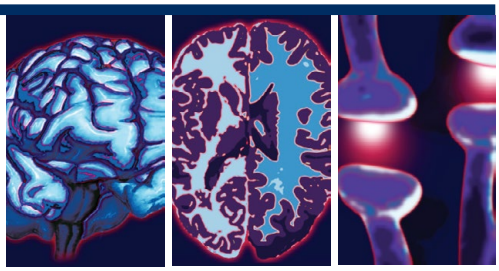
Editorial



Neuropsychiatry Syndromes Following Ischemic Stroke

Hua Jun Liang[1], Yan Liang[1], Gabor S. Ungvari[2,3], Wai Kwong Tang[1,4*]

Stroke is the leading cause of chronic disability [1]. Many people who survive a cerebrovascular accident live with the chronic and disabling consequences [2]. Stroke survivors commonly report a low sense of well-being caused by restricted physical functions and the general psychological sequelae of disability coupled with a variety of specific neuropsychiatric complications [3]. Neuropsychiatric conditions following stroke include cognitive decline, depression, anxiety, fatigue, apathy, aggression and emotional lability. Mania, post-traumatic stress disorder, and psychosis are rare but have also been reported following a stroke. Emotional and/or cognitive disturbances impede recovery from stroke and increase mortality. The prevalence of post-stroke neuropsychiatric complications varies across studies due to different methods and populations, the time frame of the assessments, and the instruments used. Post-stroke depression (PSD) is the most extensively investigated neuropsychiatric syndrome. Major and minor depression affects 9-24% of patients post-stroke. The prevalence of PSD is lower in the community compared to rehabilitation settings or outpatient clinics. PSD tends to be chronic: depressive symptoms found at 3 months post-stroke are also evident at 12 months in around 40-66% of patients. A number of factors, although not in all studies, are associated with PSD: female sex, age, pre-morbid personality, stressful life events, poor social support, history of depression, severity of the index stroke, stroke location, impaired cognition, and genetic factors related to the functions of the serotoninergic system and brain-derived neurotropic factor. The severity and location of stroke are associated with a brief period of PSD occurring not long after the index stroke, while persistent PSD starting some time later is more likely to be associated with vascular burden, ongoing neurological symptoms at 1-year follow up, severity of PSD at baseline, and poor social support.

Anxiety is frequently concurrent with PSD, affecting 20-28% of stroke survivors. The prevalence of post-stroke anxiety is stable over a five-year period following stroke. Post-stroke fatigue is part of PSD symptomatology, but can also occur separately. Fatigue is reported by 20-65% of stroke survivors in the first 2 years. Another frequent neuropsychiatric complication of stroke is apathy, which develops in 19-55% of patients within 5 years post-stroke. Apathy also has a chronic course. Similar to fatigue, apathy may occur as a complex symptom of PSD or as a separate syndrome. Aggression occurs in 17-23% of patients in the first week and in 32% 3-12 months after stroke. Emotional lability, comprising embarrassing, sudden bursts of unprovoked sobbing or laughter, develops in 12-34% of stroke survivors at 2-4 months post-stroke. The risk factors for these post-stroke emotional disturbances are essentially the same as for PSD: female sex is associated with anxiety and fatigue; younger age with anxiety; older age with fatigue and apathy; low education level with apathy; poor social support with anxiety and emotional liability; premorbid personality problems, diabetes mellitus, and recurrent stroke with aggression and fatigue; depressive symptoms with anxiety, fatigue, aggression, and emotional liability; restricted independence with anxiety,

[1]Department of Psychiatry, Chinese University of Hong Kong, Hong Kong SAR, China

[2]School of Psychiatry & Clinical Neurosciences, University of Western Australia, Perth, Australia

[3]University of Notre Dame Australia/Marian Centre, Perth, Australia

[4]CUHK Shenzhen Research Institute, Hong Kong, China

[†]Author for correspondence: Wai Kwong Tang, Department of Psychiatry, Shatin Hospital, Shatin, N.T., Hong Kong SAR, China, Tel: +852-2636-7760; Fax: +852-2648-3394; Email: tangwk@cuhk.edu.hk

fatigue, apathy, aggression, and emotional liability; and cognitive impairment with anxiety, apathy, and aggression. Genetic variants in the dopaminergic and serotoninergic systems have been related to post-stroke aggression and emotional liability, respectively.

Although the role of lesion location in post-stroke neuropsychiatric syndromes is still uncertain, most findings point to regions thought to be responsible for regulating emotions. Frontal lobe lesions have been associated with PSD, anxiety, aggression, and emotional liability; thalamic lesions with emotional liability; basal ganglia pathology with depression, fatigue, aggression, and emotional liability; and vascular brainstem insult with apathy, aggression, and emotional liability. Left-sided lesions are more likely to result in PSD and aggression, while right-sided lesions have been connected with anxiety and apathy.

Post-stroke neuropsychiatric syndromes occur frequently and adversely affect recovery from stroke, thus their prevention, early recognition, and treatment have major implications for all aspects of stroke survivors' lives. Both antidepressants and psychological interventions are effective for treating and preventing PSD. To date, only a few explorative treatment response studies have investigated other neuropsychiatric syndromes. Antidepressants appear to be effective in alleviating post-stroke anxiety, aggression, and emotional liability, but not fatigue. The central stimulant modafinil seems to be effective for treating post-stroke fatigue and the neurotropic nefiracetam could be the treatment choice for apathy. Non-pharmacological interventions, such as Cognitive and Graded Activity Training, can alleviate post-stroke fatigue, and interventions involving stimulating activities show promise for treating post-stroke apathy.

To conclude, post-stroke neuropsychiatric syndromes are common and adversely influence patients' physical and psychological condition, daily activities, and quality of life. Much work is needed to elucidate the course and pathomechanisms, and to find effective preventive and therapeutic measures for the neuropsychiatric complications of stroke.

References

1. Soler EP, Ruiz VC (2010) Epidemiology and risk factors of cerebral ischemia and ischemic heart diseases: similarities and differences. Current cardiology reviews 6: 138-149.
2. Carod-Artal FJ, Egido JA (2009) Quality of life after stroke: the importance of a good recovery. Cerebrovasc Dis 1: 204-214.
3. Robinson RG (1997) Neuropsychiatric consequences of stroke. Annu Rev Med 48: 217-229.