Vitamin D deficiency in Saudi patients with systemic lupus erythematosus

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ABSTRACT

الأهداف: تحديد مستوى فيتامين (د) لدى مجموعة من المرضى السعوديين المصابين بمرض الذئبة الحمراء (SLE) مقارنة بمجموعة (الضبط) من الأفراد الأصحاء.

الطريقة: أجري البحث في مستشفى جامعة الملك عبدالعزيز – جدة، اشتملت الدراسة على 165 مصاب بالذئبة الحمراء و 214 فردا سليما تم قياس فيتامين (د) مخبريا لدى جميع الأفراد الذين اشتملت عليهم الدراسة. تم اعتبار مستوى فيتامين (د) كافيا في المصل عند مستوى أعلى75 نانومول / ليتر (30 نانوجرام / ملليتر)، و ناقصا عند مستوى أقل من 50 نانومول / ليتر (أقل من 20 نانوجرام / ملليتر)، وغير كافيا عند مستوى يتراوح بين 75–50 نانومول / ليتر (30–20 نانوجرام / ملليتر).

النتائج: أظهرت النتائج وجود نقص وعدم كفاية في مستوى فيتامين (د) في مجموعة التحكم عند حوالي98.8% مقابل 55% و 89.7% مقابل 20%($p<0.0001$)، وكافيا لدى 2 (1.2%) من مرضى الذئبة الحمراء SLE مقارنة بحوالي 96 (45%) من أفراد مجموعة الضبط على التوالي($p<0.0001$). كان متوسط مستوى فيتامين (د) لدى مجموعة المرضى المصاحبون بنقص في مستوى فيتامين (د) 13.6±22.3 مقارنة بحوالي 17.5±44.5 من أفراد مجموعة الضبط $p<0.0001$ و9.5±19.1 مقارنة6.7±22.9 من أفراد مجموعة الضبط ($p=0.0152$). لم يوجد أي اختلاف إحصائي كدليل في المرضى الذكور و الإناث المصابين بمرض الذئبة الحمراء SLE مع الأخذ بالاعتبار بنقص وعدم كفاية مستوى فيتامين (د).

خاتمة: أثبتت دراستنا هذه عن وجود نقص في مستوى فيتامين (د) لدى المرضى السعوديين المصابين بمرض الذئبة الحمراءSLE . وعليه نقترح إمدادهم بجرعات علاجية من فيتامين (د)، مع تقييم دوره الفعال في علاج مرضى الذئبة الحمراءSLE.

Objectives: To determine vitamin D status among Saudi patients with systemic lupus erythematosus (SLE) versus matched control group.

Methods: Hospital-based cohorts of 165 SLE patients and 214 SLE-free volunteers were recruited at King Abdulaziz University Hospital in Jeddah, Kingdom of Saudi Arabia between January 2006 and June 2008. Serum levels of 25-hydroxyvitamin D [25(OH)D] were measured. Vitamin D sufficiency is defined as a serum level of 25(OH)D ≥75nmol/L (≥30ng/ml). A level ranging between >50 to <75nmol/L (>20 to <30ng/ml) is considered as vitamin D insufficiency, whereas ≤50nmol/L (≤20ng/ml) as vitamin D deficiency. Both deficiency and insufficiency are considered to comprise vitamin D inadequacy.

Results: The prevalence of SLE patients with 25(OH) D inadequacy and deficiency was higher than in the control group: 98.8 versus 55%, 89.7 versus 20% ($p<0.0001$). Only 2 (1.2%) SLE patients had adequate levels of 25(OH)D compared to 96 (45%) of control group ($p<0.0001$). The mean serum levels (nmol/L) of 25(OH)D in SLE patients with vitamin D inadequacy and deficiency in comparison to the control group were 22.3 ± 13.6 versus 44.5 ± 17.5 ($p<0.0001$) and 19.1 ± 9.5 versus 22.9 ± 6.7 ($p=0.0152$). No significant differences were evident in female and male patients with SLE with respect to the mean serum levels of 25(OH)D and prevalence of its deficiency.

Conclusion: Vitamin D inadequacy is highly prevalent in Saudi patients with SLE. Vitamin D supplementation and its evaluation in the treatment of SLE should be considered.

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Systemic lupus erythematosus (SLE) is a complex autoimmune disease involving multiple organs and tissues. Based on a community survey in Al-Qassim area of Saudi Arabia, the prevalence of SLE has been estimated to be 19.28 per 100,000.[1] Although the cause remains uncertain, several hereditary and environmental factors have been postulated to play a role in the development of SLE disease.[2-4] Recently, vitamin D deficiency has been implicated as a potential environmental factor triggering several autoimmune disorders such as SLE,[5,6] rheumatoid arthritis,[7,8] type 1 diabetes[9] and multiple sclerosis.[10] Serum levels of 25-hydroxyvitamin D [25(OH)D] were inversely correlated with the severity of SLE disease.[6] The prevalence of 25(OH)D deficiency is high in middle eastern countries.[11] Several recent studies from Saudi Arabia revealed low serum levels of 25(OH)D in Saudi population.[11-13] Many studies have highlighted an association between SLE and vitamin D deficiency in different ethnic groups.[14] This study's aim was to assess vitamin D status in Saudi patients with SLE.

Methods. A hospital-based cohort study was carried out at King Abdulaziz University Hospital (KAUH) in Jeddah, Kingdom of Saudi Arabia between January 2006 and June 2008. A total of 165 patients (148 female) with a diagnosis of SLE who attended the outpatient rheumatic clinic at KAUH and 214 age-matched SLE-free volunteers (122 female) as control group were recruited. All participants in this study were Saudi nationals. Patients with SLE enrolled in this study fulfilled at least 4 of the 11 diagnostic criteria for SLE suggested by the American College of Rheumatology.[15] All patients with SLE were positive for anti-nuclear antibodies (ANA) and antibody to double-stranded DNA antigen (anti-dsDNA) (**Table 1**). However, healthy volunteers (control group) were negative for ANA and anti-dsDNA. Patients with SLE and control group had normal liver and kidney function tests; those with abnormal liver and kidney function tests were excluded from the study. Patients with SLE were on corticosteroids and/or azathioprine and/or hydroxychloroquine treatment. This study was approved by The Bioethical and Research Committee, Faculty of Medicine, King Abdulaziz University Hospital, Saudi Arabia. Informed patient consent was received from all study participants.

Blood samples were collected from all participants to measure the 25(OH)D. The serum level of 25(OH)D was measured by competitive protein binding assay using kits (Immunodiagnostic, Bensheim, Germany). Anti-nuclear antibodies were estimated by indirect immuno-fluorescence antibody technique using the standard immunofluorescence on HEp-2 human epithelial cells (IMMCO Diagnostic Inc., Buffalo, NY, USA). Samples were considered positive if the nuclear or cytoplasmic staining was positive at a dilution of ≥1:80. All positive ANA samples were investigated for anti-dsDNA by standard enzyme linked immunosorbent assay (ELISA) kits (Immulisa., IMMCO Diagnostic Inc., Buffalo, NY, USA).

Vitamin D sufficiency is defined as a serum level of 25(OH)D ≥75 nmol/L (≥30 ng/ml). A level ranging between >50 to <75 nmol/L (>20 to <30 ng/ml) was considered as a relatively insufficiency of vitamin D, whereas ≤50 nmol/L (≤20 ng/ml) as vitamin D deficiency.[16] Vitamin D inadequacy includes both vitamin D deficiency and insufficiency.

Data were analyzed using the Statistical Package for the Social Sciences (SPSS Version 14.0). Descriptive statistics such as percentage, mean ± standard deviation were used to describe study variables. Fisher's exact test was used to test the difference between proportions, and 2-tailed t-test was used for continuous variables. A p-value of less than 0.05 was considered statistically significant.

Results. The baseline demographic and laboratory characteristics of 165 SLE patients and 214 control group are presented in **Table 1**. The mean±SD age of SLE patients and control group was 27.8 ± 8.8 years and 27.9 ± 5.1 years. Among SLE group, females comprised 89.7% of the total number of SLE patients with a 8.7:1 female to male ratio. Vitamin D status in SLE patients and control group is presented in **Table 2**. Mean serum levels (nmol/L) of 25(OH)D were significantly lower among SLE patients (23.5±13.7) compared to the matched control group (64.5±28.9, $p<0.0001$). The prevalence of SLE patients with 25(OH)D inadequacy and deficiency was significantly higher than in the control group: 98.8% versus 55% and 89.7% versus 20% ($p<0.0001$). Only 2 (1.2%) SLE patients had adequate (sufficient) 25(OH)D levels compared to 96 (45%) of the control group ($p<0.0001$). The mean serum levels of 25(OH)D in SLE patients with vitamin D inadequacy and deficiency in comparison to control group were 22.3±13.6 versus 44.5±17.5 ($p<0.0001$) and 19.1±9.5 versus 22.9±6.7 ($p=0.0152$). No significant differences were evident with respect to the mean serum levels of 25(OH)D and prevalence of its deficiency in female and male patients with SLE and vitamin D deficiency (**Table 3**).

Discussion. The current study provides insights into vitamin D status and SLE among Saudi patients. In this study, prevalence values and serum mean levels of 25(OH)D were compared only to the data reported from other studies that used the cut-off values for

Table 1 - Demographic characteristics of patients with systemic lupus erythematosus (SLE) and control group.

Characteristics	SLE group (n=165)	Control group (n=214)
Gender		
Male	17	92
Female	148	122
Nationality		
Saudi	165	214
Non-Saudi	0	0
Mean age (years) ± SD	27.8 ± 8.8	27.9 ± 5.1
Median age (years)	26	26
Range (years)	15 - 45	23 - 45
Antinuclear antibody test		
Positive	165	0
Negative	0	214
Antibody to double-stranded DNA antigen test		
Positive	165	0
Negative	0	214

defining vitamin D status recommended by Holick.[16] Vitamin D inadequacy along with the substantial reduction in serum levels of 25(OH)D were found to be highly prevalent in Saudi patients with SLE. Such high prevalence (98.8%) is comparable to a Spanish study (90%),[17] and is greater than reported in the USA (65%).[18] Moreover, the prevalence of vitamin D deficiency in SLE patients (89.7%) was higher than that reported in Canada (56%)[19] and among African-Americans and Caucasians in the USA (67%).[5] However, it is slightly lower than that reported in African-Americans (95%).[20] Despite homogeneity of age, gender, and cut-off value for vitamin D inadequacy, the prevalence of vitamin D inadequacy among female healthy control group in the current study was 56.5% (data not shown). This prevalence is approximately double than that recently reported among young healthy women in Saudi Arabia (30%).[13] The reasons for the disparity between findings of these studies are uncertain. Recent reports have stressed methodological considerations in assay, and inter-laboratory variations, even when using the

Table 2 - 25-hydroxyvitamin D status in patients with systemic lupus erythematosus (SLE) and control group.

25-hydroxyvitamin D	SLE patients (n=165)		Control group (n=214)		Mean difference	95% CI	*P*-value
	n (%)	Mean ± SD*	n (%)	Mean ± SD*			
Deficiency (≤50 nmol/L) Range of serum level	148 (89.7)	19.1 ± 9.5 2 - 48	43 (20.1)	22.9 ± 6.7 11 - 35	-3.80	-6.86, -0.74	0.0152
Insufficiency (>50 - <75nmol/L) Range of serum level	15 (9.1)	53.9 ± 2.7 51 - 61	75 (35.0)	56.9 ± 5.5 51 - 74	-3.0	-5.89, -0.10	0.0427
Inadequacy (<75nmol/L) Range of serum level	163 (98.8)	22.3 ± 13.6 2 - 61	118 (55.1)	44.5 ± 17.5 11 - 74	-22.2	-25.85, -8.55	<0.0001
Sufficiency (≥75nmol/L) Range of serum level	2 (1.2)	128 ± 73.5 76 - 180	96 (44.9)	89.1 ± 19.5 76 - 210	38.9	9.40, 68.39	0.0103
Overall mean Range of serum level	165 (100.0)	23.5 ± 13.7 2 - 180	214 (100.0)	64.5 ± 28.9 11 - 210	-41.0	-45.79, -36.21	<0.0001

*Serum mean levels of 25-hydroxyvitamin D [25(OH)D] in nmol/L ± standard deviation (2.5 nmol/L = 1.0 ng/ml)

Table 3 - Gender-based difference with respect to prevalence of 25-hydroxyvitamin D [25(OH)D] deficiency in patients with systemic lupus erythematosus (SLE) and control group.

Gender	SLE patients with 25(OH)D deficiency			Control group with 25(OH)D deficiency		
	n (%)	Mean ± SD*	Range	n (%)	Mean ± SD*	Range
Female	132/148 (89.2)	19.0 ± 9.3	5 - 48	33/122 (27.0)	22.8 ± 6.9	11 - 35
Male	16/17 (94.1)	19.5 ± 11.6	2 - 44	10/92 (10.9)	23.3 ± 6.4	17 - 35
P-value	1.000	0.844		0.003	0.834	

*Serum mean levels of 25-hydroxyvitamin D [25(OH)D] in nmol/L ± standard deviation (2.5 nmol/L = 1.0 ng/ml)

same assay procedures. Efforts to standardize assays and to improve accuracy and reproducibility have been recommended.[21] Nevertheless, the current study along with Al-Turki et al[13] findings support that low serum concentration of 25(OH)D is not only confined to patients with SLE, but it is highly prevalent among SLE-free apparently healthy women population in Saudi Arabia.

Approximately 90% of SLE patients who were vitamin D deficient had serum levels of 25(OH)D <10ng/ml (**Table 2**), which were far lower than that reported in studies with different ethnic background populations,[5,13,19,20,22] but slightly higher than that reported in SLE-free Arab-American women.[23] In a study of Passeri et al,[24] a total of 76% patients with osteoporosis had a serum level of 25(OH)D <30nmol/L (<12ng/ml). Serum levels of 25(OH)D <10ng/ml trigger the secondary hyperparathyroidism and thereby exert negative impact on skeletal system.[16,21] Restoring serum 25(OH)D to optimal levels >75nmol/L (>30ng/ml) may be required to maximize intestinal calcium absorption and to prevent secondary hyperparathyroidism induced-skeletal disorder.[16,21] It is important to note that the majority of SLE patients in this study had serum level of 25(OH)D <25nmol/L (<10ng/ml). It is well known that the incidence of SLE is higher in female than male. Nonetheless, the current study showed no significant gender-based differences in levels of 25(OH)D ≤50nmol/L (≤20ng/ml) in patients with SLE (**Table 3**). Multiple risk factors for vitamin D deficiency in SLE patients include a lack or limited exposure to sunlight, chronic use of corticosteroids, and deterioration of kidney function as most patients with SLE may have renal involvement. Patients with SLE are frequently photosensitive[17] and lack of their exposure to sunlight, a prerequisite for vitamin D biosynthesis, may contribute to the development of vitamin D deficiency. With respect with the effects of corticosteroids on vitamin D metabolism, contradictory reports have been published. Corticosteroid-treated patients have shown a reduction[25] in serum levels of $1,25(OH)_2D_3$ or no change,[26,27] and a reduction in level of 25(OH)D[26-28] or no change.[29] Systemic lupus erythematosus-related renal involvement may inhibit the conversion of 25(OH)D in the kidney to its biologically active form $1,25(OH)_2D_3$ via inhibition of 1-α hydroxylase.[16,21] This risk factor can be ruled out since all patients in this study were free from kidney and liver dysfunctions. Another factor related to low serum level of 25(OH)D is obesity which is associated with low level of 25(OH)D as a result of sequestration of the latter in body fat.[16,30] A 35.6% overall prevalence of obesity in Saudi Arabia[31] (female 44%, male 26.4%) would have contributed to 25(OH)D inadequacy in both SLE and healthy population in this study. Moreover, cultural and religious practice of wearing clothes that cover the entire body (veiled and unveiled women), preventing direct sunlight exposure may have contributed to the high prevalence of low serum 25(OH) levels, particularly in young women in Saudi Arabia,[12,13] United Arab Emirates,[32] Jordan,[33] Kuwait,[34] and Arab-Americans.[23]

The current study has the following limitations: a) correlation of low serum levels of 25(OH)D with variables related to vitamin D deficiency such as serum calcium, serum inorganic phosphate, serum parathyroid hormone and alkaline phosphatase have not been evaluated; and b) the relation between low serum levels of 25(OH)D with SLE severity and drug treatment regimens used have not been explored.

In conclusions, health policy decision makers should pay attention to the high prevalence of vitamin D inadequacy and the low levels of 25(OH)D among SLE patients and healthy population in Saudi Arabia. In view of the immunomodulatory effects of calcitriol, and hypovitaminosis D - related health consequences, a national strategy for vitamin D supplementation, and its role in treatment of SLE should be evaluated.

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Related topics

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