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Effectiveness of Integrating Cognitive Remediation Program into Everyday Clinical Practice of Schizophrenia at Psychiatric Rehabilitation Settings

Alexander John[1], Helen Ayres[2], Milan Dragovic[2], Kim Yeak[2]
[1]University of Western Australia, Australia, [2]Bentley Health Service, Australia

Abstract

Cognitive deficits (CD) in schizophrenia are recalcitrant to treatment as usual. Whilst there has been considerable interest in recent years for evaluating the efficacy of cognitive remediation (CR) programs in schizophrenia at research settings, scant attention has been paid to evaluate the effectiveness of CR programs at everyday clinical practice settings.

Method: We evaluated retrospectively short-term cognitive, occupational and accommodation outcomes of consecutive patients with schizophrenia admitted over a 5 year period at a tertiary-care inpatient public psychiatric rehabilitation facility in Western Australia. The Brief Assessment of Cognition in Schizophrenia (BACS) was utilised to assess cognition. Patients were divided into 3 groups based on their participation in the neuroplasticity based auditory CR program of PositScience; those who did not participate (non-trainers), those who completed less than 20 hours of training (incomplete-trainers) and those who completed 20 or more hours of training (complete- trainers).

Results: The mean age of the patients was 32.1 years, 68.5% were males, nearly 80% had treatment-resistant illness, 65% were on clozapine and comorbidity was highly prevalent (72%). Thirty-seven patients were classified as non-trainers, 17 as incomplete-trainers and 34 as complete- trainers. The 3 groups did not differ in measured demographic and clinical parameters. Compared to their admission scores, complete- trainers had significantly improved scores at discharge on verbal memory (p=0.012), motor speed (p=0.009) and the composite score (p=0.006). Furthermore, 24%, 22% and 36% of patients changed from unemployed to the employed group in the non-trainers, incomplete-trainers and complete-trainers groups respectively from admission to discharge.

Conclusion: Our study demonstrates that CR program can be integrated effectively into the interventions provided for people with schizophrenia at public psychiatric rehabilitation settings. Significant improvements in cognitive and functional outcomes revealed in this study indicate the need for further translational research in the field of CR in schizophrenia.

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A family of primary familial brain calcification due to mutation in platelet-derived growth factor-B gene

Teruo Hayashi, Giovanni Coppola, Andrea Legati, Tadashi Nishikawa
Seiwakai Nishikawa Hospital, Japan

Abstract

Primary familial brain calcification (PFBC) is a neuropsychiatric disorder characterized by abnormal deposits of calcium in the basal ganglia and cerebellum. PFBC can present with a spectrum of symptoms resembling those seen in dementia and schizophrenia. Mutations in some genes have been found to cause PFBC: namely, the *SLC20A2* gene that codes for the sodium-dependent phosphate transporter and the *PDGFRB* gene that codes for the platelet-derived growth factor (PDGF) receptor β. A recent study found that mutations of *PDGFB*, which encode the ligand peptide PDGF-B for the PDGF receptor β, also cause PFBC. Here we report the first Japanese family of PFBC carrying a mutation of *PDGFB*. CT scans revealed a symmetrical calcification over the basal ganglia in two members of the family. One family member complained auditory hallucination at 16 years old, and had been treated for schizophrenia. The other family member complained memory and gait disturbances in his late 60s. The mutation in *PDGFB* (c.445C>T, p.Arg149*) that causes the substitution of an arginine with a stop codon at amino acid 149 of the PDGF-B protein (p. Arg149*) was consistently detected in both cases. No mutations in *SLC20A2* were detected. This finding indicates that the PDGF pathway plays a crucial role in pathogenesis of PFBC, and that dysfunction of the PDGF signaling may lead to psychiatric symptoms that are associated with dementia and/or schizophrenia.

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The relationship between MMN and COMT Val108/158Met genotype in schizophrenia

Sho Horikoshi,Tetsuya Shiga, Hiroshi Hoshino, Haruka Ochiai, Keiko Kanno-Nozaki, Kazuko Kanno, Haruka Kaneko, Itaru Miura, Hirooki Yabe
Department of Psychiatry, Fukushima Medical University

Abstract

Background: Mismatch negativity (MMN) is a component of auditory event-related potentials that reflect automatic change detection in brain, showing qualities of endophenotypes in schizophrenia. MMN deficiency is one of the robust findings in the patients, and reflects cognitive and functional decline.

The Catechol-O-methyltransferase (COMT) is a key enzyme involved in regulating dopamine transmission within the prefrontal cortex. Preliminary study suggested that COMTVal108/158Met genotype is related to cognitive function in schizophrenia. Both related to cognitive function, however, no studies have reported the relationship between MMN and COMTVal108/158Met genotype in schizophrenia. In this study, we examined the relationship between them.

Method: Duration MMN was measured, and COMTVal108/158Met polymorphism was detected by polymerase chain reaction-restriction fragment length polymorphism in 49 schizophrenia patients. (Val/Val, 21; Met carriers 28). The amplitude and latency of MMN were compared between the Val/Val and Met carriers.

Results: The MMN amplitudes in schizophrenia patients were no difference between Val/Val and Met carriers. The MMN latency of Met carriers was shorter than that of Val/Val.

Conclusions: It is known that the enzyme containing Met has less activity and presumably greater synaptic dopamine than the Val/Val enzyme. Therefore, these results mean that the dopamine activity in prefrontal cortex accelerates pre-attentive auditory change detection.

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Association between coding single nucleotide polymorphisms in ADAMTS20 gene and schizophrenia in a Korean population

Hae Jeong Park, Jong Woo Kim, Youngjong Kim
Kyunghee Univ. Hospital, Republic of Korea

Abstract

Objective: In support of the neurodevelopmental hypothesis of schizophrenia, patients with schizophrenia have been shown to have a higher incidence of minor physical anomalies (MPAs) than healthy controls, especially in the craniofacial region. A disintegrin-like and metalloprotease with thrombospondin type 1 motif, 20 (ADAMTS20) has been implicated in craniofacial abnormalities and in particular the development of cleft lip and