ORIGINAL ARTICLE

Role of Vitamin D in Dementia in a sample of Egyptian patients

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Abstract

Background: ***In order for the body to absorb calcium and phosphate, vitamin D—a steroid hormone—is essential. Two primary forms of vitamin D, vitamin D3 and vitamin D2, make up the inactive form of vitamin D. The skin produces vitamin D3, also known as cholecalciferol, when exposed to ultraviolet light, vitamin D3 supplements, or foods that are fortified with vitamin D.***

Aim and objectives: ***To define the relation between vitamin D and dementia among the Egyptian population.***

Patients and methods: ***This was a retrospective, case control study that was conducted at El-Hussein University hospital and Kobri El-koba military complex on 50 patients with a diagnosis of dementia and control group of 50 healthy individuals of similar age and gender.***

Results: ***The present study's most noteworthy discovery was that the dementia group had significantly reduced serum 25(OH)D levels (20.88±5.91 ng/ml) in comparison to the control group (41.28±7.21 ng/ml).***

Conclusion: ***Our study demonstrated compelling evidence for an association between low vitamin D levels and an increased risk of dementia among the Egyptian population.***

Keywords: Vitamin D; Dementia; Egyptian patients

1. Introduction

In order for the body to absorb calcium and phosphate, vitamin D—a steroid hormone—is essential. Two primary forms of vitamin D, vitamin D3 and vitamin D2, make up the inactive form of vitamin D. The skin produces vitamin D3, also known as cholecalciferol, when exposed to ultraviolet light, D3 supplements, or fortified foods. Ergocalciferol, or vitamin D2, is found in foods that are fortified with it or in supplements. In the liver, these two vitamin D forms are converted into 25-hydroxyvitamin D, which is then stored for later use.[1]

Vitamin D can passively diffuse its way across the blood-brain barrier (BBB) and into the brain. Upon binding to the vitamin D receptor (VDR), the active form of 1,25(OH)D affects gene expression. A vitamin D receptor (VDR) is located in several brain regions, including the thalamus, amygdala, orbitofrontal cortex, cingulate, hippocampus, and glial cells.[2]

Several neurological and mental diseases, such as schizophrenia, epilepsy, Parkinson's disease, multiple sclerosis, and dementia, are linked to hypovitaminosis D. Multiple hypothesized pathways exist for how hypovitaminosis D may influence various diseases. Neuronal death is one way this happens. This kind of cell death is brought on by hypovitaminosis D, which slows the neuronal cell cycle and reduces cytochrome C expression. One protein that helps get pro-apoptotic factors going is cytochrome C.[3]

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In a randomized trial conducted on healthy people, those who took 4,000 IU of vitamin D daily for 18 weeks had better visual memory than those who took 400 IU daily; however, no improvements were observed in verbal memory or any other cognitive domains.[4]

Determining the association between vitamin D and dementia in the Egyptian population is the primary goal of this research.

2. Patients and methods

Half of the participants in this retrospective case control research had a dementia diagnosis, while the other half were healthy adults of the same age and gender. They were recruited from the neurology clinic of Kobri El-Koba Military hospital and Al-Azhar University hospital, Egypt.

Inclusion criteria

Patient with a diagnosis of dementia and aged at least 60 years at the time of assessment.

Exclusion criteria:

Brain injury or post-concussive syndrome, history of or diagnosis of any psychiatric or substance use disorder, other causes for hypovitaminosis D such as obesity, chronic kidney disease, Crohn's disease, celiac disease, cystic fibrosis, and limited exposure to the sun, and insufficient serum volumes for the vitamin D assay to be performed.

All patients were subjected to:

All aspects of the patient's medical history, including their age, gender, smoking status (nonsmoker or current smoker), educational background (high school dropout, high school graduate, some college, vocational training, college graduate), and level of physical activity (sedentary, moderate, regular, or athletic) were gathered through interviews and questionnaires.[5]

As a stand-in for actual sun exposure, we relied on engaging in outside activities. The Zutphen Study's validated physical activity questionnaire was used to measure these levels.[6]

Walking, cycling, gardening, other sports, and hobbies were some of the topics covered in the questionnaire. We tallied up the hours spent engaging in various outdoor pursuits over the course of a week to arrive at the overall amount of time spent outdoors.

Patients' self-reported histories of cardiovascular events (such as stroke, diabetes mellitus type 2, myocardial infarction, and heart failure) were cross-checked with ongoing record reviews in the research database.[7]

The Center for Epidemiology, a widely used and validated tool, was used to measure neuropsychiatric and general health factors, including height, weight, and body mass index (BMI in kg/m2), as well as symptoms of depression. Scale for Depression (CES-D); a score of 16 or above is seen as indicative of symptoms of depression.[8]

Laboratory examination includes serum calcium, kidney function, lipid profile, and serum 25(OH)D measurement.

Assessment of dementia:

At baseline, all participants were evaluated for dementia. If a participant had a score above 27, it was considered normal. Mild cognitive impairment was defined as a score between 22 and 27, and severe cognitive impairment was defined as a score below 22. Cognitive impairment was further confirmed if a participant had either (1) a subjective memory complaint or (2) an objective impairment in one of four cognitive domains: memory, executive function, language, or visuospatial function.[9]

Among the many benefits of the MoCA are its simplicity and sensitivity in screening for moderate cognitive impairment, Alzheimer's disease, and dementia. The MoCA has undergone extensive validation across a variety of languages and therapeutic contexts. The duration of the MoCA's administration is one of its drawbacks.

Outcomes:

Dementia diagnosis:

The MoCA-based dementia was given to the patient. Appointments were suggested to participants with lower scores at the two hospitals that received the highest number of dementia incident cases: Kobri El-Koba Military Hospital and Al-Azhar University Hospital.

Patients with dementia who did not undergo neuroimaging or whose medical records did not have enough information to establish a subtype diagnosis were categorized as having undefined dementia.[10]

Dementia due to any cause includes Alzheimer's disease, vascular dementia, and frontotemporal dementia.[11] Lewy body dementia[12], a variety of dementias, including Parkinson's disease and dementia.[13]

Cognitive impairment:

We used the MoCA's performance as a secondary outcome to define cognitive impairment.[14] In most cases, the MoCA test outperforms the MMSE in identifying mild cognitive impairment and early stages of dementia. This is derived from contrasting the two tests' sensitivity and specificity, which measure their capacity to accurately detect illness and its absence, respectively.

The MoCA has a sensitivity of 90% and a specificity of 87% in identifying MCI, according to a research published in BMC Geriatrics in 2015. In comparison, the MMSE is 100% specific

and has a sensitivity of 18%[15] It was only in cases where MMSE was not present that it proved to be more effective.

Statistical Analysis

I used IBM SPSS Statistics for Windows, Version 23.0, which was published by IBM Corp. in 2015, to compile, tabulate, and analyze all of the data. The mean±SD and median (range) were used to represent quantitative data, whereas numbers and percentages were used to represent qualitative data. Two groups were compared using a t-test, which compares normally distributed variables. To compare proportions between two qualitative factors, the chi-square (x2) test was utilized. The association between the several study variables was evaluated using the Spearman correlation coefficient. A number close to 1 indicates strong correlation, whereas a value near 0 indicates weak correlation. A positive sign indicates direct correlation, and a negative sign indicates inverse correlation. The ROC curve was also utilized. There was no one-sided test. Statistical significance (S) was denoted by a p-value<0.05, whereas statistical insignificance (I) was denoted by a p-value≥0.05.

3. Results

Table 1. Demographic data of the studied groups.

		CONTROL GROUP (N=50)	CASES GROUP (N=50)	P-VALUE
AGE(YEARS)	Mean±SD	74.7±6.6	76.9±7.43	0.12
	Range	61-82	64-89	
BMI(KG/M²)	Mean±SD	27.22±1.88	26.94±1.78	0.448
	Range	24-31	24-31	
SEX	Male	24(48%)	15(30%)	0.06
	Female	26(52%)	35(70%)	
HISTORY OF TYPE 2 DIABETES	Yes	5(10%)	11(22%)	0.104
	No	45(90%)	39(78%)	
HISTORY OF STROKE	Yes	2(4%)	12(24%)	
	No	48(96%)	38(76%)	0.004
HISTORY OF HEART FAILURE	Yes	1(2%)	5(10%)	0.094
	No	49(98%)	45(90%)	
HISTORY OF MYOCARDIAL INFARCTION	Yes	3(6%)	7(14%)	0.186
	No	47(94%)	43(86%)	
PSYCHOSIS	Yes	4(8%)	12(24%)	0.029
	No	46(92%)	38(76%)	

BMI: Body Mass Index, SD: Standard deviation

There was no significant difference between the studied groups regarding to demographic data except history of stroke and psychosis that were higher in cases than control group,(table 2; figures 1&2).

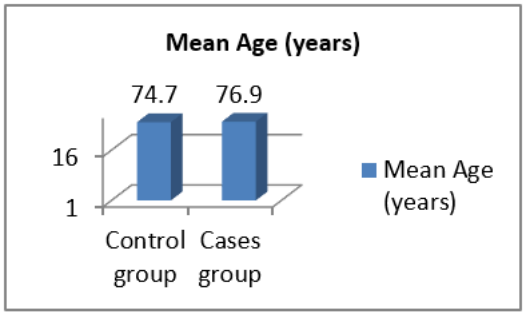

Figure 1. Age of the studied groups.

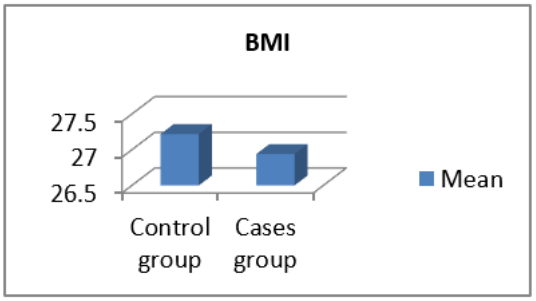

Figure 2. BMI of the studied groups.

Table 2. Blood pressure of the studied groups

		CONTROL GROUP (N=50)	CASES GROUP (N=50)	P-VALUE
SBP (MMHG)	Mean±SD	135.9±10.23	137.8±11.34	0.381
	Range	115-150	115-155	
DBP (MMHG)	Mean±SD	81.9±9.25	82.8±8.64	0.616
	Range	65-95	70-95	

SBP: Systolic blood pressure, DBP: Diastolic blood pressure

There was no significant statistical difference between the studied groups regarding to blood pressure,(table 2; figure 3).

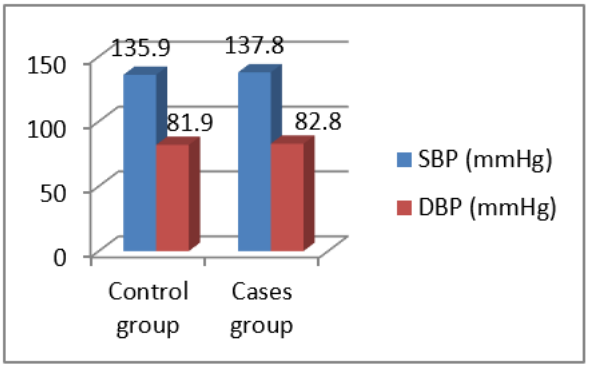

Figure 3. Blood pressure of the studied groups.

Table 3. Outdoor activity, hours per week of the studied groups.

		CONTROL GROUP (N=50)	CASES GROUP (N=50)	P-VALUE
OUTDOOR ACTIVITY, HOURS PER WEEK	Mean±SD	11.92±2.75	6.25±1.82	0.001
	Range	6-16	4-10	

As compared to the control group, the research groups engaged in significantly less outdoor activity,(table 3; figure 4).

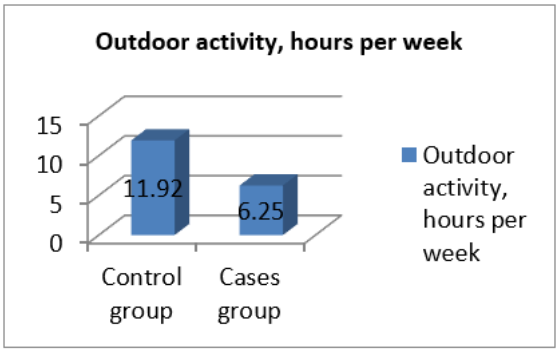

Figure 4. Outdoor activity, hours per week of the studied groups.

Table 4. Lipid profile of the studied groups.

		CONTROL GROUP (N=50)	CASES GROUP (N=50)	P-VALUE
CHOLESTEROL (MG/DL)	Mean±SD	175.42±31.68	203.64±35.41	0.001
	Range	119-246	136-269	
TRIGLYCERIDES (MG/DL)	Mean±SD	124.04±26.19	149.88±23.44	0.001
	Range	73-174	111-212	
HDL (MG/DL)	Mean±SD	51.08±10.18	50.54±9.78	0.787
	Range	34-68	33-68	
LDL (MG/DL)	Mean±SD	99.53±33.65	123.13±37.07	0.001
	Range	32.8-168.2	46-188.4	

HDL :High-density lipoprotein, LDL: Low-density lipoprotein.

Cases compared poorly with controls in terms of cholesterol, triglycerides, and low-density lipoprotein (LDL), while there was no such difference in terms of high-density lipoprotein (HDL),(table 4; figure 5).

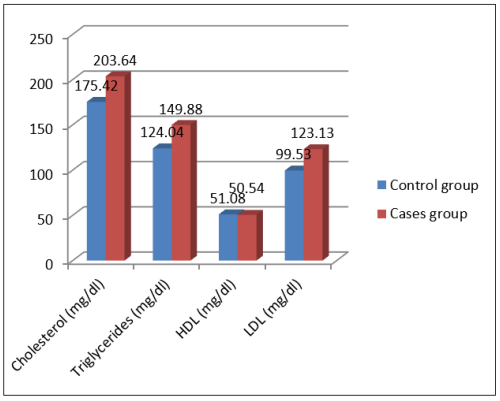

Figure 5. Lipid profile of the studied groups.

Table 5. Serum 25(OH)D and calcium of the studied groups.

		CONTROL GROUP (N=50)	CASES GROUP (N=50)	P-VALUE
SERUM 25(OH)D, (NG/ML)	Mean ± SD	41.28±7.21	20.88±5.91	
	Range	27-59	10-31	0.001
SERUM CALCIUM, (MG/DL)	Mean±SD	9.24±0.88	9.13±0.83	0.532
	Range	7.2-10.7	7.4 -10.7	

HDL: High-density lipoprotein, 25(OH)D:25 hydroxy vitamin D3-Calcifediol

While there was no statistically significant difference in blood calcium levels across the groups, there was a significant difference in serum 25(OH)D levels, which were lower in the case group compared to the control group, (table 5; figure 6).

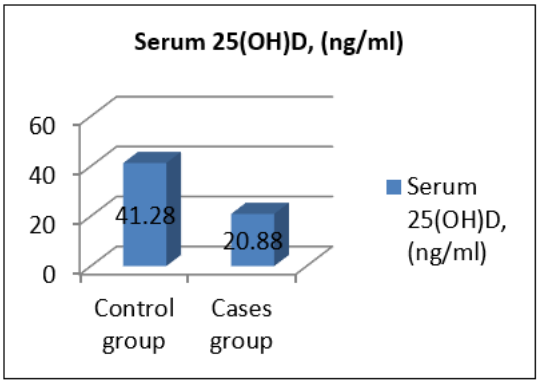

Figure 6. Serum 25(OH)D of the studied groups.

Table 6. MoCA of the studied groups.

		CONTROL GROUP (N=50)	CASES GROUP (N=50)	P-VALUE
MOCA	Mean±SD	28.84±0.81	20.38±4.16	0.001
	Range	28-30	13-27	

MoCA:Montreal Cognitive Assessment

There was significant difference between the studied groups regarding to MoCA score that was lower in cases than control group (table 6).

Table7. ROC curve of 25(OH)D for prediction of dementia.

	CUT-OFF	AUC	SENSITIVITY	SPECIFICITY	PPV	NPV	P-VALUE
SERUM 25(OH) D	30.5	0.992	95.9%	94.1%	94%	96%	0.001

AUC:area under the curve

At a cut off 30.5, serum 25(OH)D showed sensitivity 95.9%, specificity 94.1%, PPV 94%, NPV 96% and AUC 0.992 in prediction of dementia. So, serum 25(OH)D can be used in prediction of dementia, (table 7; figures 7&8).

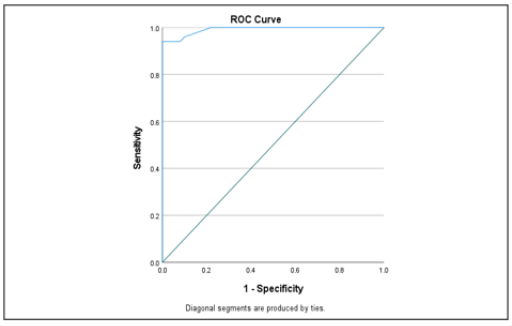

Figure 7. ROC curve of 25(OH)D for prediction of dementia.

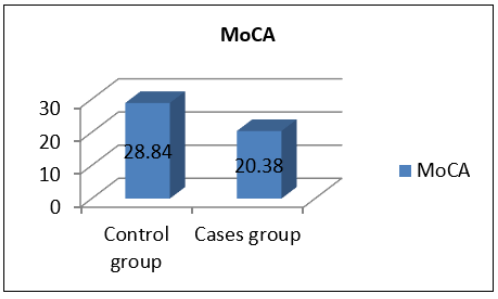

Figure 8. MoCA of the studied groups.

Table 8. Relation between Serum 25(OH) D and severity of cases:

		MILD COGNITIVE IMPAIRMENT (36)	MODERATE COGNITIVE IMPAIRMENT (14)	P-VALUE
SERUM 25(OH)D, NMOL/L	Mean±SD	23.61±4.38	13.85±2.53	0.001
	Range	15-31	10-19	

Serum 25(OH) D was significantly lower in moderate cognitive impairment than mild cognitive impairment,(table 8; figure 9).

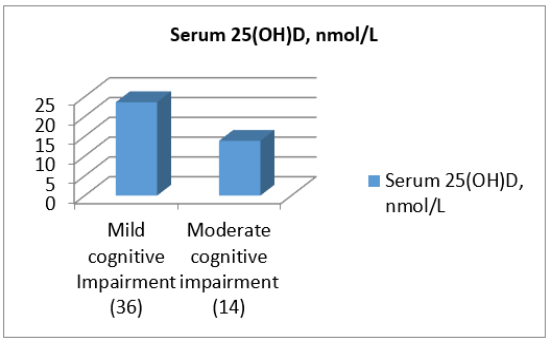

Figure 9. Relation between Serum 25(OH) D and severity of cases.

4. Discussion

Changes in thinking, conduct, and ability to carry out daily tasks are hallmarks of dementia, a neurological disease. [16]

The present study investigated the relationship between vitamin D levels and dementia among Egyptian individuals.

This was a retrospective, case control study that was conducted at El-Hussein University hospital and Kobri Elkoba military complex on 50-patients with a diagnosis of dementia and control group of 50 healthy individuals of similar age and gender.

Our results revealed significant differences between the dementia and control groups in terms of age, history of stroke, Psychosis, serum 25(OH)D levels, cholesterol, triglycerides, and LDL cholesterol.

The dementia group had an average age of 76.9±7.43 years. This finding is consistent with numerous previous studies that have reported an increased risk of dementia with advancing age.[17]

Our results showed that the dementia group differed significantly from the control group with respect to age, history of stroke, depressive symptoms, blood 25(OH)D levels, cholesterol, triglycerides, and LDL cholesterol.

With an average age of 76.9±7.43 years compared to 68.76±5.33 years in the control group, the dementia group had a noticeably older demographic.

Our findings are consistent with the vast amount of literature demonstrating that the probability of developing dementia rises with advancing age.[17]

Our results showed that the dementia group differed significantly from the control group with respect to age, history of stroke, depressive symptoms, blood 25(OH)D levels, cholesterol, triglycerides, and LDL cholesterol.

Dementia patients tended to be older than those in the control group, with an average age of 76.9±7.43 years compared to 68.76±5.33 years.

Our findings are consistent with the vast amount of literature demonstrating that the probability of developing dementia rises with advancing age.[17]

The percentage of those having a history of stroke was much greater in the dementia group (24% vs. 4% in the control group).

Previous studies have shown a substantial correlation between cerebrovascular disorders like stroke and an elevated risk of dementia, including vascular dementia and Alzheimer's disease. This finding is in line with those findings.[18]

The findings corroborate other studies that demonstrated a reciprocal relationship between the two illnesses; more especially, that depression can be both a cause and an effect of dementia.[19]

The most notable finding of the current analysis was that the dementia group had substantially lower serum 25(OH)D levels (20.88±5.91 ng/ml) than the control group (41.28±7.21 ng/ml).

Consistent with previous meta-analyses, we observed that low vitamin D levels were associated with an increased risk of dementia.[20] Ten separate cohort studies comprised this investigation, and over 20,000 participants were a part of them.

Elseesy et al.,[21] discovered that vitamin deficiencies are common among those with major depressive disorder (MDD). In terms of vitamin D levels, a mean value of 8.14±6.69 ng/ml was indicative of a deficiency in 94.6% of our sample. Adequate vitamin D levels are defined as 30 ng/ml or higher. This provides further evidence that vitamin D insufficiency might be a sign of major depressive disorder and that it could be used alongside other medications to treat the condition.

Researchers from Kasr Al-Aini University's neurology department examined 50 healthy adults aged 60 and up to find out whether there was a connection between cognitive decline, serum vitamin D levels, and polymorphisms in the vitamin D receptor genes (VDR-Apal and VDRTaql). A battery of psychometric tests tailored to distinct cognitive domains was administered to the study participants. Twenty-two percent of the participants lacked adequate vitamin D levels. This supports our hypothesis that low vitamin D levels may play a role in the development of dementia.[22]

In a sunny nation like Egypt, vitamin D levels should be high. In order to prevent the potential for erroneous conclusions on low vitamin D levels due to hospital confinement, this study relied on outpatient clinic recruitment rather than inpatient ward recruitment. Only 23.7% of 59 top executive managers in Egypt were found to have 25(OH)D deficiency, defined as 20 ng/dl or below, according to a cross-sectional study.[23]

Tay et al.,[23] who discovered vitamin D to be

associated with a higher incidence of cognitive decline in SLE patients.[24]

Cholesterol, triglyceride, and low-density lipoprotein (LDL) cholesterol levels were substantially greater in the dementia group than in the control group. Our results show that serum 25(OH)D was negatively correlated with these lipid parameters, which may indicate a relationship between vitamin D status, lipid metabolism, and neural function.

These results corroborate those of earlier research that linked dyslipidemia to an increased likelihood of developing dementia, especially Alzheimer's disease.[16]

Blood 25(OH)D and molecular oxygen consumption were positively correlated in our research.

This came in agreement with Hussein et al.,[25] individuals who stated that low vitamin D levels were associated with impaired executive function.

In contrast, Elseesy et al.,[21] cognitive measures measuring attention, memory, and executive functioning were not significantly related to vitamin D levels.

Previous studies have shown a detrimental link between vitamin D levels and body mass index (BMI), but our data reveal no such relationship. Perhaps our small sample size explains this.

A past study by Perez-Lopez and colleagues reported the crucial role of vitamin D in maintaining cognitive function. Another study also suggests that the vitamin D receptors are present in the brain regions and are responsible for cognitive functions and memory development. The roles of Vitamin D in neurotropism, neurotransmission, neuroprotection, and neuroplasticity may explain vitamin D deficiency involvement in the progression of AD.[26]

Elseesy et al.,[21] discovered comparable results. This is likely due to the fact that their Major Depressive Disorder (MDD) patients with cognitive dysfunction were equally vitamin D deficient and had similar BMI levels, which prevented them from drawing statistically meaningful conclusions.

The area under the curve (AUC) of 0.992, sensitivity of 95.9%, and specificity of 94.1% at a cut-off value of 30.5 ng/ml, the ROC curve analysis proved that serum 25(OH)D levels could successfully differentiate between individuals with and without dementia. According to this discovery, vitamin D may be useful as a biomarker for evaluating the likelihood of dementia and initiating preventative measures.

It is critical to note that the study has certain limitations that prevent it from demonstrating causal correlations. These drawbacks include its retrospective and case-control design.

4. Conclusion

Our results provide strong evidence that low vitamin D levels are associated with an increased incidence of dementia in the Egyptian population.

Disclosure

The authors have no financial interest to declare in relation to the content of this article.

Authorship

All authors have a substantial contribution to the article

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Conflicts of interest

There are no conflicts of interest.

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