The neurocognitive dysfunction in adult patients with bipolar I disorder type

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Abstract

To study the strategies of optical and spatial activities, perceptual integrity of perception in patients with bipolar I disorder type – and analyze the effectiveness of pharmacotherapy. The initial analysis revealed impairment in executive functioning, modality-unspecific disorders of selective memory and attention in both manic and depressed bipolar patients. These disturbances are, in neuropsychological aspect, pathognomonic for dysfunction of the right hemisphere of the brain on the background of the left hemisphere’s general hyperactivity [8, 9]. Patients with inadequate response to treatment (a mood stabilizer, an antidepressant) showed signs of dominance of right and bilateral functional disorders of the brain.

Kew words: neurocognitive dysfunction, bipolar I disorder type.

Affective disorders are among the most common groups of psychiatric disorders in developed countries. Bipolar affective disorder (or mood disorder) include manic, hypomanic, depressive, mixed episode and is associated with specific neurocognitive deficits consistent with neuropathology in cerebello-striatal-prefrontal neural networks [1, 2]. The visuospatial and executive function are the basic cognitive functions that ensure full implementation of individual mental activity. The visuospatial representation is fully actualized by the right hemisphere of the brain and provides implementation of somatognostic, metric, structural and topological parameters of mental activity.

Patients with bipolar disorder had cycling through episodes of mania, depression, and euthymia. They demonstrated fluctuations in energy, social behavior, mood and cognitive function. An important factor of this disorder is the relationship between mood and cognition. The nature of cognitive deficits in patients with bipolar disorder and how these deficits relate to the clinical symptoms and neuropathology of the bipolar disorder is not clearly known so far. Few researchers have investigated cognitive functioning in patients with manic-depressive illness [3]. The majority of neuropsychological studies have been conducted in patients with bipolar disorder type 1 (the history of at least one of the manic episode). Neurocognitive dysfunction associated with verbal and visual memory, and speech disorders were identified. The early onset of BP I is linked to a high risk of subsequent cognitive dysfunction. Untreated depressive patients have a marked impairment of visual recognition of the facial expressions, multiple disturbances of attention and speech. Neurocognitive deficits have been reported basing on tests of short-term memory, verbal and visual recognition memory, spatial working memory and immediate or delayed response [4-6]. Patients with mania have been studied in certain aspects of learning and memory, visuospatial ability and executive function. In a study performed by Taylor & Abrams (1986), tests of attention, visuospatial function and memory were administered to patients with mania, approximately half of whom exhibited moderate or severe global cognitive impairment. With respect to memory processes, Bunney & Hartmann (1965) detected memory loss during manic states in a patient with regular manic-depressive cycles over every 48 hours. Furthermore, Henry et al (1971) reported impaired serial word list learning during mania, with decrements in performance directly related to the increasing severity of illness [3]. Murphy et al (1999) in their recent findings have suggested that patients with bipolar disorder in the manic phase of their illness are impaired in tests of pattern and spatial recognition memory and have a delayed visual recognition. In an attempt to explain the observed memory deficits, Henry et al (1971) proposed that memory impairment may at least sometimes be due to altered patterns of verbal association. Andreasen & Powers (1974) made a similar conclusion in their findings: relative to the control subjects, the memory structures of patients with mania were loose. A specific neuropsychological profile for mania could prove fruitful for a more general investigation of mood and cognition. Some features of cognitive deficit might be common for the both poles of this disorder. These studies determine whether the impairments observed in mania can be explained by the factors specific to the manic state or whether they are, alternatively, due to global pathology and more general problems, such as psychosis or disordered thought [3]. E.Kraepelin (1921) distinguished manic depression from schizophrenia based on its relapsing and remitting course.

Patients with affective illness, unlike those with dementia praecox, were thought to have remission with cognition mostly unimpaired. Recent studies of patients...
in the euthymic phase of bipolar disorder have challenged this view. Some of the BP patients are suffering from psychological and social difficulties while the extent of their neuropsychological impairment remains unclear.

Bipolar affective disorder (BD) is characterized by fluctuating affect and mood, include manic, hypomanic, depressive, mixed episode and is associated with specific neurocognitive deficits consistent with neuropathology in cerebello-striatal-prefrontal neural networks [1, 2]. There was a significant reduction over time in overall capacity bilaterally in the anterior and posterior cingulate gyri, medial prefrontal cortices, inferior and middle frontal cortices, precentral gyri, right inferior parietal cortex, left precuneus and cuneus, and cerebellum. A significant reduction in dynamic range over time was found in the following regions in the right brain: lingual and inferior occipital cortices, hippocampal region, inferior frontal cortex, insula, inferior parietal and superior temporal cortices, precentral gyri, and cerebellum [7]. It appears that there is a positive correlation between the symptoms of psychotic disorders, age of onset and duration of the disease, the severity of cognitive impairment, and with the risk of dementia. Correlations between obstetric complications, infection with herpes simplex virus type 1 (HSV-1) and childhood trauma were detected.

Bipolar disorder is a polymorphic disease with different outcomes. Some patients noted at the outset more severe clinical course. Other patients with frequent recurrences had a good functional recovery in remission. Rubinstein et al (2000) found asymptomatic patients with bipolar disorder (in remission for at least 4 months) to show deficits on tests of visuospatial recognition memory; response latency, but not accuracy, on four distinct tests of executive function, was also impaired. Other investigators have reported evidence of residual impairment as well [3].

The similarities between the organic dementia and bipolar spectrum disorders have real diagnostic and therapeutic problems. Low social activity, fatigue, and lack of initiative, impaired sleep-wake cycle, delusions are observed in both of these states. These symptoms, apathy, anti-social behavior, cognitive impairment are similar to frontal lobe dysfunction [8]. These neuroimaging studies have demonstrated changes in the parenchyma, hippocampus and amygdala, expansion of the right lateral ventricle, changes in the associative cortex (prefrontal, anterior cingulate and dorsolateral prefrontal core).

Patients with bipolar disorder are generally receiving a combination of medications – including mood stabilizers, antidepressants, neuroleptics and benzodiazepines – that may or may not influence neuropsychological performance. Bipolar disorder treatment of neurocognitive dysfunction is defective without specific violations and verification pathogenesis and etiological causes. A comprehensive understanding of the manic, depressed and euthymic phases of bipolar disorder and their association with neurocognitive dysfunction is important for the successful management and possible remedy of this debilitating disorder.

To study the strategies of optical and spatial activities, perceptual integrity of perception – coordinate, metric, structural, topological, projective representations in patients with bipolar disorder – and analyze the effectiveness of pharmacotherapy.

**MATERIALS AND METHODS**

20 manic bipolar patients and 20 depressed bipolar patients aged 28 to 50 years were assessed as for visuospatial function memory, attention, planning, and working memory (executive functions) using the Rey-Osterrieth Complex Figure Test (ROCF) and Taylor Complex Figure test. The research was carried out under sensitization conditions. The assessment was performed prior to the treatment and 12 weeks after the treatment start. Treatment: mood stabilizer (valproic acid, quetiapine), antidepressants.

**RESULTS AND DISCUSSIONS**

We revealed failures of dynamic components of mental activity of varying severity in all the patients, impaired visual-spatial syntheses of various degrees in 90% of manic BD patients and in 85% of depressed BD patients with relapse of BD. 35% manic BD patients had moderate impairment of visuospatial and executive function, 60% of patients had mild impairment of visuospatial and executive function. In 25% of depressed BD patients we revealed mild impairment, 50% of patients had moderate impairment of visuospatial and executive function. The initial analysis revealed impairment in executive functioning, modality-unspecific disorders of selective memory and attention in both manic and depressed bipolar patients. In nonspecific memory deficits, also noted at the stabilization period, we observed minor improvements after psychotropic treatment. Bipolar-depressed and manic patients differed from each other with respect to the nature of their memory impairment. Depressed patients were more impaired than manic patients at tests of visuospatial response and fine motor skills. Both groups were characterized as for the integrity of perception, randomness of strategy copying, flaws in metric-form calculation of distances, angles, proportions, failures of to copy geometric objects and individual elements, failures of joining lines and points of intersection. These disturbances are, in neuropsychological aspect, pathognomonic for dysfunction of the right hemisphere of the brain on the background of the left hemisphere’s general hyperactivity [8, 9]. Patients with inadequate response to treatment (a mood stabilizer, an antidepressant) showed signs of dominance of right and bilateral functional disorders of the brain.

**CONCLUSION**

The visuospatial and executive dysfunction in BD is an issue of profound clinical and research interest that warrants further investigation. Its evaluation in BD may be an objective criterion of prognosis and response to treatment. It is important to develop individual-treatment strategies in BD in future research that would be able to define the neurofunctional state of the brain structures in a better way and to determine their functional impact.

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