The relationship between depression and both dementia and cerebrovascular pathology has, for good reason, received much attention from researchers and clinicians alike. This presentation focused on relevant findings of neuroanatomical pathways and associated monoaminergic abnormality in vascular depression, apathy, cognitive dysfunction, and pathway linking depression, apathy to cognitive dysfunction, and dementia.

We examined the relationships between post-stroke depression (PSD), functional recovery, cognitive functioning, and lesion location, after separating PSD into two core symptom dimensions: Affective (depressive) and apathetic (loss of interest). These two core symptom dimensions appear to have different underlying neuroanatomical mechanisms, and appear to exert different effects on cognitive functioning and functional recovery. Among the patients with higher depressive scores, the lesion overlap centered on the brainstem, left basal ganglia, and left frontal cortex. Among the patients with higher apathy scores, the lesion overlap centered on the brainstem and bilateral striatum. And apathy was a better predictor of poor functional recovery after a stroke than depression although apathy and depression both affect negatively on cognitive functioning. It is therefore important that studies of PSD consider the two symptoms dimensions separately.

Next, we investigated relationship between abnormality of neuroanatomical pathways, and monoaminergic abnormality in PSD patients. Findings indicated that depression and apathy scores did not correlate with monoamine, and or metabolite values. However, the decrease of NA&DA and the increase of NA&DA turnover were related to lesions in the brainstem, whereas the increase in NA&DA and the decrease in NA&DA turnover were related to cortical and/or striatum lesions. The data on 5-HT turnover showed an opposite tendency to NA&DA turnover. Results of our studies on vascular depression and apathy may indicate catecholamine (NA&DA) and serotonin, both of which are anatomically and functionally interconnected and could respectively influence apathetic, and affective symptoms of depression after stroke.

**Speaker 4: Hochang Lee, USA**

**Testing Vascular Depression hypothesis:**

**Neuropsychiatric Outcomes after Heart Surgery (NOAHS) Study**

**Abstract**

Depression after coronary artery bypass graft (CABG) surgery is common (up to 40%) and increases risk of cardiac morbidity and mortality in the first year by more than two fold. However, current scarcity of data on etiopathogenesis of post-CABG depression hampers development of prevention or treatment strategies of post-CABG depression. “Vascular Depression” hypothesis posits that cerebrovascular disease predisposes, precipitates, or perpetuates late-onset depression and implicates etiopathogenesis and treatment strategies that are different from idiopathic, early-onset depression. Conspicuous similarities in demographics, longitudinal course, and presence of vascular risk factors exist between post-CABG depression and vascular depression. The NIMH-sponsored Neuropsychiatric Outcomes after Heart Surgery (NOAHS) study utilizes Transcranial Doppler ultrasound (TCD) to detect and quantify the location and severity of Intracranial Atherosclerosis (ICA), as well as to assess for other putative pre-CABG risk factors (e.g. pre-CABG depression, neuroticism, low social and support) for post-CABG depression in CABG surgery patients at the time of cardiac catheterization. The NOAHS study tests the Vascular Depression Hypothesis by follow the subjects over the subsequent 12 months to assess for incidence, symptomatology and course of post-CABG depression. Confirming these predictions will support the hypothesis that post-CABG depression is a form of Vascular Depression, thus laying the foundation for risk stratification with a mobile, bedside tool and development of etiologically-based prevention strategies to reduce morbidity and mortality associated with post-CABG depression.

**CP05: Schizophrenia**

**Speaker: Gerhard Grunder, Germany**

**Speaker: Sung Wan Kim, Republic of Korea**

**Abstract**

Early intervention services for the first episode schizophrenia are shown to be effective for symptomatic and functional outcomes. Early intervention has two objectives: The first is to prevent the onset of schizophrenia in individuals at ultra-high risk. The second is to provide effective treatment to people in the early stages of schizophrenia, with the goal of reducing the ultimate severity of the illness. The functioning of patients with schizophrenia spectrum disorders often declines within 3–5 years of the onset of this illness and plateaus thereafter. Therefore, the first 3–5 years of this disorder have been described as a critical period during which the future course and prognosis of the patient is determined. The guidelines of the National Institute for Health and Care Excellence (NICE) in the UK recommend that a full range of pharmacological, psychological, social, occupational, and educational interventions be provided for people with psychosis. A key element of the psychological interventions for people with first-episode psychosis is cognitive–behavioural treatment (CBT). There is less evidence for early intervention for people with early psychosis in Asian countries compared with Western countries. This presentation provides an overview of early intervention services for psychosis. Leading Korean early intervention programs in Gwangju Bukgu Mental Health Center are demonstrated. Group cognitive-behavioral therapy (CBT), intensive cognitive behavioral case management with smartphone application, and nutritional support have been conducted for early psychosis. Those early intervention services were effective for the improvement of psychotic symptoms and functional impairment in patients with early psychosis. Integration of psychosocial treatment based on community mental health center and pharmacotherapy based on hospital is important for comprehensive treatment for psychosis.

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