. This syndrome, defined as denial of hemiparesis or hemianopsia, is a common disorder verified in 17-28% of all patients with acute brain stroke. There are different reports on frequency of delirium in acute stroke, from 24 to 48%, and it is more frequent in hemorrhagic then ischemic stroke. Post stroke depression (PSD) is one of the more frequent consequences on the stroke, and the prevalence of PSD has ranged from 5 to 63% of patients in several cross-sectional studies, peaking three to six months after a stroke.

Key words: aphasia – unilateral neglect – anosognosia – delirium – depression – stroke

* * * *

Introduction

Behavioural neurology is discipline which was revived in the 1960s by Norman Geschwind with his description of the disconnection syndromes (Geschwind 1965, Cummings & Mega 2003). This discipline is cornerstone of neuropsychiatry or clinical neuropsychology which includes both the psychiatric manifestations of neurological illness (primary brain-based disorders) and neurobiology of «idiopathic» psychiatric disorders (Geschwind 1965, Rizzo & Eslinger 2004; Cummings 1995). Neurological primary brain disorders provoke broad spectrum of brain pathophysiology that cause deficit in human behaviour, and the magnitude of neurobehavioral-related problems is a world wide health concern. Although there are recognized specialists in behavioural neurology/neuropsychology, the field is not a conventional discipline but rather a broad sphere of interest to which experts in different disciplines, medical and nonmedical, have made important contributions (Rizzo & Eslinger 2004).

Speech disorders of aphasic type, unilateral neglect, anosognosia (deficit disorders), and delirium and mood disorders (productive disorders) in urgent neurology, first of all in acute phase of stroke are more frequent disorders than it verified in routine exam, not only in the developed and large neurological departments (Sinanović et al. 2006; Sinanović 2007).

Speech disorders. Aphasias may be defined differently, and according to most accepted neurological

or/and neuropsychological definition, aphasia is loss or impairment of linguistic communication produced by brain dysfunction. It can be manifested by impairment of all language modalities – abnormalities of spontaneous verbal output, disturbances of comprehension of spoken or written language, repetition, naming, reading and writing. For practical use classification of aphasias according to fluency, comprehension and abilities of naming it seems to be most suitable (nonfluent aphasias: Broca's, transcortical motor, global and mixed transcortical aphasia; fluent aphasias: anomic, conduction, Wernicke's, transcortical sensory, subcortical aphasia). Aphasia is common consequence of left hemispheric lesion and most common neuropsychological consequence of stroke, with prevalence of one third of all stroke patients in acute phase although exist reports on greater frequency. Spontaneous recovery is most remarkable in first three months after stroke onset (Sinanović et al. 2006; Sinanović 2007; Sinanović 2005; Pederson et al. 1995; Laska et al. 2001; Godefroy et al. 2002; Engelter et al. 2006).

Unilateral neglect. Unilateral neglect refers to inattention to half the universe, with hemineglect contralateral to the brain lesion. Hemineglect means the neglect of sensor stimuli (visual, auditory, tactile and olfactory) coming from the contralateral side in relation to cerebral lesion, which means the loss of attention or responses to objects or people that are situated on the opposite side to the cerebral lesion, i.e. it implies the neglect of the half of the visual field on the contralateral

side in relation to the cerebral lesion (Cummings 1995; Cummings & Mega 2003). It is a disorder that mostly effects the patient after the lesion of the right hemisphere, and in general the left-side neglect is the most widespread neuropsychological deficit after the lesion of the right cerebral hemisphere (Jehkonen 2002). Reports on the incidence of visual neglect vary and they range from 13 to 85% (Jehkonen 2002; Kerkhoff 2001; Sinanović & Vidović 2004; Sinanović & Vidović 2005; Sinanović 2007; Stone et al. 1993; Parton et al. 2004; Vidović et al. 2004; Pederson et al. 1996). Visual neglect is more noticeable and lasting after the lesion of the right hemisphere than the lesion of the left hemisphere, and it is mostly caused by a cerebrovascular insult (infarct or haemorrhage affecting a large area – up to two thirds of the right hemisphere) (Jehkonen 2002; Sinanović 2007).

Neglect is characterized by the loss of orientation, perception or reaction to the stimuli which are located predominantly on the opposite side to the cerebral lesion, and the developed disorder cannot be explained by a sensor or motoric deficit (Sinanović et al. 2006). Different aspects of space may be neglected without the inclusion of any other: the space of the figure of the patient; the space directly around the body of the patient; distant space or the representation of the space as such (Stone et al. 1993).

Anosognosia. Anosognosia is defined as the loss of awareness of motoric visual or cognitive inability of patients with neurological disorders. It may occur independently, at verbal and non-verbal behavioural level, which, for example, means that the patient, who verbally reacts to his or her hemiplegia, may accept to stay in bed, but another patient who verbally accepts the existing hemiplegia may try to walk, thus clearly showing inconsistent behaviour (Cummings & Mega 2003; Sinanović & Vidović 2005; Sinanović et al. 2006). Anosognosia is on the second place as neuropsychological syndrome of stroke in right hemisphere, characterized by the denial of the motor, visual or cognitive deficit. According to different reports, in case of brain stroke in the right hemisphere anosognosia ranges from 28 to 85%, while in case of brain stroke in the left hemisphere from 0 to 17% (Sinanović & Vidović 2005; Vidović et al. 2004; Pederson et al. 1996; Vidović et al. 2006; Berti et al. 1996; Meador et al. 2000). This syndrome, defined as denial of hemiparesis or hemianopsia, is a common disorder verified in 17-28% of all patients with acute brain stroke.

Often is present together with unilateral neglect. The disorder varies in severity from mild underestimation of the degree of weakness to complete denial of ownership of paralyzed limbs. Anosognosia has been observed in patients with lesions of the right or left parietal and frontal lobes (more frequent of the right than of the left side). The most common expression of anosognosia is denial or minimization of the disease (stroke, for

example) or existing neurological deficit, for example hemiparesis or hemianopsia (Sinanović & Vidović 2005; Sinanović 2007; Stone et al. 1993; Parton et al. 2004; Vidović et al. 2004; Pederson et al. 1996). Anosognosia may occur with hemiplegia, cortical blindness (Antonov syndrome), hemianopsia, dementia and aphasia. It may occur without generalized intellectual damage, confusion or diffusional cerebral damage (Berti et al. 1996; Meador et al. 2000).

Visual neglect and anosognosia are often combined in the acute phase of brain stroke; however, they can be clearly separated by the use of some diagnostic methods. Anosognosia may be selective – the patient with more than one neurological problem may be anosognostic to one problem but not to the others. Some patients with hemineglect in the acute phase of CVI are not “aware” of this disorder, this being the reason why it is sometimes difficult to make distinction between anosognosia and neglect in some cases. However, most of the chronic patients with neglect syndrome have certain insight in their deficit and its consequences (Meador et al. 2000).

Delirium. Delirium, synonymous with the acute confusional state, is a condition of relatively abrupt onset and short duration whose major behavioural characteristics is altered attention. It is acute reversible mental disorders characterized by confessional state with disorientation for time or place (Cummings & Mega 2003). Other behavioural abnormalities frequently coexist including mood and emotional alterations, illusions, hallucinations with increased or decreased psychomotor activity (Cummings 1995; Cummings & Mega 2003; Rizzo & Eslinger 2004; Sinanović et al. 2006; Sinanović 2007; Anonymous 1994). There are different reports on frequency of delirium in acute stroke, from 24 to 48%, and it is more frequent in hemorrhagic then ischemic stroke (Sinanović et al. 2006; Sinanović 2007). Delirium is not stable state. The level of consciousness may be reduced or may fluctuate between drowsiness and hypervigilance, but the patient is unable to maintain attention for any substantial period of time. There are two types of clinical picture – hyperactive and hypoactive type (Gustafson et al. 1993; Henon et al. 1999). The principal effort in the management of the patient in delirium is directed at identifying and treating the underlying disease process (Anonymous 1999).

Depression. Depression following a stroke is also referred as post stroke depression (PSD). It is one of the more frequent consequences on the stroke, and has negative consequences on the recovery of motor and cognitive deficits, as well as the mortality risks associated with stroke (Kanner 2005; Berg et al. 2003; Maree et al. 2005; Ibrahimagić 2003; Ibrahimagić et al. 2005). The prevalence of PSD has ranged from 5 to 63% of patients in several cross-sectional studies, peaking three to six months after a stroke (Kanner 2005;

Maree et al. 2005; Ibrahimagić 2003; Ibrahimagić et al. 2005; Sinanović 2007; Sinanović 2007) The systematic review of 51 studies (reported in 96 publications) conducted between 1977 and 2002 by Maree et al. (2005) showed that frequencies of depression varied considerably across studies, but the pooled estimate was 33% (95% confidence interval, 29% to 36%) of all stroke survivors experiencing depression.

Depression is usually consequence of stroke from the acute phase to at least 2 to 3 years after stroke (Berg et al. 2003). However, a consensus on the course and associated factors of depression has not been reached. It has been accepted that DS is most common in disorders producing dysfunction of the left frontal lobe, the temporal lobes, or the left caudate nucleus (Cummings 1995), and in some studies it was shown that there is a higher risk of depression when the lesion is located in the left hemisphere (Ibrahimagić 2003; Sinanović 2007; Kauhanen et al. 1999), but other studies were not able to replicate these findings (Johnson et al. 2006), and systematic review by Carson et al (2000) also could not support this hypothesis. The presence of cortical atrophy and enlarged ventricles has been suggested as a potential risk factor for PSD.

Major and minor depression are the most frequently recognized expressions of PSD, but various investigators have proposed the existence of another type of depression in stroke patients, referred to as vascular depression (VD)(Kanner 2005; Sneed et al. 2006). This is a late onset (after 65 years) depressive disorder identified in patients that may have had overt or silent stroke(s) or sub cortical bilateral white matter ischemic disease (Kanner 2005). The symptoms of VD consist of mood abnormalities, neuropsychological disturbances with impairment of executive functions, a greater tendency to psychomotor retardation, poor insight and impaired activities of daily living. In general, the clinical manifestations of PSD are similar to those of idiosyncratic late onset depression, but psychomotor retardation may be more frequently identified.

REFERENCES

1. Anonymous. *Diagnostic and Statistical Manual of Mental Disorders*. 4th ed. American Psychiatric Association: Washington DC, 1994.
2. Anonymous. American Psychiatric Association. Practice guidelines for treatment of delirium. *Am J Psychiatry* 1999; 156: 1-20.
3. Berg A, Palomaki H, Lehtihalames M, Lonnqvist J & Kaste M: Poststroke depression. An 18-month follow-up. *Stroke* 2003; 34: 138-148.
4. Berti A, Ladavas E & Della Corte M: Anosognosia for hemiplegia, neglect dyslexia, and drawing neglect: Clinical findings and theoretical considerations. *Jorunal of the International Neuropsychological Society* 1996; 2: 426-440.
5. Caeiro L, Ferro JM, Santos CO & Figueira ML: Depression in acute stroke. *J Psychiatry Neurosci* 2006; 31: 377-383.
6. Carson AJ, MacHale S, Allen K, Lawrie SM, Dennis M, House A & Sharpe M: Depression after stroke and lesion locate: a systematic review. *Lancet* 2000; 356: 122-126.
7. Cummings J: Neuropsychiatry. In: Simpson GM (ed). *Manual of Psychiatric Disorders*. New York: Impact Communications Inc., 1995: 109-136.
8. Cummings JL & Mega MS: *Neuropsychiatry and Behavioral Neuroscience*. Oxford: University Press, 2003.
9. Engelter ST, Gostynski M, Papa S, Frein M, Born C, Ajadacic-Gross V, Gutzwiler F & Lyer PA: Epidemiology of aphasia attributable to first ischemic stroke: incidence, severity, fluency, etiology, and thrombolysis. *Stroke* 2006; 37(6): 1379-1384.
10. Geschwind N: Disconnection syndromes in animal and man. *Brain* 1965; 88: 237-294.
11. Godefroy O, Dubois C, Debachy B, Leclerc M & Kreisler A: Vascular aphasias: main characteristics of patients hospitalized in acute stroke units. *Stroke* 2002; 33(3): 702-705.
12. Gustafson Y, Olsson T, Asplund K & Hagg E: Acute confusional state (delirium) soon after stroke is associated with hypercortisolism. *Cerberovasc Dis* 1993; 3: 33-38.
13. Henon H, Lebert F, Durieu I, Godefroy O, Lucas C, Pasquier F & Leys D: Confusional state in stroke. Relation to preexisting dementia, patient characteristic, and outcome. *Stroke* 1999; 30: 773-779.
14. Ibrahimagić OC: Depression, anxiety and cortisol level after ischemic stroke. Master thesis. Medical Faculty University of Tuzla, 2003.
15. Ibrahimagić OC, Sinanović O & Smajlović D: Anxiety in acute phase of ischemic stroke and myocardial infarction. *Med Arh* 2005; 59: 366-369.
16. Jehkonen M: The Role of Visual Neglect and Anosognosias in Functional Recovery After Right Hemisphere Stroke. Academic Dissertation. Faculty of Social Sciences, University of Tampere, 2002.
17. Johnson JL, Minarik PA, Nystrom KV, Bautista C & Gorman MJ: Poststroke depression incidence and risk factor: an integrative literature review. *J Neurosci Nurs* 2006; 38 (Suppl 4): 316-327.
18. Kanner AM: Depression in Neurological Disorders. Skodsborg: The Lundbeck Institute, 2005.
19. Kauhanen ML, Korpelainen JT, Hiltunen P, Brusin E, Mononen H, Maatta R, Nieminen P, Sotaniemi KA & Myllyla VV: Poststroke depression correlates with cognitive impairment and neurological deficits. *Stroke* 1999; 30: 1875-1880.
20. Kerkhoff G: Spatial hemineglect in humans. *Prog Neurobiol* 2001; 63: 1-27.
21. Laska AC, Hellblom A, Murray V, Kahan T & Von Arbin M: Aphasia in acute stroke and relation to outcome. *J Intern Med* 2001; 249: 413-422.
22. Maree LH, Yapa C, Parag V & Anderson CS: Frequency of depression after stroke. A systematic review of observational studies. *Stroke* 2005; 36: 1330-1343.
23. Meador KJ, Loring DW, Feinberg TF, Lee GP & Nichols ME: Anosognosia and asomatognosia during intracarotid amobarbital inactivation. *Neurology* 2000; 55: 816-820.
24. Parton A, Malhotra P & Husain M: Hemispatial neglect. *J Neurol Neurosurg Psychiatr* 2004; 75: 13-21.

25. Pederson PM, Jorgensen HS, Nakayama H, Raaschou HO & Olsen TS: *Apasia in acute stroke: incidence, determinants, and recovery.* Ann Neurol 1995; 38: 659-666.
26. Pedersen PM, Jorgensen HS, Nakayama H, Raaschou HO & Olsen TS: *Frequency, determinants and consequences of anosognosia in acute stroke.* J Neurol Rehab 1996; 10: 243-250.
27. Rizzo M & Eslinger PJ: *Principles and Practice of Behavioral Neurology and Neuropsychology.* Philadelphia: W.B. Saunders Company, 2004.
28. Sinanović O & Vidović M: *Neuropsihologija sindroma unilateralnog zanemarivanja.* Defektologija 2004; 8: 5-14.
29. Sinanović O: *Afazije.* U: Sinanović O, Smajlović Dž i saradnici. *Osnove neuropsihologije i neurologije ponašanja.* Tuzla. Univerzitet u Tuzli, 2005: 47-67.
30. Sinanović O & Vidović O: *Anozognozija i unilateralno zanemarivanje.* U: Sinanović O, Smajlović Dž i saradnici. *Osnove neuropsihologije i neurologije ponašanja.* Tuzla. Univerzitet u Tuzli, 2005: 98-102.
31. Sinanović O: Vidović M, Smajlović Dž. *Najčešći neuropsihološki poremećaji u akutnom cerebrovaskularnom inzultu.* Liječ Vjesn 2006; 128 (Supl 6): 20-21.
32. Sinanović O: *The most frequent neuropsychological disorders in urgent neurology.* Med Arh 2007; 61(Suppl 2): 10.
33. Sinanović O: *Anozognozija i unilateralno zanemarivanje i cerebrovaskularna bolest.* Neuro Croat 2007; 56(Suppl 1): 171-187.
34. Sinanović O: *Anosognosia and unilateral neglect in acute first-ever stroke.* J Neural Transm 2007; 114: CXXIV-CXXV.
35. Sinanović O: *Organically originated depressive syndromes.* Neurol Croat 2007; 56 (Suppl 5): 67-72.
36. Sinanović O: *Psychopharmacotherapy organically originated depressive syndromes.* Psychiatr Danub 2007; 19:390-391.
37. Sneed JR, Roose SP & Sackeim HA: *Vascular depression: A distinct diagnostic subtype?* Biol Psychiatry 2006; 60: 1304-1305.
38. Stone SP, Halligan PW & Greenwood RJ: *The incidence of neglect phenomena and related disorders in patients with acute right or left hemisphere stroke.* Age Ageing 1993; 22:46-52
39. Vidović M, Sinanović O & Smajlović S: *Hemineglect, anosognosia and somatoparesis in patient with ischemic stroke.* Neurol Croat 2004; 53(Suppl 1): 136-137.
40. Vidović M, Sinanović O, Smajlović DŽ & Zonić L: *Neglect and anosognosia in acute cerebrovascular disease.* Neurol Croat 2006; 55 (Suppl 2): 167-168.

Correspondence:

Osman Sinanović
Department of Neurology, University Clinical Center Tuzla
Medical Faculty University of Tuzla
75000 Tuzla, Bosnia and Herzegovina
E-mail: osman.sinanovic@ukctuzla.ba