

Lipid profile in β -thalassemia major children and its correlation with various parameters

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ABSTRACT

Background: Beta-thalassemia is considered to be the most frequent hereditary blood disorder worldwide. Lipid abnormalities have been detected in different types of beta-thalassemia thus pre-disposing them to pre-mature atherosclerosis. **Objectives:** To study the lipid profile in beta-thalassemia major (β -TM) children and establish its co-relation with various parameters. **Materials and Methods:** A total of 100 diagnosed patients of β -TM age 1–18 years and 100 subjects in the control group matched for age and sex were enrolled. For detecting a minimum difference of at least 20 mg/dl of triglyceride between the two groups, the required sample size was 81 subjects in each group, which was further, enhanced and rounded off to 100 subjects in each group. Univariate and multivariate regression analysis was done to establish a correlation of lipid profile with various parameters. **Results:** Total cholesterol (TC) was lower in thalassemia patients as compared to controls 104.45 versus 117.97 ($p < 0.001$). Triglycerides (TG) values were higher in thalassemia patients as compared to controls 155.96 versus 73.95 ($p < 0.001$). Low high-density lipoprotein (HDL) cholesterol was observed in thalassemia patients as compared to controls 36.33 versus 43.85 ($p < 0.001$). No statistically significant difference was observed between low-density lipoprotein (LDL) values between patients and controls 55.89 versus 54.53 ($p < 0.817$). The pro-atherogenic TC: HDL ratio was significantly higher (3.21 ± 0.88 vs. 2.91 ± 1.06) in patients as compared to controls ($p < 0.001$). By multivariate regression analysis, we observed that there was a significant decrease in TC with advancing age ($p = 0.002$, $\beta = -0.283$) and with a decrease in hemoglobin ($p = 0.028$, $\beta = 0.425$) but TC was not related to hematocrit, gender, liver enzymes, serum bilirubin, chelating agents and serum ferritin. LDL cholesterol also had significant negative correlation with age ($p = 0.015$, $\beta = -0.240$) and positive correlation with hemoglobin ($p = 0.033$, $\beta = 0.211$). HDL cholesterol and TG were not related to age, gender, hemoglobin, hematocrit, liver enzymes, chelating agents, and serum ferritin. **Conclusion:** β -TM patients have hypertriglyceridemia and hypocholesterolemia. Thus, early detection of these abnormalities will help to prevent cardiovascular complications.

Key words: Beta thalassemia major, High-density lipoprotein, Low-density lipoprotein, Total cholesterol, Triglycerides

Beta-thalassemia is the most common single-gene disorder in the Indian population [1,2]. It is an autosomal recessive heterogeneous hemolytic blood disorder, as a result of inadequate beta globin chain synthesis. Decreased production of one or more globin chains is due to different globin gene mutations resulting in ineffective hematopoiesis, increased hemolysis, and early-onset anemia [3]. It has a high prevalence in the Indian subcontinent, Middle East, Central Asia, and Mediterranean countries [4,5]. In India, 3.9% are beta thalassemia carrier and 16,200 are expected beta thalassemia syndromes per year out of 1270 million population [6]. About 10% of the world's total thalassemics are born in India every year.

The mainstay of treatment of thalassemia is regular blood transfusions. The major complications of blood transfusion are those related to the transmission of infectious agents or the development of iron overload [7]. Various endocrine, cardiac, and hepatic diseases may occur depending on excessive iron-loading. With the increasing average age and because of promptly managed siderotic complications, by use of chelating agents, now various non-siderotic complications are being increasingly recognized.

Recent literature shows strong evidence that children with thalassemia Major (TM) are at risk of developing subclinical atherosclerosis [8]. Studies have also revealed strong affiliation of dyslipidemia with premature atherosclerosis as an emerging complication in these patients. Subclinical atherosclerosis begins early in life and may evolve into coronary heart disease

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later [9,10]. Free radical production is increased in patients with iron overload. TM patients are subject to per-oxidative tissue injury [11]. Variants of lipoproteins, cholesterol, and triglycerides (TG) in TM patients show marked oxidative modification due to free radical injury that could represent an event leading to pathogenesis [12]. Hypertriglyceridemia along with cardiac iron overload is responsible for many cardiovascular complications seen in these patients. In India, there is not enough evidence regarding this, so we conducted this study.

MATERIALS AND METHODS

The study was a single center tertiary hospital-based observational study, carried out in the Thalassemia unit of SMS Medical College Jaipur. A total of 100 diagnosed patients of TM aged 1–18 years and 100 healthy controls matched for age and gender from the pediatric outpatient department were included in the study. The sample size was calculated at 90% study power, alpha error 0.05 assuming a standard deviation (SD) of 39 mg/dl of triglyceride between patient and control. $p < 0.05$ was considered as significant. For detecting a minimum difference of at least 20 mg/dl of triglyceride between the two groups, the required sample size was 81 subjects in each group, which was further, enhanced and rounded off to 100 subjects in each group.

All TM children who were transfusion dependent at the rate of once or twice a month and were on regular iron chelation agents such as deferasirox, deferiprone, or both agents for at least 1 year were included in the study. Those children having any previous cardiac event, on lipid-lowering medications, hypothyroidism, renal failure, and diabetes mellitus were excluded from the study. Informed consent was taken from all study participants, both patients and controls. Approval of the Institutional Ethics Committee was obtained for the study.

TM children who came for blood transfusion were asked to come on empty stomach next time. On the next visit, blood was obtained for complete blood count, lipid profile such as total cholesterol (TC), high-density lipoprotein (HDL) cholesterol, TG, low-density lipoprotein (LDL) cholesterol, liver enzymes, bilirubin, and serum ferritin.

Categorical variables were presented in number and percentage (%) and continuous variables were presented as mean \pm SD and median. The normality of data was tested by the Kolmogorov–Smirnov test. If the normality was rejected, then non-parametric test was used. Quantitative variables were compared using the independent t-test/Mann–Whitney test (when the data sets were not normally distributed) between the two groups. Qualitative variables were compared using the Chi-square test. Univariate and multivariate linear regression was used to find the significant factors affecting CL, LDL, triglyceride, and HDL. The $p < 0.05$ was considered statistically significant. The data were entered in MS EXCEL spreadsheet and analysis was done using Statistical Package for the Social Sciences version 21.0.

RESULTS

Out of 100 cases in our study, 62 were male and 38 were female as compared to 61 males and 39 females in the control group. The mean age of cases was 7.74 ± 4.02 years and the mean age of controls was 7.26 ± 4.05 years, as shown in Table 1.

The hemoglobin values were significantly lower in cases (9.21 ± 1.98 mg/dl) as compared to controls (10.55 ± 1.3 mg/dl), ($p < 0.001$). The mean hematocrit values in cases and controls were found to be 28.19 ± 5.82 and 33.86 ± 4.46 , respectively. Aspartate transaminase (AST), alanine aminotransferase (ALT), and serum bilirubin values were significantly higher in patients as compared to controls, as shown in Table 2.

After comparing lipid profile between cases and controls, TC and HDL were significantly low in cases as compared to controls while serum TG and TC: HDL ratio were significantly higher in cases as compared to controls. No significant difference was observed between the mean values of LDL between cases and controls ($p = 0.817$), as shown in Table 3. On univariate regression analysis of TC, we observed that it is related to age, hemoglobin, hematocrit, AST, and gender but in multivariate regression analysis, it had significant negative correlation only with age ($\beta = -1.809$, $p = 0.002$) and positive relation to hemoglobin ($\beta = 5.517$, $p = 0.028$) but not to gender, liver enzymes, serum ferritin, and chelating agents. In multivariate regression analysis, LDL also had significant negative co-relation to age ($p = 0.015$, $\beta = -0.906$) and positive relation with hemoglobin ($p = 0.033$, $\beta = 1.594$) but was not related to gender, hematocrit, liver enzymes, serum bilirubin, chelating agents, and serum ferritin (Table 4).

In univariate regression analysis, TG were found to be significantly related to total bilirubin and ferritin but in multivariate regression analysis, TG did not correlate with age, gender, hemoglobin, hematocrit, liver enzymes, serum bilirubin, chelating agents, and serum ferritin. HDL cholesterol also did not correlate with age, gender, hemoglobin, hematocrit, liver enzymes, serum bilirubin, chelating agents, and serum ferritin, as shown in Table 5.

DISCUSSION

In our study, we observed that β -thalassemia patients had lower TC, low HDL, high TG, and high TC: HDL ratio as compared to control subjects. No significant difference was observed between

Table 1: Comparison of demographic characteristics between cases and controls

Demographic profile	Case (n=100)	Control (n=100)	p-value
Age (in years)			
Mean \pm SD	7.74 \pm 4.03	7.26 \pm 4.05	0.37
Median (IQR)	7 (4–10)	6.5 (4–10)	
Range	1–18	1–16	
Gender			
Female	38 (38%)	39 (39%)	0.88
Male	62 (62%)	61 (61%)	

IQR: Interquartile range

Table 2: Comparison of laboratory parameters between cases and controls

Laboratory parameters	Case (n=100)	Control (n=100)	p-value
Hemoglobin (g/dl)			
Mean±SD	9.21±1.98	10.55±1.3	<0.001
Median (IQR)	9 (8.200–10.100)	10.45 (9.950–11.300)	
Range	4–18	7.2–14.1	
Hematocrit			
Mean±SD	28.19±5.82	33.86±4.46	<0.001
Median (IQR)	28.05 (25.60–30.400)	33.5 (32–34.900)	
Range	12.1–52.9	22.4–54	
ALT			
Mean±SD	58.68±44.81	30.99±19.47	<0.001
Median (IQR)	43 (28–71)	28 (19–41.500)	
Range	15–212	10–186	
AST			
Mean±SD	59.07±32.92	42.41±24.1	<0.001
Median (IQR)	48.5 (36.500–67)	40 (27–50)	
Range	21–185	15–189	
Total serum bilirubin			
Mean±SD	1.23±0.84	0.47±0.35	<0.001
Median (IQR)	0.98 (0.695–1.530)	0.43 (0.330–0.525)	
Range	0.28–5.33	0.17–3.4	

IQR: Interquartile range; AST: Aspartate transaminase; ALT: Alanine aminotransferase

Table 3: Comparison of lipid profile between cases and controls

Lipid profile	Case (n=100)	Control (n=100)	p-value
TC (mg/dl)			
Mean±SD	104.45±25.69	117.97±27.95	<0.001
Median(IQR)	101.5 (85.500–120)	116 (96.500–136.500)	
Range	51–187	52–199	
TG (mg/dl)			
Mean±SD	155.96±58.04	73.95±22.62	<0.001
Median(IQR)	141.5 (116–185.500)	70.5 (60–92)	
Range	60–323	24–124	
HDL cholesterol (mg/dl)			
Mean±SD	36.33±23.44	43.85±13.8	<0.001
Median(IQR)	33 (27–40)	42 (35–55)	
Range	16–233	18–80	
LDL cholesterol (mg/dl)			
Mean±SD	55.89±15.09	55.89±15.09	0.81
Median(IQR)	55 (47.750–62)	56 (40–69)	
Range	22–112	11–194	
TC: HDL ratio			
Mean±SD	3.21±0.88	2.91±1.06	<0.001
Median(IQR)	3 (2.662–3.945)	2.64 (2.137–3.389)	
Range	0.52–5.22	1.29–7.39	

TC: Total cholesterol; IQR: Interquartile range; HDL: High-density lipoprotein; LDL: Low-density lipoprotein; TG: Triglycerides

the LDL values between the two groups. Vefic Arica *et al.* [11] in Turkey found lower TC, HDL, and LDL values, and high level of TG as compared to control subjects. These results were in agreement with the studies conducted by Shareif *et al.* [13] in Egypt and Mashalli *et al.* [14] in Iraq.

Liver is the earliest site of iron deposition in regularly transfused TM patients and a common cause of morbidity. Iron overload occurs both in hepatocytes and reticuloendothelial cells. This iron overload leads to increased free radical production in these patients through Fenton's reaction. These free radicals

Table 4: Univariate and multivariate linear regression to find the significant factors affecting TOTAL CL and LDL

Variables	Total CL				LDL			
	Univariate		Multivariate		Univariate		Multivariate	
	Beta coefficient (95% CI)	p-value	Beta coefficient (95% CI)	p-value	Beta coefficient (95% CI)	p-value	Beta coefficient (95% CI)	p-value
Age	-2.212 (-3.412-1.011)	<0.01	-1.809 (-2.959-0.658)	0.002	-1.044 (-1.784-0.305)	0.006	-0.906 (-1.634-0.179)	0.015
HB	4.284 (1.829-6.74)	<0.001	5.517 (0.606-10.427)	0.028	1.932 (0.446-3.417)	0.01	1.594 (0.131-3.058)	0.033
HCT	1.151 (0.297-2.004)	0.00	-0.746 (-2.409-0.918)	0.376	0.427 (-0.087-0.94)	0.10	-	-
AST	-0.175 (-0.328-0.023)	0.02	-0.101 (-0.243-0.041)	0.162	-0.028 (-0.122-0.065)	0.54	-	-
ALT	-0.092 (-0.206-0.021)	0.10	-	-	-0.01 (-0.078-0.058)	0.77	-	-
SBrT	-3.105 (-9.23-3.02)	0.3	-	-	-3.178 (-6.83-0.47)	0.08	-	-
SBrD	-20.96 (-43.12-1.20)	0.06	-	-	-5.87 (-19.51-7.76)	0.39	-	-
Gender	-13.96 (-24.13-3.78)	0.00	-7.93 (17.57-1.69)	.10	-6.38 (-12.54-0.227)	0.04	-4.03 (-10.08-2.0)	.10
CHT								
Deferasirox	-11.67 (-36.73-13.39)	0.35	-	-	-5.86 (-20.13-8.399)	0.41	-	-
Deferiprone	-6.6 (-27.79-14.59)	0.49	-	-	-4.5 (-12.66-3.66)	0.23	-	-
Both	-5.903 (-13.374-1.569)	0.11	-	-	-3.09 (-8.76-2.58)	0.26	-	-
Ferritin	9.72 (-9.05-28.51)	0.30	-	-	2.64 (-9.15-14.45)	0.65	-	-

HCT: Hematocrit; HB: Hemoglobin; AST: Aspartate transaminase; ALT: Alanine aminotransferase; LDL: Low-density lipoprotein

Table 5: Univariate and multivariate linear regression to find the significant factors affecting TG and HDL cholesterol

Variables	TG				HDL cholesterol	
	Univariate		Multivariate		Univariate	
	Beta coefficient (95% CI)	p-value	Beta coefficient (95% CI)	p-value	Beta coefficient (95% CI)	p-value
Age	-1.817 (-4.686-1.051)	0.22	-	-	-0.311 (-1.504-0.882)	0.60
HB	0.621 (-5.255-6.497)	0.83	-	-	1.896 (-0.459-4.251)	0.11
HCT	0.799 (-1.192-2.791)	0.42	-	-	0.403 (-0.402-1.207)	0.32
AST	-0.151 (-0.503-0.201)	0.39	-	-	-0.065 (-0.21-0.08)	0.37
ALT	-0.216 (-0.472-0.04)	0.09	-	-	-0.054 (-0.159-0.051)	0.30
SBrT	-13.766 (-27.405--0.127)	0.04	-12.18 (-25.722-1.362)	0.07	1.312 (-4.442-7.066)	0.65
SBrD	-41.049 (-91.35-9.252)	0.10	-	-	-11.75 (-32.878-9.377)	0.27
Gender	-10.718 (-34.469-13.033)	0.37	-	-	2.237 (-7.53-12.005)	0.65
CHT						
Deferasirox	36.816 (-16.828-90.46)	0.17	-	-	-1.558 (-23.9-20.784)	0.89
Deferiprone	11 (-8.841-30.841)	0.23	-	-	-2.2 (-8.574-4.174)	0.44
Both	15.159 (-6.389-36.707)	0.15	-	-	1.109 (-6.592-8.81)	0.76
Ferritin	45.473 (3.79-87.156)	0.03	41.05 (-0.474-82.573)	0.05	-3.186 (-21.53-15.158)	0.73

HCT: Hematocrit; HB: Hemoglobin; AST: Aspartate transaminase; ALT: Alanine aminotransferase; HDL: High-density lipoprotein; TG: Triglycerides

then accumulate in the heart, liver, and other organs and cause extensive tissue damage [15].

The underlying mechanism of hypocholesterolemia in thalassemia includes decreased production secondary to liver injury, increased consumption due to amplified erythropoiesis, and an augmented uptake of cholesterol by histiocytes [16,17]. By multivariate regression analysis, we found that TC had a significant negative correlation with age and positive relation to hemoglobin but not related to gender, liver enzymes, serum ferritin, and chelating agents. This decrease in cholesterol values with age can be explained by increasing liver damage by iron deposition with advancing age. Hartman *et al.* [18] in their study found that TC correlated with diagnosis (beta-thalassemia major [β -TM] or intermedia) but not age, sex, hemoglobin, or ferritin levels. This difference may be explained by a small number of subjects in their study and racial and ethnic differences. Sedigheh *et al.* [19] in their study found that TC had a correlation with TG, ALT, and iron but no correlation was seen between TC and age, glucose, insulin, and ferritin. In their study, 5.1% of patients had diabetes mellitus in contrast to our study where none of the cases had diabetes mellitus.

Hypertriglyceridemia was observed in TM patients as compared to controls. The proposed mechanism for hypertriglyceridemia is due to a reduction in lipolytic activity [16]. This is because triglyceride lipase enzyme activities both hepatic and extrahepatic were found to be significantly lower in β -TM patients [16]. Through multivariate regression analysis, we further observed that TGs did not correlate with age, gender, liver enzymes, serum ferritin, and chelating agents. Papanastasiou *et al.* [20] observed a positive correlation between triglycerides and age, triglycerides and ferritin. This difference maybe because of the differences in mean age group between the two studies (10.2 ± 3.5 vs. 7.74 ± 4.03); as well as racial and ethnic differences. Mahmoud *et al.* [21] also observed a positive correlation between TG and frequency of blood transfusion ($r=0.4$, $p=0.006$) and serum ferritin ($r=0.4$, $p=0.020$) but their study included a small number of subjects as compared to our study. While Khaleel *et al.* [22] and Amal *et al.* [23] found no correlation between serum ferritin and TG similar to our study.

HDL and LDL cholesterol are important parameters of the lipid profile. Various studies have established that the risk for ischemic myocardial infarction is high with low HDL levels regardless of normal TC levels [17]. Low HDL is considered as a predictive factor of cardiovascular risk stratification in patients with β -TM patients. We found low HDL cholesterol in TM patients as compared to controls but no significant difference between LDL values between patient and controls. Through regression analysis, we found that HDL had no significant correlation with age, gender, hemoglobin, hematocrit, liver enzymes, chelating agents, and serum ferritin while LDL had a significant negative correlation with age and positive relation with hemoglobin. Amal *et al.* [23] observed a significant negative correlation between HDL and serum ferritin and a positive significant correlation between serum ferritin and LDL. This difference might be because their study included only 64 thalassemia patients and included both thalassemia major and intermedia patients.

The pro-atherogenic ratio, TC: HDL was significantly higher in our thalassemic patients as compared to controls. This is in concordance with the study conducted by Ferdaus *et al.* [24]. This ratio also predicts the risk of coronary heart disease regardless of the LDL and HDL levels. Limitation of this study is that a larger sample size is required for the better understanding and establishment of this cause-effect relation of lipid profile in patients with β -TM.

Thus, β -TM patients have low TC and HDL and a high level of TGs and TC/HDL ratio. Early detection of these patients with a deranged lipid profile is required to avoid thrombotic and atherogenic complications. Patient with deranged lipid profile should undergo dietary and lifestyle modifications and maybe start on lipid-lowering agents such as statins or other agents if not controlled by dietary or lifestyle modifications.

CONCLUSION

From our study, we can conclude that children suffering from β -TM have dyslipidemia and may possess a premature increased risk of atherosclerotic cardiovascular disease. Hence, a blood lipid profile should be carried out regularly in patients with β -TM.

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