

Behavioral Dysexecutive Syndrome after Stroke

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Received: February 19, 2016; **Accepted:** February 21, 2016; **Published:** February 25, 2016

Dysexecutive syndrome (DES) is defined as an impairment of executive functions constituting of two domains: behavioral dysexecutive syndrome (BDES) and cognitive dysexecutive syndrome (CDES) which are not accompanied always [1]. A growing body of studies demonstrated that BDES is a common post-stroke neuropsychiatric comorbidity. The prevalence of BDES in stroke survivors varies ranging from 3% to 25% possibly attributed to the lack of standardized diagnosis methods and variances in study sample and study mode.

Post-stroke BDES comprises varieties of clinical presentations, the most prevalent of which are anosognosia and hypoactivity with apathy-abulia [2]. The clinical course of BDES in stroke population has not yet fully elucidated. Some studies showed that there was only a minor decrease of prevalence of BDES several months after stroke, suggesting the possible chronicity of BDES. Possible clinical correlates of behavioral symptoms in stroke are global cognitive impairment, executive dysfunction, premorbid personality and psychopathology and stroke severity. Despite BDES is also a possible predictor of poor post-stroke physical function and can increase the burden of caregivers, it is still often underestimated and untreated. Furthermore, the treating methods for BDES are limited and lack of high quality supporting evidences. Some studies suggested that antipsychotic drugs might be effective in controlling behavioral dysexecutive problems such as agitation, apathy and disinhibition. The methods of psychosocial treatments varies including caregiver education, aromatherapy, exercise and behavioral intervention whereas their effectiveness is still under debated.

The neuroanatomical pattern of post-stroke BDES is rarely studied. Lesion studies demonstrated that disruption to frontal-subcortical circuit (FSC) is the pivotal cause of BDES [3]. First of all, frontal lobe is treated as the key component of FSC. Frontal lesion and reduced frontal volume contribute to behavioral disturbances. Particularly, abnormalities of orbitofrontal cortex (OFC) and medial prefrontal cortex (MPC) involving the reward representation, response selection and behavioral flexibility are closely correlated with apathy, disinhibition and other dysexecutive syndromes in patients with neurologic diseases. Further, Basal ganglia are involved in motivated behavior, behavioral switching theory of mind. Basal ganglia lesions can lead to apathy, abulia, disinhibition, irritability and labile behavior. An abrupt of BDES can be observed in those thalamic stroke patients with complex syndromes varying according to the nuclei affected. However,

very few structural brain imaging studies have been published on BDES or behavioral symptoms in stroke. The existing studies found associations between BDES/behavioral symptoms and infarcts in the right hemisphere, anterior capsule, thalamus, and WMH. These studies have many limitations such as small sample size, biased sampling, lack of standardized assessment of BDES and rude classifications of lesion location. Therefore, the studies of high quality with a more advanced method to investigate neuroanatomical lesion pattern are keenly demanded.

Except for the neuroanatomical abnormalities, cerebral hemodynamics and metabolism are also suggested as the possible mechanisms of BDES after stroke. There are a great deal of evidences supporting the role of reduced cerebral blood flow (CBF) in CDES whereas limited studies reporting the relationship of CBF and BDES. Subjects with reduced CBF in frontal lobe presented a worse behavioral executive function. On the other hand, an improvement of executive function was observed when CBF was augmented, which possibly implicated that augmentation of focal CBF could be a promising treating method of BDES. Meanwhile, several studies showed that dysexecutive function

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Citation: Liang Y, Liang HJ, Ungvari GS, et al. Behavioral Dysexecutive Syndrome after Stroke. *Stroke Res Ther.* 2016, 1:1.

was associated with lower metabolic level in frontal lobe. Further investigations on role of CBF and cerebral metabolism in BDES in stroke population are suggested to apply.

To conclude, the existing literatures on BDES and stroke suggest that BDES is one of the most common post-stroke psychiatric comorbidity and a combined neuroanatomical and neurobiological lesion accounted to stroke substantially serves as the underlying mechanisms of post-stroke BDES. Standardized diagnosis criteria and a deeper understanding of the mechanism of post-stroke BDES is urgently needed , which may benefit to

recognize BDES in stroke survivors as early as possible and select the appropriate treatment, therefore, result in a better outcome of stroke.

Acknowledgement

This paper is supported by the following grants:

1. Health and Medical Research Fund, reference number is 02130726
2. Direct Grant for Research, , reference number is 2013.1.072

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