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ORIGINAL ARTICLE

ASSOCIATION BETWEEN VITAMIN D DEFICIENCY AND COGNITIVE FUNCTION IN THE ELDERLY PATIENT IN A TERTIARY CARE HOSPITAL, BANGLADESH

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Abstract:

Background: *Recent research has shown vitamin D deficiency as a risk factor for cognitive impairment. The study's objective was to determine the association between vitamin D insufficiency and cognitive function in the elderly patient.* ***Methods:*** *This case control study was conducted in the department of neurology, Sir Salimullah Medical College, Mitford, Dhaka, Bangladesh from July 2022 to June 2023. Fifty cognitive impairment patients and 50 matched healthy control subjects were participated in the study. We evaluated subjects with cognitive function with available serum Vitamin D [2S (OH) D] levels. Descriptive statistics means and standard deviations and logistic regression analyses were performed by SPSS 25.* ***Results:*** *The mean age of patients was 70.7 +/-6.3 years and the control was 61.480 +/ -7.15 years (p =0.613). Twenty-three subjects (46%) had severe cognitive impairment (SCI); 16 (32%) moderate cognitive impairment (MoCI); and 11 (22%) mild cognitive impairment (MCI). The overall prevalence of severe Vitamin D deficiency was 76%, being more frequent in the severe cognitive impairment group (47.4%), followed by the moderate cognitive (31.6%) and the MCI (21%) (p < 0.001) groups. Executive function was measured by the Clinical Dementia Rating (CDR) (6.2+/- 1.01, 3.8+/- 1.0, 2.3+/-0.5; p = <0.10) respectively which were statistically significant. The same differences were noted between these groups in the Katz Index of independence in Activities in Daily Living (K Index) (4.0+/- 0.7, 2.6+/-0.5, 1.5+/-0.5; p = <0.10); verbal fluency (4.8 ± 1.3, 9.0 ± 2.3, 11.6+/-1.9 words, respectively; p < 0.01), and clock-drawing test scores (3.6 +/- 1.4, 6.2 ± 1.5 , 7.4 +/-1.4 points; p = <0.01) respectively. Cognitive status measured by MMSE was statistically significant with the sufficiency of Vitamin D group (11.8 ± 5.8 vs. 12.5 ± 4.7; p < 0.01).Executive function was measured by the Clinical Dementia Rating (CDR) was statistically significant with the sufficiency of Vitamin D group (4.6 ± 1.8 vs. 4.5 ± 2.1; p = 0.01) ; respectively, and statistically significant differences were observed when comparing with the cognitive impairment groups. In the univariate logistic regression analysis, the Vitamin D deficiency and insufficiency groups were significantly associated with MoCI (OR, 95% confidence interval [CI] 3.438 [1.256, 8.078]; p < 0.01 respectively, and OR, 95% CI 4.350 [1.258, 7.072]; p < 0.01, respectively). In the group with SCI, when compared with the sufficiency of Vitamin D control group, significant associations were also observed for the deficiency and insufficiency states (OR, 95% CI 5.953 [1.157, 15.792]; p < 0.01 and OR, 95% CI 5.125 [1.197, 15.434]; p = <0.01, respectively).* ***Conclusion:*** *Serum Vitamin D deficiency was associated with severe cognitive impairment; but not with the moderate cognitive impairment. So our study does not indicate that Vit D is a direct risk factor to cognitive decline, rather Vit D could be a covariable factor.*

Keywords: *vitamin D deficiency, cognitive function*

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Introduction:

Dementia affects around 50 million people globally.1 These numbers are expected to reach 82 million by 2030 and 152 million by 2050, with the majority of people arriving from low- and middle-income nations.2 According to recent studies, vitamin D plays an essential role in preventing and/or lowering the risk of a range of neurodegenerative illnesses, including dementia, in addition to its well-established role in bone and calcium metabolism. It is believed that 40-80% of geriatric patients have low serum vitamin D levels.3

Other functions of vitamin D in the body include cell growth modulation, neurogenesis, neuroprotection, detoxification, immunological function regulation, and inflammation reduction.4 Vit D receptors are located in the human cortex and hippocampus, which are crucial areas for cognitive functioning, and an absence or malfunction has been associated with neurodegenerative dementia such as Alzheimer disease (AD). Indeed, in elderly patients with AD, Vit D deficiency is very common with an incidence of 70% to 90%.[5] Several studies have established an association between decreased cognitive function and decreased serum levels of vitamin D [6-9]. Both vitamin D receptor (VDR) and1á-hydroxylase, the enzyme that converts 25-hydroxyvitaminD [25OHD] to its active form 1, 25-dihydroxyvitaminD (1,25OHD) are present in increased concentrations in regions of the brain essential for cognition.[10]

Recent research has connected vitamin D deficiency to cognitive deterioration or dementia. Despite a few suggestions, the association between serum vitamin D levels and cognitive performance remained unclear. A variety of neuroprotective mechanisms have been found, including enhanced amyloid-beta peptide phagocytosis, modulation of neurotrophins and calcium homeostasis, anti-inflammatory and antioxidant action, implying that vitamin D may play an important role in dementia prevention.[11,12] In the normal brain, vitamin D neuroprotection is mostly achieved by the Vit D receptor, a nuclear receptor that works in tandem with the retinoid X receptor to control gene transcription.[13]

Due to its neuroprotective properties, vitamin D deficiency may result in impaired memory and cognitive abilities. The human cortex and hippocampus contain vitamin D receptors, which are essential for cognitive function. Neurodegenerative diseases like Alzheimer's disease (AD) have been linked to the lack or failure of these receptors. In fact, vitamin D insufficiency affects 70% to 90% of older AD patients.[14.]

As Vit D has a neuroprotective function; the insufficiency of Vit D may lead to decreased memory and cognitive function. Vit D receptors are located in the human cortex and hippocampus, which are crucial areas for cognitive functioning, and an absence or malfunction has been associated with neuro-degenerative cognitive function such as AD. Indeed, in elderly patients with AD, Vit D deficiency is very common with an incidence of 70% to 90%.[14]

The average life expectancy of the people of Bangladesh has risen in 2025; life expectancy is projected to be around 74.07 to 75.2 years, according to report by the Bangladesh Bureau of Statistics (BBS). As the average life expectancy rises, dementia has been on the rise in our nation, which is a serious public health concern. However, because there are no national dementia registries, it is unknown how widespread dementia is in Bangladesh. In our prospective study, there is no information on vitamin D insufficiency and cognitive performance in older patients. In order to avoid cognitive decline, active medical intervention is necessary due to the expanding geriatric population and longer life expectancy. Finding modifiable factors linked to cognitive decline is so important. Dementia has been increasing in our country which is a major issue of public health concern currently as the average life expectancy increasing. Investigating the connection between serum vitamin D levels and overall cognitive performance in elderly Bangladeshis was the aim of this investigation. The results of this study could lead to new research on dementia prevention and alternative management.

Methods:

This case-control study was conducted on 50 impaired cognitive patients and 50 healthy individuals as the control group who will attend in the Department of Neurology of Sir Salimullah Medical College & Mitford Hospital, Dhaka, Bangladesh during one year (2022-2023) will be included in the study. Both groups were randomly selected and divided to two groups, according to their age (older than 60 and above).They were also classiûed in terms of education in three groups: higher than an secondary , secondary, primary education and illiterate. Dementia in accordance with inclusion criteria were having a diagnosis of cognitive function decline with MMSE and patient with above the age of 60 years or above. Exclusion criteria were based on the common criteria for this topic and those that were speciûcally related to this study, such as a history of bone disease or fractures, renal or liver failure, a history and current risk of debilitating diseases, such as malignancies and some neurological disorders, such as Multiple Sclerosis (M.S), patients treated with corticosteroids or those who recently used medical supplements and those who refused to continue being in the study.

All data including demographic information (age, gender, marital status, education, and occupation), serum vitamin D levels of the two groups, cardinal features, duration (time between the onset of symptoms until diagnosis and entry in the study, causes of decline cognitive function in the elderly and relevant laboratory investigations will done and recorded.

We evaluated Vit D level of all dementia patients. Blood samples were collected from each participant in sodium citrate tubes. Blood samples obtained after the patient had fasted for 12 hours and rested for at least 15 minutes were centrifuged and stored at "80°C until analyzed. Serum levels of 25(OH) D were measured by radioimmunoassay (RIA kit; DiaSorin, Stillwater, Minnesota): intraassay and interassay coefficients of variation were 8.1% and 10.2%, respectively. The result was read with a specific apparatus provided with the kit. This analysis will be done in standard local laboratory. The serum Vit D levels were measured by nanogram per milliliter (ng/mL), and the results were evaluated with 95% confidential interval.

After inclusion in the study information on patient socio-demographics, clinical characteristics of impaired cognitive function in the elderly and relevant laboratory investigations including Vit D will done. All the data will be collected and stored in Case Report Forms (CRF).

Data analysis:

Continuous variables were shown as mean (SD) or medians (quartiles) depending on the normal or non-normal distribution of data, while categorical variables were represented as percentages. Student's t-test was applied for normal distribution test, while the asymmetrically distributed variables were compared using the Kruskal–Wallis, chi-squared test, Univariate and Multivariate Regression Analysis was employed for potential confounding factors. All statistical analyses were performed with SPSS for Windows, version 25.0 (SPSS Inc., Chicago, IL). P-value <0.05 was considered statistically significant.

Operational definition:

Cognitive assessment:

The Mini-Mental State Examination (MMSE) or Folstein test is a 30-point questionnaire that was used extensively in clinical and research settings to measure cognitive impairment.[13] Cognitive function was assessed by trained neurologists who were blinded to the subjects' clinical presentations and laboratories using of the Mini-Mental State Examination (MMSE). Scores for the MMSE range from 0 to 30 [15]. Lower scores indicate greater cognitive impairment, and cognitive impairment was defined by an MMSE. Any score of 24 or more (out of 30) indicates a normal cognition. Below this, scores can indicate severe (d"9 points), moderate (10–18 points) or mild (19–23 points) cognitive impairment.

Katz Index:

The Katz Index of Independence in Activities of Daily Living, commonly referred to as the Katz ADL, is the most appropriate instrument to assess functional status as a measurement of the client's ability to perform activities of daily living independently[16]. The Index ranks adequacy of performance in the six functions of bathing, dressing, toileting, transferring, continence, and feeding. Clients are scored yes/no for independence in each of the six functions. A score of 6 indicates full function, 4 indicate moderate impairment, and 2 or less indicates severe functional impairment.

Frontal Assessment Battery (FAB):

The FAB is a brief tool that can be used at the bedside or in a clinic setting to assist in discriminating between dementias with a frontal dysexecutive phenotype and Dementia of Alzheimer's Type (DAT)[17]. The FAB has validity in distinguishing Fronto-temporal type dementia from DAT in mildly demented patients (MMSE > 24). Total score is from a maximum of 18, higher scores indicating better performance.

Verbal fluency test:

The verbal fluency test (VFT) is a short screening test that evaluates cognitive function. It's often used by physicians and other practitioners if there is some concern that the person may have cognitive impairment.[18] To score the VFT, count up the total number of animals or words that the individual is able to produce.[3]ÿþ A score of under 17 indicates concern, although some practitioners use 14 as a cutoff. Typically, if someone scores less than 17, the test administrator will use additional tests to further evaluate cognition.

Clock drawing test (CDT):

A clock drawing test (CDT) is a neurological test used for the assessment of cognitive impairment based on sketches of a clock completed by a patient[19]. Usually, a medical expert assesses the sketches to discover any deficiencies in the cognitive processes of the patient. The Interpretations of CDT score are if score 10 suggests that cognitive impairment is unlikely, 8 or 9 must be interpreted clinically, <8 indicates cognitive impairment and <5 indicates prominent impairment.

Vitamin D level assessment:

The normal serum reference range for total 25-hydroxyvitamin D is 30–100 ng/ml, and in this study,

vitamin D insufficiency was defined as a serum level <30 ng/ml, and vitamin D deficiency was defined as a serum level <20 ng/ml[20] Patient's written consent had been taken. This study protocol was approved by ethical committee of Sir Salimullah Medical College, Dhaka, Bangladesh.

Results:

Sociodemographic and Heath characteristics of participants are shown in Table 1. 50 patients with acute cognitive impairment (24 male and 26 female) and 50 control subjects (26 male and 24 female) were included in the study. The age of patients ranged from 60 to 84 years with a mean of 70.7 +/-6.3 years 52% (n = 136) were female. While the age of control subjects ranged from 26 to 70 years with a mean of 61.480 +/ -7.15 years (p =0.613). (Table 1 & 2). Among controls 24 (48%) were females and 26 (52%) were males. Twenty three subjects (46%) had Severe cognitive impairment; 16 (32%) had moderate cognitive impairment; and 11 (22%) had mild cognitive impairment. Thirty patients (60%) had diploma, 52% (n = 26) had hypertension diagnosis, 42% (n = 21) had dyslipidemia, 36% (n = 18) had diabetes, 20% (n = 10) had hypothyroidism, and 14% (n = 7) had atrial fibrillation. The overall prevalence of severe Vitamin D deficiency was 76%, being more frequent in the severe cognitive impairment group (47.4%), followed by the moderate cognitive (31.6%) and the mild cognitive impairment (21%) (p<0.001) groups.

Table I

Sociodemographic and Heath characteristics of participants

Characteristics	Total)	Cognitive Impairment			
	(n=50	Severe(n=23)	Moderate(n=16)	Mild(n=11)	P value
Age (mean, SD)	70.7 (6.3)	72.1 (5.3)	70.5 (8.3)	69.9 (5.9)	0.01
Sex (n, %)					<0.01
Male	24 (48)	11 (48)	9 (56)	4 (36)	
Female	26 (52)	12 (52)	7 (44)	7 (64)	
Education (n, %)					0.02
Diploma	30 (60)	12 (52)	10 (63)	8 (73)	
Under diploma	9 (18)	4 (17)	4 (25)	1 (9)	
Illiterate	11 (22)	7 (30)	2 (13)	2 (18)	
Past illness history (n, %)					
HTN	26 (52)	11 (48)	7 (44)	8 (73)	0.09
DM	18 (36)	8 (35)	8 (50)	2 (18)	0.40
HYPOTHYROIDISM	10 (20)	3 (13)	4 (25)	3 (27)	0.08
Dyslipidemia	21 (42)	11 (48)	4 (25)	6 (55)	0.13
AF	7 (14)	4 (17)	1 (6)	2 (18)	0.87
Vitamin D level (n, %)					<0.01
Deficiency	38 (76)	18 (78)	12 (75)	8 (73)	
Insufficiency	12 (24)	5 (22)	4 (25)	3 (27)	

Cognitive Impairment Status: Normal cognition: >= 24 points, Severe: ≤9 points, Moderate:10–18 points, Mild: 19–23 points.

Table II

Demographic characteristics for control group (N=50)

Characteristics	Total (N=50)	Percentage
Age (mean, SD)	61.480 (7.1493)	
Gender		
Male	26	52.0
Female	24	48.0
Vitamin D level		
Deficiency	3	6.0
insufficiency	25	50.0
Sufficiency	22	44.0

Among 50 control subjects (26 male (52%) and 24(48%) female) were included in the study. The age of control subjects ranged from 26 to 70 years with a mean of 61.480 +/-7.15 years (p =0.613).

Table: III

Executive Functions status of the participants

Executive Functions (mean, SD)	Total (n=50)	Cognitive Impairment			
		Severe(n=23)	Moderate(n=16)	Mild(n=11)	p Value
CDR	4.6 (1.9)	6.2 (1.01)	3.8 (1.0)	2.3 (0.5)	<0.01
K Index	3.0 (1.2)	4.0 (0.7)	2.6 (0.5)	1.5 (0.5)	<0.01
Verbal Fluency	7.7 (3.3)	4.8 (1.3)	9.0 (2.3)	11.6 (1.9)	<0.01
Clock Drawing Test	5.3 (2.1)	3.6 (1.4)	6.2 (1.5)	7.4 (1.4)	<0.01
FAB	8.9 (3.7)	6.4 (2.9)	10.6 (3.1)	11.7 (2.7)	<0.01

*Kruskal–Wallis, *Chi-square test, K Index: Katz Index of independence in Activities in Daily Living, FAB: Frontal Assessment Battery, CDR: Clinical Dementia Rating.

In Table: III, Executive function was measured by the Clinical Dementia Rating (CDR) (6.2+/- 1.01, 3.8+/- 1.0, 2.3+/-0.5; p = <0.10) respectively, and statistically significant differences were observed when comparing with the cognitive impairment groups. The same differences were noted between these groups in the Katz Index of independence in Activities in Daily Living (K Index) (4.0+/- 0.7, 2.6+/-0.5, 1.5+/-0.5; p = <0.10); verbal fluency (4.8 ± 1.3, 9.0 ± 2.3, 11.6+/-1.9 words, respectively; p < 0.01), and clock-drawing test scores (3.6 +/- 1.4, 6.2 ± 1.5 , 7.4 +/-1.4 points; p = <0.01) respectively.

In Table IV , Cognitive status was measured by the MMSE, and statistically significant differences were observed when comparing the deficiency group with the sufficiency of Vitamin D group (11.8 ± 5.8 vs. 12.5 ± 4.7; p < 0.01).Executive function was measured by the Clinical Dementia Rating (CDR), and statistically significant differences were observed when comparing the deficiency group with the sufficiency of Vitamin D group (4.6 ± 1.8 vs. 4.5 ± 2.1; p = 0.01) ; respectively, and statistically significant differences were observed when comparing with the cognitive impairment groups. The same differences were noted between these groups in the K Index (3.1 ± 1.2 vs. 4.5 ± 2.1; p = 0.01); verbal fluency (7.4 ± 3.4 vs. 8.5 ± 3.1; p = <0.01);, respectively , and clock-drawing test scores (5.2 ± 2.2 vs. 5.5 ± 2.1; p = 0.02);respectively, FAB (8.8 ± 3.8 vs. 9.3 ± 3.4; p < 0.01).

Table-IV

Participants' cognitive performance according to serum Vitamin D levels

Cognitive Status	Deficiency (n=38)		Insufficiency (n=12)		P Value
	Mean	Standard Deviation	Mean	Standard Deviation	
MMSE	11.8	5.8	12.3	4.7	<0.01
Executive functions					
CDR	4.6	1.8	4.5	2.1	0.01
K Index	3.1	1.2	2.7	1.1	0.01
Verbal Fluency	7.4	3.4	8.5	3.1	<0.01
Clock Drawing Test	5.2	2.2	5.5	2.1	0.02
FAB	8.8	3.8	9.3	3.4	<0.01

Serum Vitamin D levels: sufficiency: > 30 ng/mL, Insufficiency: 21-29 ng/mL, Deficiency: d" 20 ng/Ml. Means and SD presented. *Kruskal–Wallis: mini-mental state examination, K Index: Katz Index of independence in Activities in Daily Living, FAB: Frontal Assessment Battery, CDR: Clinical Dementia Rating.

Table V

Univariate and Multivariate Regression Analysis

Cognition	OR[95% CI]	P Value	Adjusted OR[95% CI]	P Value
Moderate				
Gender				
Male	1.639 [1.360, 7.46]	0.04	1.604 [1.367, 7.019]	0.02
Female (ref)				
Education				
Diploma	1.391[1.063, 2.446]	0.02	1.429 [1.070, 2.614]	0.03
Under diploma	1.91 [1.057, 14.534]	0.01	1.143 [1.077, 16.947]	0.01
Illiterate (ref)				
Vitamin D level				
Deficiency	3.438 [1.256, 8.078]	<0.01	4.350 [1.258, 7.072]	<0.01
Insufficiency (ref)				
Severe				
Gender				
Male	1.992 [1.400, 9.914]	<0.01	2.250 [1.465, 10.883]	<0.01
Female (ref)				
Education				
Diploma	1.205 [1.003, 10.894]	0.01	1.250 [1.143, 10.940]	0.01
Under diploma	3.402 [1.165, 70.112]	0.02	4.000 [1.211, 75.659]	0.01
Illiterate (ref)				
Vitamin D level				
Deficiency	5.953 [1.157, 15.792]	<0.01	5.125 [1.197, 15.434]	<0.01
Insufficiency (ref)				

OR: Odds ratio from univariate analysis, Adjusted OR: Odds ratio after adjusting for sex and education. Vitamin D sufficiency: > 30 ng/mL, Insufficiency: 21-29 ng/mL, Deficiency: ≤20 ng/mL, ref: Reference Category.

In Table: V, the univariate logistic regression analysis, the Vitamin D deficiency and insufficiency groups were significantly associated with moderate cognitive impairment (OR, 95% confidence interval [CI] 3.438 [1.256, 8.078] ; p < 0.01 respectively, and OR, 95% CI 4.350 [1.258, 7.072]; p < 0.01, respectively). In the group with severe cognitive impairment ,when compared with the sufficiency of Vitamin D control group, significant associations were also observed for the deficiency and insufficiency states (OR, 95% CI 5.953 [1.157, 15.792]; p < 0.01 and OR, 95% CI 5.125 [1.197, 15.434]; p = <0.01, respectively). After adjusting for age, sex, and years of education, the association remained significant But In the severe cognitive impairment group, the association also remained significant in both the deficiency (OR:, 95% CI 1.992 [1.400, 9.914] p < 0.01) and 2.250 [1.465, 10.883]; p < 0.01.(Table IV). But in the moderate cognitive impairment group in the association remained insignificant in moderate cognitive impairment in both the deficiency group (OR, 95% CI 1.639 [1.360, 7.46]; p= 0.04) and 1.604 [1.367, 7.019]; p= 0.02.

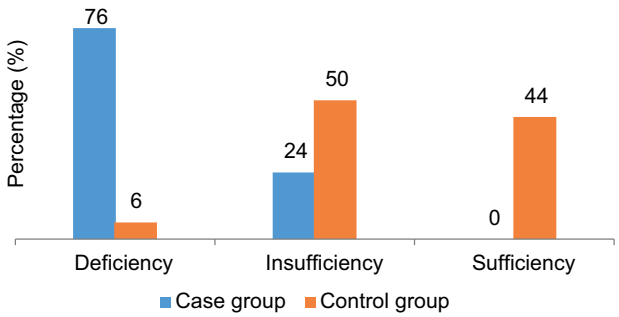

Figure 1: *Vitamin D levels among Case and Control group (for each group, n=50)*

Discussion:

Vitamin D is essential for many physiological functions, including immune response modulation and brain development regulation which is a secosteroid hormone,. By influencing the production of proinflammatory cytokines and lowering the load of oxidative stress, vitamin D has anti-inflammatory and neuroprotective effects in the brain. Additionally, vitamin D plays a part in the immune cell 20's removal of amyloid plaques. Consequently, there is mounting preclinical data about vitamin D's potential to inhibit amyloid buildup and, consequently, cognitive deterioration 21. Numerous research have examined the connection between vitamin D and cognitive function, and many of them have found a positive link.

This study observed various signs of cognitive decline. Age, gender, education level, hypertension, diabetes, hypothyroidism, and dyslipidemia were all linked to cognitive impairment. Cognitive impairment risk variables include age, gender, education period, annual income, alcohol usage, smoking, depression, obesity, and cardiovascular disease. 30 . As a result, several examinations should be conducted to determine the source of the deterioration in cognitive function. We also discovered that numerous baseline features are associated with vitamin D insufficiency. These findings suggest that a variety of factors influence both VitD levels and cognitive performance. In addition to the individuals' health status, their socioeconomic status was linked to their VitD levels.

This outcome is consistent with earlier VitD research. After adjusting for the covariates, this study revealed a relationship between VitD and cognitive function.

Llewellyn et al. linked VitD levels to cognitive abilities like the MMSE East Boston Memory test in a cross-sectional study of 3325 senior Americans 65 and older. Participants with vitamin D deficiency had a higher OR of cognitive impairment, according to this study's adjusted multivariate logistic model.31. Over a mean of 4.8 years, a recent prospective study of 318 older adults revealed a significant annual decline in verbal memory (immediate and delayed word list recall) in those with moderate and severe serum vitamin D deficiencies (12 to <20 and <12 ng/mL, respectively) compared to those with sufficient levels (20 ng/mL).32.

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However, using the MMSE to test cognitive status, we found statistically significant differences between the vitamin D deficiency and sufficiency groups (11.8 ± 5.8 vs. 12.5 ± 4.7; $p < 0.01$). Using questionnaires like the Katz Index of Independence in Activities of Daily Living (ADL) 34, 35, functional assessments measure the capacity to carry out ADLs. Functional evaluations are useful for determining the severity of dementia and for giving patients and their caregivers the right direction. Global staging scales can be used in clinical practice to measure both cognitive and functional deficiencies.

Similar results were found in our study when evaluating executive function using the Clinical Dementia Rating (CDR). There were statistically significant differences between the deficiency group and the sufficiency of vitamin D group (4.6 ± 1.8 vs. 4.5 ± 2.1; $p = 0.01$); similarly, there were statistically significant differences between the cognitive impairment groups. The K Index (3.1 ± 1.2 vs. 4.5 ± 2.1; $p = 0.01$), verbal fluency (7.4 ± 3.4 vs. 8.5 ± 3.1; $p = <0.01$), clock-drawing test scores (5.2 ± 2.2 vs. 5.5 ± 2.1; $p = 0.02$), and FAB (8.8 ± 3.8 vs. 9.3 3.4; $p < 0.01$) showed the similar disparities between these groups.

Only one French study has examined vitamin D insufficiency in preclinical stages or MCI; it found a strong correlation between low vitamin D levels with all forms of MCI (HR 25.4, 95% CI 3.2-201.2, $p = 0.002$)41. The idea that the course of cognitive impairment could be altered in the early phases of the illness is supported by this data. Since vitamin D functions as a neurosteroid—that is, it crosses the blood-brain barrier and binds to its receptor in neurons—it has an effect on MCI through the same processes as dementia. Hypovitaminosis D may contribute to brain dysfunction and cognitive decline by reducing defensive systems.

Thus, it makes sense to think that low vitamin D levels during the early or preclinical stages (MCI) contribute to the development of cognitive disorders[42]. According to our study, foundoup's vitamin D shortage was more pronounced (76%) in cognitive impairment, while the control group's vitamin D sufficiency and insufficiency were both 94%.

A meta-analysis of 20,750 people from various nations revealed a significant link between vitamin D level and cognitive impairment (HR 1.24, 95% CI 1.14-1.35; p < 0.001).44. A cohort research of 1200 older Chinese people with a 2-year follow-up found a link between low Vitamin D levels and poorer global cognitive performance (HR: 2.89, 95% CI 1.36-6.14; p = 0.004).[44]. Another multi-ethnic longitudinal research of older adults with a 4.8-year follow-up found that individuals with Vitamin D insufficiency were more likely to experience cognitive impairment. The Cardiovascular Health Study conducted in the United States has the longest follow-up period.

Vitamin D deficiency was linked to an increased risk of all-type cognitive function impairment (HR: 2.25, 95% CI 1.2-4.1, p = 0.002) and AD (HR: 2.2, 95% CI 1-4.8, p = 0.008) among the 658 cognitively healthy adults in this study [46].

The vitamin D deficiency and insufficiency groups were significantly linked to moderate cognitive impairment in our study, according to the univariate logistic regression analysis (OR, 95% confidence interval [CI] 3.438 [1.256, 8.078]; p < 0.01, respectively, and OR, 95% CI 4.350 [1.258, 7.072]; p < 0.01, respectively).

Significant correlations were also found for the deficiency and insufficiency states in the severe cognitive impairment group when compared to the vitamin D sufficiency control group (OR, 95% CI 5.953 [1.157, 15.792]; p < 0.01 and OR, 95% CI 5.125 [1.197, 15.434]; p = <0.01, respectively). The connection persisted even after controlling for years of schooling, sex, and age. However, The association continued to be significant in the severe cognitive impairment group for both the deficit (OR:, 95% CI 1.992 [1.400, 9.914] p < 0.01) and 2.250 [1.465, 10.883]; p < 0.01.However, in both the deficit group (OR, 95% CI 1.639 [1.360, 7.46]; p = 0.04) and the mild cognitive impairment group (1.604 [1.367, 7.019]; p = 0.02), the association remained negligible. The association between serum vitamin D concentrations and cognitive impairment found in our study is in agreement with previous evidence that analysed vitamin D in quartiles[47-49].

This study's strength is its statistical control of potentially confounding demographic factors, such as clinical comorbidity, socioeconomic level, and prior medical history. After adjusting for factors that could have an impact on either VitD level or cognitive performance, there is no correlation between VitD and cognitive function in this study. Despite the fact that these findings contradict those of other research, our findings are significant for comprehending the connection between vitamin D and cognition because this is a large population study and the data includes a variety of demographic and cognitive measures.

Limitations:

This study has certain limitations. First, vitamin levels and cognition are influenced by a number of confounding factors, a prospective study or randomized controlled trials are required to examine how changes in vitamin D levels affect cognitive performance. Second, the participants' consumption of vitamin D supplements was not examined. Many postmenopausal women and older men may take vitamin D as a supplement because it is commonly used to prevent and treat osteopenia and osteoporosis. However, we did not look into supplement consumption in this study. Third, because the study excluded patients with dementia or those who could not fully comprehend the questionnaire; it did not examine the association between VitD and dementia.

Conclusion:

Serum vitamin D deficiency was significantly higher in patients with severe cognitive impairment over 60 years old compared to controls, based on logistic regression analysis with adjustments for gender, age, and education. However, there was no significant association between moderate cognitive impairment and serum vitamin D levels. So, while our data does not show that vitamin D is a direct risk factor for cognitive impairment, it may be a covariable factor.

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Conflict of Interest:

No author has any conflict of interest to disclose for this manuscript. The authors themselves are responsible for their ideas and views expressed in this article, which do not necessarily represent the views, decisions or policies of the institutions with which they are affiliated.

Ethical Approval:

The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). This study was approved by the Institutional Review Board of the Sir Salimullah Medical College. Written informed consent was taken from all the patients before taking part of the study.

Author Contributions

Aminur Rahman contributed to the concept and design. Aminur Rahman, Abul Hasnat Md. Russel performed data collection and compilation. Aminur Rahman ,Sabina Yasmin, Mohammed Nazmul Huq contributed in data analysis and manuscript writing. All authors revised and approved the manuscript.

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