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Vitamin D3 Deficiency's Impact on Atrial Fibrillation in Hyperthyroidism Patients

Iqbal Hanash Dhefer [1]

[1]Department of Medical Laboratory Techniques, AL-Suwaira Technical Institute, Middle Technical University, Waist, Iraq

Correspondence: Iqbal Hanash Dhefer
Email: dr.iqbalhanash@mtu.edu.iq

Website: https://djm.uodiyala.edu.iq/index.php/djm

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Abstract

Background: Atrial fibrillation (AFi) is more common as people get older. Additionally, a further indicator of the occurrence of AFi is subclinical hyperthyroidism, which is linked to a 3-fold increased risk. Lack of vitamin D causes the renin-angiotensin-aldosterone pathway to become active, which has an impact on the cardiovascular system.
Objective: To examine the association between 25-hydroxyvitamin D3 insufficiency and AFi and hyperthyroidism cases with valvular and nonvalvular AFi that required treatment at our medical center.
Patients and Methods: Samples taken from 200 cases of AFi (50-65 years old) divided into: group A cases with nonvalvular AFi and group B cases with valve AFi, and 100 healthy individuals with sinus rhythm who were age-matched chosen as control groups. Standard biochemical measurements, including levels of 25-OHvit.D3,the hormone of the thyroid gland, and parathyroid hormone, were made.
Results: Cases in group A had decreased 25-OHvit.D3 levels compare to those in the control and B groups ($P \leq 0.05$).In comparison to the control group, the patients in groups A and B had larger left atriums and greater systolic pulmonary artery pressures.
Conclusion: Thus, the study shows a connection between nonvalvular AFi cases with hyperthyroidism and 25-OHvit D3 deficiency.
Keywords: 25-hydroxyvitamin D3, Atrial fibrillation, Valvular, Parathyroid hormone, Hyperthyroidism.

Introduction

Among heart rhythm disorders, atrial fibrillation (AFi) is the most prevalent. In the population as a whole, the prevalence of (AFi) is predicted to range from 0.4% to 15% and to rise with age. The risk of mortality, heart failure, and stroke is all raised by atrial fibrillation, which is a significant public health concern [1]. Up to about 15% of hyperthyroid patients experience AFi, compared to 4% prevalence in the population as a whole [2]. Atrial fibrosis, atrial dilatation, and diminished atrial muscle mass are frequently seen in AFi. The renin-angiotensin-aldosterone system is assumed to be primarily responsible for these physiopathologic alterations [3]. Additionally, it is well recognized that calcium is crucial for AFi and electrophysiologic reorganization. By reducing the atrial refractory duration and the action potential time, intracellular calcium excess inside the atrial myocytes contributes to the onset and maintenance of Afi [4]. The sarcoplasmic reticulum (SR) releases More calcium readily spontaneously when angiotensin is present, and it also encourages the proliferation of fibroblasts, both of which are crucial for the growth and maintenance of AFi. Age, gender, valvular heart disease, congestive heart failure, and ischemic heart disease are hazard issues for AFi in hyperthyroid patients that are similar to those in the population as a whole [5]. Vitamin D impacts heart function both directly and indirectly. Additionally, vitamin D controls blood pressure and guards against heart hypertrophy by suppressing renin activity [6]. Given this knowledge, we surmised that AFi might be associated with 25-hydroxyvitamin D3 insufficiency. The current study aiming to examine the association between 25-hydroxyvitamin D3 insufficiency, AFi , hyperthyroidism cases with both valvular, and nonvalvular AFi of the routine patients at our medical center.

Patients and Methods

Two hundrad hyperthyroid patients between the ages of 50 and 65 who were diagnosed with persistent (AFi) visited the cardiology outpatient clinic and the National Diabetes Center/AL-Mustansiriyah University in Baghdad between December 2022 and July 2023. The Center Ethical Committee approved the trial since physicians make diagnoses for every patient. The cases were divided into two groups: group A cases with nonvalvular (AFi) and group B cases with valvular (AFi), and 100 healthy individuals with sinus rhythm who were age-matched were chosen as control groups (SiR). The patients were required to sign formal informed consent forms in accordance with the research protocol that was certified by the ethical council of the University of the Middle Technical, Institute of AL-Suwaira Technical, Medical Laboratory Department, Iraq. Patients with other diseases were omitted from this study. The cases' blood pressure was gauged. Patients classified as hypertensive were those with a systolic blood pressure ≥140 mm Hg and/or a diastolic blood pressure ≥90 mm Hg, as well as those on antihypertensive medications.

Parathyroid hormone (PTH),T3,T4,25-OHVitD3,phosphorus, and calcium analysis in serum was carried out on Roche Modular E 170 Analytic System equipment utilizing the electrochemiluminescence technique. For the winter season, the reference values are 25-OHVit. D3 was set at 30 to 60 ng/mL, and for the summer season, at 30 to 130 ng/mL[1,7].

The protocol for transthoracic echocardiographic assessment was achieved by a 3.5 MHz transducer on a Vivid 7 Pro TTE. The test is conducted by a sonographer, a professional skilled in the use of ultrasound technology, who then provides the results to the physician. The European Association of Echocardiography and the American Society of Echocardiography instructions were followed for all echocardiographic examinations[8]. The patient remained in the position of lateral decubitus throughout the whole recording of the echocardiographic images.international units should be used in this section and throughout the manuscript.

Statistical Analysis

The SPSS version 22 were used to conduct the statistical analyses. Categorical data were shown as percentage values, whereas

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numerical variables were displayed as the mean ± SD. A P value≤ 0.05 was chosen as the cutoff for significance in all statistical studies.

Results

Parameters such as body mass index [BMI], gender distribution, and age did not vary statistically between the three groups, but the biochemical factors revealed that group A cases had higher PTH, T3, T4, TSH,Ca, and Pi levels than other groups, while having lower 25-OH vit D3 levels than group B and the control group, as seen in Table (1).

The mean LA diameter of the two case groups with AFi was substantially larger than that of the members of the SiR group when the initial conventional echocardiographic parameters of those in the group were assessed ($P \le 0.05$). As indicated in Table (2), the average systolic PA of groups A and B diagnosed with AFi was significantly greater than that of the persons in the SiR group ($P \le 0.05$). There was no significant difference in mean (edi), (esy) LV diameter, posterior wall and interventricular septum thickness between the all groups.

All biochemical markers were evaluated for potential linkage; the only significant correlation found was between PTH and 25-OH vitamin D3. Table (3) shows a substantial negative association between Upth and 25-OH vitamin D3 and a non-significant negative correlation between PTH and each of T3 and T4.

Table (1): Comparing of the biochemical characteristics of (AFi) cases and (SiR) groups.

Item	Patients (AFi)		Control	P value
	Group A No. 100	GroupB No. 100	(SiR) No. 100	
BMI kg/m^2	23.1±1.22	21.95±1.75	24.35 ±2.48	0.09
Age Year	62.51 ±5.8	61.5 ± 5.05	61.35 ±5.44	0.07
Hb g/dl	15.2 ± 1.4	13.7 ±1.15	14.2 ±1.89	0.057
Cre mg/dL	0.91 ±0.24	0.71 ± 0.1	0.84 ± 0.21	0.06
25-OH vit.D3, ng/Ml	5.22 ±2.30	10.1 ±4.28	12.10 ±4.63	0.01*i
TSH, mcIU/mL	1.52 ±0.98	1.27 ±0.95	1.61 ±0.54	0.081
T3(nmol/L)	3.12±0.23	2.0±0.369	1.296±0.36	0.03*
T4(nmol/L)	199.1±25.1	181.2±31.1	103.9±23.6	0.00*
PTH, pg/ml	88.6±25.1	80.3±33.9	75.27±29.3	0.03*i
Ca (mg/dL)	9.02±0.52	8.99±0.68	8.7±0.12	0.09
Pi mg/dL	3.6±0.21	3.2±0.51	3.0±0.11	0.08

AFi, atrial fibrillation, SiR, sinus rhythm, Hb, hemoglobin ,Cre, creatinine, TSH, thyroid-stimulating hormone, 25-OH vit.D3,25hydroxyvitaminD,T3,triiodothyronine,T4,thyroxin,Ca,calcium,Pi,phosphorous. (*) significant p value ≤ 0.05, (i) non-significant for group B against control.

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Table (2): Comparison of traditional echocardiographic characteristics of two (AFi) and SiR groups.

Item	Patients (AFi)		Control	P value
	Group A No. =100	Group B No. =100	(SiR) No. =100	
LA	45.1 ±4.1	47.9±3.1	36.6 ±3.4	0.002*
RA	32.6 ±3.6	39.0±3.8	30.9±3.6	0.08
PA	35.0±2.96	40.1±2.9	29.5±2.9	0.004*
LV-ej	63.6 ±3.77	60.4±4.1	63.9 ±4.2	0.002*
LV -sy	29.1 ±3.56	27.9±1.1	28.7±2.6	0.07
LV-edi	45.1± 3.23	46.8±2.2	44.9 ±3.9	0.054

LA: Left atrium(diameter, mm), RA: Right atrium(diameter, mm),PA, pulmonary artery (systolic pressure, mm Hg), LV, left ventricular(%), ej: ejection fraction , esy : end-systolic diameter(mm) ,edi : end-diastolic diameter(mm). (*) significant p value ≤ 0.05.

Table (3): Reduction in tumor sizes in three treatment protocols The relationship between PTH and 25-OH vitamin D3, T3, and T4 levels in hyperthyroid patients.

Correlation	Pearson Correlation	Significant (2-tailed)
PTH and 25-OH vitamin D3	-0.22	0.02*
PTH and T3	-0.14	0.32
PTH and T4	-0.12	0.43

Table (4) displays the outcomes of the forward progressive logistic regression test. The independent primary indicators of AFi in group A were found to be the LA diameter and 25-OH vitamin D3 level.

Table (4): Analysis of independent risk factors for AFi using logistic regression (Group A).

Character	HR(CI :95%)	P-value
PTH	1.75 (0.45–1.98)	0.51
LAdiameter	3.11(1.712-2.751)	0.02*
LV-ej	1.16 (0.37–1.43)	0.45
25-oH vit.D3	0.91(0.652-0.865)	0.01*

As demonstrated in Table (5), systolic PA, LA, and RA diameters were the independent primary indicators of (AFi group B) and predisposes to the development of (AFi). Heart failure, myocardial infarction, myocardial fibrosis, and hypertensive cardiac disorders are all linked to the renin-angiotensin-aldosterone pathway [10].

Table (5): Analysis of independent risk factors for AFi (Group B) using logistic regression.

Character	HR(CI :95%)	P-value
PA systolic pressure	1.75 (1.412-2.311)	0.03*
LA diameter	2.58 (1.911-2.790)	0.00*
RA diameter	1.86 (1.458-2.312)	0.004*

Discussion

Primary hyperaldosteronism causes an increase in the incidence of Afi [11]. Angiotensin II, which is created locally in primary hyperaldosteronism, is linked to fibrosis in the reactive tissues and myocyte death [12,13].

Angiotensin II receptor blockers and angiotensin-converting enzyme inhibitors, which are RAAS inhibitors, have been shown to protect against AFi by this mechanism. These medications had an 18% reduction in new-onset AF. Among patients with heart failure, the rate can reach 43% [14,15]. RAAS blockage reduces the likelihood of AFi recurrence and failure rates following cardioversion [16]. Reentry cycles are made easier by AFi's reduced effectiveness refractory period and lower atrial impulse velocity [17,18,19,20]. Action potential duration, shorter action potential plateau time, refractory period, and wavelength—the distance traversed by the electrical impulse throughout the refractory period—are all effects of the decline in calcium channels of the L type in the short term as well as the long term. These alterations are typical of Afi [21]. Another study revealed that vitamin D serves purposes aside from those related to bone metabolism [22]. According to the previous studies 25-OH vit. D3 deficiency may impact the onset of inflammatory bowel disease, autoimmune disorders, rheumatoid arthritis, some cancers, psoriasis, multiple sclerosis, diabetes, stroke, cardiac failure and infectious diseases like pneumonia and tuberculosis [23,24]. Supplementation with vitamin D is also effective in these people [23] by blocking RAAS and PTH, vitamin D maintains blood pressure homeostasis [24]. The research demonstrated that vitamin D and its equivalents could lower levels of angiotensin II and renin [16]. Calcium is crucial for both AFi initiation and electrophysiological modification [25]. By reducing the atrial refractory action and potential duration, intracellular calcium excess inside the atrial myocytes contributes to the growth and ongoing functioning of the AFi[4].We suggested that 25-OH vit.D3 insufficiency might be connected to AFi, given the significance of RAAS in the pathological process of AFi and the harmful regulating role that vitamin D plays for renin[16].

In current reasearch, three groups were contrasted using 25-OHvit. D3 patients with nonvalvular AFi disease had considerably lower 25-OHvit,D3 levels than patients without (AFi),and those with valvular (AFi) disease had higher 25-OHvit. D3 levels ($P \leq 0.05$). It is well established that vitamin D decreases levels of pro-inflammatory cytokines and raises levels of IL-10[6]. This result has led to the hypothesis that 25-OH vit. D3 insufficiency may contribute to the emergence and maintenance of AFi. Under conditions of normal atrial action potential, normal ventricular function and structure and transforming growth factor B1increases atrial fibrosis. Additionally, it increases conduction heterogeneity and AFi vulnerability [26]. According to certain theories, vitamin D insufficiency makes people more susceptible to AFi via increased TGFB1expression, conduction heterogeneity, and atrial fibrosis. In the start of AFi and electrophysiological remodeling, calcium is crucial [27]. PTH raises intracellular calcium levels by decreasing cardiomyocyte cellular calcium absorption and sarcoplasmic reticulum calcium reuptake [28]. In contrast to the assertion made by Rienstra et al. that vit. D3 status was unrelated to incident (AFi), our investigation found a link between 25-OH vit. D3 insufficiency and nonvalvular (AFi) [29]. In line with the findings of the literature, it was discovered in our investigation that both the LA and RA diameters were related to AF. PTH levels of individuals who had AFi were much greater than PTH concentrations of individuals without AFi when three groups were examined. This finding implies that AFi contributes to intracellular calcium overload by causing hyperparathyroidism due to vit.D deficiency. However, given the limited sample size and short follow-up period, the link between 25-OHVit.D3 and AFi needs to be verified in bigger, well-designed investigations.

Conclusions

Our investigation thus demonstrated a link between nonvalvular (AFi) and 25-OHVit.D3 insufficiency in hyperthyroid patients. However, it was discovered that both the control group and individuals with valvular AFi mitral valve disease had comparable vitamin D levels. This circumstance shows that valvular heart disease in these individuals causes AFi. Additionally, 25-OHVit.D3 insufficiency may contribute to nonvalvular AFi in patients with hyperthyorid.

Recommendations

To better understand the link between nonvalvular (AFi) and 25-OHVit.D3 insufficiency in hyperthyroid patients, the quantity of samples must be increased, and the disease must be researched in more than one location.

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Ethical clearance: The examination configuration is approved by the ethical committee of National Diabetes Center and cardiology outpatient clinic approved and the research protocol that was certified by the ethical council of the University of the Middle Technical, Institute of AL-Suwaira Technical, Medical Laboratory Department, Iraq. This study was conducted according to the approval of College of Medicine/ University of Diyala and in accordance with the ethical guidelines of the Declaration of ethical committee of the College (Document no.2023IHD801).

Conflict of interest: Nil

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تأثير نقص فيتامين د٣ على الرجفان الأذيني لدى مرضى فرط نشاط الغدة الدرقية

اقبال حنش ضفير [١]

الملخص

خلفية الدراسة: الرجفان الأذيني (AFi) أكثر شيوعًا مع تقدم الأشخاص في السن. بالإضافة إلى ذلك، هناك مؤشر آخر لحدوث الـ AFI وهو فرط نشاط الغدة الدرقية تحت الإكلينيكي، والذي يرتبط بزيادة خطر الإصابة بثلاثة أضعاف. يؤدي نقص فيتامين د إلى تنشيط مسار الرينين-أنجيوتنسين-الألدوستيرون، مما يؤثر على نظام القلب والأوعية الدموية.

اهداف الدراسة: لتقييم العلاقة بين قصور ٢٥-هيدروكسي فيتامين د٣ وحالات الـ AFI وفرط نشاط الغدة الدرقية مع الـ AFI الصمامي وغير الصمامي الذي يتطلب العلاج في المركز الطبي.

المرضى والطرائق: عينات مأخوذة من ٢٠٠ حالة رجفان اذيني AFi اعمارهم (من (٥٠-٦٥ سنة مقسمة إلى: حالات المجموعة أ مع AFi غير الصمامي وحالات المجموعة بAFi مع صمام ، و١٠٠ فرد سليم مع إيقاع الجيوب الأنفية الذين كانوا متطابقين مع العمر تم اختيارهم كمجموعات مراقبة. تم إجراء قياسات كيميائية حيوية قياسية، بما في ذلك مستويات ٢٥-هيدروكسي فيتامين د٣ , هرمون الغدة الدرقية، وهرمون الغدة الجار الدرقية.

النتائج: انخفضت الحالات في المجموعة (أ) من مستويات ٢٥-هيدروكسي فيتامين د٣ مقارنة بتلك الموجودة في المجموعتين الضابطة والمجموعة ب ($P \geq 0.05$) وبالمقارنة مع المجموعة الضابطة، كان لدى المرضى في المجموعتين أ وب الأذين الأيسر أكبر وضغط انقباضي وضغوط الشريان الرئوي أكبر.

الاستنتاجات: وهكذا، أظهرت الدراسة وجود علاقة بين حالات AFI غير الصمامية مع فرط نشاط الغدة الدرقية ونقص مستويات ٢٥-هيدروكسي فيتامين د٣.

الكلمات المفتاحية: -هيدروكسي فيتامين د٣، الرجفان الأذيني، الصمامات، هرمون الغدة الدرقية، فرط نشاط الغدة الدرقية.

البريد الالكتروني: dr.iqbalhanash@mtu.edu.iq

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[١] المعهد التقني الصويرة - الجامعة التقنية الوسطى - واسط - العراق