P159: Low Cognitive Reserve as a Risk Factor for Delirium in Elderly: A Case-Control Study

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Objective: Cognitive Reserve (CR) developed from observation that several individuals show fewer cognitive impairment compared to others with the same brain injuries or neuropathology. Cognitive reserve is a potentially modifiable characteristic. Most of studies on cognitive reserve were conducted on chronic progressive diseases such as dementia. This study aims to define the role of cognitive reserve in geriatric delirium cases.

Methods: This case-control study was conducted in the acute geriatric inpatient of Cipto Mangunkusumo Hospital, Jakarta, Indonesia on June to September 2019 that consisted of 33 subjects with delirium and 33 controls. The measurement of cognitive reserve was done using the Indonesian adaptation of Cognitive Reserve Index questionnaire (CRIq) with 3 subscales, i.e. Education, Work Activity and Leisure Time.

Results: We found that the CRIq scores of delirium patients were lower compared to the non-delirium controls both on total and each subscores, with a statistically significant mean difference ($p<0,01$). Patients with low-medium cognitive reserve also more likely to develop delirium compared to those with medium-high cognitive reserve (OR 9; 95% CI 2.86 to 28.22).

Conclusion: Low cognitive reserve may serve as a risk factor for delirium in the elderly. The measure of CRI in the geriatric inpatients unit can be used to determine those at risk of developing delirium. Further research are warranted to elaborate potentially modifiable variables of cognitive reserve to minimize the risk of delirium.

Keywords: cognitive reserve, delirium, elderly.

P161: Heterogeneity and Clinical Uncertainty of BPSD Therapeutics

Authors: Lon S. Schneider, Rebecca Howard

Objective: BPSD is typically treated as a singular entity. Yet it is heterogeneous and challenges simple phenotyping by behavioral inventory. Some investigators recognize BPSD more as 'obstreperous,' disruptive behavior, or unwanted behavior. Others conceptualize it as a neuropsychiatric entity with an underlying pathobiology, or as the expression of an unmet need. Treatments for BPSD have been challenging since before the first clinical trials with chlorpromazine.

Methods: We systematically reviewed interventional studies to understand the successes, limitations, and knowledge gaps in terms of methodology that might misinform practice. Questions addressed included: What do these studies look like? How is BPRS operationalized, and does it vary between studies? What interventions have been tested? How are we measuring eligibility and outcomes? Are there methodological factors that influence the outcomes and validity of these trials? Are the trials methods fit for purpose and how can we better test interventions?

Results: From a search yielding 6497 candidate studies, we included 474 of which 413 were randomized, 340 parallel group, 197 double-blinded, 51 unblinded. About 30% were in nursing homes only and 20% outpatient only. Most NH studies were drug studies; most outpatient studies were non-pharmacological. Over time, study durations consolidated to 6–12-week treatment periods and samples grew exceptionally large, involving 400 to 1200 participants.

Of studies that specified a target, 171 were for 'agitation.' 50 investigated sleep disturbance, 25 apathy, 25 depression, 21 psychosis. 150 described only 'BPSD' or 'neuropsychiatric symptoms.' Two-thirds of the agitation

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studies were single drug interventions; most used a scale score cut-off to define agitation. Important characteristics, secular trends in design, and quality of the BPSD studies will be detailed.

Conclusions: The important trends in methods for interventions and assessment of BPSD are not necessarily toward quality. Eligibility criteria have become designed for convenience, are misspecified relying on the same scales used for outcomes, although randomization is the rule, allocation concealment and treatment blinding is poor. There is marked autoregression of outcomes. Studies have become larger and designed to detect small effects even when clinical meaning is uncertain. BPSD studies need reconsideration and a few simple fixes to better discover effective treatments. Only a little care is needed to improve the quality and reliability of studies. This includes study management that is independent of patient selection and outcomes and from most procedures, and truly blinded assessments.

P168: Resilience and cortical thickness in the medial orbitofrontal cortex in Japanese older cancer survivors: A population-based cross-sectional study

P173: Structural Changes in the Hippocampal Subfields in Early-Onset Mild Cognitive Impairment

Author: Seok Woo Moon

Objective: The aim of this study was to examine the structural change in the hippocampal subfields in early-onset (EO) mild cognitive impairment (MCI) patients associated with the APOE ε4 carrier state.

Methods: This study had 50 subjects aged 55-63 years, all of whom were diagnosed with amnestic MCI at baseline via the Korean version of the Consortium to Establish a Registry for Alzheimer's Disease Assessment Packet (CERAD-K). The EO-MCI patients were divided into the MCI continued (MCIcont) and Alzheimer's disease (AD) converted (ADconv) groups 2 years later. The volumes of hippocampal subfields were measured for all the subjects. The calculations were based on the change of the volumes between the 2-year-interval brain Magnetic resonance image (MRI) scans between MCIcont and ADconv groups according to the Apolipoprotein ε4 (APOE ε4) carrier state.

Results: There was a significant correlation between APOE ε4 allele and structural changes in several hippocampal subfields. The volume reduction in cornus ammonis 1 (CA1) field and subiculum, especially in the APOE ε4 carriers. The significance was more prominent in ADconv group.

Conclusion: These results suggest that the possession of APOE ε4 allele may lead to significantly greater predilection for the structural changes in hippocampal subfields, showing significant changes, especially in the ADconv patients compared with MCIcont patients.

KEY WORDS: Early-onset · Mild cognitive impairment · Hippocampus · APOE ε4 · Atrophy.

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