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NEUROCOGNITIVE DEFICIT CHANGES IN RELATION TO THE COURSE OF SCHIZOPHRENIA AND SCHIZOPHRENIA SPECTRUM DISORDERS: 5-YEAR FOLLOW-UP STUDY

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SUMMARY

Cognitive deficit is present in most of schizophrenia cases and even better explains functional outcomes then positive and negative symptoms. There have been less consensus regarding the long-term course of cognitive functioning after onset of the illness.

In our study we used a neuropsychological test battery based on Luria`s systematic approach in testing of patients at their first episode of schizophrenia and schizophrenia spectrum disorders and during 5-year follow-up. The results indicated that patients with various types of course of schizophrenia and schizophrenia spectrum disorders and hence, good and poor outcomes demonstrated different patterns of dynamic of cognitive decline during the follow-up.

Key words: *schizophrenia - schizophrenia spectrum disorders – neurocognitive deficits- 5 year follow-up*

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Introduction

Cognitive deficit is a core feature of schizophrenia mostly grasping memory, psychomotor processing, attention, thinking, and executive functioning. The existing literature suggests that cognitive deficit is already present in the prodromal phase of the illness (Hawkins et al. 2004) and is detected at the onset (Gopal et al. 2005).

However, there are inconsistencies in the evidence of the cognitive decline over time and these suggest the possibility of different patterns of deficit according to heterogeneity of the illness. While one group of researchers reported the interconnections of cognitive deficits with illness duration and more prominent cognitive dysfunction during the course of illness (Braw et al. 2007), the others demonstrated fluctuations of the cognitive deficit changes being worse in acute states and decreasing in remission (Carlsson et al. 2005). Moreover, many studies showed the stability of cognitive dysfunction and no connection with changes in the clinical state (O`Carroll et al. 2000; Elevag et al. 1997). Recent studies have been focused on the differentiation of cognitive functioning in relation to the diagnostic categories (Harrow et al. 2000) and the course of schizophrenia (Joyce et al. 2005) which reveal cognitive heterogeneity in schizophrenia and schizophrenia spectrum disorders.

To address the question whether patients with schizophrenia and schizophrenia spectrum disorders with the various courses of illness and especially with good and poor outcomes could reveal the heterogeneity of cognitive decline after the first psychotic episode during 5-year follow-up we performed the following study.

Methods

58 patients with a first episode of schizophrenia and schizophrenia form disorders who have been treated within the integrated program of the Early Intervention Centre in the Moscow Research Institute of Psychiatry were included in the study. The patients were assigned to subgroups with good (group 1, n=25, age-28.1$\pm$6.2 years, female-37.5%, mean years of education-14$\pm$3.8, F.25, F.20) and poor outcomes (group 2, n=33, age-32.9$\pm$5.8 years, female-45.5%, mean years of education-13$\pm$2.3 years, F.20, F.21, F.25) on the basis of clinical and social characteristics. Good outcome patients were those who had experienced 1-2 psychotic episodes followed by 1, 5-4 years of stable remission close to recovery and rather high social adjustment. Poor outcome was determined in patients with continuous course of illness with severe negative symptoms in remission and the need of social support. All patients were treated in an outpatient setting. Psychopathological symptoms were rated on the Positive and Negative Symptoms Scale (PANSS; Kay et al. 1987). The control group consisted of 25 healthy comparisons matched by age, sex, and education level to the 1st group.

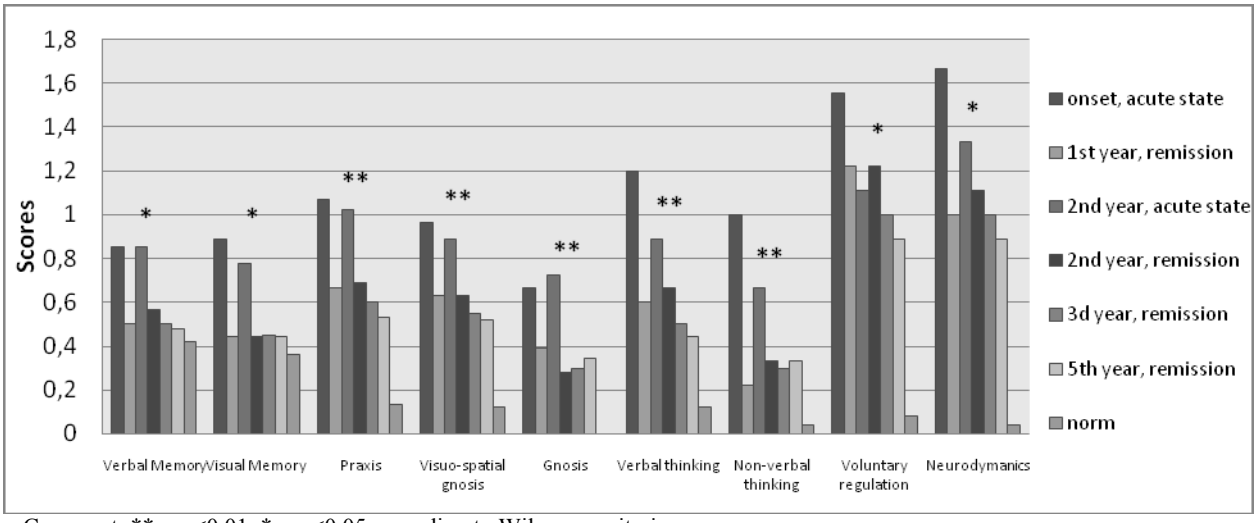
Both patients and controls underwent neuropsychological testing based on Luria`s systematic approach (Luria 1966) during 5 years follow-up. The neuropsychological test battery included examination of verbal memory, visual memory, spatial-motor skills, tactile and visio-spatial recognition, audio recognition, visual recognition, thinking, and general evaluation of neurodynamics and voluntary regulation (executive functioning). Assessment of patients was done twice in the acute state and twice in remission on the first and second years of observation and then annually in remission.

Results

According to PANSS scores (negative symptoms subscale) during the follow-up, group1 demonstrated a significant decrease of negative symptoms over time while in the group 2 rates significantly increased at the follow-up.

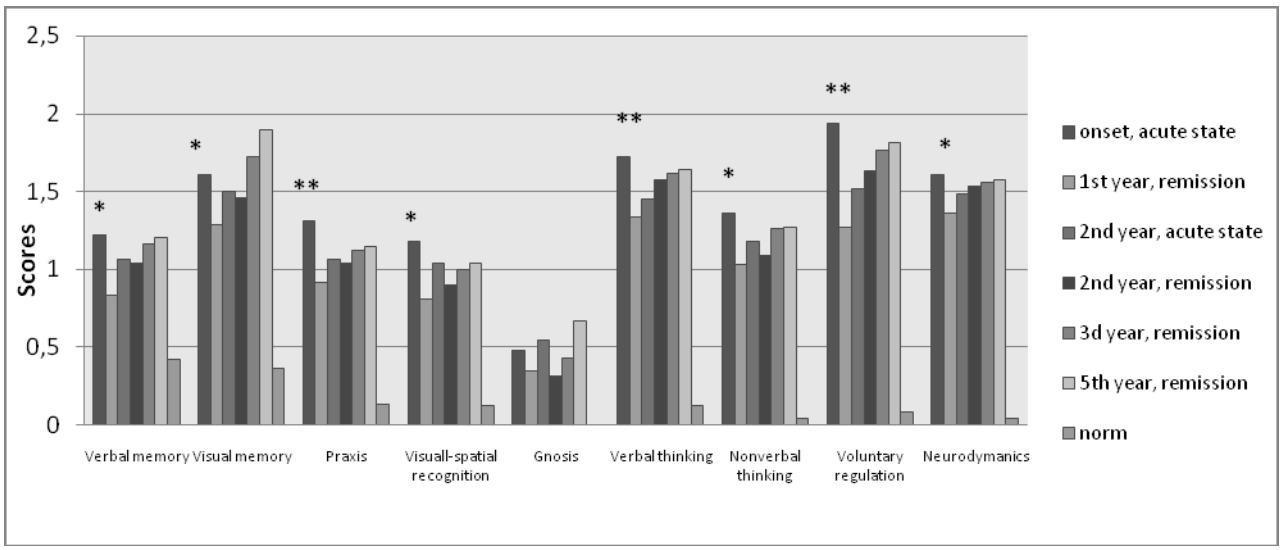
Compared with healthy subjects, the overall patients group had significantly worse qualitative characteristics of neurocognitive functions with relatively similar cognitive profile at the onset. Neurocognitive deterioration in patients of the 1st group fluctuated being worse during onset and relapse and improved in remission. Parameters of praxis, gnosis, visual-spatial recognition, verbal thinking and non-verbal thinking were changeable depending on the phase of the disease (basic cognitive deficit), while verbal and visual memory, neurodynamics and executive functioning, remained impaired even in five years, though insignificantly improving in remission (Variable_cognitive symptoms) (Figure 1).

Patients of the 2nd group presented a deficit of the main cognitive functions (verbal and visual memory, gnosis, spatial motor skills, verbal and non-verbal thinking, attention, neurodynamics and executive functioning) during the first 2 years which corresponded to the worsening of clinical state over time. Later on these variables remained stable with further decline of visual memory and visual gnosis by the end of 5 year follow-up. The third year of the follow-up was characterized by the cognitive "plateau"(stabilization of impaired cognitive functions) However, we detected further decline of visual memory and visual gnosis by the end of 5 year follow-up (Figure2).



Comment: ** - p <0.01, * - p <0.05 according to Wilcoxon criterion

Figure 1. Dynamics of the neurocognitive functioning of the group 1during 5-year follow-up



Comment: ** - p <0.01, * - p <0.05 according to Wilcoxon criterion

Figure 2. Dynamics of the neurocognitive functioning of the group 2 during 5-year follow-up

Conclusions

The following results indicate different courses of cognitive deterioration in the follow-up period in patients with good and poor outcomes after the first psychotic episode and during 5-year follow-up. Patients with favorable outcome demonstrate a stable basic neurocognitive deficit with insignificant improvement in long term follow-up. Other neurocognitive features are changeable along with the particular phase of the disease. Results of the patients with poor outcome are consistent with the notion of "biological toxicity of the first 2 years of illness (Birchwood et al., 1998). Neurocognitive deterioration is related to the relapses and unfavourable course of the illness, therefore, the peculiarities of cognitive impairment at the baseline and during the course of the illness in patients with poor outcome could have a predictive value for the poor prognosis.

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