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Study of Cardiac Abnormalities in Pediatric Patients with Sickle Cell Disease

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Abstract
Background: *Sickle cell disease is an autosomal inherited recessive disease as a result of beta globin gene mutation in the form of substitution of a glutamic acid protein with a valine protein at position 6 result in formation of abnormal hemoglobin S with normal functions except when deoxygenated inside the erythrocyte transformed to polymerization reducing its flexibility and distorts its shape but presence of hemoglobin F, reduce and interferes with formation of polymerization .This abnormal hemoglobin S result in closure of the microvasculature [27] . This lead to ischemia and reperfusion injury to vital organs the presence of abnormal hemoglobin within the red blood cells result in hemolytic anemia. The chronic anemia is stressed the cardiovascular system by closuring small pulmonary artery and myocardial hemosiderosis that cause multiple anatomical and functional changes.*
Objective: *Cardiac assessment of patients with sickle cell anemia in pediatrics age group both anatomical and functional using (M mode, 2 dimensional and Doppler echocardiography).*
Study Design: *Forty-four patients with Sickle cell disease (mean age 10 ± 3.6 years), 26 males and 18 females, admitted to AlMalekKhalid hospital, Al-khareg K.S.A. with proven diagnosis of Sickle cell anemia were included in the study. In addition, thirty tow healthy children with (18 males and 14 females) age and sex matched were served as a control group. All children were subjected to history taking, clinical examination, and routine laboratory investigations as well as M mode, 2 dimensional and Doppler echocardiography.*
Results: *Patients with Sickle cell disease had higher left ventricular end diastolic dimension, left atrial dimension, stroke volume and left ventricular mass compared to children of control group. Mild pulmonary hypertension with mean pulmonary artery systolic pressure (PASP)of 30 ± 6.6 mmHg were observed in patients with Sickle cell disease.*
Conclusion: *Chamber dilations, left ventricular hypertrophy with increased left ventricular mass associated with left ventricular diastolic dysfunction secondary to chronic anemia with volume overload and sickle cell cardiomyopathy confirm the evidence of the literature in characterizing a sickle cell disease in pediatrics*
Keywords*: Sickle cell disease – Congestive heart failure - Cardiac functions – Echocardiography.*

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I. Introduction

Sickle cell disease is an autosomal inherited recessive disease as a result of beta globin gene mutation in the form of substitution of a glutamic acid protein with a valine protein at position 6 result in formation of abnormal hemoglobin S with normal functions except when deoxygenated inside the erythrocyte transformed to polymerization reducing its flexibility and distorts its shape but presence of hemoglobin F, reduce and interferes with formation of polymerization .This abnormal hemoglobin S result in closure of the microvasculature [27]. This lead to ischemia and reperfusion injury to vital organs the presence of abnormal hemoglobin within the red blood cells result in hemolytic anemia. The chronic anemia is stressed the cardiovascular system by closuring small pulmonary artery and infiltration of hemosiderin in the myocardial muscles that cause several changes both anatomical and functional. Sickle cell disease is a chronic disease affecting 1.5 child of every 900 African children, American children [1]. More than 80 % adult patients with Sickle cell anemia suffering from cardiomegaly due to recurrent congestive cardiac failure [2]. Sickle cell disease patients with abnormal hemoglobin S inside the red cells are associated with hemolytic anemia and occlusion of the blood vessels by polymerization of deoxyhemoglobin S inside the sickled cells leading to erythrocyte adhesion to the endothelial cell wall, affecting flow of blood in the vascular system with the result of occlusion of many vessels [3]. Most Sickle cell patients show abnormal finding of the heart because of increasing cardiac output as a compensatory mechanism, also most of severe Sickle cell patients usually have hyperdynamic stats of heart with increased

ejection fractions and a cardiac index that considerable exceeding the normal ranges [4]. Diastolic dysfunction is the most important marker sings appear early in most Sickle cell patients with cardiovascular complications before the development of congestive heart failure, enlargement of the heart, ischemia of the muscles of the myocardium, dysfunction of both ventricles and pulmonary artery hypertension [1]. Furthermore, most patients with Sickle cell anemia developed hemosiderosis of the myocardium which is due to overload of iron from recurrent blood transfusion therapy so the Sickle cell patients at higher risk to [5]. Survive into adulthood has been more common, they had been an increased incidence rate of chronic systems failure [6]. In our study, we are assessing anatomical and functional changes of the heart in patients with Sickle cell patients.

II. Patients And Method

This study included 44 patients with sickle cell disease (26 males and 18 females), of mean ages 10±3.6 years, who were admitted to Al Malek Khalid hospital of Al-khareg, Saudi Arabia during the year 2014 through 2016.

A control group with normal hemoglobin level and hemoglobin electrophoresis consist of 32 healthy children (18 males and 14 females) of mean age 10±3.5 years were studied as.

Parents of all children included in the study gave an informed written consent.

All children were subjected to the following:

1- Clinical history taking including time of onset of anemia, frequency of blood transfusion and complications related to sickle cell anemia.

2- Physical examination including heart rate, mean blood pressure and cardiac evaluation.

3- Routine laboratory investigations including hemoglobin level, hemoglobin electrophoresis and serum ferritin.

The diagnosis of Sickle cell disease was confirmed by the presence of high hemoglobin S. Hemoglobin was measured when patients were at steady state, free from any acute process that affect its level, on the same day of echocardiography, were crises free for 3 weeks prior to the study and had not been transfused in the preceding 2 months.

4- Chest X ray and electrocardiography (ECG) for the patients only.

5- Echocardiographic examination**:**

All patients with S.C.D. examined by 2D, M-mode and Color Doppler Imaging. long axis and short axis parasternal views, right ventricular outflow parasternal view, apical views (4 chambers and 5 chambers) and pulmonary blood flow were obtained in all study patients. The measurements of Doppler echocardiography included; the aortic root, left atrial dimension, left ventricular end systolic and end diastolic dimension, left ventricular posterior wall thickness and septal thickness were measured by M-mode, the left ventricle ejection fraction (EF), fractional shortening (FS) and pulmonary artery systolic pressure were calculated. Patients were recorded to have pulmonary hypertension if the estimated pulmonary artery systolic pressure (PASP) was 30 mmHg or above or tricuspid regurgitant jet velocity > 2.5 m/sec. Pulmonary hypertension was categorized as mild (PASP: 30-39 mm Hg), moderate (PASP: 40-54 mmHg) and severe (PASP: 55 mmHg or more) **[7]**.

Echocardiography was used to record LV inflow velocities. Peak flow velocities of the LV inflow in early diastole (E) and late diastole with atrial contraction (A) were measured. E/A velocity ratios for each cardiac cycle. Diastolic dysfunction was graded as normal or as mild, moderate, or severe according to E/A ratio (Mild was defined as an E/A ratio < 1.0, moderate as an E/A ratio of ≥ 1.0 and severe as an E/A ratio higher than the 95th percentiles) [8]. Systolic function was considered abnormal if the fraction of shortening was less than 30 % **[1]**. The LV mass was calculated using the ASE–cube method in Devereux modification LVM (g) = 0.8 [1.04 (septum + posterior wall + LV internal diastolic dimension) ^{3} LV diastolic dimension) ^{3}] + 0.6 and was normalized for the body surface area (BSA) (LV mass index) **[9]**. As LV mass index is overestimated in the presence of volume overload, we calculated LV mass/volume index. This index allows for the elimination of those with a falsely calculated hypertrophy due to enlarged left ventricle but normal ventricular and septal wall thickness. LV hypertrophy (LVH) was defined as LV mass/volume index >1.15gm /ml.

III. Statistical Analysis

Data was expressed as mean ± standard deviation (X ± SD) or percentage (%). The means of two groups were compared using student "t"-test. Data were carried out with the Statistical Package for Social Sciences (SPSS), version 10 software. P-value less than 0.05 was considered statistically significant for all comparison.

IV. Results

In this study, statistical analysis of clinical and laboratory characters of the patients with Sickle cell disease detected that heart rate, blood pressure, fetal hemoglobin ratio and serum ferritin were higher significant

in patients with Sickle cell anemia more than in the controlled children while lower in significance of hemoglobin HB concentrations were observed. With auscultation of (11) cardiac patients (25 %) an ejection systolic murmur heard more in the 3rd left parasternal intercostals space. On chest X ray examination (24) patients with SCD enlargement of the heart were detected (54.4%) and on electrocardiography examinations of (5) cardiac patients with Sickle cell disease ECG changes of left ventricular hypertrophy were present in 11.4 % of patients with Sickle cell disease as shown in (**Table 1**). **(Table 2)**: discussed that the patients with Sickle cell anemia had left ventricular end diastolic dimension, left atrial dimension, stroke volume and left ventricular mass a statistically higher more than that of the controlled group. Significantly higher Pulmonary artery systolic pressure (PASP) and Peak flow velocities of the LV inflow in early diastole (E wave amplitude) was noticed in patients with Sickle cell anemia in comparing to controlled children ($P < 0.05$) but late diastole with atrial contraction (A wave) revealed no difference between the patients and controlled groups so the statistical analysis were not significant as shown in (**Table 3**).

Table (1). Clinical and laboratory results of patients with SCD and control group

Variable parameters	Patients group (n =44)	Control group (n =32)	P value
Age	10 ± 3.6 years	10 ± 3.5 years	> 0.05 (NS)
Sex	26 M /18 F	18 M / 14 F	> 0.05 (NS)
Heart rate (bpm)	94 ± 10	84 ± 8	< 0.05(S)
Hemoglobin level (gm/dl)	8.4 ± 4	12.4 ± 2.6	< 0.001 (HS)
Blood pressure (mmHg)	94 ± 12 / 64 ± 9	84 ± 11 / 58 ± 7	< 0.001 (HS)
Past medical history: Congestive heart failure Acute chest syndrome Sepsis or bacteremia	6 (13.6 %) 11 (25 %) 5 (11.4 %)		
Blood transfusion (≥ 8 / year)	24 (54.5 %)		
Cardiac murmur	11 (25 %)		
Hemoglobin S (%)	68.2 ± 16. 8		
Hemoglobin F (%)	9.9 ± 8.3	2.2±1.2	< 0.001 (HS)
Serum ferritin (ng/ml)	966 ± 212	86 ± 20	< 0.001(HS)
Cardiomegaly in chest X – ray	24 (54.5%)		
Abnormal ECG	5 (11.4 %)		

NS: Non-significant S: Significant HS: Highly significant

Table (2). Echocardiographic changes of patients with SCD and control group

variable parameters	Patients group (n =44)	Control group (n =32)	P value
Left atrial dimension	20 ± 4.4	15 ± 3.6	< 0.05 (S)
Aortic root dimension	19.0 ± 4.1	17.5 ± 2.8	> 0.05 (NS)
LVIDd	35 ± 2.2	26. 7 ± 3.2	< 0.001 (HS)
LVIDs	22.4 ± 3.5	21.7 ± 3.1	0.07 (NS)
IVSd	8.0 ± 0.5	7.2 ± 2.0	0.59 (NS)
IVSs	10.8 ± 2.3	9.7 ± 4.2	> 0.05 (NS)
LVPWd	6.8 ± 1.2	6.8 ± 1.5	0.46 (NS)
LVPWs	9.9 ± 1.5	9.1 ± 2.2	0.64 (NS)
LV mass /volume index (gm/ml)	1.25 ± 0.20	1.05 ± 0.2	< 0.001 (HS)
SV	30.5 ± 9.4	22.9 ± 4.2	< 0.001 (HS)
FS %	31.6 ± 5.4	30 ± 6	0.65 (NS)
EF %	66.2 ± 7.4	65.1 ± 3.8	0.51 (NS)

NS: Non-significant S: Significant HS: Highly significant

IVSs: Interventricular septum in systole
LVIDd: Left ventricular internal dimension in diastole
IVSd: Interventricular septum in diastole
LVIDs: Left ventricular internal dimension in systole
LVPWd: LV posterior wall thickness in diastole
LVPWs: LV posterior wall thickness in systole

LVM: Left ventricular mass
SV: Stroke volume
FS: Fractional shortening
EF: Ejection fraction

Table (3). Results of echocardiography of patients with SCD and control groups

variable parameters	SCD (n = 44)	Control Groups (n=32)	P value
Pulmonary artery systolic pressure	30 ± 6.6	24 ± 3.6	<0.05 (S)
E (cm / sec)	114 ± 20	102 ± 15	< 0.05 (S)
A (cm / sec)	50 ± 18	45 ± 11	> 0.05 (NS)
E / A	2.26 ± 0.7	2.20 ± 0.5	> 0.05 (NS)

NS: Non-significant S: Significant E: Peak flow velocities of the LV inflow in early diastole

A: late diastole with atrial contraction

V. Discussion

Sickle cell disease is a chronic disease with chronic anemia so patients with Sickle cell anemia suffering from cardiomegaly due to recurrent congestive cardiac failure [2]. Sickle cell disease patients with abnormal hemoglobin S inside the red cells are associated with hemolytic anemia and occlusion of the blood vessels by polymerization of deoxyhemoglobin S inside the sickled cells leading to erythrocyte adhesion to the endothelial cell wall, affecting flow of blood in the vascular system with the result of occlusion of many vessels [3]. Most Sickle cell patients show abnormal finding of the heart because of increasing cardiac output as a compensatory mechanism, also most of severe Sickle cell patients usually have hyperdynamic stats of heart with increased ejection fractions and the cardiac index that exceeding the normal ranges [4]. Diastolic dysfunction is the most important marker sings appear early in most Sickle cell patients with cardiovascular complications before the development of congestive heart failure, enlargement of the heart, ischemia of the muscles of the myocardium, dysfunction of both ventricles and pulmonary artery hypertension [1]. most patients with Sickle cell anemia developed hemosiderosis of the myocardium which is due to overload of iron from recurrent blood transfusion therapy [10]. Bulkely BH [11], recorded that dilatation and enlargement of both ventricles are common in all patients with Sickle cell anemia. Manish J, et al. [13] I reported that ejection systolic murmur found in 24 % 0f Sickle cell patients but in this study, there was a history of congestive cardiac failure had been recorded in (6) (13.6 %) patients with S.C.A. and (25%) of (11) patients with S.C.A. revealed variable degree of cardiac murmurs according to degree and severity of Sickle cell anemia.

Steinberg M, et al. [24] reported that the blood pressure normal range lower in patients with Sickle cell disease than that reported in healthy children, they and Ensing G, et al, [17] recorded that patients with Sickle cell anemia having high blood pressure more than the normal range of blood pressure (systemic hypertension) liable to elevated risk of death due to occurrence of stroke. But in our study the blood pressures of patients with S.C.A. were higher than that of the controlled group. The blood pressure range were significantly higher values in patients with S.C.D. than that which were normal values for the controlled group so contributing to increase the risk in the patients with S.C.D. to end-organ results in the heart and kidneys as discussed by Manish, et al. [13]. In our study, the abnormalities in ECG have been recorded in 5 patients (11.4 %) agree with echocardiographic information's revealed by Acar P, et al. [3]. AdnanS, et al., Covitz W, et al., Ensing G, et al, [7, 16, 17] they That the consequences of hemodynamic of patients with S.C.D. are describe the state of volume overload that well be described in early studies before. cardiac output increased as results of persistent and chronic anemia lead to increase the state of volume overload, which is revealed by dilation of all chambers of heart with change the heart rate very little. So, left ventricular dilatation results in systolic wall stress and compensated by left ventricular hypertrophy.

Manish, et al. [13] revealed that the impairment of both (left and right) ventricles were no significant systolic function when study occurred on 140 patients with S.C.D. these results agree with our study which found that the systolic function is within ideal limits as the ejection fraction in the patients with S.C.D. showed no significant difference comparing with the controlled group. But a recent study by Roberto F, et al. [18] recorded that systolic function in the patients with S.C.D. was decreased very mild; however, Wei Du, et al. & Al-Saad HI, et al. [1-19] they recorded that systolic function of the patients with S.C.D. were normal.

Wei Du, et al. [1] recorded that parietal stress reduced because of volume over load as a compensating mechanism which lead to hypertrophy and increased stiffness of the ventricle. In this study, the index of left ventricular mass is higher significantly in comparison with the controlled group. Wong W, et al. [2] they recorded that volume over load of the heart with significantly increasing mass of left ventricle related to the degree and severity of anemia.

In contrast, Lima WH, et al. [12] revealed in their study that normality of the mass left ventricle. Bulkely BH [11] and Covitz W, et al. [16]. They reported that hypertrophy of left ventricle in patients with Sickle cell disease prominently early leading to myopathy of myocardium. In this study and Wei Du, et al. [1] found that Pulmonary artery systolic pressure (PASP) in patients with S.C.A. was statistically higher and significant in comparison with controlled group. PASP recorded mild pulmonary hypertension in patients with S.C.D. due to hemolysis with micro-vascular occlusion and left ventricular hypertrophy and stiffness. AdnanS, et al. [7], Manish, et al. [13] and Pignatelli R, et al. [20] they recorded that the prevalence of pulmonary hypertension was high in patients with S.C.D. and the mainly secondary causes of pulmonary hypertension in patients with S.C.D. were vasculopathy, stress of chronic hypoxia, increased viscosity of blood with thromboembolism, increased load of right ventricle and acute syndrome of chest. Our study revealed impairment the inflow of the left ventricle in early diastole (E) in the patients with S.C.D. are statistically difference in significance in comparison with the controlled group. Also, in late diastole the inflow of left ventricle was impaired during contraction of the atrium (A) with difference non-significant when comparison with the controlled group. This revealed stiffness of the left ventricle to the inflow of mitral valve in spite of normality of E/A ratio by Acar P, et al. [3]. Different studies by Wei Du, et al. and Acar P, et al. [11, 13]. They revealed that diastolic function abnormality and impairment of both E and A value with normality of E/A ratio.

Roberto F, et al. [18] they discussed that in patients with S.C.D. systemic hypertension and diastolic dysfunction due to over load of iron and vaso-occlusion of micro-vasculature liable to damage of the myocardium. In conclusion of our study, echocardiography detects cardiac abnormality in form of dilatation of the cardiac chambers, hypertrophy of left ventricle, increased mass of left ventricle in patients with Sickle cell anemia associated with dysfunction of diastolic left ventricle. So, we advise regular echocardiographic follow up of the patients with S.C.D. for early discovered of cardiac abnormalities secondary to the disease. Also, further studies are advised for a recent modality in assessment of cardiomyopathy in sickle cell disease.

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1. التغيرات المرضية بالأشعة التلفزيونية علي القلب في الأطفال المصابين بمرض الخلايا المنجلية

مقدمة الدراسة:
مرض الخلايا المنجلية من الأمراض الوراثية الناتجة عن وجود هيموجلوبين "س" غير طبيعي في كرات الدم الحمراء والذي يسبب الكثير من المشاكل خاصة علي الجهاز الدوري و التنفسي نتيجة فقر الدم المزمن وتكرار حدوث جلطات في الشرايين الرئوية الصغيرة وترسيب بقايا الحديد في عضلة القلب.
الهدف من الدراسة:
تهدف الدراسة الي تقييم التغيرات التشريحية و الوظيفية علي القلب بإستخدا م الاشعة التلفزيونية علي القلب في الأطفال المصابين بمرض الخلايا المنجلية .
خطة الدراسة:
تم دراسة 44 طفلا مصابين بمرض الخلايا المنجلية تتراوح أعمارهم من 5 الي 15 سنة 26 طفل و 18 طفلة وتم مقارنتهم بمجموعة ضابطة 32 طفل و طفلة لا يعانون من مرض الخلايا المنجلية. وقد تم الفحص الطبي الكامل لجميع الأطفال وعمل الفحوصات الآتية:
* التحاليل اللازمة وتشمل: (نسبة الهيموجلوبين – الفصل الكهربائى للهيموجلوبين - نسبة الفريتين في الدم).
* أشعة عادية علي الصدر.
* أشعة تلفزيونية علي القلب.
* تخطيط كهربائي للقلب.
نتائج الدراسة:
الأطفال المصابين بمرض الخلايا المنجلية يعانون من تضخم في عضلة القلب وإرتفاع في ضغط الشريان الرئوي.
التوصيات:
وتوصي الدراسة بوجوب العناية بالأطفال المصابين بمرض الخلايا المنجلية و الفحص الدوري لهم بالأشعة التلفزيونية والتدخل المبكر السريع لعلاج مضاعفات الخلايا المنجلية علي القلب.

*Saber A M El-Sayed. "Study of Cardiac Abnormalities in Pediatric Patients with Sickle Cell Disease." IOSR Journal of Dental and Medical Sciences (IOSR-JDMS) 16.8 (2017): 53-58