Clinical Neurology and Neuroscience
2022; 6(1): 6-13
http://www.sciencepublishinggroup.com/j/cnn
doi: 10.11648/j.cnn.20220601.12
ISSN: 2578-8922 (Print); ISSN: 2578-8930 (Online)

Correlation Between Serum 25-hydroxyvitamin D Level and Cognitive Function Among the Elderly in Enugu State, Nigeria

Ezra Agbo[1, 2], Ifeyinwa Nnakenyi[1], Collins Amadi[3, 4, *], Chituru Orluwene[3], Chika Okwor[1], Aloysius Aleke[5], Chidozie Agu[5]

[1]Department of Chemical Pathology, University of Nigeria Teaching Hospital (UNTH), Enugu, Nigeria

[2]Department of Chemical Pathology, Federal Medical Center, Owerri, Nigeria

[3]Department of Chemical Pathology, Rivers State University, Port Harcourt, Nigeria

[4]Department of Chemical Pathology, PAMO University of Medical Sciences, Port Harcourt, Nigeria

[5]Everight Diagnostic and Laboratory Services Ltd, Owerri, Nigeria

Email address:

collins338@yahoo.com (C. Amadi)

[*]Corresponding author

To cite this article:

Ezra Agbo, Ifeyinwa Nnakenyi, Collins Amadi, Chituru Orluwene, Chika Okwor, Aloysius Aleke, Chidozie Agu. Correlation Between Serum 25-hydroxyvitamin D Level and Cognitive Function Among the Elderly in Enugu State, Nigeria. *Clinical Neurology and Neuroscience*. Vol. 6, No. 1, 2022, pp. 6-13. doi: 10.11648/j.cnn.20220601.12

Received: January 4, 2022; **Accepted**: January 19, 2022; **Published**: January 26, 2022

Abstract: *Background*: Low serum vitamin D is now implicated in many disease conditions among the elderly including cognitive impairment. Cognitive impairment is a common disease among elderly people causing much financial and social burden to the elderly, their families and caregivers. Therefore, the study aimed to determine the relationship between serum 25-hydroxyvitamin D (25(OH)D) concentration and cognitive function among elderly people in Enugu State, South-eastern Nigeria. *Methods*: This was a cross-sectional survey of subjects aged ≥ 60 years with cognitive impairment in communities and old People's home in Enugu State in Nigeria and age- and sex-matched controls. The subjects were further divided into those with severe cognitive impairment and those with mild cognitive impairment using the Mini-Mental State Examination (MMSE). Serum 25(OH)D and parathyroid hormone (PTH) were assayed by enzyme-linked immune-sorbent assay while serum calcium, inorganic phosphate, and albumin were determined using the spectrophotometric method. Student t-test was used to compare mean values obtained, while Pearson correlation was used to determine relationships between continuous data. A p-value<0.05 was considered to be statistically significant. *Result*: One hundred and four (104) patients comprising of 58 males and 46 females were recruited for the current study. Among the study subjects compared to those of the controls, there were significantly decreased levels of serum 25(OH)D (p=0.0001) and the adjusted calcium (p=0.0001) concentrations but significantly increased level of serum PTH (p=0.0001) and inorganic phosphate (p=0.005) concentrations. Also, the mean values of serum 25(OH)D and adjusted calcium were significantly lower, serum PTH values were significantly higher while serum inorganic phosphate concentrations showed no significant difference for those with severe cognitive impairment when compared to values of those with mild cognitive impairment and controls. Additionally, there was also significant positive correlations between serum 25(OH)D and cognitive function (r=0.504, ρ<0.05). *Conclusion*: The study findings suggest that decreased serum Vitamin D concentration is associated with diminished cognitive function among the elderly and vice versa. Hence, the determination of Vitamin D status among elderly patients presenting with impairment of cognitive function is highly recommended. However, further studies are needed to validate findings from the present study.

Keywords: Elderly, Cognitive Function, Vitamin D, 25(OH)D

1. Introduction

The role of vitamin D deficiency in many bone diseases have been established for decades. In recent years, researchers have implicated low vitamin D status in many non-bone disease conditions such as the decline in cognitive function (cognitive impairment), hypertension and cardiovascular diseases, immune disorders, cancers, diabetes mellitus, etc [1]. This is because vitamin D receptor (VDR) is expressed in virtually all human cells and so its activation (or deactivation) regulates the functioning of almost all body tissues/organs [2]. The elderly (persons who according to the United Nations are aged 60 years and above) [3] are more prone to vitamin D deficiency due to the following risk factors: diminished sunlight exposure, decreased dietary intake, reduced skin thickness, impaired intestinal absorption, and impaired hydroxylation in the liver and kidneys [4, 5]. This partly explains why the above-mentioned chronic disease conditions associated with low vitamin D status are more common among elderly people.

One of the chronic non-bone diseases associated with vitamin D deficiency, which is very common among elderly people, is cognitive impairment. Cognitive function involves all intellectual processes of perception, thinking, reasoning, and remembering [6]. Therefore, cognitive impairment refers to a complex neurological disease condition in which there is global and irreversible decline in cognitive function that could undermine daily functioning [7]. When cognitive impairment is very severe, it is usually referred to as dementia. But when mild, it is usually called mild cognitive impairment (MCI) [8]. Cognitive impairment is fast becoming an increasing medical problem as older people now represent a very significant fraction of the world's population, and it is predicted to be worse in developing countries. Statistically, about 35.6 million people are currently living with dementia worldwide [9], and that the number will nearly double every 20 years, with the figure getting to 115.4 million by 2050, with the majority living in developing countries [10]. Of the total number of people with dementia around the world, 57.7% lived in developing countries in 2010 [11] and a significant increase to 70.5% by 2050 is expected [12].

Researchers have argued that the presence of VDR even in the central nervous system (CNS) explains why vitamin D deficiency is associated with cognitive impairment. Two mechanisms have been suggested as possible means of how vitamin D deficiency causes this disorder: (a) by causing specific neuronal apoptosis through decreasing the expression by cytochrome and decreasing the cell cycle of neurons, and/or (b) through its association in the neurotrophic factors such as nerve growth factors (NGF), brain-derived neurotrophic factor (BDNF), glial cell-derived neurotrophic factor (GDNF), which are proteins that are involved in the growth and survival of developing neurons, and also maintenance of mature neurons [13, 14].

Hence, the study aimed to determine the relationship between serum 25-hydroxyvitamin D level and cognitive function among elderly people in Enugu State.

2. Materials and Methods

2.1. Study Design, Location, and Site

The research was a cross-sectional study of serum 25-hydroxy vitamin D and cognitive function of elderly people. The study was carried out in the Department of Chemical Pathology, University of Nigeria Teaching Hospital (UNTH), Ituku/Ozalla Enugu, Nigeria. Subjects were recruited through a medical outreach program that was conducted at St. Paul Old People's Home, Agbani Road, Enugu, an urban center, and from three other rural communities of Enugu State: Ituku community in Awgu Local Government Area (LGA), and Akpakume and Nze communities both in Udi LGA.

2.2. Ethical Consideration

Ethical clearance was obtained from the Human Research Ethics Committee (HREC) of UNTH Enugu before the commencement of the study, and permission was sought from the Management of the Old People's Home and also from community leaders of the rural communities that were involved in the research. The research was performed according to the Declaration of Helsinki and confidentiality of results was maintained.

2.3. Informed Consent

Oral and written informed consent was obtained from each participant before enrolment into the study. But for those who were mentally deranged, their caregivers' approval was obtained.

2.4. Study Population

The study population was made up of subjects (n=52) from the study area who satisfied the inclusion criteria mentioned below.

2.5. Eligibility Criteria

2.5.1. Inclusion Criteria

1) Age 60 years and above.
2) Male and female.
3) Mini-mental state examination (MMSE) scores 0 – 23 for test subjects.
4) MMSE scores 24 –30 for control subjects.

2.5.2. Exclusion Criteria

1) Hypertensive (Blood Pressure>140/90 mmHg or being on any anti-hypertensive).
2) Diabetic mellitus patient (random plasma glucose>11.1mmol/L or on anti-diabetic medication).
3) Renal disease patient (previous history of renal disease and/or proteinuria).
4) Chronic liver disease (previous history of yellowness of sclera and/or past medical history of liver disease).

5) History of head injury or trauma before decline of cognition.
6) Family history of cognitive impairment.
7) History of any chronic illness before decline of cognition (e.g. HIV/AIDS).
8) Anyone who is on vitamin D supplementation.

2.6. Sample Size Determination

The sample size was determined using the formula described by Araoye, 2004 [15].

2.7. Data Collection

During the program questionnaires were administered by an interviewer to obtain information about their biodata, previous/current medical, drug, and family histories.

The blood pressure of the subjects was measured using a stethoscope (Littmann®) and sphygmomanometer (Accoson®) after they had rested for at least 10 minutes, to exclude those that were hypertensive.

The random blood glucose of the subjects was determined using glucometer (Accu-Chek® USA). Also, urinalysis was carried out by the use of Combi-9 strips (Accu-Answer®) to rule out kidney and liver diseases.

2.8. Clinical Assessment of Cognitive Function

This was done using the Mini-Mental State Examination (MMSE), which is a 30-point questionnaire used to measure cognitive impairment (attached as appendix III). The score obtained was then interpreted as previously described [16, 17].

2.9. Specimen Collection, Processing, and Laboratory Assay

2.9.1. Specimen Collection and Processing

5 ml of venous blood specimen was collected into plain tubes and was allowed to clot and retract, after which it was centrifuged at 3000 rpm for 10 minutes using a standard centrifuge (Labofuge II®, United Kingdom). Then serum samples were obtained for measurements of 25-hydroxy vitamin D, parathyroid hormone, inorganic phosphate, calcium, and albumin concentrations. Sera were stored frozen (-20°C) until analyses were performed. Repeated freeze-thaw cycles were avoided.

2.9.2. Assay of 25-Hydroxy Vitamin D

Serum 25(OH) Vitamin D was analyzed using the enzyme-linked immunosorbent assay (ELISA) as described by Holick in 2009 [18].

2.9.3. Assay of Parathyroid Hormone

Serum intact parathyroid (PTH) was also analyzed using the enzyme-linked immunosorbent assay (ELISA) as described by Mallette in 1991 [19].

2.9.4. Determination of Inorganic Phosphate Concentration

Serum inorganic phosphate was assayed using the phosphomolybdate ultraviolet method as described by Goodwin in 1970 [20].

2.9.5. Determination of Calcium Concentration

Serum calcium was assayed using the Arsenazo-III-spectrophotometric method as described by Janssen *et al* in 1991 [21].

2.9.6. Determination of Albumin Concentration

Serum albumin was analyzed using the Bromocresol green (BCG) method as described by Leonard *et al* in 1971 [22].

2.9.7. Albumin-Adjusted Calcium Concentration

The albumin-adjusted calcium concentration was calculated using the following formula:

$$\text{Albumin-adjusted calcium concentration} = \text{Measured Ca concentration (mmol/L)} + 0.02\,(40 - [\text{Alb}]\ (\text{g/L}))$$

2.10. Data Analysis

This was done using Statistical Package for Social Sciences (SPSS) version 25.0 (IBM®, USA). Serum 25-hydroxyvitamin D (25(OH)D) concentrations had a non-parametric distribution while results of parathyroid hormone (PTH), inorganic phosphate, total calcium, albumin, and adjusted calcium were found to be parametric. Results of 25(OH)D were first log-transformed. Then results of all the analytes were presented as mean ± SD. Student t-test and analysis of variance (ANOVA) were used to compare the mean results of each analyte between two groups and among three groups, respectively. Pearson's correlation coefficient was used to determine the relationship between 25(OH)D and each of cognitive function, adjusted calcium, PTH, and inorganic phosphate respectively among the test subjects. A ρ-value of<0.05 was considered statistically significant.

3. Results

3.1. Demographic and Biochemical Parameters of Study Participants

Results of serum biochemical parameters; 25(OH)D, PTH, inorganic phosphate, total calcium, albumin and adjusted calcium of the 52 subjects as well as those of 52 controls are presented as mean±SD in Table 1. Results of subjects **(**52) were: 25(OH)D: 25.7±5.9 ng/mL, adjusted calcium: 2.16±0.17 mmol/L, PTH: 98.4±12.5 pg/mL and inorganic phosphate: 1.39±0.29 mmol/L and those of controls (52) were: 25(OH)D: 45.6±13.1 ng/mL, adjusted calcium: 2.40±0.09 mmol/L, PTH: 34.2±10.7 pg/mL and inorganic phosphate: 1.14±0.15 mmol/L.

Concentrations of 25(OH)D, and adjusted calcium were significantly ($p<0.05$) lower while levels of PTH and inorganic phosphate were significantly ($p<0.05$) higher for subjects when compared to their controls.

3.2. Biochemical Parameters of Subjects with Severe Cognitive Impairment, Those with Mild Cognitive Impairment and Control Group

Table 2 shows results of biochemical parameters; 25(OH)D, PTH, inorganic phosphate, total calcium, albumin

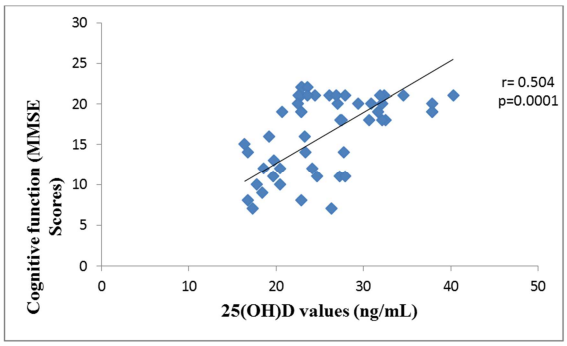
and adjusted calcium presented as mean±SD. Results of subjects with severe cognitive impairment (22) were: 25(OH)D: 21.4±3.8 ng/mL, adjusted calcium: 2.05±0.13 mmol/L, PTH: 128.5±20.1 pg/mL and inorganic phosphate: 1.48±0.29 mmol/L, subjects with mild cognitive impairment (30) were: 25(OH)D: 27.5±5.4 ng/mL, adjusted calcium: 2.17±0.16 mmol/L, PTH: 77.1±32.6 pg/mL and inorganic phosphate: 1.35±0.27 mmol/L and those of controls (52) were: 25(OH)D: 45.6±13.1 ng/mL, adjusted calcium: 2.40±0.09 mmol/L, PTH: 34.2±10.7 pg/mL and inorganic phosphate: 1.14±0.15 mmol/L. Concentrations of 25(OH)D and adjusted calcium were significantly ($p<0.05$) lower, PTH levels were significantly ($p<0.05$) higher while inorganic phosphate concentrations were not significantly ($p>0.05$) higher for subjects with severe cognitive impairment when compared to the other two groups.

3.3. Biochemical Parameters of Male and Female Subjects

Table 3 shows results of serum biochemical parameters; 25(OH)D, PTH, inorganic phosphate, total calcium, albumin and adjusted calcium of male and female subjects presented as mean±SD. Results of male subjects (29) were: 25(OH)D: 28.3±6.4 ng/mL, adjusted calcium: 2.32±0.14 mmol/L, PTH: 98.4±13.6 pg/mL and inorganic phosphate: 1.12±0.23 mmol/L and those of female subjects (23) were: 25(OH)D: 31.7±9.2 ng/mL, adjusted calcium: 2.30±0.11 mmol/L, PTH: 100.9±15.3 pg/mL and inorganic phosphate: 1.13±0.26 mmol/L. Concentrations of 25(OH)D, adjusted calcium PTH and inorganic phosphate showed no statistical significant difference ($p>0.05$) between the two groups.

3.4. Correlation Between 25(OH)D and Cognitive Function (Mmse Scores)

Pearson correlation coefficients and p-values for the relationship between 25(OH)D and each of cognitive function (MMSE Scores) were r=0.504, p=0.0001 among the subjects as presented in Figure 1 below.

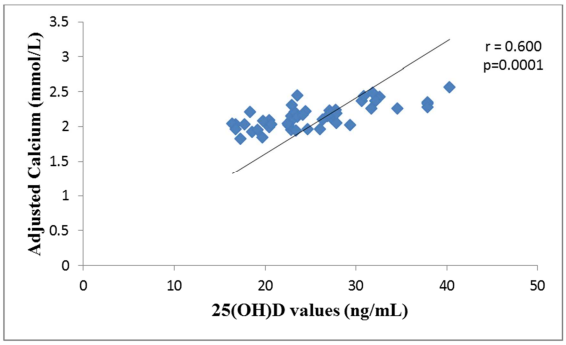


Meaning: Positive correlation that is statistically significant (p=0.0001)

Figure 1. *Correlation between 25(OH)D and cognitive function (MMSE Scores) of subjects.*

3.5. Correlation Between 25(OH)D and Adjusted Calcium

Pearson correlation coefficients and p-values for the relationship between 25(OH)D and adjusted calcium among the subjects were r=0.600, p=0001 as presented in Figure 2 below.

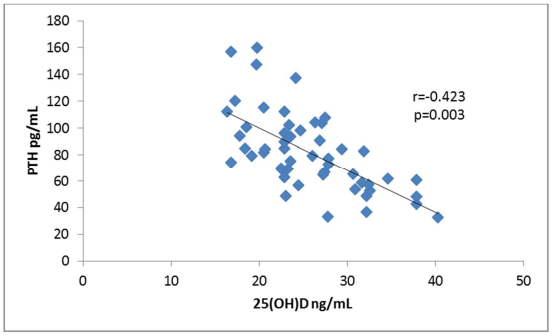


Meaning: Positive correlation that is statistically significant (p=0.0001)

Figure 2. *Correlation between 25(OH)D and adjusted calcium concentrations of subjects.*

3.6. Correlation Between 25(OH)D and Pth

Pearson correlation coefficients and p-values for the relationship between 25(OH)D and PTH (r=-0.423, p=0.003) among the subjects are presented in Figure 3 below.

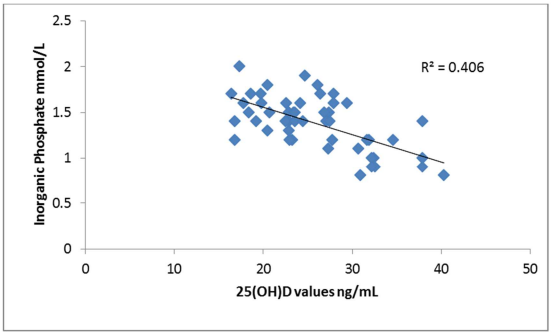


Meaning: Negative correlation that is statistically significant (p=0.003)

Figure 3. *Correlation between 25(OH)D and PTH concentrations of subjects.*

3.7. Correlation Between 25(OH)D and Inorganic Phosphate

Pearson correlation coefficients and ρ-values for the relationship between 25(OH)D and the inorganic phosphate among the subjects were r=-0.382, p=0.045 among the subjects as presented in Figures 4 below.



Meaning: Weak negative correlation that is statistically significant (p=0.045)

Figure 4. *Correlation between 25(OH)D and inorganic phosphate concentrations of subjects.*

Table 1. *Demographic and Biochemical Parameters of Study Participants.*

Parameters	Control M±SD	Test M±SD	p-value
Number (n)	52	52	NA
Age (years)	71.8±12.4	73.6±11.3	0.386
25(OH)D (ng/mL)	45.6±13.1	25.7±5.9	0.000*
PTH (pg/mL)	34.2±10.7	98.4±12.5	0.000*
Inorganic Phosphate (mmol/L)	1.14±0.15	1.39±0.28	0.005*
Total Calcium (mmol/L)	2.27±0.12	2.10±0.13	0.000*
Albumin (g/dL)	53.2±10.7	40.3±12.8	0.021*
Adjusted Calcium (mmol/L)	2.40±0.09	2.16±0.17	0.000*

M±SD: Mean ± standard deviation; *Statistically significant; NA: not applicable.

Table 2. *Biochemical Parameters of Subjects with Severe Cognitive Impairment, Those with Mild Cognitive Impairment and Control Group.*

Parameter	Control (MMSE score 24-30)	Mild Cognitive Impairment (MMSE score 18-23)	Severe Cognitive Impairment (MMSE score 0-17)	p-value
	M±SD/n	M±SD/n	M±SD/n	
Number (n)	52	30	22	NA
25(OH)D (ng/mL)	45.6±13.1	27.5±5.4	21.4±3.8	0.000*
PTH (pg/mL)	34.2±10.7	77.1±32.6	128.5±20.1	0.014*
Inorganic Phosphate (mmol/L)	1.14±0.15	1.35±0.27	1.48±0.29	0.133
Total Calcium (mmol/L)	2.27±0.12	2.14±0.10	2.09±0.18	0.014*
Albumin (g/dL)	53.2±10.7	48.2±11.5	38.8±16.2	0.046*
Adjusted Calcium (mmol/L)	2.40±0.09	2.17±0.16	2.05±0.13	0.032*

M±SD: Mean ± Standard Deviation; *Statistically significant; NA: not applicable.

Table 3. *Biochemical Parameters of Male and Female Subjects.*

Parameter	Male M±SD/n	Female M±SD/n	p-value
Number (n)	29	23	NA
25(OH)D (ng/mL)	28.3±6.4	31.7±9.2	0.162
PTH (pg/mL)	98.4±13.6	100.9±15.3	0.205
Inorganic Phosphate (mmol/L)	1.12±0.23	1.13±0.26	0.374
Total Calcium (mmol/L)	2.27±0.10	2.23±0.13	0.192
Albumin (g/dL)	42.6±10.5	51.2±13.7	0.012*
Adjusted Calcium (mmol/L)	2.32±0.14	2.30±0.11	0.214

M±SD: Mean ± Standard Deviation; *Statistically significant; NA: not applicable.

4. Discussion

Low serum vitamin D concentration has been implicated in many disease conditions among elderly people, including cognitive impairment. These diseases are common among the elderly because of high-risk factors for low Vitamin D common to this age group, which include diminished sunlight exposure, decreased dietary intake, reduced skin thickness, impaired intestinal absorption, and impaired hydroxylation in the liver and kidneys [4]. Also, vitamin D receptor (VDR) is present in virtually all cells of the body, including the brain, and vitamin D is involved in not only calcium metabolism but in cell signaling and gene expression in all body cells [23].

Findings from this study showed that serum 25-hydroxy vitamin D (25(OH)D) concentrations among subjects were significantly lower when compared to controls, and there was a positive correlation between concentrations of serum 25(OH)D and cognitive function (MMSE scores) among subjects ($r=0.540$, $p=0.0001$). This is similar to findings of other studies done in the United States and Europe, where patients with dementia/AD were reported to have low serum vitamin D levels when compared to healthy controls [24, 25]. Also, in a large longitudinal study of over five years of follow-up, it was observed that vitamin D deficient individuals experienced cognitive declines that were two to three times faster than those with adequate vitamin D levels and so the study concluded that low vitamin D among the elderly is associated with significant decline in cognition [26]. The similarity in these findings has been attributed to the presence of vitamin D receptors (VDR) in the brain and the role of vitamin D in cell signaling and gene expression [23].

From this study, it was discovered that adjusted calcium concentrations among test subjects were significantly lower when compared to control subjects. This is similar to a finding in another study in which it was reported that increases in calcium and magnesium are linked to an increase in cognition scores [27]. It was postulated that the likely explanation may not be unconnected with the role calcium plays in cell signal transduction and neurobiology [27].

Also vitamin D is a major hormone that helps increase serum calcium concentration. Therefore, at low vitamin D levels, as found in the subjects, serum calcium levels are expected to be decreased.

It was also observed that parathyroid hormone (PTH) levels among subjects were significantly higher when compared to controls. This is similar to findings from a ten-year longitudinal study of elderly people in Finland [28]. It was observed that elevated PTH indicated a 2-fold risk of at least 4-point decrease in Mini-Mental State Examination within the first year of follow-up, which remains significantly elevated even after controlling serum calcium. The high PTH also predicted cognitive decline within a five-year follow-up, but the association disappeared at ten years. The explanation suggested is that it may likely be as a result of feedback response to counter the effect of hypocalcemia from low vitamin D levels.

Also, the inorganic phosphate concentrations among subjects were significantly higher when compared to controls. This is similar to findings from a study of phosphorus and cognition in the elderly population in India, where it was observed that decreased in phosphorus concentration is linked to increase in cognition score and vice versa [29]. The counteracting roles of calcium and phosphate in neurotransmission and cell signal transduction was suggested as the likely explanation. Also, at low levels of vitamin D, as observed in the subjects, serum calcium is usually lowered with a concomitant increase in inorganic phosphate concentration that in turn triggers the release of PTH as a feedback response.

Comparison of mean values of biochemical parameters among a group with severe cognitive impairment, group with mild cognitive impairment, and control group showed that serum 25(OH)D and adjusted calcium concentrations were significantly lower while PTH levels were significantly higher whereas inorganic phosphate concentrations showed no significant difference. This is similar to findings from other studies where low vitamin D levels, low calcium concentrations, and high PTH levels were found to be associated with dementia and to correlate with severity of the cognitive decline in patients [24, 28]. The suggested explanation is likely because of the interconnectivity between 25(OH)D and cognition and the interplay of vitamin D and PTH on calcium metabolism.

This link becomes even stronger as cognition declines further [28].

Among the subjects, it was observed that serum 25(OH)D concentrations showed a significant positive correlation with adjusted calcium concentrations ($r=0.600$, $p=0.0001$), significant negative correlation with PTH levels ($r=-0.423$, $p=0.003$), and no significant correlation with inorganic phosphate concentrations ($r=-0.281$, $p=0.055$). These findings are similar to observations from other studies that suggest a link between low levels of vitamin D and poor global cognitive function and strong associations between low serum vitamin D concentrations and all-cause dementia [24, 30]. However, in this study the correlation between 25(OH)D and PTH levels was not as strong as found in other studies [24, 28]. This may be attributed to differences in races and weather conditions as they carried out their researches with Caucasians and in temperate regions. Nonetheless, there is a dearth of information on local studies correlating 25(OH)D with PTH, calcium, or inorganic phosphate.

Again, it was observed that there was no significant difference in 25(OH)D, adjusted calcium, PTH, and inorganic phosphate concentrations among male and female subjects. Although, it is well established that female sex hormones play some roles in vitamin D and calcium metabolism [31], the likely explanation could be due to the age range of female subjects for this study. At age ≥60 years, the female subjects are postmenopausal; so effects of these female hormones are waned off resulting in some biochemical parameters showing patterns similar to their male counterparts [31].

Additionally, serum 25(OH)D concentration of controls (apparently healthy elderly people) was 46.6±13.1 ng/mL, which could be extrapolated to mean that the reference interval of serum 25(OH)D for this population is 20.9-72.3 ng/mL (mean±1.96SD) (although the number of participants was 52, which is less than 120 minimum required for determination of reference interval) [32]. This value is much lower than 30-100 ng/mL recommended serum 25(OH)D reference interval for the general population by Endocrine Society [33].

The Negroid skin is poor in vitamin D production [34], in addition to other risk factors for low vitamin D status common to these elderly people. Therefore, there is a need for local study of their age-specific reference interval for serum 25(OH)D to avoid overestimation of low vitamin D status among them.

The study was limited by a number of factors that should be acknowleged. First, it was a cross-sectional study therefore cause-effect conclusion could not be drawn. For example, observed low vitamin D status among the test subjects could be the cause of the cognitive impairment or due to reduced sunlight exposure from decreased outdoor activities resulting from cognitive impairment. Secondly, the extent of sunlight exposure in the subjects could not be determined although effort was made to exclude those with minimal outdoor activities by the use of questionnaire. Also amount of vitamin D-rich meal consumed by the subjects was difficult to estimate although effort at ascertaining good appetite was made by the use of questionnaire.

5. Conclusion

The study findings suggest that decreased serum Vitamin D concentration is associated with diminished cognitive function among the elderly and vice versa. Hence, determination of Vitamin D status among elderly patients presenting with impairment of cognitive function is highly recommended. However, further studies are needed to validate findings from the present study.

6. Recommendation

In view of the above findings, the following recommendations are made:

1. Foods rich in vitamin D should be integral part of elderly people's diets.
2. Vitamin D and calcium supplementation should be part of routine geriatric care.
3. Vitamin D and calcium supplementation should be part of the management for cognitive impairment among the elderly.
4. Increased research work on vitamin D in our localities should be encouraged.

Disclosure Statement

The authors declare that they do not have any conflicts of interest.

Authors' Contributions

All the authors were involved substantially in the concept and design of the study, data acquisition, analysis and interpretation of the data, drafting the article, revising the article critically for its intellectual content, and in the final approval of the version to be published.

Data Availability

The data analyzed and used in this study may be shared with other researchers on reasonable request provided the data comply with the same standards as the main dataset.

Acknowledgements

Both authors extend their appreciation to all the resident doctors in the Departments of Chemical Pathology, UNTH for their professional assistance during the conduct of the study.

References

[1] Oduwole AO, Renner JK, Disu E, Ibitoye E, Emokpae E. Relationship between vitamin D levels and outcome of pneumonia in children. *West Afr J Med*. 2010; 29 (6): 373-378.

[2] Holick MF. Vitamin D deficiency. *N Engl J Med.* 2007: 266-281.

[3] Suzman R, Haaga JG. World demography of ageing In: Harrison's Principles of Internal Medicine, 18th edn. Longo DL, Fauci AS, Kasper DL, Jameson JL, Loscalzo J (eds). McGraw Hill, New York. 2012.

[4] Viljakainen HT, Palssa A, Karkkainen M. How much vitamin D the elderly need? *J Am Coll Nutr*. 2006; 25: 429-435.

[5] Wichers IS, Van Schoor NM, Boeke AJ, Visser M, Deeg DJ, Smit J. Vitamin D status predicts physical performance and its decline in older persons. *J Clin Endocrinol Metab*. 2007; 6: 7-8.

[6] Ogunniyi A, Gureje O, Baiyewu O, Unverzagt FW, Hall KS, Oluwole S *et al*. Profile of dementia in a Nigerian community – types, pattern of impairment, and severity rating. *J Natl Med Assoc*. 1997; 89 (6): 392-396.

[7] Ochayi B, Thacher TD. Risk factors for dementia in central Nigeria. *Aging Mental Health*. 2006; 10 (6): 616-620.

[8] Velkoff VA, Kowal PR. Ageing in sub-Saharan Africa: changing demography of the region: panel on policy research and data needs to the challenge of ageing in Africa. *National Academic Press*, Washington USA. 2006.

[9] Uwakwe R, Ibeh CC, Modebe AI, Bo E, Ezeama N, Njelita I *et al*. The epidemiology of dependence in older people in Nigeria: prevalence, determinants, informal care, and health service utilization. *J Am Geriatr Soc*. 2009; 57: 1620-1627.

[10] Adebowale SA, Atte O, Ayemi O. Elderly well-being in a rural community in north-central Nigeria, Sub-Saharan Africa. *Pub Health Res*. 2012; 2 (4): 92-101.

[11] World Health Organisation. Dementia: a public health priority. WHO, Geneva. 2012.

[12] Garrett MJ. Health futures: a handbook for health professionals. WHO, Geneva. 1999.

[13] Vinh-Quoc-Luong K, Thi-Hoang-Nguyen L. Vitamin D and Parkinson's disease. *J Neuroscien Res*. 2012; 90 (12): 2227-2236.

[14] Garcion E, Wion-Barbot N, Moutero-Menei CN, Berger FN, Wion D. New clues about vitamin D functions in the nervous system. *Trends Endocrinol Metab*. 2002; 13 (2): 100-105.

[15] Araoye MO. Research methodology with statistics for health and social sciences. *Nathadex*. 2004; 115-121.

[16] Ekenze OS, Onwuekwe IO, Ezeala BA. Profile of neurological admissions at UNTH Enugu. *Nig J Med*. 2010; 19 (4): 419-422.

[17] Folstein MF, Folstein SE, McHugh PR. Mini-mental state: a practical method for grading the cognitive state of patients for the clinician. *J Psychiatr Res*. 1975; 12: 189-198.

[18] Holick MF. Vitamin D status: measurement, interpretation and clinical application. *Ann Epidemiol*. 2009; 65 (6): 1309-1317.

[19] Mallete LE. The parathyroid polyhormones: new concepts in the spectrum of peptide hormone action. *Endocrinol Rev*. 1991; 12: 110-117.

[20] Goodwin JF. Phosphate by phosphomolybdate-ultraviolet method. *Clin Chem*. 1970; 16 (19): 776-778.

[21] Janssen JW, Helbing AR. Arsenazo III: an improvement of the routine calcium determination in serum. *European J Clin Chem*. 1991 (1): 3-4.

[22] Leonard PL, Persaud J, Motwani R. Albumin methods. *Clin Chem Acta*. 1971; (35): 409-410.

[23] Buell JS, Dawson-Hughes B, Scott TM, Weiner DE, Dallal GE, Qui WQ *et al*. 25-Hydroxyvitamin D, dementia and cerebrovascular pathology in elders receiving home services. *Neurology*. 2010; 74: 18-26.

[24] Dursun E, Gezen-Ak D, Yilmazer S. A novel perspective for Alzheimer's disease: vitamin D receptor suppression by amyloid-beta and preventing the amyloid-beta induced alterations by vitamin D in cortical neurons. *J Alzheimer's Dis*. 2011; 23: 207-219.

[25] Annweiler C, Schoff AM, Allali G, Bridenbaugh SA, Kressig RW, Allain P *et al*. Association of vitamin D deficiency with cognitive impairment in older women: cross- sectional study. *Neurology*. 2010; 74: 27-32.

[26] Millner JW, Harvey DJ, Beckett LA, Green R, Farias ST, Reed BR *et al*. Vitamin D status and rates of cognitive decline in a multi-ethnic cohorts of older adults. *JAMA Neurol*. 2015; 72 (11): 1295-1303.

[27] Basheer MP, Pradeep-Kumar KM, Sreekumaran E, Ramakrishna T. A study of Magnesium, Calcium and Phosphate level and cognition in the elderly population of South India. *Alexandria J Med*. 2016; 52 (4): 11-14.

[28] Bjorkman MP, Sorva AJ, Tilvis RS. Does elevated parathyroid hormone concentration predict decline in older adults? *Aging Clin Exp Res*. 2010; 22 (2): 164-169.

[29] Li T, Xie Y, Bowe B, Xian H, Al-Aly Z. Serum phosphorus level and risk of incident dementia. *PLoS*. 2017; 12 (2) 125-127.

[30] Llewellyn DJ, Langa IA, Langa KM, Muniz-Terrera G, Phillips CL, Cherubini A *et al*. Vitamin D and risk of cognitive decline in elderly persons. *Arch Intern Med*. 2010; 170: 1135-1141.

[31] Nordin BE, Morris HA, Osteoporosis and vitamin D. *J Cell Biochem*. 1992; 49: 19-25.

[32] Bellera CA, Hanley JA. A method is presented to plan for the required sample size when estimating regression-based reference limits. *J Clin Epidemiol*. 2007; 60 (6): 610-615.

[33] Holick MF, Garabechian M. Vitamin D: photobiology, metabolism, mechanism of action, and clinical application. *Am Soc Bone Miner Res*. 2006; 129-137.

[34] Wolpowitz D and Gilchrest BA. The vitamin D questions: how much do you need and how should you get it? *J Am Acad Dermatol*. 2006: 54: 301-317.