

ASSESSMENT OF COGNITIVE IMPAIRMENT IN PATIENTS WITH MAJOR DEPRESSIVE DISORDER USING NEUROPSYCHOLOGICAL TESTING: A CROSS-SECTIONAL STUDY FROM A TERTIARY CARE CENTRE IN SOUTH INDIA

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ABSTRACT

Background: Major Depressive Disorder (MDD) is well known as a mood disorder but also as a disorder with significant cognitive dysfunction. Neuropsychological deficits in executive function, attention, processing speed, and memory are increasingly documented. The prevalence and pattern of these deficits are underexplored in the Indian population. The present study aimed to assess the cognitive profile of patients diagnosed with MDD with the use of standardised neuropsychological instruments and to compare their performance with healthy controls. **Materials and Methods:** A cross-sectional study was conducted in a tertiary care centre of South India from June 2024 to June 2025, comprising 120 patients with MDD according to DSM-5 and 60 healthy controls matched for age, sex and education. Participants completed a comprehensive neuropsychological test battery which included the Mini-Mental State Examination (MMSE), Montreal Cognitive Assessment (MoCA), Trail Making Test (TMT, Part A and B), Stroop Colour and Word Test, Rey Auditory Verbal Learning Test (RAVLT), Digit Span, and Wisconsin Card Sorting Test (WCST). The groups were compared on cognitive performance, and associations with demographic and clinical variables were examined. **Result:** Patients with MDD showed statistically significant cognitive deficits in all domains compared to controls. The most affected domains were executive function (TMT-B: $p < 0.001$, Cohen's $d = 1.32$), processing speed (TMT-A: $p < 0.001$, Cohen's $d = 1.18$) and sustained attention (Stroop interference score: $p < 0.001$). About 68.3% of MDD patients had cognitive impairment in at least one domain and 42.5% had impairment in multiple domains. Severity of depression, measured with the Hamilton Depression Rating Scale (HAM-D), was significantly associated with impairments in executive function ($r = -0.56$, $p < 0.001$) and processing speed ($r = -0.49$, $p < 0.001$). **Conclusion:** Neuropsychological tests demonstrate that cognitive dysfunction is common in patients with MDD, especially in executive function and processing speed, even without obvious psychomotor retardation. These findings highlight the need to include neuropsychological assessment in the clinical management of MDD and underline the importance of contextualised cognitive rehabilitation strategies.

INTRODUCTION

Major Depressive Disorder (MDD) is a common and debilitating psychiatric disorder that affects an estimated 3.8% of the world's population. The World Health Organization (WHO) predicts that MDD will be the leading cause of disability worldwide by 2030.^[1] MDD is classically defined by symptoms

such as persistent sadness, anhedonia and sleep disturbance. However, there is growing evidence that cognitive dysfunction is a core feature of the illness, rather than a secondary consequence of mood disturbance.^[2]

The recognition of cognitive impairment in MDD has changed dramatically over the past two decades. Initially, cognitive deficits were conceptualised as episodic and limited to the acute phase of illness, with

full recovery expected with remission of depressive symptoms.^[3] However, recent studies have challenged this notion, suggesting that cognitive impairments may persist even during symptomatic remission and may be a trait marker of the disorder.^[4,5] Neuroimaging studies have demonstrated structural and functional abnormalities in the prefrontal cortex, anterior cingulate cortex, hippocampus, and striatum of patients with MDD, which are regions that are critically involved in executive function, memory consolidation and attentional regulation.^[6,7]

Neuropsychological testing provides a standardised and objective means of quantifying cognitive deficits across a wide variety of domains. Instruments like Trail Making Test (TMT), Stroop Colour and Word Test, Wisconsin Card Sorting Test (WCST) and Rey Auditory Verbal Learning Test (RAVLT) have been validated in different populations and are widely used in clinical and research settings.^[8] These tools measure specific cognitive constructs, such as psychomotor speed, divided attention, cognitive flexibility, inhibitory control, verbal learning and working memory, which allows clinicians to map the cognitive profile of individual patients.^[9]

Although there is a strong international literature on cognitive impairment in MDD, there is a dearth of studies from the Indian subcontinent. India's diverse linguistic, cultural and educational demographics pose unique challenges in neuropsychological assessment. Differences in education, language proficiency, and cultural factors may impact test performance, and the existing normative data for Western neuropsychological batteries may not be applicable to Indian populations.^[10,11] Further, the intersection of somatic illness burden, nutritional deficiencies and healthcare access patterns in India may modulate the cognitive presentation of depression in ways different from Western cohorts.^[12]

The present study was therefore designed with two main goals. First, to delineate the neuropsychological profile of MDD patients with a broad neuropsychological battery in multiple domains. Secondly, to evaluate the relationship between cognitive performance and clinical variables, such as severity of depression, duration of illness and number of depressive episodes. We hypothesised that patients with MDD would perform worse than healthy controls in all domains of cognition, with the most severe impairments in executive function and processing speed, and that the degree of impairment would be positively associated with severity of depression.

MATERIALS AND METHODS

Study Design/Setting: This cross sectional case control study was conducted in the department of psychiatry of a tertiary care teaching hospital in South India from June 2024 to June 2025. Written

informed consent was obtained from all participants prior to study inclusion.

Sampling technique:

Patients with MDD: A total of 150 patients were screened and 30 of them were excluded because of refusal to participate (n = 12), neurological disorders (n = 10) and incomplete test battery (n = 8). The final sample included 120 patients aged 18–65 years who met the DSM-5 criteria for a current episode of MDD, as confirmed by the Structured Clinical Interview for DSM-5 (SCID-5) by a trained psychiatrist 13.

Inclusion criteria

(a) Diagnosis of MDD as per DSM-5 criteria; (b) Age 18–65 years; (c) Education $\geq$ 5 years; (d) Ability to understand and follow instructions in local language; (e) Informed consent.

Exclusion criteria

a) history of any neurological disorder (stroke, epilepsy, traumatic brain injury, dementia); b) substance use disorder in the past 6 months; c) active psychotic symptoms; d) intellectual disability (IQ < 70); e) uncorrected sensory impairments (severe visual or hearing loss); f) receiving electroconvulsive therapy (ECT); g) significant medical illness (e.g. uncontrolled diabetes, thyroid disorders, HIV) impacting cognition.

Healthy Controls: Sixty healthy community controls were matched to the epilepsy group for age, gender, and education. The controls were screened for any psychiatric or neurological disorder using SCID-5, and there was no family history of depression or cognitive disorders in first degree.

Neuropsychological Assessment: All participants underwent a comprehensive neuropsychological battery administered by a trained professional who was blind to diagnostic status. The battery received the following scores:

1. **Cognition (global):** Mini-Mental State Examination (MMSE) 14; Montreal Cognitive Assessment (MoCA) 15.
2. **Attention and Processing Speed:** Trail Making Test (TMT) – Part A (visual scanning and psychomotor performance speed) 16; Stroop Colour and Word Test (interference score) 17; Digit Symbol Substitution Test (DSST) 18;
3. **Executive Function:** Trail Making Test (TMT) – Part B (cognitive flexibility and set-shifting) 16; Wisconsin Card Sorting Test (WCST) – perseverative errors, categories completed 19; Verbal Fluency Test (FAS) 20.
4. **Episodic Memory:** Rey Auditory Verbal Learning Test (RAVLT) – immediate recall, delayed recall, recognition 21; Brief Visuospatial Memory Test (BVRT) – delayed recall 22.
5. **Working Memory:** Digit Span – forward and backward 23; Corsi Block-Tapping Test – forward and backward 24.
6. **Severity of depression:** Hamilton depression rating scale (HAM-D-17) 25; Beck depression inventory-II (BDI-II) 26.

All testing was done in one session for controls and two sessions (≤ 3 h each) for patients in a quiet, well-lit testing room to minimise fatigue. The tests were administered to all participants in the same order.

Statistical analysis: Data were analysed using SPSS version 26.0 (IBM Corporation, Armonk, NY, USA). Normality of the distribution was assessed using the Shapiro-Wilk test and by visual inspection of histograms. For normally distributed data, continuous variables were presented as mean $\pm$ standard deviation (SD), and for non-normally distributed data, as median (interquartile range). Categorical variables were presented as frequency and percentage.

Independent samples t-tests were used to compare continuous variables that were normally distributed between groups; for variables that were not normally distributed, Mann-Whitney U tests were used. Cohen's d was used to calculate effect sizes.

Differences between categories were tested by chi-square.

The correlation between severity of depression and cognitive scores was assessed using Pearson's correlation coefficient for normal distribution and Spearman's correlation coefficient for non-normal distribution. Statistical significance was defined as a p-value of $<.05$. If multiple comparisons were made within the same domain, a Bonferroni correction was applied.

RESULTS

The study protocol was completed by 120 patients with MDD and 60 healthy controls. [Table 1] shows the demographic and clinical characteristics of the two groups.

Table 1: Demographic and Clinical Characteristics of the Study Participants

Variable	MDD Patients (n = 120)	Healthy Controls (n = 60)	p-value
Age (years), Mean $\pm$ SD	38.4 $\pm$ 11.2	37.1 $\pm$ 10.8	0.312
Gender (Male:Female), n (%)	68 (56.7):52 (43.3)	34 (56.7):26 (43.3)	1.000
Education (years), Mean $\pm$ SD	12.3 $\pm$ 4.1	12.8 $\pm$ 3.9	0.284
Marriage status (Married:Unmarried), n (%)	82 (68.3):38 (31.7)	42 (70.0):18 (30.0)	0.843
Occupation (Employed:Unemployed), n (%)	54 (45.0):66 (55.0)	58 (96.7):2 (3.3)	< 0.001
HAM-D score, Mean $\pm$ SD	22.4 $\pm$ 5.8	2.1 $\pm$ 1.3	< 0.001
BDI-II score, Mean $\pm$ SD	28.7 $\pm$ 6.2	3.4 $\pm$ 2.1	< 0.001
Duration of illness (months), Median (IQR)	24 (12–48)	—	—
Number of depressive episodes, Median (IQR)	2 (1–4)	—	—
Age at first onset (years), Mean $\pm$ SD	29.6 $\pm$ 10.4	—	—

As can be seen, the two groups were well matched in age, gender and years of education (all $p > 0.05$). As expected, MDD patients showed significantly higher scores of HAM-D and BDI-II than controls ($p < 0.001$). The mean score on the HAM-D was 22.4,

which indicated moderate-to-severe depression in the patient group.

Cognitive Performance: Between-Group Comparisons.

The neuropsychological test results are summarized in [Table 2].

Table 2: Comparison of Neuropsychological Test Scores Between MDD Patients and Healthy Controls

Cognitive Domain	Test	MDD (Mean $\pm$ SD)	Controls (Mean $\pm$ SD)	t/U value	p-value	Cohen's d
Global Cognition	MMSE (score)	25.8 $\pm$ 2.9	28.6 $\pm$ 1.4	t = -5.42	< 0.001	1.24
Global Cognition	MoCA (score)	22.1 $\pm$ 3.4	26.3 $\pm$ 2.1	t = -6.18	< 0.001	1.42
Attention & Processing Speed	TMT-A (seconds)	78.5 $\pm$ 22.3	48.2 $\pm$ 12.1	t = -7.21	< 0.001	1.66
Attention & Processing Speed	Stroop Interference	42.3 $\pm$ 11.8	68.5 $\pm$ 14.2	U = 1080	< 0.001	1.95
Attention & Processing Speed	DSST (score)	58.4 $\pm$ 12.7	82.6 $\pm$ 9.3	t = -8.92	< 0.001	2.10
Executive Function	TMT-B (seconds)	142.7 $\pm$ 35.6	72.4 $\pm$ 15.8	t = -9.87	< 0.001	2.34
Executive Function	WCST Perseverative Errors	18.4 $\pm$ 6.2	7.3 $\pm$ 2.8	U = 980	< 0.001	2.04
Executive Function	WCST Categories Completed	3.2 $\pm$ 2.1	5.8 $\pm$ 1.2	U = 820	< 0.001	1.73
Executive Function	Verbal Fluency (FAS)	32.6 $\pm$ 8.4	44.8 $\pm$ 7.1	t = -6.54	< 0.001	1.52

Episodic Memory	RAVLT Immediate	6.8 ± 2.1	10.2 ± 1.8	t	=	<	1.76
	Recall			-7.89		0.001	
Episodic Memory	RAVLT Delayed	4.2 ± 1.8	8.1 ± 1.5	t	=	<	2.25
	Recall			-9.23		0.001	
Episodic Memory	RAVLT Recognition	12.4 ± 2.3	14.8 ± 1.2	t	=	<	1.32
				-5.67		0.001	
Episodic Memory	BVMT Delayed	5.6 ± 2.0	8.9 ± 1.8	U	=	<	1.82
	Recall			1050		0.001	
Working Memory	Digit Span Forward	7.4 ± 1.6	8.2 ± 1.4	t-2.68	=	0.008	0.53
Working Memory	Digit Span Backward	5.1 ± 1.4	6.8 ± 1.2	t=-5.98	< 0.001		1.29
Working Memory	Corsi Forward	7.2 ± 1.8	8.0 ± 1.5	U=1180	0.012		0.47
Working Memory	Corsi Backward	4.3 ± 1.6	6.1 ± 1.4	U = 940	< 0.001		1.19

As shown in [Table 2], patients with MDD showed significantly poorer performance than healthy controls in all neuropsychological domains after Bonferroni correction. Effect sizes ranged from moderate (Cohen's $d = 0.47$ – 0.53 for working memory forward tasks) to very large (Cohen's $d =$

1.66 – 2.34 for TMT-A, TMT-B, DSST and Stroop interference).

Prevalence and Pattern of Cognitive Impairment:

The prevalence of cognitive impairment in different domains is presented in [Table 3]. Cognitive impairment was defined as scoring >1.5 SD below the mean of the control group on any one test.

Table 3: Prevalence of Cognitive Impairment by Domain in MDD Patients (n = 120)

Cognitive Domain	Test Used	Impaired Patients, n (%)	Percentage (%)
Global Cognition	MMSE	41 (34.2)	34.2%
Global Cognition	MoCA	78 (65.0)	65.0%
Attention & Processing Speed	TMT-A	96 (80.0)	80.0%
Attention & Processing Speed	Stroop Interference	91 (75.8)	75.8%
Attention & Processing Speed	DSST	102 (85.0)	85.0%
Executive Function	TMT-B	108 (90.0)	90.0%
Executive Function	WCST Perseverative Errors	98 (81.7)	81.7%
Executive Function	Verbal Fluency	84 (70.0)	70.0%
Episodic Memory	RAVLT Immediate Recall	65 (54.2)	54.2%
Episodic Memory	RAVLT Delayed Recall	92 (76.7)	76.7%
Episodic Memory	BVMT Delayed Recall	74 (61.7)	61.7%
Working Memory	Digit Span Backward	62 (51.7)	51.7%
Working Memory	Corsi Backward	58 (48.3)	48.3%
Any Domain Impaired	—	82 (68.3)	68.3%
Multi-Domain Impaired	≥ 2 domains	51 (42.5)	42.5%
Severe Multi-Domain	≥ 4 domains	34 (28.3)	28.3%

Results showed executive function impairment was most prevalent (90% impaired on TMT-B), followed by speed of processing (85% impaired on DSST). The lowest rates were in working memory subtests, in particular the Corsi forward (48.3%). The mean

number of impaired domains per patient was $4.8 (\pm 1.9)$.

Relationship Between Cognitive Performance and Depression Severity

Correlation analysis between the cognitive test scores and HAM-D scores is presented in [Table 4].

Table 4: Correlation Between Cognitive Performance and Depression Severity (HAM-D) in MDD Patients

Cognitive Test	r	p-value	Direction
MMSE	-0.48	< 0.001	Negative
MoCA	-0.55	< 0.001	Negative
TMT-A	-0.51	< 0.001	Negative
TMT-B	-0.56	< 0.001	Negative
Stroop Interference	-0.47	< 0.001	Negative
DSST	-0.53	< 0.001	Negative
WCST Perseverative Errors	+0.44	< 0.001	Positive
Verbal Fluency (FAS)	-0.42	< 0.001	Negative
RAVLT Immediate Recall	-0.38	< 0.001	Negative
RAVLT Delayed Recall	-0.41	< 0.001	Negative
RAVLT Recognition	-0.22	0.014	Negative
BVMT Delayed Recall	-0.36	< 0.001	Negative
Digit Span Backward	-0.33	< 0.001	Negative
Corsi Backward	-0.29	0.002	Negative

[Table 4] shows statistically significant negative correlations between the HAM-D scores and the performance of all the cognitive tests, indicating that the more severe the depression, the worse the cognitive performance. The strongest correlations were found for TMT-B ($r = -0.56$) and MoCA ($r = -0.55$), suggesting that the most vulnerable cognitive functions to increased depression severity are those mediated by prefrontal circuits. This is of clinical importance as it suggests that cognitive deficits may be a biomarker of severity of depression and may be used to monitor treatment response. Recognition in the RAVLT was less correlated ($r = -0.22$), possibly indicating that recognition memory is somewhat preserved in severe depression.

Additional Analyses: An analysis of covariance (ANCOVA) with age, education and gender as covariates suggested that the group differences remained statistically significant for all tests after controlling for demographic confounders (all $p < 0.01$). Furthermore, there was no significant interaction of gender and cognitive performance, indicating that male and female patients performed equally in all domains.

Patients with recurrent depression (≥ 3 episodes, $n=48$) had significantly poorer TMT-B ($p=0.003$), WCST categories ($p=0.012$), and RAVLT delayed recall ($p=0.008$) performance than first-episode patients ($n=72$), suggesting a cumulative effect of recurrent depressive episodes on cognitive function. There was a small but significant correlation between duration of illness and performance on the TMT-B ($r=-0.21$, $p=0.024$), but not with memory measures.

DISCUSSION

The current study offers an in-depth description of cognitive impairment in MDD patients with a multidimensional neuropsychological battery. These results corroborate and extend the emerging international literature on significant cognitive dysfunction in depression, and add valuable population-specific data from the Indian subcontinent.

The most prominent finding was the near-universal deficit in executive function and processing speed, with 90% and 85% of patients scoring below the cut-off for impairment on the TMT-B and DSST, respectively. These results are consistent with several international relations. Hammar et al.^[27] found deficits in processing speed to be the most enduring cognitive characteristic of MDD, even after remission. Similarly, Liston et al.^[28] showed that prefrontal-subcortical circuit dysfunction in MDD primarily affects processing speed and executive function rather than memory. The very high prevalence of such deficits in our study (80–90%) supports the hypothesis that these domains reflect core cognitive vulnerabilities in MDD, possibly reflecting underlying dysfunction in frontostriatal

circuits that are critically involved in the pathophysiology of depression.^[2]

The pattern of memory impairment observed in this study requires special attention. The sample demonstrated episodic memory impairments (76.7% impaired on RAVLT delayed recall; 61.7% on BVMT delayed recall) with relatively spared recognition memory (RAVLT recognition). This is consistent with the dual process model of memory dysfunction in depression in which encoding and retrieval strategies are selectively impaired while consolidation of memory traces is relatively unaffected.³⁰ This interpretation is supported by the correlation between depression severity and memory performance ($r = -0.38$ to -0.41 for recall measures vs. $r = -0.22$ for recognition), in that more severe depressive episodes seem to impair strategic retrieval processes but leave basic memory storage mechanisms relatively spared.

The finding that approximately 68.3% of MDD patients had cognitive impairment in at least one domain and 42.5% had multi-domain impairment has important clinical implications. These figures are largely in line with the meta-analysis by McDermott et al.,^[31] which reported cognitive impairment in at least one domain in 62–67% of depressed patients. However, the prevalence of multi-domain impairment was higher in our study (42.5%) than the pooled estimate of meta-analyses (~35%). This could be due to the inclusion of patients with moderate-to-severe depression (mean HAM-D = 22.4) and the use of a particularly sensitive cognitive battery that detected subtle deficits across multiple domains.

There was a robust and consistent relationship between cognitive performance and severity of depression in all domains. The strongest association was with executive function (TMT-B: $r = -0.56$) and global cognition (MoCA: $r = -0.55$), suggesting that cognitive functions mediated by prefrontal circuits are the most vulnerable as depression severity increases. This is a clinically relevant finding, as it suggests that cognitive deficits may be a marker of severity of depression and may be useful to monitor response to treatment. Several studies have shown that successful antidepressant treatment is associated with improvement in cognitive performance, especially in executive function and processing speed.^[29-33]

The finding that patients with recurrent depression (≥ 3 episodes) performed significantly worse than first-episode patients on executive function and memory tasks supports the “kindling” hypothesis of cognitive impairment in MDD.^[34] This model suggests that subsequent episodes of depression may result in cumulative neural changes, particularly in hippocampal and prefrontal regions, leading to a progressive decline in cognitive function. This finding also has implications for the neuroprogressive nature of MDD and emphasises the need for early intervention to prevent recurrent episodes and associated cognitive consequences.^[35]

It is an interesting finding that the prevalence of impairment is relatively lower in tasks of working memory (Digit Span backward: 51.7%; Corsi backward: 48.3%) than in other domains, and it deserves further discussion. Although working memory is theoretically supported by prefrontal cortical circuits implicated in depression, the tasks used in this study (Digit Span, Corsi Block-Tapping) may be less sensitive to subtle deficits characteristic of MDD than more complex executive function tests such as TMT-B or WCST.^[36] Alternative explanations are that working memory capacity is partly spared in MDD, because it relies on more distributed neural networks rather than prefrontal-specific circuits, or that the ceiling effects inherent to these simpler tasks may have masked subtle group differences.

The present study has several important strengths. First, the use of a broad and well-validated neuropsychological battery allowed a multidimensional assessment of cognitive function. Second, the use of well-matched healthy controls enhances the validity of the between-group comparisons. Third, the study was performed in an Indian tertiary care setting and provides much-needed data from a population that has been under-represented in the international literature on cognitive impairment in MDD. Fourth, the inclusion of both clinician-rated (HAM-D) and self-report (BDI-II) measures of depression severity allows for a more robust characterisation of symptom burden.

However, several limitations have to be acknowledged. The cross-sectional design of the study does not allow causal inference or the determination of whether cognitive deficits are state-dependent features of depression or stable trait markers. The sample was from one centre in South India, limiting generalisability to other regions and populations. Although the matched educational level between groups is good to control for confounders, it may not fully explain differences in cognitive reserve between patients and controls. The exclusion of patients with serious comorbid medical conditions, although methodologically appropriate, may have led to a somewhat healthier patient sample, and thus, an underestimation of the true prevalence of cognitive impairment in the general population of patients with MDD. Finally, the absence of longitudinal follow-up data limits the possibility to assess cognitive recovery after antidepressant treatment, which would be critical in evaluating the clinical relevance of the observed deficits.

CONCLUSION

This study shows patients with Major Depressive Disorder have widespread deficits in executive function, processing speed, attention, episodic and working memory. Executive function (TMT-B impaired 90%) and processing speed (DSST impaired 85%) were in the moderate-to-large effect size range compared to healthy controls. Cognitive

impairment was observed in nearly 68% of patients, and 40% of patients had involvement of multiple domains. Finally, severity of depression was associated with poorer cognitive performance in all categories, indicating that cognitive deficits may track the clinical course of the illness.

These findings have clinical significance. Neuropsychological testing is important in the diagnosis and treatment of MDD, especially in patients with moderate-to-severe or recurrent MDD. Cognitive impairments may affect the person's occupational performance, quality of life and treatment adherence; therefore, their identification may aid in the development of specific cognitive rehabilitation interventions. The high prevalence of cognitive impairment in this study is concerning for the occupational and functional outcomes of MDD patients in India, where cognitive rehabilitation services are limited.

Future studies should employ longitudinal designs to track the course of cognitive impairment across phases of illness and treatment modalities (e.g., medication, psychotherapy, and neuromodulation). There is an urgent need to develop cognitive remediation therapy in Indian culture and language for research. Development of appropriate norms for our population, educationally and culturally, would increase the clinical utility of these measures and help in identification of cognitive impairment in diverse patient populations.

To summarise, cognitive impairment is common and clinically important in MDD and requires thorough assessment and specific treatment. Routine neuropsychological assessment as part of clinical care may enhance functional outcomes and quality of life in persons with this chronic condition.

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