

Treating the Cognitive Impairment in Bipolar Patients

Michel Bourin*

Department of Neurobiology of Mood Disorders, University of Nantes, France

An important syndrome of bipolar disorder is the cognitive impairment that is observed in the patients it must be considered as a part of the illness.

Bipolar disorder is a high priority research field, due to its pervasiveness and high economic and personal (suicidality, impaired function, quality of life) costs and the limited evidence base to inform therapeutics. Mood stabilizers and second-generation antipsychotics are partially effective regarding cognitive impairment of mood disorders.

What is red in literature are descriptions of mood elevation that reflect symptoms of high energy, lessened need for sleep, feelings of euphoria, grandiosity, impulsivity, elevated libido, etc. But when patients are on the depressed end of the mood spectrum, they are presenting with low energy, low self-esteem, feelings of sadness, loss or emptiness, suicidal ideation, pervasive pessimism, low motivation, and all the other experiences we associate with feeling depressed. But bipolar disorder patients exhibit cognitive impairments even during euthymic states. There seems to be a fairly broad consensus in the research literature that for some with bipolar disorder, the presence of cognitive deficit is not just a reflection of mood intensity, but an enduring element of the illness itself. Yet, in my opinion, deficit of concentration even weak is linked with a small mood disorder and often it is the aura of a depressive episode coming a week later. So for now, it is almost impossible to use cognitive measures to establish a "discontinuity" (between normal and pathological) and serve as diagnostic criteria for bipolarity. The nature of the cognitive deficits observed after the onset of the bipolar problem appears to be stable. On lack of data in patients with a first thymic episode. However, the hypothesis of cognitive deterioration in the early stages of clinical expression of bipolarity cannot be considered. This deterioration associated with the beginning of the debate fits well with studies that showed normal or even superior cognitive functions before the onset of the disorder.

They are positive correlation between cognitive deficit and higher bipolar depression or hypomania or mania. This means that patients with histories of depressive or hypomanic or manic episodes are experiencing more aspects of cognitive deficit. There are also more and more findings that point out those patients which are well compliant with their treatment with mood stabilizers have less cognitive impairment. It is well known that patients who have experienced a more difficult course of their disorder because of residual symptoms, or treatment noncompliance and/or unhealthy lifestyle choices suffer more cognitive impairments, and although a growing number of studies have demonstrated the benefits of psychotherapy as an add-on to pharmacological treatment, its effectiveness appears to be less compelling in severe presentations of the disorder. New interventions have attempted to improve cognitive functioning in BD patients, but results have been mixed. Stimulants drugs such as methylphenidate, modafinil and armodafinil have been used as adjuncts in bipolar patients mainly depressed but as well in euthymic patients with encouraging results. But it is often case reports and not double blind controlled studies. There are not evaluation of efficacy and safety [1,2].

Treatment approaches should consider not only euthymia as a goal but also cognitive and functional improvement of patients with such

a complex disorder [3]. Functional remediation and psychoeducation among psychological interventions may help to enhance functioning. Social cognition impairment, especially impaired Theory of Mind (ToM) [4], might also play an important role in bipolar patients' every-day functioning, The combination of cognitive enhancers and cognitive/functional remediation programs may help in improving cognitive and functional impairments. Early interventions are essential to prevent cognitive deficits and disability.

There is no established efficacious treatment for cognitive dysfunction in bipolar disorder. This may be partially due to lack of consensus regarding the need to screen for cognitive impairment in cognition trials or which screening criteria to use. Neurocognitive dysfunction in BD patients has been related to several clinical factors, but data on the effect of medication are relatively scarce and inconsistent. Neurocognitive dysfunction in bipolar patients could be a possible marker of underlying pathophysiology, but most studies are heterogeneous regarding the psychopharmacological treatment. Non-pharmacological strategies, such as cognitive remediation and exercise, are increasingly studied in patients with mood disorders [5].

My own practice experience for years leads to differentiate bipolar patients presenting with Attention-Deficit/Hyperactivity Disorder Symptoms in childhood with other bipolar patients. Often these bipolar 2 patients are impaired with major cognitive symptoms most of them are responding with 30 mg of methylphenidate in combination with their usual drug mood stabilizer treatment. On the other hand, other patients with stable mood but still cognitive impairment or other with depressed mood with anhedonia or even with anhedonia alone, were treated with methylphenidate in combination.

Despite the growing interest in strategies to manage cognitive function, there are few trials testing either pharmacological or non-pharmacological modes of interventions. We need to have controlled clinical trials in this field and of course to have validated tools to assess specifically cognitive impairment in bipolar patients [6].

Finally, cognitive impairment which is more often an attention deficit is a marker of non-euthymia in the patient, so it is interesting to consider methylphenidate as a help to mood stabilizers.

References

1. Szmulewicz AG, Angriman F, Samamé C, Ferraris A, Vigo D, et al. (2017) Dopaminergic agents in the treatment of bipolar depression: A systematic review and meta-analysis. *Acta Psychiatr Scand* 135: 527-538.

*Corresponding author: Michel Bourin, Department of Neurobiology of Mood Disorders, University of Nantes, 98, rue Joseph Blanchart, 44100, Nantes, France, Tel: 33240734511; E-mail: michel.bourin@univ-nantes.fr

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2. Berk M, Dodd S, Kauer-Sant'anna M, Malhi GS, Bourin M, et al. (2007) Dopamine dysregulation syndrome: Implications for a dopamine hypothesis of bipolar disorder. *Acta Psychiatr Scand Suppl* 434: 41-49.
 3. Emilien G, Septien L, Brisard C, Corruble E, Bourin M (2007) Bipolar disorder: How far are we from a rigorous definition and effective management? *Prog Neuropsychopharmacol Biol Psychiatry* 31: 975-996.
 4. Lecce S, Ceccato I, Rosi A, Bianco F, Bottiroli S, et al. (2017) Theory of mind plasticity in aging: The role of baseline, verbal knowledge and executive functions. *Neuropsychol Rehabil* 7: 1-16.
 5. Sanchez-Moreno J, Martinez-Aram A, Vieta E (2017) Treatment of functional impairment in patients with bipolar disorder. *Curr Psychiatry Rep* 19: 3.
 6. Burdick KE, Ketter TA, Goldberg JF, Calabrese JR (2015) Assessing cognitive function in bipolar disorder: Challenges and recommendations for clinical trial design. *J Clin Psychiatry* 76: e342-e350.