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Cognitive impairment among an Egyptian sample of patients with schizophrenia and bipolar disorders: a comparative study

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Abstract

Background The cognitive profile among patients with schizophrenia (SZ) and bipolar disorder (BD) has varied widely across different studies. The aim of the current study was to compare different cognitive domains using psychometric and neurophysiological tests in patients with SZ to those with BD. A case–control study was conducted on 30 BD, 30 SZ and 30 age and sex matched control group. Each subject was submitted to the following: Wechsler Adult Intelligence Scale-3rd edition (WAIS-III), Montreal cognitive assessment scale (MoCA), Brief Visuospatial Memory Test-Revised (BVMT-R), Memory Assessment Scales (MAS), and the P300 event related potential (ERP).

Results SZ and BD patients had significantly lower total and subscales of WAIS-III scores than the control group. SZ patients had significantly higher deterioration index (DI) than controls, while absence of such significant between BD and controls. SZ patients reported significantly lower MoCA scores and subitems, especially in visuospatial, naming, attention, delayed recall, and orientation subtests than controls. Only visuospatial and delayed recall scores were significantly decreased in BD than controls. SZ patients performed poorer on BVMT-R subscales than the control group. Both SZ and BD groups had lower mean values of all subscales except verbal assessment in the four memory tests. P300 latencies and amplitude had no significant difference among the three groups, although the BD group had a shorter P300 latency.

Conclusion Patients with SZ and BD had significantly lower scores on various cognitive function domains in comparison to controls with more affection in SZ. The frequency of mood episodes, disease duration, and education level must be considered.

Keywords Bipolar disorder, Schizophrenia, Cognition, Cognitive impairment

Background

Cognitive dysfunction is frequently observed in both schizophrenia (SZ) and bipolar disorder (BD) [1] and is significantly associated with impaired psychosocial functioning and diminished quality of life [2]. The extent of cognitive impairment varies widely across different studies and patients, ranging from mild to severe impairment resembling dementia [3]. As a result, cognitive dysfunction has become an essential measure of treatment outcomes for SZ and BD [4].

Recent research over the last two decades has provided clear evidence that BD is also associated with cognitive deficits and functional impairment, even in euthymic

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patients [5–7], while the severity of cognitive deficits in BD is less severe than in SZ, data suggests that the patterns of cognitive deficits in the two disorders are similar. These findings contradict other observations, such as the existence of a connection between BD, creativity, and premorbid academic achievement [8, 9].

However, methodological limitations such as the use of cross-sectional design, the inclusion of patients with chronic illnesses, and the lack of premorbid cognitive development information can limit our capacity to show possible cognitive variations between SZ and BD. One theory proposes that early cognitive abnormalities are specific to SZ [10, 11] since they have been seen during the prodromal stage and initial episode of SZ but not in BD condition. Another theory suggests SZ is characterized by cognitive abnormalities crucial for communication and social functioning (social cognition). Theory of mind (ToM) and other social cognitive deficits are more distinct in SZ than non-social cognitive abnormalities [12]. Some authors have also suggested that premorbid intellectual development issues can differentiate SZ from BD [13, 14]. There is significant neurobiological and clinical data overlap between SZ and BD [15].

Moreover, both illnesses share susceptibility genes. Nonetheless, familial, and genetic studies have provided evidence for schizophrenia- and BD-specific genetic risk factors [16, 17].

Furthermore, research suggests that BD with psychotic symptoms has cognitive deficits similar to SZ, notably in some respects such as episodic and working memory, executive, and attentional skills [18]. Some investigations, for example, showed no disparities in the severity of deficits between BP psychosis and SZ, especially in affective individuals with poor outcomes [19]. Others have discovered that people with SZ and BD with psychotic characteristics have a comparable neurocognitive profile, however the severity is greater in SZ patients [20].

Our objective was to evaluate cognitive function in patients with BD and SZ and compare it to a control group of healthy individuals. Additionally, we aimed to determine if each disorder exhibits a distinct pattern of cognitive dysfunction.

Methods

Participants and procedures

A case–control study was done at the outpatient clinic of the Psychiatry Department at Assiut University Hospital between August 2018 and July 2019. Patients diagnosed with schizophrenia and bipolar I disorder according to Diagnostic and Statistical Manual of Mental Disorders (DSM-5) [21] diagnostic criteria, aged between 18 and 60 years, were included in the study. For schizophrenia patients, the inclusion criteria required recurrent episodes, current full remission, while bipolar disorder patients should be in full remission. All patients must complain on their medication at least 6 months. Moreover, bipolar individuals were required to be lacking in acute mood symptoms for at least one month prior to enrolment, with euthymia defined as a 1-month score of 7 or less [22] on the Hamilton Depression Rating Scale 17-item (HAMD) [23] and Young Mania Rating Scale (YMRS) [24]. Exclusion criteria included comorbid substance use or personality disorders, medical illness or neurological disease, and illiterate subjects.

The recruited patients consisted of 2 groups (30 per group): group1 with BP and group 2 consisted of SZ. Additionally, a control group (group 3) was included, consisting of healthy volunteers.

Assessment tools

Psychiatric interview

Participants underwent a standardized psychiatric interview using the semi-structured Interview based on DSM-5 diagnostic criteria prepared by researchers to collect psychiatric and medical history upon enrolled to the study. Controls also underwent the interview to ensure the absence of psychiatric or personality disorders.

Neuropsychological assessment

Wechsler Adult Intelligence Scale-3rd edition (WAIS-III)

This test measures intelligence in adults and older adolescents, evaluating verbal, performance, total intelligence quotient (IQ), and deterioration index (DI). Various classifications are used based on IQ scores [25].

Montreal Cognitive Assessment Scale (MoCA)

This test is a brief 30-point assessment that measures various aspects of cognitive function. It includes tasks for short-term memory recall, visuospatial abilities, executive functions (alternation, phonemic fluency, and verbal abstraction), attention, concentration, working memory, language skills, and orientation to time and place [26].

Brief Visuospatial Memory Test-Revised (BVMT-R)

This test assesses visual memory through tasks involving recall, learning, and recognition of figures presented on a plate. Participants reproduce the figures from memory and recognize target and non-target figures [27].

Memory Assessment Scales (MAS)

This set of tests assesses short-term, verbal, and visual memory through tasks that test recall and recognition directly and after a period following stimulus presentation. The battery includes 12 subscales, covering verbal span, list learning, prose memory, visual span,

visual recognition, visual reproduction, and names-face recognition [28].

Neurophysiological assessment: event-related potential (ERP) test, P300 wave

The test was conducted using electromagnetic stimulation (EMS) biomedical equipment. Before running the test, the subjects were placed in a semi-dark acoustically quiet room, and they were allowed to adapt to the surrounding environment for a suitable duration. An oddball paradigm was applied, where the subjects had to recognize an infrequent target signal in a chain that included "frequent" signals. The stimuli were applied at a specific intensity and with varying interstimulus intervals. captured using scalp electrodes implanted at three different locations: central (Cz), frontal (Fz), and parietal (Pz), as well as their average value. The measured variables included P300 wave latency and amplitude. The test was repeated twice for each subject, and the mean result was obtained for analysis [29].

Statistical analysis

Continuous variables were reported as mean and standard deviation, whereas categorical variables were expressed with numbers and percentages. The normal distribution of continuous variables was tested using the Kolmogorov–Smirnov test and Q-Q Plots. The chi-square test was used to compare categorical variables, and the Mann–Whitney test was used for non-parametric variables, while ANOVA was used for normally distributed data. The spearman correlation coefficient was used to evaluate association factors of cognitive impairment among participants. A significance level of $P < 0.05$ was set for all statistical analyses, and the analysis was performed using SPSS 27 software (IBM Armonk, NY, USA).

Results

Sociodemographic data

There were no significant differences regarding demographic data between the three studied groups. Patients with BD had a mean 4.2 ± 3.3 episodes. Details illustrated in Table 1.

Neuropsychological assessment

The Wechsler Adult Intelligence Scale-3rd edition (WAIS-III)

There were significant differences between the three groups in total, verbal, performance, and DI. These differences were due to: The total score, verbal, and performance assessment of the WAIS-III in SZ patients were significantly poor compared to control group. Furthermore, SZ patients exhibited a higher DI than control as depicted in Table 2.

Table 1 Demographic and clinical data among BD, SZ, and control groups

	BD (1) (*n* = 30)	SZ (2) (*n* = 30)	Control (3) (*n* = 30)	*P* value
Age, years mean ± SD	31.43 ± 5.98	33.47 ± 8.7	30.03 ± 10.5	0.22
***P* value**	(1 vs. 2) 0.29	(2 vs. 3) 0.079	(1 vs. 3) 0.27	
Female *n* (%)	10 (33.3%)	6 (20%)	9 (30%)	0.48
Marital status *n* (%)				0.62
Single	13 (43.4%)	16 (53.3%)	13 (43.4%)	
Married	16 (53.3%)	13 (43.4%)	17 (56.6%)	
Divorced	1 (3.3%)	1 (3.3%)	0 (0%)	
Educational level *n* (%)				0.46
Primary school	8 (26.7%)	9 (30%)	5 (16.7%)	
Secondary/high school	22 (73.3%)	21 (70%)	25 (83.3%)	
Age at onset, years			–	0.78
mean ± SD	22.6 ± 6.4	23.1 ± 6.4		
median [IQR]	21 [15–42]	22 [12–35]		
Disease duration, years			–	0.29
mean ± SD	5.73 ± 4.2	7.73 ± 6.5		
median [IQR]	5 [1–19]	6 [1–26]		
Number of attacks in BD				
mean ± SD	5.4 ± 5.9	____	____	
median [IQR]	3.50 [1–30]			

Abbreviations: SD Standard deviation, *BD* Bipolar disorder, *SZ* Schizophrenia, *n (%)* Number (percentage)

Table 2 Wechsler adult intelligence scale-3rd edition score among BD, SZ patients, and control group

	BD (1) (*n* = 30)	**SZ (2) (*n* = 30)**	**Control (3) (*n* = 30)**	***P* value***
IQ				
•Verbal subset	80.9 ± 8.2	75.1 ± 5.1	88.6 ± 5	**< 0.001**
P value	(1 vs 2) < **0.001**	(2 vs 3) < **0.001**	(1 vs 3) < **0.001**	
• Performance subset	88.9 ± 10.3	81.6 ± 5.8	93.1 ± 5.7	**< 0.001**
P value	(1 vs 2) < **0.001**	(2 vs 3) < **0.001**	(1 vs 3) **0.02**	
• IQ total	82.7 ± 9.2	76.2 ± 4.6	88.9 ± 5.3	**< 0.001**
P value	(1 vs 2) < **0.001**	(2 vs 3) < **0.001**	(1 vs 3) < **0.001**	
• Deterioration index (DI)	7.7 ± 5.9	16.8 ± 8.4	8.7 ± 4.6	**< 0.001**
P value	(1 vs 2) < **0.001**	(2 vs 3) < **0.001**	(1 vs 3) 0.21	

Abbreviations: *BD* Bipolar disorder, *SZ* Schizophrenia, *SD* Standard deviation, *IQR* interquartile range

On the other hand, BD patients showed lower scores compared to the control group on the total and verbal scores of the WAIS-III ($P < 0.001$ for each), and the performance score ($P = 0.02$). However, there was no significant difference between BD patients and the control group in terms of the DI. Comparison between SZ versus BD showed that: SZ had significantly lower scores than BP in total score, verbal and performance scores and higher deterioration index than BP group as illustrated in Table 2.

Montreal Cognitive Assessment Scale score (MoCA)

There were significant differences between the three groups in total and all subitems scores except in Language subitems score. These significant were due to:

> Patients with SZ had significantly lower total score of the MoCA scale compared to control group particularly in visuospatial, naming, attention, delayed recall, and orientation subtests. However, BP had significantly lower scores than controls in visuospatial and delayed recall only.
> Comparison between SZ versus BD showed that SZ was significantly more affection than BP in total score, visuospatial, naming, attention, abstraction, and orientation as illustrated in Table 3.

Brief Visuospatial Memory Test-Revised (BVMT-R)

Table 4 demonstrated that there were significant differences between the three groups in all items of BVMT-R measures except in learning and % retained and Rec. Response Bias. These differences come from SZ patients performed worse than healthy controls in the same subscales of BVMT-R. Moreover, the BD group had poor scores in compared to the control group on trial 2 and total recall only. In comparison between the two patients' groups: SZ group had more affection in trial 1, 2, 3, total recall, and delayed recall with higher RDI than BP.

Memory Assessment Scale (MAS)

Table 5 show that BD and SZ groups performed significantly worse than the control group on all four memory assessment tests ($P < 0.001$) with significantly more affection in SZ than BP in all subscales except in verbal assessment were recorded.

Neurophysiological assessment: event-related potential (ERP) test, P300

The P300 latencies and amplitudes were within normal ranges of patient's groups, with no significant differences between the three groups at different points of assessment recorded. However, BD patients displayed a significantly shorter P300 latency compared to SZ at the Pz point of assessment ($P = 0.024$) (Table 6).

Correlation studies

Table 7 presents the correlations observed between various clinical variables and psychometric scales, as well as event-related potentials (ERPs), among individuals diagnosed with mood disorders. An increase in the number of years of education was found to be associated with higher scores on MOCA ($r = 0.521$, $p = 0.003$), total IQ ($r = 0.456$, $p = 0.022$), and all subscales of the BVMT-R, while no such correlation with other psychometric tests. Furthermore, an increase in the number of episodes experienced by the patients inversely correlated with total scores on the MOCA ($r = -0.709$, $p = 0.0001$), as well as performance on trial 1 ($r = -0.412$, $p = 0.024$), trial 3 ($r = -0.377$, $p = 0.040$), total recall ($r = -0.408$, $p = 0.025$), and delayed recall ($r = -0.395$, $p = 0.031$). On the other hand, an increase in the duration of illness was associated with a decrease in the total score on the MOCA

Table 3 Montreal cognitive assessment scale score among BD, SZ, and control groups

Parameter	BD (1) (*n* = 30)	SZ (2) (*n* = 30)	Control (3) (*n* = 30)	*P* value Between the 3 groups (ANOVA)
MoCA				
• Total score	27.1 ± 1.5	24.10 ± 1.4	27.3 ± 1.8	**< 0.001**
P value (Mann–Whitney)	(1 vs 2) < **0.001**	(2 vs 3) < **0.001**	(1 vs 3) 0.44	
• Visuospatial	4.8 ± 0.4	3.77 ± 0.7	4.4 ± 0.5	**< 0.001**
P value (Mann–Whitney)	(1 vs 2) < **0.001**	(2 vs 3) < **0.001**	(1 vs 3) **0.002**	
• Naming	2.87 ± 0.3	2.53 ± 0.5	2.97 ± 0.2	**< 0.001**
P value	(1 vs 2) **0.005**	(2 vs 3) < **0.001**	(1 vs 3) 0.17	
• Attention	4.60 ± 0.8	3.93 ± 1.2	4.57 ± 1.0	**0.02**
P value	(1 vs 2) **0.02**	(2 vs 3) **0.046**	(1 vs 3) 0.88	
• Language	2.73 ± 0.5	2.87 ± 0.3	2.90 ± 0.3	0.24
P value	(1 vs 2) 0.30	(2 vs 3) 0.69	(1 vs 3) 0.16	
• Abstraction	2.10 ± 0.3	1.97 ± 0.2	2.00 ± 0.1	**0.03**
P value	(1 vs 2) **0.045**	(2 vs 3) 0.32	(1 vs 3) 0.08	
• Delayed recall	4.23 ± 0.5	3.93 ± 0.8	4.67 ± 0.5	**< 0.001**
P value	(1 vs 2) 0.12	(2 vs 3) < **0.001**	(1 vs 3) **0.002**	
• Orientation	5.60 ± 0.8	5.03 ± 0.9	5.87 ± 0.3	**< 0.001**
P value	(1 vs 2) **0.004**	(2 vs 3) < **0.001**	(1 vs 3) 0.11	

Abbreviations: *BD* Bipolar disorder, *SZ* Schizophrenia, *SD* Standard deviation, *IQR* Interquartile range, *MOCA* Montreal Cognitive Assessment Scale

($r = -0.588$, $p = 0.001$), whereas an early onset of illness was associated with an more deterioration of the total IQ score ($r = -0.456$, $p = 0.011$).

In the case of individuals diagnosed with schizophrenia, the correlations observed between clinical variables, psychometric scales, and ERPs had no significant correlation between different parameters with each other.

Discussion

The current study's main findings include patients with SZ and BD had significantly lower scores on the total, verbal, and performance assessment of the WAIS-III compared with controls. However, SZ patients exhibited a higher Deterioration Index (DI) compared with controls; however, there was no difference in DI between BD patients and controls.

Previous research utilizing the WAIS-III to assess cognitive function has consistently demonstrated that SZ patients perform worse than BD and control individuals in verbal and performance domains. Compared to controls, BD patients also exhibit lower scores on overall IQ, verbal, and performance assessment subtests [30]. Another meta-analysis comparing the intellectual capacity of BD and SZ patients before and after illness onset with a control group found that SZ patients experienced a significant decline in premorbid intellectual function, whereas BD individuals did not [31]. Additionally, SZ were found to have slightly lower general intelligence than BD, with both groups displaying significantly lower general intelligence than healthy individuals [32]

In this study, individuals with SZ exhibited significantly lower total scores on the MoCA assessment, particularly in the visuospatial, naming, attention, delayed recall, and orientation subtests, as well as all subscales of the BVMT-R assessment, compared with controls. On the other hand, individuals with BD had significantly lower scores than controls in visuospatial and delayed recall subtests, as well as on trial 2 and total recall only. Moreover, the SZ group demonstrated more impairment in trials 1, 2, 3, total recall, and delayed recall, as evidenced by higher RDI scores than the BD group.

Several systematic reviews and meta-analyses have been performed to compare cognitive domains in SZ and BD, yielding contradictory results. One of this meta-analysis reported that patients with SZ performed worse than BP. BD patients performed worse in 9 of 11 cognitive domains, including verbal fluency, verbal working memory, delayed visual memory, executive control, and mental speed. SZ patients also performed worse in verbal memory, intelligence, and concept information, albeit with minor effect sizes [33].

Previous research has identified lower IQ, executive function, visual memory, language, and linguistic expertise in SZ patients, with varying patterns of decline at different ages. However, impairments in verbal memory,

Table 4 Brief visuospatial memory test-revised data differences among BD, SZ patients, and control group

	BD (1) (*n* = 30)	SZ (2) (*n* = 30)	Control (3) (*n* = 30)	*P* value
BVMT-R				
• Trial 1	2.50 ± 1.2	1.13 ± 1.2	2.93 ± 1.5	**< 0.001**
P value	(1 vs 2) < **0.001**	(2 vs 3) < **0.001**	(1 vs 3) 0.10	
• Trial 2	3.13 ± 1.3	2.10 ± 1.3	3.73 ± 1.5	**< 0.001**
P value	(1 vs 2) **0.003**	(2 vs 3) < **0.001**	(1 vs 3) **0.03**	
• Trial 3	3.73 ± 1.3	2.57 ± 1.5	4.33 ± 2.0	**0.001**
P value	(1 vs 2) **0.009**	(2 vs 3) < **0.001**	(1 vs 3) 0.11	
• Total recall	9.37 ± 3.8	5.80 ± 3.2	11.00 ± 5.0	**< 0.001**
P value	(1 vs 2) **0.001**	(2 vs 3) < **0.001**	(1 vs 3) **0.04**	
• Learning	1.23 ± 0.9	1.50 ± 0.9	1.40 ± 0.3	0.57
P value	(1 vs 2) 0.19	(2 vs 3) 0.62	(1 vs 3) 0.53	
• Delayed recall	2.90 ± 1.3	1.80 ± 1.2	3.33 ± 1.1	**0.001**
P value	(1 vs 2) **0.002**	(2 vs 3) < **0.001**	(1 vs 3) 0.14	
• % Retained	73.30 ± 29.5	59.33 ± 35.8	69.53 ± 29.3	0.22
P value	(1 vs 2) 0.08	(2 vs 3) 0.24	(1 vs 3) 0.50	
• Rec. hits	4.27 ± 0.6	4.03 ± 0.6	4.53 ± 0.7	**0.01**
P value	(1 vs 2) 0.12	(2 vs 3) **0.002**	(1 vs 3) 0.12	
• Rec. false alarm	1.47 ± 0.9	1.93 ± 0.9	1.37 ± 1.0	**0.049**
P value	(1 vs 2) 0.05	(2 vs 3) **0.03**	(1 vs 3) 0.68	
• RDI	2.77 ± 1.3	2.00 ± 1.1	3.17 ± 1.4	**0.002**
P value	(1 vs 2**) 0.02**	(2 vs 3) **0.001**	(1 vs 3) 0.22	
• Rec. response bias	0.45 ± 0.1	0.50 ± 0.1	0.47 ± 0.2	0.47
P value	(1 vs 2) 0.29	(2 vs 3) 0.63	(1 vs 3) 0.62	

Abbreviations: *BD* Bipolar disorder, *SZ* Schizophrenia, *%* percentage, *Rec* Recognition, *RDI* Recognition discrimination index

working memory, processing speed, and visuospatial ability were present from the early stages and remained stable.

In contrast, BD patients displayed worse performance in IQ, verbal knowledge, and language, but only in verbal functions and at different ages. Deficits in verbal memory, processing speed, and executive function also remained stable. SZ and BD patients experienced cognitive impairment in general and specialized functions following the initial episode, but the specific nature of the impairments varied with age. For effective cognitive rehabilitation, focusing on specific adult functions may be the most successful approach [3].

Additionally, some studies have reported that patients with SZ performed worse than BD in visual learning, working memory, and attention persistence, while other studies failed to confirm these findings [34, 35]. Conversely, meta-analysis on executive dysfunction tests indicated that the kind of cognitive deficits did not differentiate between BD and SZ [36] However, Jenkins et al. found that the BD group with psychosis outperformed the SZ group in processing speed, attention, visual, and working memory [34].

Genetic factors have been linked to brain function and cognitive deficiencies, although the precise mechanisms remain unknown [37, 38], while the structural and functional differences in various brain regions between SZ and BD patients are still unclear, studies suggested that neurodevelopmental variables may play a role in cognitive deficits in SZ and may be in a subgroups of BD patients [39].

A meta-analysis encompassing all available research found that individuals with BD generally performed better than SZ patients. The differences in cognitive impairment profiles observed in previous studies may be attributed to using different cognitive function assessment instruments. Other factors that could potentially explain the contradictory findings in cognitive function assessment in euthymic BD patients include the number of episodes, chronicity of the illness, length of hospitalization, and types of antipsychotic medication [40], comorbidities such as

Table 5 Memory assessment scale data differences between groups

	BD (1) (*n* = 30)	SZ (2) (*n* = 30)	Control (3) (*n* = 30)	*P* value*
• Short-term memory	68.70 ± 10.0	62.87 ± 6.9	79.43 ± 7.4	**< 0.001**
P value**	(1 vs 2) **0.02**	(2 vs 3) < **0.001**	(1 vs 3) < **0.001**	
• Verbal assessment	80.00 ± 6.3	76.83 ± 6.3	85.50 ± 5.6	**< 0.001**
P value**	(1 vs 2) 0.06	(2 vs 3) < **0.001**	(1 vs 3**) 0.002**	
• Visual assessment	66.03 ± 5.2	62.67 ± 2.8	77.30 ± 9.3	**< 0.001**
P value**	(1 vs 2) **0.008**	(2 vs 3) **< 0.001**	(1 vs 3) < **0.001**	
• Total score	70.00 ± 5.2	66.20 ± 4.4	79.00 ± 7.6	**< 0.001**
P value**	(1 vs 2) **0.006**	(2 vs 3) < **0.001**	(1 vs 3) < **0.001**	

Abbreviations: *BD* Bipolar disorder, *SZ* Schizophrenia

* ANOVA test

** Mann–Whitney test

Table 6 P300 latency and amplitude among BD, SZ and control groups

Parameter	BD (1) (*n* = 30)	SZ (2) (*n* = 30)	Control (3) (*n* = 30)	*P* value
P300 latency, ms				
• Fz	301.2 ± 30.5	318.3 ± 37.6	313.22 ± 24.9	0.099
P value	(1 vs 2) 0.058	(2 vs 3) 0.62	(1 vs 3) 0.077	
• Cz	298.1 ± 27.4	314.3 ± 38.1	308.2 ± 25.9	0.19
P value	(1 vs 2) 0.065	(2 vs 3) 0.56	(1 vs 3) 0.15	
• Pz	299.6 ± 28.2	320 ± 38.5	310.2 ± 25.9	0.09
P value	(1 vs 2) **0.024**	(2 vs 3) 0.35	(1 vs 3) 0.12	
P300 amplitude, mv				
• Fz	9.98 ± 4.3	12.68 ± 7.7	13.20 ± 8.2	0.51
P value	(1 vs 2) 0.34	(2 vs 3) 0.81	(1 vs 3) 0.31	
• Cz	9.73 ± 4.9	12.55 ± 7.6	12.44 ± 7.5	0.36
P value	(1 vs 2) 0.15	(2 vs 3) 0.89	(1 vs 3) 0.33	
• Pz	9.91 ± 5.0	12.65 ± 7.4	12.78 ± 7.9	0.20
P value	(1 vs 2) 0.22	(2 vs 3) 0.99	(1 vs 3) 0.24	

Abbreviations: *BD* Bipolar disorder, *SZ* Schizophrenia, *ms* Millisecond, *mv* Microvolt, *Cz* Central, *Fz* Frontal, *Pz* Parietal

substance misuse, definitions of euthymia, and the specific cognitive tests employed. Additionally, factors that may introduce bias in assessing cognitive function, the representativeness of the samples, the specific cognitive domains assessed, and the recruitment methods employed [41].

Regarding memory assessment, both BD and SZ groups performed significantly worse than the controls across all four subscales ($P < 0.001$). Patients with SZ had significant impairment more than patients with BD in all subscales except verbal assessment.

Some studies have suggested that SZ and BD differ in working memory [42, 43]. Working memory is recognized as a core cognitive domain that is impacted by major mental illness [44], so it serves as a suitable endophenotypic marker for distinguishing between SZ and BD [45]. Another investigation assessing the cognitive profiles of people with early-onset SZ and pediatric-onset BD showed the prevalence of abnormalities in numerous domains in SZ and a comparable but less severe of cognitive impairment in BD [46]. Conversely, another study found no significant reduction in the Working Memory index, indicating that illness duration may impact cognitive decline.

The study also employed event-related potentials (ERPs), specifically the P300 component, to assess cognitive function. The P300 ERP is a late cognitive-related signal linked to attention and memory functions [47].

P300, an indicator of the process of discriminating between significant and insignificant brain stimuli, is dissected into two fundamental components. These two basic components are known as amplitude and delay. While the amplitude of the P300 changes due to the inability of the targets, the latency changes due to the difficulty of distinguishing the target stimulant from the standard stimulants. In most individuals with a psychiatric disease and reduced cognitive skills, latency is often shortened while P300 amplitude decreases.

This study observed no significant differences in P300 latencies and amplitudes between the three groups at different assessment points. However, BD had significantly shorter P300 latency compared with controls and compared with SZ group separately. This is due to changes in the information processing in the brain [48] as BD had increased physiological arousal and rapid information process. Previous research has indicated that BD and SZ patients have lower P300 amplitudes than healthy individuals [49, 50] suggesting that P300 amplitude alone may not distinguish between these disorders. These findings support the notion that lower amplitude and delayed latency of P300 may be implicated in the pathogenesis of both BD and SZ, irrespective of psychotic history [51, 52]. In the current study, absence of significant correlation between P300 parameters and different clinical variables in both ZS and BD could be due to the normal P300 latency values ranging between 250 and 350 ms were established in adults by Kraus et al. [53]. However, wider latency ranges, were also reported [54] and this wide range of normal values as well as the small sample size of each group may explain the absence of such correlation and may explain why P300 is not routinely used in clinical practice.

While early results suggested that SZ and BD could potentially be spectrum of psychoses [36] investigations have shown that SZ has higher grey matter atrophy [55] and worse global connectivity than BD. Poor global connectivity could lead to worse verbal learning, processing speed, and attention performance in SZ [36, 56].

In the present study, an increased number of episodes in individuals with mood disorders and a longer duration of illness were found to be associated with a decline in cognitive function. Previous research has consistently reported that heightened cognitive dysfunction, particularly in areas such as attention and memory, is frequently observed alongside greater symptom severity [57]. Additionally, it has been suggested that frontal dysfunction may be linked to a prolonged duration of illness [58].

In the context of mood disorders, higher scores on cognitive function assessments were observed in individuals with more years of formal education. Previous studies have consistently demonstrated a positive correlation between the level of formal education and cognitive

Table 7 Correlation between clinical variables and psychometrics scales, and ERP among mood disorder patients

		Years of education	Age of onset	Duration of illness in years	Number of episodes
Total score of MOCA	*r*	0.521	0.125	−0.588	−0.709
	P value	0.003*	0.510	0.001*	0.0001*
Total IQ score	*r*	0.456	0.440	−0.212	−0.325
	P value	0.011*	0.015*	0.262	0.080
Total score of MAS	*r*	0.245	−0.257	−0.258	−0.227
	P value	0.191	0.170	0.168	0.227
Trial 1	*r*	0.392	0.352	−0.291	−0.412
	P value	0.032*	0.056	0.119	0.024*
Trial 2	*r*	0.468	0.318	−0.235	−0.301
	P value	0.009*	0.087	0.211	0.106
Trial 3	*r*	0.369	0.200	−0.358	−0.377
	P value	0.045*	0.290	0.052	0.040*
Total recall	*r*	0.443	0.253	−0.319	−0.408
	P value	0.014*	0.177	0.085	0.025*
Learning	*r*	0.029	−0.050	−0.156	−0.033
	P value	0.877	0.791	0.411	0.861
Delayed recall	*r*	0.456	0.308	−0.241	−0.395
	P value	0.011*	0.098	0.200	0.031*
Percent retained	*r*	0.126	0.360	0.043	−0.169
	P value	0.506	0.051	0.822	0.372
Recognition hits	*r*	0.258	0.306	−0.154	−0.328
	P value	0.169	0.100	0.418	0.076
Recognition false alarm	*r*	−0.304	−0.138	0.067	0.221
	P value	0.102	0.468	0.726	0.241
Recognition Discrimination Index	*r*	0.368	0.251	−0.118	−0.318
	P value	0.045*	0.180	0.533	0.087
Recognition response bias	*r*	−0.168	0.007	0.015	0.083
	P value	0.374	0.970	0.938	0.662
P 300 average latency	*r*	−0.280	−0.249	0.343	0.158
	P value	0.134	0.184	0.064	0.404
P300 average amplitude	*r*	0.148	−0.051	0.161	−0.041
	P value	0.436	0.790	0.394	0.830

* Significant *p* value

abilities [59]. Furthermore, it has been noted that education pertaining to mental health plays a significant role in fostering educational resources, cognitive skills, and social integration [60].

Our study has several limitations, such as a small sample size, we did not control for the types, dosage, or duration of psychotropics, duration of untreated [61], possible biological etiology [62], and the study design being an observational case–control study with no follow-up data.

In conclusion, patients with SZ and BP had significantly lower scores on various cognitive function domains compared with the control group with more affection in SZ. The importance of considering factors such as number of mood episodes, duration of illness, education, and specific cognitive domains in understanding the cognitive profiles of these psychiatric conditions. Further studies are warranted to investigate the underlying mechanisms of cognitive dysfunction in both SZ and BD patients, which may help in developing better treatment strategies for these patients.

Abbreviations

SZ	Schizophrenia
BD	Bipolar disorder
Cz	Central
Fz	Frontal
Pz	Parietal

ERP	Event-related potential
MoCA	Montreal Cognitive Assessment Scale
WAIS-III	Wechsler Adult Intelligence Scale-3rd edition
BVMT-R	Brief Visuospatial Memory Test-Revised
MAS	Memory Assessment Scales
DI	Deterioration Index
RDI	Recognition Discrimination Index

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EK, NG, KE, GA, and BF recruited participants, analysis, and interpreted data, and were the contributors in writing the manuscript. All authors revised data interpretation, read and approved the final manuscript.

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Availability of data and materials
All data generated or analyzed during this study are available from corresponded on request.

Declarations

Ethics approval and consent to participate
The Aswan University faculty of medicine's institutional review board approved the study for ethical reasons. To participate in the study, all subjects provided written informed permission. All participant personal information was kept private, and all patient personal data was anonymized immediately following data collection. We verified that all processes were carried out in accordance with the applicable norms and regulations.

Consent for publication
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The authors have no conflicts of interest to declare that are relevant to the content of this article.

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