

21st World Congress on Neurology and Therapeutics March 15-17, 2018, London, UK - Structural and functional MRI correlates of post stroke depression

Wai Kwong Tang

The Chinese University of Hong Kong, Hong Kong

Depression is common following an acute stroke. Post Stroke Depression (PSD) has notable impacts on the function recovery and quality of lifetime of stroke survivors. Many studies have explored the association between lesion location and therefore the incidence of PSD. For instance, lesions in lobe, basal ganglia and deep substantia alba are related with PSD. Furthermore, cerebral microbleeds and functional changes in brain networks have also been implicated within the development of PSD. During this study, evidences of such association between the above structural and functional brain changes and PSD are going to be reviewed. Specifically, PSD is said to frontosubcortical circuit infarcts, vascular markers of huge and little vessel diseases also as abnormal substantia alba integrity and functioning connectivity in various brain regions. PSD has been linked with poorer recovery of function and cognition, yet our understanding of potential mechanisms is currently limited. Alterations in resting-state functional MRI are investigated to a limited extent. Post stroke, patients frequently experience motor, sensory, cognitive, and behavioural changes, all of which can impact recovery. Changes to a stroke survivor's mood also are common, with depression because the most often reported psychiatric disorder following ischemic stroke. Poststroke depression (PSD) is estimated to affect approximately one-third of survivors, compared to about one-sixth of the nonstroke population. PSD is related to poorer recovery prospects, including increased disability, worse cognitive outcomes, decreased quality of life, and increased risk of mortality. In particular, PSD negatively impacts response to rehabilitation in acute and subacute phases of recovery. One approach has been to re-

search the connection between lesion location and depression; however, despite an outsized number of studies, findings are equivocal. These findings suggest that lesion location alone is unlikely to be an informative biomarker related to PSD. In the broader literature of depressive disorder, the disorder is not considered to be caused by independent, localised changes within specific brain regions but is thought to be partially due to disruption of communication between areas. Several meta-analyses of fMRI cohort studies of depressive disorder have found changes in brain activation and connectivity. Findings highlight alteration of brain regions according to the present system-level models of depression. It may therefore be useful to look at biomarkers of PSD using resting-state methods that specialise in intrinsic brain activity and whole brain. To date, PSD studies of resting-state changes have not been widely employed, have focused on functional connectivity from specific regions, e.g., within the default mode network (DMN) and anterior cingulate, and have included participants of varying times post stroke. Results from these studies have been inconsistent. For example, Lassalle-Lagadec et al. found correlations at 10 days post stroke between Depression score and therefore the left middle temporal cortex and precuneus and at 3 months with the neostriatum. Vicentini et al. failed to find any regional correlations of the posterior cingulate with a depression score during a cohort of chronic stroke survivors. However, our understanding of the potential mechanisms underlying the negative impact of depressive symptoms on recovery and rehabilitation is currently limited. Determining factors which can assist within the identification of those "at risk" of developing poststroke de-

pression may aid within the recovery process and/or prediction of response to rehabilitation. The value of biomarkers of stroke recovery that specialise in brain structure and performance has recently been highlighted in consensus-based recommendations. Neuroimaging markers of depression could also be used to provide new insight into neural mechanisms underlying depression, to predict the likelihood of future depressive symptoms, and/or to predict readiness to interact in treatment or treatment response. All are important reasons to spot stroke survivors with underlying vulnerabilities which will be “at risk” of developing depression. Of the 63 stroke survivors, 38 were classified as “low-depressive symptoms” and 25 as “high depressive symptoms.” Six had a past

history of depression. We found interaction effects across frequency bands in several brain regions that differentiated the 2 groups. The broadband analysis revealed interaction effects within the left insula and therefore the left superior lobe. The subband analysis showed contrasting fALFF response between the 2 groups within the left thalamus, right caudate, and left cerebellum.

Conclusions: We provide evidence that fALFF is sensitive to changes in poststroke depressive symptom severity and implicates frontostriatal and cerebellar regions, according to previous studies. The utilization of multiband analysis might be an efficient method to look at neural correlates of depression after stroke.