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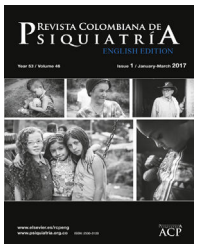

Review Article

Neuropsychological aspects of bipolar disorder

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ABSTRACT

Bipolar disorder (BD) is a chronic condition with serious consequences on the health and functionality of patients who suffer from it, with a high heritability and segregation, and aprevalence of between 1% and 2%. Neuropsychological deficits have been implicated as a very important issue related to BD prognosis, so a review was conducted of these deficits, the related factors and their functional consequences. It has been determined that the presence of neuropsychological deficits can vary in patients with BD according to their mood state, with a great influence of depressive symptoms on the cognitive variability of patients with respect to the general population and differences with respect to patients in the manic phase. In euthymic patients, the most affected cognitive domains are those of memory, attention, and executive function, associated with a more severe disease, sociodemographic vulnerability factors, and stable over time. A relationship has been found between poor cognitive performance, especially executive dysfunction, and objective functional deficit. Furthermore, cognitive differences have been outlined between BD and other serious mental illnesses that are described in this review.

Aspectos neuropsicológicos del trastorno afectivo bipolar

Palabras clave:
Trastorno afectivo bipolar
Manía
Depresión
Eutimia
Neuropsicología

RESUMEN

El trastorno afectivo bipolar (TAB) es una entidad crónica con graves efectos para la salud y funcionalidad de los pacientes que la presentan, con una alta carga de heredabilidad, segregabilidad, y con una prevalencia que oscila entre 1% y el 2%. Las alteraciones neurpsicológicas son características importantes relacionadas con su pronóstico, por lo cual se hizo una revisión narrativa sobre estas alteraciones, los factores asociados y sus consecuencias funcionales. Se ha determinado que la presencia de alteraciones neuropsicológicas puede variar en los pacientes con TAB de acuerdo con la fase anímica en que se encuentren, con

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Cognición
Funcionalidad

una gran influencia de los síntomas depresivos en la variabilidad cognitiva de los pacientes respecto a la población general, con diferencias respecto a los pacientes en fase maníaca. En pacientes eutímicos los dominios cognitivos más afectados son los de memoria, atención y función ejecutiva, asociados con una mayor gravedad de enfermedad, factores sociodemográficos de vulnerabilidad y sin interacción con el tiempo de evolución. Se ha encontrado una relación entre el pobre rendimiento cognitivo, especialmente la disfunción ejecutiva y el déficit funcional objetivo; adicionalmente, se han perfilado diferencias cognitivas entre el TAB y otras enfermedades mentales graves que se describen en la revisión.

Introduction

The global prevalence of bipolar disorder (BD) is estimated at 1%–2%, according to the World Mental Health Survey.[1] However, underdiagnosis must be taken into account, as reportedly more than one third of diagnoses of depressive disorders correspond to BD.[2] There are various markers of disease burden, such as comorbidity with substance use (around 50%)[3] and other psychiatric entities (up to 75%), in addition to an increase by up to 25 times in suicidal behaviour, comparing patients with BD to the general population,[1] and an increase in mortality due to cardiovascular causes up to two to three times that of the general population. In Colombia, the prevalence of bipolar I disorder (BD-I) is estimated at 1.3%, and that of bipolar II disorder (BD-II) is estimated at 0.2%, with a strong association with factors of psychosocial adversity.[4]

The disease burden of BD, measured in healthy life years (HLY) lost annually in the world, has been estimated at around 9.9 million, 1.5 million of them 25–29 years of age. This represents enormous economic costs for states, most of them indirect.[5] Colombia lacks projections of expenses associated with BD, beyond estimates for outpatient drug treatment.[6] Research on BD has sought new explanations and factors that contribute to disability and increase BD costs and disease burden. In this regard, BD is believed to be a condition with intercritical manifestations that can be chronic; in fact, just one third of those who emerge from an acute episode recover full functioning, and current research on neurocognition and abnormalities thereof in BD has found associations with patient functioning.[7] Hence this review of the evidence and report of the most important findings on neuropsychological performance in patients with BD, factors associated with this and the functional consequences thereof. The review concludes with a summary of methodological recommendations from the most renowned expert groups for optimising and conducting empirical studies in this field.

Methods

A literature search was conducted in December 2019, with no time limits, using the following databases: Scopus, APA PsycNet, AccessMedicine, AccessMedicina, Embase, JAMA Network, Lilacs, Scielo, Ovid and PubMed. The MeSH terms "Bipolar Disorder" and C̈ognitionẅere used, connected with the AND Boolean operator. The titles and abstracts of the articles retrieved were reviewed, and those that were eligible were subsequently read in their entirety to determine the quality and pertinence of the information reported. Next, a narrative review of the literature was conducted.

Cognitive abnormalities reported in BD and phases thereof (cross-sectional studies)

Mania

Patients experiencing their first manic episode were found to have deficits in executive function, with no examination of other domains; during subsequent remission, evidence was found of deficits in working memory,[8] and interaction between these deficits and poor mechanisms of emotional regulation was seen to be linked to more severe manic episodes.[9] One study of subjects during a manic episode found cognitive deficits compared to healthy controls, especially in the areas of verbal memory and executive function, but surprisingly found no association between psychotic signs and worse neuropsychological performance.[10] Moreover, when patients with predominantly manic polarity were compared to those with predominantly depressive polarity and healthy controls, they were found to have deficits in executive function, verbal fluency and short-term memory; factors such as number of episodes and history of psychosis had no impact.[11]

Depression

Patients having a depressive episode showed worse cognitive performance evaluated using the Brief Assessment of Cognition in Affective Disorders (BAC-A), and in weighted affective processing also showed deficits due to depressive bias.[12] Among patients with a moderate to severe depressive episode, those with BD-I had greater deficits in categorical fluency and general intelligence quotient (IQ) than those with BD-II and controls; furthermore, in BD-I, there was a higher proportion of patients in a range of cognitive disability than in BD-II (68.4% versus 37.5%), predominantly affecting processing speed as well as general IQ and decline in IQ compared to calculated premorbid IQ, influenced by age.[13] One study reviewed proposed that mild to moderate depression would be correlated with worse performance in executive function, processing speed and attention, with a moderate strength of

association, and even noted that depressive symptoms would account for up to 24% of the difference in cognitive performance between patients with BD and healthy controls.[14]

Euthymia

Patients with BD show a subtle tendency towards worse cognitive performance than healthy controls. Subgroups have been reported: one with intact cognition (43.5%); one with selective decline primarily in working memory, executive function and processing speed (33.3%); and one with overall decline (23.3%).[15] The greatest decline tended to occur in executive function and working memory, indirectly associated with a history of psychosis.[16] Said influence of a history of psychosis was repeated in general cognitive performance, and the author of one meta-analysis reported a much higher proportion of patients with BD and severe cognitive deficits compared to healthy controls, with a history of psychosis exerting a moderate influence.[17] Strikingly, in a prior study, the same author did not find a link between a history of psychosis and neuropsychological deficits.[18]

There is debate as to the influence of clinical factors on cognitive performance. For example, numbers of hospitalisations and episodes of mania/hypomania have been associated with worse performance in terms of memory, attention and executive function.[19,20] The Grupo de Investigación en Psiquiatría [Psychiatry Research Group] (GIPSI) of the Universidad de Antioquia [University of Antioquia] reproduced those results in the domains of attention and executive function, although the group did not reproduce the influence of a history of psychosis on neurocognitive performance,[21] which has indeed been reported in other studies.[16,17,22,23] In addition, there is conflicting evidence on the association between cognitive status and age; some studies have linked an early onset (before age 22) to worse performance,[24] while there is evidence of a late onset (after age 40) being linked to greater deficits.[25] Previously, Martino et al.[26] had reported an association between BD in patients over 60 years of age and worse psychomotor speed, verbal memory, executive function and extrapyramidal symptoms, the latter two being linked to worse psychosocial functioning. Another group arrived at similar findings on cognitive performance in patients over 60 years of age with BD with an onset before age 50, as these patients had deficits in the domains of attention, memory and verbal fluency.[27] The same study team reported the influence of premorbid IQ on poor general neurocognitive performance, in addition to the association of vascular risk factors, particular to advanced ages, with attention deficits in patients with BD.[28] Sleep has been examined in association with cognitive deficits in patients with BD; in fact, one of the most recent studies indicated that said factor would be determinant of or potentially cause patients' cognitive problems.[29] Most authors have not reported treatment to have an impact on cognition, although there have been reports of a link between treatment and antipsychotics with worse general performance,[18,30] while one study produced evidence of potential neuroprotective effects of treatment with lamotrigine, especially in the domains of fluency and verbal memory, with a moderate effect size.[31]

Among sociodemographic factors associated with cognitive performance, the most commonly reported has been years of training, associated with general cognition,[24,32] executive function[33] and memory.[20] Making good use of leisure time, autonomy and occupational performance have been correlated with good cognitive performance.[33] Another factor examined was biological sex; healthy men had better performance in visual memory and paired associative learning than women, but when this comparison was made in patients with BD, said difference disappeared and the two sexes showed equally low performance.[34] Moreover, it has been proposed that a model combining male sex with other variables, such as low estimated IQ, poor executive function and a family history of mood disorders may account for up to 27.6% of variance in patients with BD with respect to emotional intelligence.[35]

Quality of life was examined in a study using the 36-Item Short Form Survey (SF-36), which correlated quality of life with cognitive reserve (CR) and found that a large CR was associated with a good quality of life in the physical domain, while the relationship was inverse in the mental domain.[36] It has also been found that patients with a subjective decline in cognitive capacity reported a lower quality of life, this being more significant for patients with a chronic course.[37] Regarding psychosocial functioning, there is evidence of the association thereof with cognitive domains such as verbal memory.[38]

Specific cognitive domains affected in BD

The deficits most commonly reported in studies were in executive function, attention and memory.[18,38] The evidence on specific cognitive deficits in BD is presented below.

General intelligence

The data were conflicting. One study found no significant deficits in intelligence,[16] while another reported a difference in IQ (97.33 ± 7 versus 112.54 ± 11.5) between patients with BD and healthy controls.[20] One author reported that negative symptoms and executive dysfunction were associated with a lower IQ.[39]

Executive function

The international literature has extensively reported executive dysfunction in patients with BD.[16,20,39,40] This finding has been reproduced in Colombia in the Universidad de Antioquia and the Universidad de San Buenaventura [University of San Buenaventura],[21,41] and memory dysfunction has been found to be an apparent epiphenomenon of executive dysfunction.[42] In this regard, differential analysis of executive function has yielded the finding that patients with BD show a focal deficit in what is known as dorsolateral function, with dysfunction in the subdomains of initiation, sustained attention, localisation of attention and planning.[39] Reports of executive dysfunction in a range of disability (cut-off at –1.64 standard deviations) have been as high as one out of every three euthymic patients,[33] with evidence of a late onset of the disorder adversely affecting executive function.[25]

Memory

Patients with BD showed deficits on memory tests compared to the general population. Said deficit was correlated with fewer years of education and larger numbers of manic/hypomanic episodes and hospitalisations.[20] There have also been reports of discrepant findings in different memory modalities in BD. One study found a decline in working memory and determined that a history of psychosis was correlated with worse performance in said domain, while other cognitive domains did not show that influence.[16] Another study showed that patients with BD had deficits in immediate and short-term memory, and indicated that neuropsychological profile and interaction between memory and executive function may be phenotypical markers of BD.[39]

Attention

Compared to the general population, attention deficits have been documented using neuropsychological tests in patients with BD. This deficit has been associated with numbers of manic/hypomanic episodes and hospitalisations.[20] Analysis of attention deficits and their association with other clinical characteristics has revealed that patients with late-onset BD (in one study, defined as BD with an onset after age 40) showed greater impairment of the domain of attention.[25] The neuropsychology group of the Universidad de Antioquia also found deficits in attention, with a large effect size,[41] and one study reported low performance specifically in sustained attention in patients with BD.[43] From the evidence reviewed, it is concluded that there is no clear association between attention deficits and poor functioning in euthymic patients with BD.

Other neuropsychological domains

Studies on processing speed have yielded conflicting findings. Some have not reported significant deficits,[16] while there is evidence of moderate to severe deficits.[38] An association has been shown between deficits in this domain and functioning measured using validated instruments.[44] One of the authors mentioned reported that the patients with BD in their study showed no deficits in the domain of verbal fluency,[16] while another author did report this in a group of patients with late-onset BD, in addition to the fact that these patients, as they were more affected, also showed poor performance on tests that evaluate naming.[25]

Longitudinal studies on cognitive function in BD

Until now, the primary objective of this sort of study has been to report changes over time in cognitive functioning, as in a study by Santos et al.[45] with 80 patients with BD (22.5% losses) and 40 healthy controls, with a five-year period of clinical follow-up and neuropsychological assessment using the MATRICS Consensus Cognitive Battery (MCCB) and the California Verbal Learning Test (CVLT); that study's results reported stable chronic deficits in memory, verbal learning, processing speed and executive function, with no association with clinical factors. Another study reported very similar findings in data for 76 patients with BD-I and 40 healthy controls, also with a five-year clinical follow-up period, and evaluated the neuropsychological profile of the participants also using the MCCB; the findings showed significant deficits in general neurocognition as well as the domains of working memory, visual memory, processing speed, attention and executive function in patients with BD, with no overwhelming evidence of neuroprogression, beyond non-significant decline, in patients with larger numbers of manic/hypomanic episodes in general cognition and working and visual memory.[46]

Another study, which had 31 patients with BD paired by sex and age with healthy controls undergo the Cambridge Neuropsychological Test Automated Battery (CANTAB) on two occasions, with a two-year follow-up period, found significant deficits in psychomotor speed and sustained attention (executive function), without changes over time.[47] One study had similar results to the above-mentioned studies; it included 61 patients (55.7% losses) 15–25 years of age with a first episode of mania in remission and a diagnosis of BD, schizoaffective disorder (bipolar subtype) and mania secondary to substance use, in addition to 21 healthy controls (33.3% losses), with a one-year follow-up period. The group with a first manic episode had worse general cognitive performance, with an improvement in working memory and processing speed. All other deficits remained stable, except for the screening test, which worsened, although no association with clinical variables was seen. In addition, there was a learning effect and an improvement in response inhibition in the controls that did was not seen in the patients with a first manic episode.[48]

Daglas et al.[48] and other authors have found an improvement in neurocognitive performance as part of the normal course of BD in its early phases. For example, Volkert et al.[49] evaluated 35 patients having a depressive episode, 20 having a manic/hypomanic episode and 55 healthy controls with a three-month follow-up period. The results showed general cognitive impairment during the acute episode, with a predominance in psychomotor speed in the depressive episode and executive function in the manic/hypomanic episode. Following remission, there was an improvement in nearly all cognitive domains, although verbal and working memory remained markedly affected. In addition, subclinical depressive symptoms and sleep disturbances were associated with a decline in psychomotor speed, attention and verbal memory, while impairment of working memory was associated with a history of chronic psychotic symptoms. Another study yielded similar findings in adolescents 12–17 years of age with BD who had commenced medical follow-up two years earlier (n = 20), compared to healthy controls (n = 20), after a mean follow-up period of 33 months. That study found that patients with BD had worse general cognitive performance, but an improvement in verbal reasoning and working memory, and even had normal processing speed and visual motor abilities; taking lithium was associated with worse cognitive performance, and the influence of a history of psychosis was reproduced.[50] Yet another study focused on the natural history of recovery from a mood episode in patients with BD from another perspective, taking a sample of 26 patients with BD in their first hospitalisation and 20 healthy controls, who underwent two

neurocognitive evaluations 12 months apart, accompanied by monthly clinical follow-up. The study found that patients with greater executive dysfunction spent more time recovering from the acute episode, while there was a similar, though much weaker, effect for the domain of verbal fluency.[51]

Although there is some evidence on the influence of psychiatric drug treatment on cognitive performance, as reported by Lera-Miguel et al.[50] on the association between lithium and worse cognitive performance, this finding has not been consistent. For example, Daglas et al.[48] evaluated quetiapine and lithium and did not find said association in patients who were also young. However, after six years of follow-up, one study in a cohort of 28 patients with BD and 26 healthy controls found an association between treatment with lithium and worse cognitive performance in the domains of impulse inhibition, processing speed and verbal memory, stable over time and associated with worse psychosocial functioning measured using the Functioning Assessment Short Test (FAST);[52] the findings of this study were similar to those reported by Gildengers et al. in 2013.[44]

One study examined cognition in 65 patients over 60 years of age and 42 healthy controls, but had losses to follow-up that might have introduced bias, as the average IQ of the original cohort was lower than that of the cohort used (106.97 versus 119.1). The results again showed worse general cognitive performance in patients with BD, with no significant changes over time and no association with clinical variables.[53]

Efforts have been made to establish the association between clinical factors in BD and cognitive performance. One study evaluated 51 patients with BD and 39 healthy controls, with an average clinical follow-up of 73.21 months and neuropsychological evaluation on two occasions, taking into account three variables tied to clinical course — numbers of episodes, time spent ill and emotional instability — in addition to a detailed treatment record. Patients with BD had deficits in attention, verbal memory and executive functioning, with no worsening over time and no association with clinical variables, except for a statistically insignificant correlation between verbal memory and numbers of manic and hypomanic episodes.[54] These findings strongly resembled those of a study that compared 50 patients in remission with BD, 50 with schizophrenia and 51 healthy controls: a decline predominantly in attention and working memory in patients with schizophrenia and BD with a history of psychosis, with no significant changes in a five-year follow-up period and no association with psychosocial functioning.[55]

General cognitive deficits in BD, association thereof with functioning and comparison to other disorders

Some studies in patients with BD have evaluated the link between cognitive complaints and functioning and yielded conflicting findings. One study reported a significant association between perceived cognitive deficit and low motivation to work,[56] while another reported an association between subjective cognitive complaints measured using the Massachusetts General Hospital Cognitive and Physical Functioning Questionnaire (CPFQ) and functional deficits measured using the Work and Social Adjustment Scale (WSAS), with apparent mediation of depressive symptoms; they found no association between functional deficits and cognitive deficits documented through neuropsychological tests.[57] Another study examined subjective cognitive complaints in patients with BD, measured using the Cognitive Complaints in Bipolar Disorder Rating Assessment (COBRA) questionnaire and cognitive deficits measured using the Screen for Cognitive Impairment in Psychiatry (SCIP-D) objective cognitive screening instrument; comparison to functional deficits measured using the performance-based Assessment of Motor and Process Skills (AMPS) revealed a weak but consistent association between deficits in processing speed and day-to-day functioning, but no relationship between said functioning and subjective cognitive complaints.[58] This supports the concept that mentions the problems with cognitive assessment using subjective reporting by patients with BD, since there may be overestimation or underestimation of supposed deficits with mediation of mood symptoms.[7,59]

Some studies that have examined cognitive deficits measured using neuropsychological tests in BD have reported an association between functional deficits and decreased neuropsychological performance in the area of memory, especially verbal memory. For example, one measured functional performance using the FAST,[60] and another used the Multidimensional Scale of Independent Functioning (MSIF).[61] One study that examined functioning in a differentiated manner using an instrument that evaluated overall functioning (the eight-point Levenstein scale) and another that evaluated occupational and social functioning (Strauss and Carpenter scales) found low performance in memory to be associated with worse general, social and occupational performance, with a predominance of the influence of working memory on social functioning and of verbal memory on occupational functioning.[62]

The evidence reported has confirmed the central role of memory as a cognitive domain with respect to its influence on functioning; however, other cognitive domains have been reported, as in a two-year prospective cohort study that compared neuropsychological performance between patients over 50 years of age with BD (n = 47) and healthy controls (n = 22) and found in the BD cohort worse performance in overall neurocognition as well as the domains of memory, processing speed and executive function, with no evidence of neuroprogression, and an association between functioning measured using the Performance Assessment of Self-care Skills (PASS) and overall neurocognitive performance, but in a more marked way with the domains of executive function and processing speed.[44] Another study found a link between BD, low IQ, executive and memory deficits, and theory of mind shortcomings measured using the Cambridge Mindreading (CAM) Face-Voice Battery for decoding and difficulties with tasks of greater ecological validity in participants under 45 years of age.[63] Indeed, a relationship has been reported between limited functioning measured using the Schedule for Assessment of Psychiatric Disability (SAPD) and deficits in verbal memory, executive function and soft neurological signs of frontal disinhibition.[64] The association between cognitive performance, introspection and adherence to treatment in patients with BD has also been examined in search of another possible explanation for

the poor prognosis of people who suffer from the disorder, and there is evidence of said association, especially between executive dysfunction and disease awareness as a component of insight.[65–67]

The fact that there is also conflicting evidence on the association between cognitive deficits and functioning in patients with BD must not be overlooked. For example, one study found no association between low cognitive performance and functioning measured using the World Health Organization Disability Assessment Schedule (WHODAS 2.0) after comparing patients with BD and healthy controls, despite the fact that significant deficits were indeed found in overall cognition, attention, verbal memory and processing speed in patients with BD. However, in that study, patients with social cognitive deficits and poor theory of mind, measured using the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT) and the Reading the Mind in the Eyes Test (RMET), seemed to compensate for those problems through their overall cognitive reserve[68]; this was repeated in another similar study.[69] In addition, deficiencies in social cognition have been reported in patients with BD compared to healthy controls using other instruments, such as the MiniPONS, with no clear evidence of functional repercussions.[70] Moreover, in contrast to a study published by Yen et al. in 2008, one study reviewed did not find said correlation when it analysed neuropsychological deficits and adherence measured using the Medication Adherence Rating Scale (MARS).[71] In addition, cognitive deficits did not seem to be associated with poor coping strategies; one study that evaluated this using the Utrechtse Coping Lijst (UCL) found no association with subjective cognitive complaints and objective deficits measured neuropsychologically.[72] This conflicting evidence speaks to the wide phenotypic and phenomenic variability in this field of research, as well as the concept of cognitive heterogeneity, according to which up to 40% of patients with BD have normal cognitive performance, without losing sight of the fact that there are many criteria for classifying cognitive disability correlated with functioning measured by instruments. In this regard, a recent study examined the properties of various defining criteria for cognitive disability and found that the parameters of the Global Deficit Score (GDS) had the best psychometric qualities, including concurrence and convergence.[30]

Comparison of neuropsychological aspects of BD and other disorders

Neuropsychological differences between patients with BD-I and BD-II have been examined, and moderately worse performance in general neurocognition and the domains of working memory, verbal memory, sustained attention and executive function has been found in patients with BD-I compared to healthy controls, while patients with BD-II have shown intermediate performance between the other two groups, with preserved executive function compared to those with BD-I.[73] One meta-analysis reported better performance in the area of verbal memory in patients with BD-II than in patients with BD-I, with a moderate effect size, while the effect was weaker in the domains of visual memory and verbal fluency[74]; these findings were very similar to those of a prior cross-sectional study.[75]

When patients with BD have been compared to patients with schizophrenia, patients with BD have been found to show less cognitive and functional deficits, with specific impairment of memory, executive function, attention and processing speed. It should be noted that patients with BD and a history of psychosis showed more severe cognitive deficits, which were a little closer to the neuropsychological profile of people with schizophrenia, although the decline did not appear to progress.[17,23] Of the above studies, one found that both patient types showed deficits in functioning measured using the UCSD Performance-Based Skills Assessment–Brief Version (UPSA-B), in real-world functioning evaluated using the Specific Level of Functioning Scale (SLOF) and in social skills examined using the Social Skills Performance Assessment (SSPA), with worse performance by patients with schizophrenia, but with a history of psychosis also associated with worse performance in patients with BD. Unfortunately, the correlation with cognitive performance was not analysed in this study.[23] Another study looked for cognitive markers to distinguish BD from psychotic disorders taken as a spectrum, and found differences in psychomotor speed, which would be more affected in patients with schizophrenia, followed by those affected by autism spectrum disorder (ASD) and finally patients with BD, while the latter showed better performance in the domains of attention, executive function and processing speed.[76] Other studies have yielded the same findings in the domains of processing speed and executive function,[77,78] in addition to reporting greater decline in verbal memory in patients with schizophrenia and ASD (depressive subtype), upon comparison to subjects with BD, while the latter had far superior social cognition to participants suffering from psychotic disorders.[77] More descriptively, one study that sought to gain insight into aspects of executive function that could distinguish patients with primary psychotic disorders and BD with/without a history of psychosis found that, in addition to being more affected in said cognitive area, patients with schizophrenia and ASD had much higher rates of perseverative error on the Wisconsin Card Sorting Test (WCST). It also reported that patients could be grouped by performance profile on the Trail Making Test (TMT), with patients with psychotic disorders in one group and patients with BD in another;[79] here it is important to mention that prior studies had already demonstrated the relationship between perseverative error on the WCST and limited psychosocial functioning in patients with severe mental illness.[80]

Compared to depression, BD exhibited greater cognitive decline in the areas of delayed memory, executive function, language and visual motor abilities, with worse occupational performance as evidenced by reports indicating that patients with BD fail to fulfil their job duties up to 30% of the time, compared to 21% of patients with unipolar depressive disorder.[7] One study examined neuropsychological profiles in patients with BD-II and unipolar depression, both in a depressive phase, and found that patients with unipolar depressive disorder showed cognitive rigidity and affected processing speed, while patients with BD-II had a relatively intact cognitive profile, apart from those with a chronic course, who had affected verbal memory.[81] Standing in contrast to the above, a Chinese study reported mild to moderate deficits in various cognitive

domains in patients with BD-II and depressive disorder in a depressive episode, with no significant differences between the two groups.[82] BD and borderline personality disorder have also been seen to share executive deficits, these being more marked in BD[83]; however, said executive deficits were less serious in patients with BD than in others with conditions such as frontotemporal dementia, with which it has been compared from a nosological perspective.[84]

Recommendation for readers

To conclude, readers interested in gaining more in-depth knowledge of neurocognition in patients with BD are advised to refer to the work of Burdick et al.[85] and Miskowiak et al.[86] to learn about methodological aspects of research.

Conflicts of interest

The authors have no conflicts of interest to declare.

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