ASSOCIATION OF VITAMIN D STATUS AND CO MORBIDITIES IN EGYPTIAN CHILDREN WITH SICKLE CELL DISEASE (SINGLE CENTER STUDY)

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ABSTRACT

Background: *Sickle cell disease (SCD) is a class of hemoglobinopathy characterized by hemolytic anemia, increased incidence of infections and vaso-occlusion that may affect quality of life and life expectancy. Vitamin D deficiency in SCD patients may associate with increased disease severity.*

Objective: *The present study was conducted to investigate relationship between vitamin D insufficiency and severity of SCD and its related complications.*

Patient and Methods: *This was a cross-sectional study, where 50 SCD patients in steady state and 50 age and sex matched healthy controls, they were selected from outpatients clinic of El Galaa Teaching Hospital during period from May 2019 to November 2019 by simple random method ,they were subjected to anthropometric parameters, Thorough history taking, physical examination, laboratory investigations included complete blood count, reticulocyte count (Retics), aspartate transaminase (AST), alanine transaminase (ALT), lactate dehydrogenase (LDH), and total (TSB) and indirect (Indirect SB)serum bilirubin, Blood Urea Nitrogen (BUN), serum creatinine (Cr),serum 25-hydroxy vitamin D assessment.*

Results: *Vitamin D deficiency found in 60% of SCD patients and 22%of controls while severe VDD (25(OH) D level<10 ng /ml) found in12% of SCD patients and4% of controls. SCD patients classified according to serum vitamin D level to Normal group and Deficient group. There were statistically significant differences between the SCD studied groups regarding their age, anthropometric measures(P values=0.000), frequency of VOC, frequency of severe VOC, frequency of emergency room visit due to pain, frequency of infection, length of hospital admission, frequency of blood transfusion last year. As regarding laboratory investigations, hemoglobin and hematocrit values were significantly lower in VDD group (P values 0.000) while the levels of total leucocytic count, reticulocyte count, lactate dehydrogenase, total and indirect bilirubin were significantly higher in the same group (P values 0.002, 0.000, 0.000, 0.000, and 0.000, respectively).Regarding SCD related complications there was insignificant difference between two groups but bone fracture higher in VDD group.*

Conclusion: *SCD patients may have vitamin D deficiency and may benefit from vitamin D screening and supplementation that may lead to improve growth, decrease severity of disease, complications and improve quality of life.*

Keywords: *Sickle cell disease, vitamin D deficiency, Vitamin D, Anemia.*

INTRODUCTION

Sickle cell disease (SCD) is one of red cell genetic disorder characterized by chronic hemolysis, chronic inflammation, immune deficiency, recurrent infection, vaso-occlusive crises (VOC)[(1)] and associated with increased risks of serious morbidity and mortality[(2)].

SCA is caused by mutation in beta globin gene that leads to substitution of glutamic acid for valine and production of hemoglobin S (HbS). SCA patients exhibit a chronic inflammatory state and reduced length and quality of life[(3)].

Vitamin D plays essential role in human health, not only bone integrity, maintaining normal serum calcium and phosphate levels but also normal immune function, cell proliferation, differentiation, blood pressure control, secretion of insulin and lipid metabolism[(4)].

As the main source of vitamin D in humans results through skin from exposure to the sun, there are many factors affecting vitamin D status like age, race, latitude, sunlight exposure and skin pigmentation[(5)]. In recent years vitamin D deficiency (VDD) is emerges as worldwide major health problem and associated with many skeletal and non-skeletal disorders including cardiovascular diseases, respiratory disorders, asthma, stroke, heart failure, renal impairment, immunodeficiency and cancer[(6-8)].

In recent years, many researchers reported high prevalence of VDD among SCD patients[(9)]. Many predisposing factors lead to VDD such as decreased vitamin D synthesis from sunlight due to skin pigmentation and restricted outdoor activity, inadequate dietary intake as result of diminished appetite, impaired absorption due to damage of intestinal mucosa as a complication of SCD and high metabolic requirements as compensation for increased erythrocyte production due to shortened life span of red blood cells as well as renal impairment that interferes with activation of vitamin D[(10-12)]. As both SCD and VDD may have similar criteria of

pain, VDD may increase vulnerability of SCD patients to complications such as VOC, chronic pain, osteopenia, infection, renal impairment, and autoimmune disorders[13-16].

PATIENTS AND METHODS

This was a cross-sectional study carried out during the period from May 2019 to November 2019.Including 50 SCD patients and 50 healthy child, they were selected from out patient's clinic of El-Galaa teaching hospital by simple random method.

Inclusion criteria:

1. Age: 4-18 years old.
2. Sex: male or female.
3. Patients with SCD (including homozygous sickle cell anemia & sickle/beta thalassemia) diagnosed by hemoglobin electrophoresis and/or high-performance liquid chromatography.
4. Patients in steady state, defined as absence of acute episodes e.g. infection, VOC, acute chest syndrome, stroke, priapism, for at least four weeks prior to sampling[17].

Exclusion criteria:

1. Patients aged > 18 years.
2. Patients with renal impairment, chronic liver disease or malabsorption.
3. Patients on chronic transfusion therapy program or steroid therapy.
4. Patients who are taking dietary supplement containing vitamin D.
5. Patients who had blood transfusion in the last four weeks.

Ethical Considerations:

1. Informed consent was obtained willingly from all patients, control and/ or their legal guardians.
2. The study was approved by ethics committee of General Organization of Teaching Hospital and Institutes and conducted according to Helsinki declaration.
3. The patient has the right to withdraw from the study at any time.
4. The data &results of the study are confidential & the patient has the right to keep it.
5. The authors have no conflict of interest regarding the study publication.
6. The authors declared that no financial support.

All studies cases were subjected to the following:

Complete history taking including:

1. Demographic data including age and sex.
2. History of disease related events e.g. frequency of hospitalization, frequency of infections, frequency of VOC, frequency of severe VOC, defined as pain in the extremities, back, abdomen, chest, or head that led to an unscheduled clinic or emergency room visit and required hospitalization, and that could only be explained by SCD, with exclusion of hand-foot syndrome, chest syndrome, osteomyelitis, and any episode of pain that was treated entirely at home[18], frequency of blood transfusion, disease related complications, e.g. leg ulcers, stroke.
3. Transfusion history including frequency of transfusion last year.
4. Complete clinical and systemic examinations including:
    1. Vital signs.
    2. Anthropometric measurements.
    3. General examinations e.g. skin, joint, others.
    4. Chest, heart, abdomen, etc.

Laboratory evaluation including:

1. Complete blood count (CBC).
2. Reticulocytic count (Retics).
3. Renal function tests: blood urea (BUN) and serum creatinine (Cr).
4. Liver function tests:alanine transaminase (ALT), aspartate transaminase(AST) and serum bilirubin (total, indirect).
5. Lactate dehydrogenase (LDH)
6. Serum 25-hydroxy vitamin D (25 (OH) D).

Neuroimaging: Trans cranial Doppler (TCD).

Samples collection, vitamin D assay.

A blood sample collected from both patients and control by aseptic venipuncture, blood specimens left to be clot for 30 min at room temperature then centrifugation at 3000g for 10 minutes. The sera were analyzed or stored at 2-8°C till processing using Enzyme-linked immunosorbent assay (ELISA) (Immdiagnostic K EIA Bensheim and Biomedica, Wien, Austria)[19]. The 25 (OH) D levels were classified as follows: sufficiency (normal) was defined as 25(OH) D level of ≥20to 30 ng /ml,

insufficiency as <20 to 10ng /ml, severe deficiency as<10 ng /ml.

Statistical analysis:

Data was analyzed using statistical package for social sciences (SPSS) 15. Mean or median was used for continuous variables depending on the distribution of values. Associations between the outcome of critically ill children and various variables were estimated using Fisher's Exact Test and Chi-Square Tests or Mann Whitney test. Correlations between quantitative variables were done using Spearman correlation coefficient. A p value of <0.05 was considered to be statistically significant.

RESULTS

Fifty patients with established diagnosis of SCD on the basis of clinical manifestation and hemoglobin electrophoresis were included in this study; 36 (72%) of SCD patients were homozygous hemoglobin S (HBSS) and 14 (28%)patients with sickle β-thalassemia (HBSβ).

Table (1): Demographic and anthropometric data of the SCD patients and control

Variable	Patients (n=50)	Control (n=50)	P value
Age (years): mean ± SD	8.61 ± 4.26	8.84 ± 3.79	0.778
Sex: Males n(%) Females n(%)	 29 (58%) 21 (42%)	 23 (46%) 27 (54%)	0.230
Z score Wt: median (IQR)	-1.38 (-2.29 – 0.01)	0.04 (-0.62 – 0.62)	0.000
Z score Ht: median (IQR)	-2.32 (-3.15 – -0.33)	0.23 (-1.42 – 1.21)	0.000
BMI: mean ± SD	17.18 ± 2.89	19.01 ± 2.85	0.002

SCD patients had significantly lower anthropometric measurements (Z score for weight, Z score for height and body mass index) as compared to control.

Table (2): Laboratory finding in SCD patients and control

Variable	Patients(n=50)	Control(n=50)	P value
Hb (g/dl):mean ± SD	8.02 ± 1.31	12.85 ± 1.72	0.000
Hct (%):mean ± SD	23.92 ± 3.42	33.45 ± 3.96	0.000
MCV (FL): mean ± SD	80.92 ± 13.42	79.9 ± 7.58	0.640
TLC: mean ± SD	10.06 ± 4.63	7.44 ± 3.02	0.001
PLT: mean ± SD	382.43 ± 166.25	257.61 ± 109.05	0.001
Reticulocyte count (%): mean ± SD	7.89 ± 3.9	2.1 ± 0.8	0.000
LDH: mean ± SD	639.72 ± 248.13	257.28 ± 68.37	0.000
BUN: mean ± SD	13.14 ± 6.28	13.03 ± 6.31	0.932
Cr: mean ± SD	0.43 ± 0.13	0.47 ± 0.11	0.099
ALT(IU/L): mean ± SD	29.1±21.3	21.1±9.8	0.000
AST(IU/L): mean ± SD	46.3±23.3	34.4±10.1	0.001
25-OHD (ng/ml) Sufficient n(%) Deficient n(%)	 20 (40%) 30 (60%)	 39 (78%) 11 (22%)	0.000
25-OHD (ng/ml): mean ± SD >10 10-<20 20-30 <30	19.68 ± 8.35 6 (12%) 24 (48%) 13 (26%) 7 (14%)	22.4 ± 6.98 2 (4%) 9 (18%) 33 (66%) 6 (12%)	0.080 0.001

Table 2 shows statistically significant decrease in the level of hemoglobin (Hb), hematocrit (Hct) and increase in total leucocytic counts (TLC), reticulocyte count (Retics), lactate dehydrogenase (LDH), aspartate transaminase (AST), alanine transaminase (ALT) in SCD patients than control group. As regarding 25(OH) D levels, SCD patients were significantly lower than control ($p < 0.001$), **(Figure 1)**.

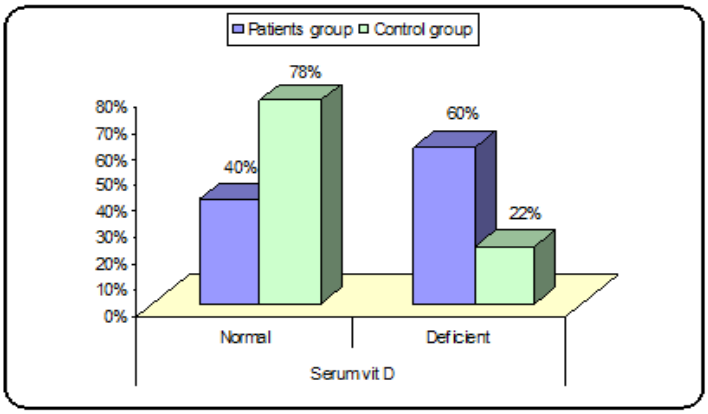

Figure (1): Comparison of serum vitamin D level between SCD patients and control

Table (3): Comparison of demographic in SCD patients groups

Variable		Sufficient vit D group n=20	Deficientvit D group n=30	P value
Age (years)	Mean ± SD Range	5.5 ± 1.95 3.3 – 11.5	10.69 ± 4.13 3.5 – 18	0.000
Sex: n (%)	Male Female	13 (65%) 7 (35%)	16 (53.3%) 14 (46.7%)	0.413
Z score Wt	Median (IQR) Range	0.45 (-0.14 – 1.05) -1.4 – 1.93	-2.2 (-2.99 – -1.49) -5.19 – -0.96	0.000
Z score Ht	Median (IQR) Range	-0.08 (-0.89 – 0.77) -3.15 – 1.2	-2.69 (-3.86 – -2.29) -5.1 – -1.21	0.000
BMI	Mean ± SD Range	19.32 ± 3.41 15 – 29.34	15.76 ± 1.12 13.67 – 17.68	0.000

Sickle cell disease patients was divided into 2 groups: 'Sufficient group' with normal vitamin D level (25(OH) D level of ≥20) and 'Deficient group' with VDD (25(OH) D level of <20).

There were statistically significant decrease in anthropometric measurements (Z score for weight, Z score for height and body mass index) in VDD group (P values=0.000).

Table (4): Comparison of clinical data and co morbidities in SCD patients groups

Variable		Sufficient vit D group n = 20	Deficient vit D group n = 30	P value
Hemoglobin genotype: n (%)	SS S/B	14 (70%) 6 (30%)	22 (73.3%) 8 (26.7%)	0.797
Number of voc/last year	Median (IQR) Range	14.5 (10 – 18) 5 – 28	22.5 (12 – 29) 6 – 36	0.017
	>10 10-20 <20	3 (15%) 14(70%) 3 (15%)	3 (10%) 10 (33.3%) 7 (56.7%)	0.012
Number of severe voc/last year	Median (IQR) Range	2 (1 – 5.5) 0 – 11	7.5 (4 – 14) 0 – 18	0.000
	>10 10-20 <20	14 (70%) 5 (25%) 1 (5%)	9 (30%) 11 (36.7%) 10 (33.3%)	0.011
frequency of ER visit due to pain	Median (IQR) Range	6 (4 – 8) 2 – 15	9 (7 – 14) 4 – 25	0.002
Number of patients having Infection/last year	Positive Negative	12 (60%) 8 (40%)	28 (93.3%) 2 (6.7%)	0.004
frequency Infection/last year	Median (IQR) Range	3.5 (2 – 5) 2 – 8	9 (6 – 10) 1 – 16	0.001
Length of hospital stay (days)	Mean ± SD Range	16.55 ± 6.3 17 – 27	23.33 ± 10.07 4 – 39	0.010
frequencyhospital admission due to pain/ last year	Median (IQR) Range	2 (1 – 5.5) 0 – 11	7.5 (4 – 14) 0 – 18	0.000
Frequency of blood transfusion/ last year	Median (IQR) Range	4 (4 – 6) 2 – 8	7 (6 – 10) 2 – 12	0.001

There was statistically significant increase in frequency of VOC, frequency of severe VOC, frequency of emergency room visit due to pain, frequency of infection, length of hospital admission, frequency hospital admission due to pain, frequency of blood transfusion last year in VDD group **(Figure 2)**.

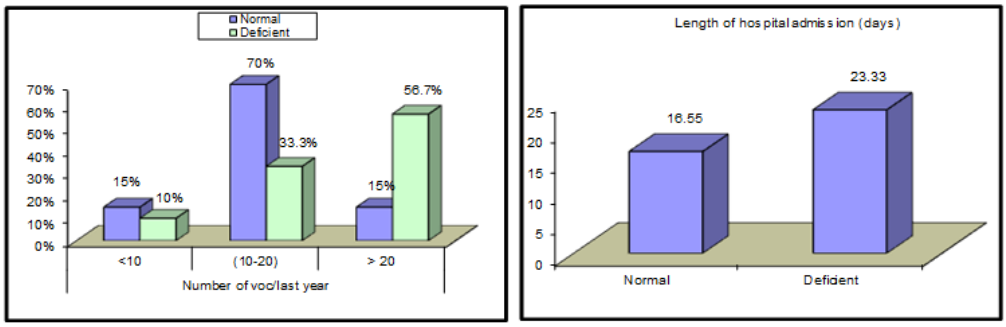

Figure (2): Comparison of clinical course between sufficient and vitamin D deficient SCD patients

Table (5): Comparison of laboratory data between sufficient and vitamin D deficient SCD patients

Variable		Sufficient vit D group n = 20	Deficient vit D group n = 30	P-value
HB	Mean ± SD	8.7 ± 0.77	7.17 ± 0.8	0.000
	Range	7.6 – 9.8	5.6 – 8.7	
HCT	Mean ± SD	26.32 ± 2.63	22.32 ± 2.94	0.000
	Range	20.1 – 30.5	18.2 – 28.5	
TLC	Mean ± SD	7.7 ± 3.55	11.63 ± 4.65	0.002
	Range	3.4 – 15.1	4.6 – 25.3	
Retics.	Mean ± SD	4.31 ± 1.74	10.28 ± 2.98	0.000
	Range	1.6 – 7.7	2.1 – 18.2	
LDH	Mean ± SD	415 ± 81.07	789.53 ± 204.15	0.000
	Range	261 – 598	453 – 1324	
BUN	Mean ± SD	13.05 ± 6.48	13.2 ± 6.26	0.935
	Range	8 – 32	8 – 32	
Cr.	Mean ± SD	0.41 ± 0.15	0.45 ± 0.12	0.361
	Range	0 – 0.6	0.05 – 0.6	
ALT	Median (IQR)	18 (14 – 25)	15 (12 – 22)	0.180
	Range	11 – 75	6 – 106	
AST	Median (IQR)	36 (31 – 46.5)	42.5 (32 – 49)	0.280
	Range	17 – 90	20 – 68	
TSB	Median (IQR)	1.6 (1.15 – 2)	4.7 (3.5 – 5.8)	0.000
	Range	0.4 – 3.5	1.6 – 7.8	
Indirect SB	Median (IQR)	0.35 (0.3 – 0.45)	2.25 (1.4 – 2.6)	0.000
	Range	0.1 – 0.7	0.3 – 3.4	

Hemoglobin and hematocrit values were significantly lower in VDD group ,while the levels of total leucocytic count,

reticulocyte count, lactate dehydrogenase, total and indirect bilirubin were significantly higher in the same group.

Table (6): Comparison of SCD related complication and Trans cranial Doppler between sufficient and vitamin D deficient SCD patients

Variable		Sufficient vit D group	Deficient vitD group	P-value
		n = 20	n = 30	
SCD related complications	Pulmonary hypertension	3 (25.0%)	4 (16.7%)	0.719
	Diabetes mellitus	2 (16.7%)	3 (12.5%)	
	Gall bladder stones	1 (8.3%)	1 (4.2%)	
	Bone disease. eg. Osteomyelitis, septic arthritis	3 (25.0%)	9 (37.5%)	
	Acute chest syndrome	0 (0.0%)	0 (0.0%)	
	Splenic sequestration crisis	1 (8.3%)	0 (0.0%)	
	Sickle cell hepatopathy	0 (0.0%)	1 (4.2%)	
	Priapism	1 (8.3%)	1 (4.2%)	
	Bone fracture	1 (8.3%)	5 (20.8%)	
TCD	1-Normal	18 (90.0%)	26 (86.7%)	0.939
	2-Conditional	1 (5.0%)	2 (6.7%)	
	3- Abnormal	1 (5.0%)	2 (6.7%)	

This table shows insignificant difference between two groups as regarding SCD complications and Trans cranial Doppler but bone fracture higher in VDD group.

Table (7): Correlation between Serum 25-OHD and clinical course of the SCD patients

Variable	Serum vit D	
	r	P-value
Age (year)	**-0.710****	**0.000**
Z-score Wt	**0.895****	**0.000**
Z-score Ht	**0.841****	**0.000**
BMI	**0.699****	**0.000**
Number of voc/last year	**-0.473****	**0.001**
Number of severe voc/last year	**-0.682****	**0.000**
Frequency of ER visit due to pain	**-0.594****	**0.000**
Frequency Infection/last year	**-0.595****	**0.000**
Length of hospital stay (days)	**-0.363****	**0.010**
Frequency hospital admission due to pain/ last year	**-0.654****	**0.000**
Frequency of blood transfusion/ last year	**-0.538****	**0.000**

This table shows statistically significant negative correlation between serum 25-OHD and age, number of VOC, number of severe VOC, frequency of emergency room visit due to pain, frequency of infection, length of hospital admission, frequency hospital admission due to pain and frequency of blood transfusion **(Figure 3)** as well as positive correlation with anthropometrics measurement.

Table (8): Correlation between Serum 25-OHD and laboratory data of the SCD patients

Variable	Serum vit D	
	r	P-value
HB	**0.819****	**0.000**
HCT	**0.498****	**0.000**
MCV	-0.208	0.148
TLC	**-0.434****	**0.002**
PLT	-0.173	0.231
Retics.	**-0.804****	**0.000**
LDH	**-0.807****	**0.000**
BUN	0.052	0.721
Cr.	-0.023	0.872
ALT	0.271	0.057
AST	-0.039	0.790
TSB	**-0.808****	**0.000**
indirect SB	**-0.759****	**0.000**

As regarding laboratory results there were statistically significant positive correlation between serum 25-OHD and hemoglobin, hematocrit and negative correlation with total leucocytic count, reticulocyte count, lactate dehydrogenase, total and indirect bilirubin.

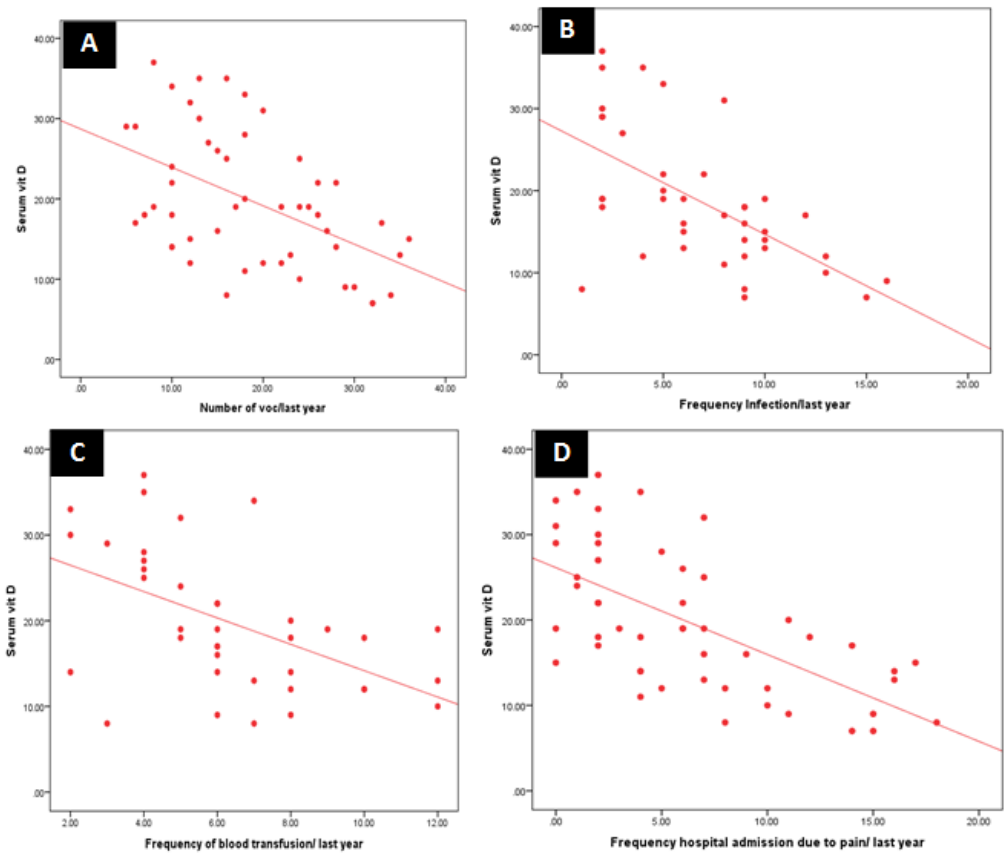

Figure (3): Correction of serum D with, A) Number of Vaso-occlusive crises, B) Frequency infection, C) Frequency of blood transfusion and D) Frequency hospital admission due to pain

DISCUSSION

Children and adolescents with sickle-cell anemia are at greater risk of developing nutritional deficiencies especially vitamins due to chronic pain, chronic inflammation, recurrent infection and frequent hospital admission. Several studies reported high prevalence of VDD in SCD children when compared with healthy control and ranged between 20%to 80% in many Middle Eastern counties [(20)].

The present study reported VDD in 60% of SCD patients and 22% of control while severe VDD was found in12% of SCD patients and4% of control that in concords with study by **Hamdy et al.** [21] that reported VDD in 60% of SCD and 26.7% of controls while severe VDD was reported in 16.2% of SCD patients and 8.3% of control and studies by **Garrido et al.** and **Ozen et al.**[22,23] revealed prevalence of VDD in SCD patients were 56.4% and 63.1%respectively.A studies by **Wykes et al.** and **AlJama et al.**[24,25] revealed high prevalence of VDD in SCD patients 91% and 82% respectively. While the prevalence of severe VDD was 12% in sickle patients on a study by **Mohammed et al.**[26].Our result showed negative correlation between age and serum 25-OHD of SCD patients, in agreement with recent studies suggested that older SCD patients associated with high vitamin D insufficiency which may be due to increased skin pigmentation, damage of intestinal mucosal and impairment of renal functions with age[21,44].

As regarding to anthropometric parameters the current study showed significant lower z score for height, z score for weight and BMI in SCD patients as compared healthy control, also there was significant difference in growth parameters between VDD and vitamin D sufficient SCD patients that in agreement with several researches that revealed growth retardation as major health problem in SCD patients. A studies by **Cipolotti et al.** and **Ozen et al.**[27,23] reported growth retardation in 24% of SCD patients and a study by **Al-Saqladi et al.**[28] reported height lower than -2 SD in 54% of SCD patients. Growth retardation in SCD patients multifactorial may be due to nutritional deficiency, chronic inflammatory process, hyper metabolic state, hypogonadism[23] and decline of vitamin D binding protein[29].

The present study detected higher frequency of VOC, severe VO C and emergency room visit due to pain, hospital admission and length of hospital stay last year in VDD group of SCD patients and correlated negatively with serum 25-OHD. This is consistent with other studies[14,21,30] revealed positive correlation between incidences of VOC and VDD in SCD patients while other study[31] can't found association between VDD and pain episodes in SCD children. Our result in concords with study

by Brown and colleague[32] suggested significant association between vitamin D insufficiency and frequency of ER visits, frequency of hospital admissions for pain crisis, and the length of hospital stay VOC is main presenting morbidity associated with SCD and may lead to frequent emergency room visit and recurrent hospital admission in about 95% of cases[33]. The mechanisms by which vitamin D might reduce pain are unclear, may be affect nervous system directly by modulation of inflammatory cytokine activation or enhance expression of SLC6A5 gene which encodes for glycine transporter 2 acts as neuronal pain pathway protein and indirect by improvement of bone health as impaired mineralization of bone allows the osteoid matrix to absorb fluid and expand, causing outward pressure on the highly innervated periosteal tissues resulting in pain syndromes[34]. Also, other studies suggested vitamin D supplementation result in improvement of pain symptoms and decreased usages of analgesic[30,35].

Researchers reported that vitamin D is essential for the integrity and proper function of both innate and acquired immunity (36). Vitamin D supports innate immunity by enhances the release of Cathelicidenone of important inflammatory mediators that promoting and accelerating antigen destruction[37] also vitamin D play important role in both cell mediated and humoral immune responses by modulating the proliferation of T lymphocytes and regulating cytokines production and regulation of B lymphocyte proliferation, production of antibodies, and cell transformation to plasma or memory cell[38].Our study reported statistically higher frequency of infection in VDD group last year, in concords with other studies that reveals increased incidence of infection among vitamin D insufficiency SCD patients[21,39] while study done by Lee and colleague[14] noticed 50% reduction in respiratory tract infection on vitamin D supplementation which can provide protective effect against respiratory complications of sickle cell disease patients and another study by **Urashima and colleagues**[40] suggested the role of vitamin D supplementation in prevention of seasonal influenza.

As regarding laboratory results, our study noticed low hemoglobin and hematocrit and high TLC, LDH and reticulocyte count in VDD group. Serum vitamin D correlate positively with hemoglobin and hematocrit but negatively with total leucocytic

count, reticulocyte count, lactate dehydrogenase, total and indirect bilirubin that in agreement with study by **Chennamashetti and colleagues**[41] observed low hemoglobin and positive correlation with vitamin D insufficiency also other studies showed significant association between low hemoglobin, hematocrit, high reticulocyte count, TSB, indirect SB, LDH and vitamin D insufficiency[21,40,42] in contrary to our result other studies reported insignificant correlation between low serum vitamin D level and hemoglobin, hematocrit, reticulocyte count and AST level[43-44]. Association between vitamin D deficiency and biomarker of hemolysis can be explained by enhanced bone marrow activity may interfere with absorption of vitamin D[45] and VDD may lead to increase RBCs hemolysis in SCD patients[46].

The present study showed higher history of bone fracture among VDD group as compared with normal serum vitamin D children with SCD that in agreement with studied by **Sadat-Ali et al.** and **Arlet et al.**[47,48] that suggested VDD in SCD patients leads to lower bone density and higher risk of bone fracture.

CONCLUSION

Vitamin D insufficiency considers as important health problem in children with SCD as may affect clinical course of the disease and exacerbate its complications.

RECOMMENDATION

Routine serum vitamin D screening to all SCD patients may result in early detection of its deficiency and consequence vitamin D supplementation may improve clinical outcome and quality of life in SCD patients. Further studies with larger sample size are needed to determine the long-term effects of vitamin D supplementation on other aspects of health and well-being in SCD patients.

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العلاقة بين مستوى فيتامين د ومضاعفات انيميا الخلايا المنجلية فى الاطفال المصريين

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خلفيـــة الدراســـة: يعتبـــرنقص فيتـــامين د مـــن اكثـــر المشـــاكل التـــى تواجـــة المرضـــى المصـــابين بانيميـــا الخلايـــا المنجليـــة حيـــث انـــة يوثربصـــورة ملحوظـــة فـــى التطـــور الاكلينيكـــى للحالـــة ويزيـــد مـــن مضاعفاتها.

الهـــدف مـــن الدراســـة: تقيـــيم العلاقـــة بـــين نقـــص فيتـــامين د وشـــدة المـــــرض ومضـــــاعفاتة للمرضـــــى المصـــــابين بانيميـــــا الخلايـــــا المنجلية.

المرضـــى وطريقـــة العمـــل: تـــم ادراج 50 مريضـــا فـــي فتـــرة ســـكون اعـــــراض المـــــرض و 50 شـــــخص ســـــليم كمجموعـــــة ضـــــابطة وخضـــــعت جميـــــع الحـــــالات إلـــــى إســـــتبيان و الفحـــــص الاكلينيكـــــى، والتحقيقات المختبرية الروتينية و تحديد نسبة فيتامين د.

النتـــائج: قـــد تبـــين نقـــص نســـبة فيتـــامين د بالـــدم لـــدى 60% مـــن الاطفـــــال المصـــــابين بانيميـــــا الخلايـــــا المنجليـــــة و22% مـــــن المجموعـــة الضـــابطة كمـــا وجـــد نقـــص شـــديد فـــى فيتـــامين د لـــدى

12% من المرضى و4% من الاصحاء وتم تقسيم مرضى انيميا الخلايا المنجلية الى مجموعتين تبعا لمستوى فيتامين د بالدم الى مجموعة ذات مستوى طبيعى ومجموعة ذات مستوى منخفض وقد وجدت علاقة ذات دلالة احصائية بين نقص نسبة فيتامين د بالدم ومعدلات الازمات المرضية والازمات المرضية الشديدة والاصابة بالعدوى وزيارة الطوارى نتيجة للالم والحجز بالمستشفى نتيجة للازمات المرضية الشديدة. ولم توجد علاقة بين نقص فيتامين د ومضاعفات المرض.

الاستنتاج: يعتبر اجراء الفحص الروتينى لنسبة فيتامين د فى الجسم لكل مرضى انيميا الخلاياالمنجلية من اهم الخطوات الواجب القيام بها للتشخيص المبكر لنقص فيتامين د وتجنب المضاعفات. قد يحسن اعطاء فيتامين د بصورة فعالة من الحالة المرضية لانيميا الخلايا المنجلية مما يودى الى حياة افضل لهولاء المرضى و مما يفضل اجراء ابحاث اخرى على عينة احصائية اكبر لمتابعة التاثيرات بعيدة المدى لاعطاء فيتامين د لهولاء المرضى ودراسة تاثيرة على ابعاد الحالة المرضية وتوفيرة حياة افضل لهولاء المرضى.