Correlation of Serum Ferritin with Lipid Profile in **B-Thalassemia** Patients

Salunkhe Vaishnavi Sandip, Rai Pooja SK, Ingale Pramod W

Abstract

Background: The present study was aimed to study alterations in lipid levels and its corelation with ferritin in Thalassemia patients. **Methods:** 50 patients of diagnosed Thalassemia in age group of 9 to 12 years were included in the study and results were compared with controls. Lipid profile and serum ferritin levels were estimated. **Results:** Significant (<0.05) decreased levels of Cholesterol, HDL-cholesterol, LDL- cholesterol, VLDL-cholesterol were found as compared to controls. Serum triglyceride & ferritin levels were significantly (<0.05) increased as compared to controls. **Conclusion:** The positive correlation between serum ferritin and Cholesterol, HDL, LDL, VLDL& negative correlation between serum ferritin & TG levels which may attribute to the future fatal cardiac complication. Thus, lipid profile monitoring as well as ferritin level are necessary to prevent or at least enhance the early detection of cardiac complications. So,ferritin can be used as a marker for prevention of cardiac complication.

Key Words

Ferritin, Lipid profile, Thalassemia

Introduction

Beta-thalassemia syndromes are a group of hereditary blood disorders characterized by reduced or absent beta globin chain synthesis, resulting in reduced Hb in red blood cells (RBC), decreased RBC production and anaemia. Beta-thalassemias are caused by point mutations or, more rarely, deletions in the beta globin gene on chromosome 11, leading to reduced (beta+) or absent (beta0) synthesis of the beta chains of haemoglobin (Hb).Most thalassemia's are inherited as recessive traits.^[1]

Three main forms have been described: thalassemia major, thalassemia intermedia and thalassemia minor. Individuals with thalassemia major usually present within the first two years of life with severe anaemia, requiring regular red blood cell (RBC) transfusions.

Department of Biochemistry, Lokmanya Tilak Municipal Medical College and General Hospital, Sion, Mumbai -22, India.

Correspondence to: Dr. Rai Pooja SK, Room No. 306 B, 3rd Floor College Building, Department of Biochemistry, Lokmanya Tilak Municipal Medical College and General Hospital, Sion, Mumbai -22, India. Manuscript Received: 21.01.2023; Revision Accepted: 30.03.2023; Published Online First: 10 January, 2024. Open Access at: https://journal.jkscience.org Beta-thalassemia is prevalent in Mediterranean countries, the Middle East, Central Asia, India, SouthernChina, and the Far East as well as countries along the north coast of Africa and in South America. The highest carrier frequency is reported in Cyprus (14%), Sardinia (10.3%), and Southeast Asia.

The total annual incidence of symptomatic individuals is estimated at 1 in 100,000 throughout the world and 1 in 10,000 people in the European Union.^[2]

Almost 25 million people in India are carriers of âthalassemia gene. The prevalence of the â-thalassemia gene in India in different regions varies between 1-17% with a mean prevalence of about 3.3%. â-thalassemia has a high frequency in certain communities such as

Copyright: © 2024 JK Science. This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 International License, which allows others to remix, transform, and build upon the work, and to copy and redistribute the material in any medium or format non-commercially, provided the original author(s) and source are credited and the new creations are distributed under the same license.

Cite this article as: Sandip SV, Rai Pooja SK, Ingale Pramod W. Correlation of serum ferritin with lipid profile in β -thalassemia patients. JK Science 2024;26(1):16-9.

Punjabis who have migrated from Gujaratis, Bhanushalis, Gujarati Khojas and Jains, West Pakistan, Lohanas, Sindhis, Bengalis.^[3]

In thalassemia major, individual is unable to make enough healthy haemoglobin and depends on frequent blood transfusions throughout the life. Frequent blood transfusions cause progressive iron overload which is the major complication of treatment. Thalassemia is a secondary iron overload condition. Iron overload is because of both due to increased absorption of iron from gut and from frequent blood transfusions.^[4]

In regularly transfused children, liver is the earliest site of iron overload and also common cause of morbidity. Iron overload occurs both in hepatocytes and reticuloendothelial cells which induces liver causing development of fibrosis and eventually cirrhosis. Liver damage in thalassemia major accounts for low serum levels of total cholesterol, low density lipoproteins (LDL) and high-density lipoproteins (HDL).^[5]

Liver iron is gold standard for determining body iron, though T2 MRI is best non-invasive method of determining liver iron.^[6] Derangement of lipid profile can be taken as indirect evidence of iron overload in liver so this study was planned to investigate the lipid pattern in thalassemia major children and to correlate their levels with serum ferritin.

Material & Methods

The study was carried out between December 2021to May 2022 on 50 samples in clinical Biochemistry department. The following parameters were analysed in Biochemistrylaboratory using fully automated biochemistry analyzer XL- 640 of Erba Manheim manufactured by Transasia Biomedicals Ltd.

The age group 9-12 with thalassemia syndrome were randomly selected from the patients attending the thalassemia clinic of the same Hospital. Clinical history and relevant data were collected from patient's files with prior permission of the attending physician. The age and sex matched 50 controls who were neither thalassemic trait nor carrier. None had any history of blood transfusion, anaemia, infection and any acute or chronic disease state.Blood samples were obtained from the study subjects after 12 hours of overnight fasting.

Data analysis was performed using SPSS statistical analysis software (SPSS version 17.0, Chicago IL, USA). Statistically significant difference was determined by theUnpaired T test. All P values are 2-sided, with values less than 0.05 considered significant.

Results

Parameters	Control (50)	Cases (50)	p value
Ferritin (μg/L)	57 ± 41	2412.88 ± 37.20	< 0.05
Cholesterol (mg/dl)	189.48± 1.6	122.55 ± 0.54	< 0.05
TG (mg/dl)	141.71 ± 0.7	232.26 ± 2.45	< 0.05
HDL-CH (mg/dl)	112.14± 0.5	27.36±1.57	< 0.05
LDL-CH (mg/dl)	119.88 ± 0.4	71.59±1.71	<0.05
VLDL-CH (mg/dl)	20.11 ± 0.3	13.11± 1.05	<0.05

Table 1: Biochemical Parameters in Thalassemia Patients

Table 1 shows a significantly (p < 0.05) increased ferritin levels in the Thalassemia patients ($2412.88 \pm 37.20 \text{ ig/L}$) as compared to the healthy controls ($57 \pm 41 \text{ ig/L}$). We also observed significantly (p < 0.05) decreased levels of cholesterol in the Thalassemia patients ($122.55 \pm 0.54 \text{ mg/d}$) as compared to the healthy individuals ($189.48 \pm 1.6 \text{ mg/d}$).

There were also a significant (p < 0.05) increase levels of Triglyceride in the Thalassemia patients (232.26 ± 2.45 mg/dl) as compared to the healthy individuals (141.71 ± 0.7). We also observed significantly (p < 0.05) decreased levels of HDL-cholesterol, LDL-cholesterol, VLDL-cholesterol in the Thalassemia patients (27.36 ± 1.57 mg/dl) (71.59 ± 1.71 mg/dl) (13.11 ± 1.05 mg/dl) as compared to the healthy individuals (112.14 ± 0.5 mg/dl) (119.88 ± 0.4 mg/dl) (20.11 ± 0.3 mg/dl) respectively.

Table 2 shows ferritin levels are negatively corelated with cholesterol, HDL-CH, LDL-CH, VLDL-CH while

Table 2: Co-relation Coefficient of ferritin with lipid profile (r-value)

Parameters	Cholesterol	TG	HDL-CH	LDL-CH	VLDL-CH
	(mg/dl)	(mg/dl)	(mg/dl)	(mg/dl)	(mg/dl)
Ferritin (µg/L) (Male)	-0.14	0.29	-0.15	-0.04	-0.19

Vol. 26 No. 1, January - March 2024



Fig.1 Showing Corelation of Ferritin with cholesterol

positively corelated with TG in the Thalassemia patients. **Discussion**

In the present study, the Thalassemiapatients were screenedfor serum Ferritin and serum lipid profile.

The main modality of treatment in beta thalassemia major is regular blood transfusion every 2–4 weekly coupled with iron chelation. Withouteffective iron chelation therapy, patients with transfusional iron overload are at risk of iron deposition in vital organs such as the liver and heart.^[7]

In our study all cases had highly significant increase in serum ferritin levels compared to the healthy controls (p<0.05). Ferritinemia was about twenty times more in patients as compared to controls. This increase in serum ferritin indicates an existing iron overload in cases, due to multiple blood transfusions and probably also due to intestinal hyperabsorption of iron.^[8]

In our study it was found that the majority of the cases had lower total cholesterol, HDL-C as well as LDL-C levels, and higher triglycerides levels in cases as compared to controls. The pathogenesis of these abnormalities can be caused by many mechanisms including plasma dilution because of anemia, accelerated erythropoiesis resulting in increased cholesterol uptake by macrophages and histiocytes of the reticuloendothelial system, defective liver functioning because of iron overload, macrophage system activation with cytokine release, and hormonal disturbances.^[9-11]

In the present study, we investigated the lipid pattern in blood of children with â Thalassemia major. It was observed that cholesterol, HDL cholesterol, LDL cholesterol & VLDL cholesterol levels were low and triglyceride level were high in cases with serum ferritin levels were >2000 ng/ml.

There was negative correlation of total cholesterol, HDL, LDL, VLDL with serum ferritin with coefficient of correlation (r=-0.14, -0.15, -0.04,-0.19 respectively) while



Fig.2 Showing Corelation of Ferritin with Triglyceride

Serum triglyceride had positive correlation with serum ferritin (coefficient of correlation r=+0.29).

Mild erythroid hyperplasia also leads to increase removal of LDL by bone marrow. Accelerated erythropoiesis and increased uptake of LDL by macrophages and histiocytosis of reticuloendothelial system are main determinants of low plasma cholesterol levels. Low total cholesterol is caused by hepatic damage and iron overload. Since these changes are signs of lipid peroxidation, the cause of phenomenon was studied.^[12] Papanastasiou et al, had shown that total cholesterol, HDL and LDL cholesterol were significantly decreased while triglycerides were significantly increased in casesas compared to controls.^[13]

Kaltwassen et al noted that increase in triglyceride was observed with increasing ferritin values and positive correlation of patient's triglyceride.^[14]Kamal *et al* noted from a study conducted in Jordon in 2008 that significantly low cholesterol, HDL, LDL were seen as compared to controls.^[15]Patne *et al* confirmed in their study that lipid abnormality occurs significantly in thalassemia children as compared to controls.^[16]Our study also supports the hypothesis given by previous authors Suman *et al* that positive correlation between serum ferritin and triglycerides and negative correlation between serum ferritin and cholesterol.^[17]

We found negative correlation between high serum ferritin and (TC), (LDL-C) which are the same result of a study ⁽¹⁸⁻²⁰⁾, while positive correlation between high ferritin and high (TG) similar to result of another study.^[19,20]

Conclusion

The present study concluded that the negative correlation between serum ferritin and Cholesterol, HDL, LDL, VLDL & positive correlation between serum ferritin & TG levels which may attribute to the future fatal cardiac complication. Thus, lipid profile monitoring as well as ferritin level are necessary to prevent or at least enhance the early detection of cardiac complications.

Financial Support and Sponsorship

Nil.

Conflicts of Interest

There are no conflicts of interest.

References

- Galanello R, Origa R. Beta-thalassemia. Orphanet J Rare Dis 2010; 5: 11.
- Flint J, Harding RM, Boyce AJ, Clegg JB. The population genetics of the hemoglobinopathies. Bailliere's Clinical Haematology 1998; 11:1-50.
- Verma I C, Choudhry V P, Jain PK. Prevention of thalassemia: A necessity in India. The Indian Journal of Paediatrics 1992; 59(6),: 649–54.
- Richa J, Sachdeva A. Iron overload and its manifestations. In Sachdeva A, Jain R, AggarwalRK, Yadav SP, Broker A. Manual of thalassemia IAP, Paediatric Hemato Oncology. 2009;76-115.
- Goldfarb A, Rachmilewitz A, Elsemberg S. Abnormal lowand high-density lipoproteins in homozygous â thalassemia an Italian multicentric study. Acta Hematol 1998;99:76-9.
- Hillenbra CMR, Loeffler B, Carville M. Evaluation on hepatic iron concentration by T2 MRI in patients with iron overload. Proc Intl Soc Mag Reson Med 2005;105:855-61.
- Bhatia P, Nagar V, Meena JS, Singh D, Pal DK. A study on the demographic andmorbidity patterns of thalassemia patients registered at a tertiary-care center of centralIndia. Int J Med Sci Public Health 2015;4:85-8
- 8. Choudhary M, Bohra VD. Iron status of thalassemic children in south Rajasthan.Int Jr SciRes 2015;4(9):380-1
- 9. Al-Quobaili FA, Abou Asali IE. Serum levels of lipids and lipoproteins in Syrian patients withbeta-thalassemia major. Saudi Med J 2004;25:871-5.
- Deiana L, Garuti R, Pes GM, Carru C, Errigo A, Rolleri M. Influence of beta â- thalassemia on the phenotypic expression of heterozygous familial hypercholesterolemia:a

study of patients with familial hypercholesterolemia from Sardinia. Arterioscler Thromb Vasc Biol 2000;20:236-43

- Shalev H, Kapelushnik J, Moser A, Knobler H, Tamary H. Hypocholesterolemia in chronic anemias with increased erythropoietic activity. Am J Hematol 2007;82:199-202.
- Scott MD, Eaton JW. Thalassemic erythrocytes. Cellular suicide arising from iron and glutathione dependent oxidation reaction. Br J Haematol 1995;91:811-9.
- 13. Papanastasiou. Expert panel on detection, evealuation and treatment of high blood cholesterol in adults (adult treatment panel III). JAMA 2001;285:2486-97.
- 14. Kaltwassen JP, Gottschalk. Erythropoietin and iron. Kidney International Supplements1999;69:49-56.
- 15. Mansi KM, Aburjaf TA. Lipid profile in Jordian children with â thalassemia major. UHOD.2008;18(2);93-8.
- Patne AB, Hisalkar PJ, Gaikwad SB. Lipid abnormalities in patients with beta thalassemia major. Int J Pharma Sci 2012;2(1):106-12.
- Suman RL, Sanadhya A, Meena P, Singh J, Jain R, Meena S. Lipid profile in children of â-thalassemia major and their correlation with serum ferritin. Int J Contemp Pediatr 2017;4:543-7.
- Daswani P, Garg K. Lipid profile in β-thalassemia major children and its correlation with various parameters. Indian J Child Health. 2021; 8(1):26-31.
- Eman Khammas ALSaadi. Disturbances of Lipid Profile, hemoglobin and Serum Ferritin Levels in Thalassemia Patients in Misan City, Amara, Iraq. J Med Chem Sci 2022; 5(5): 779-786
- Boudrahem-Addour N, Izem-Meziane M, Bouguerra K, Nadjem N, Zidani N, Belhani M, Djerdjouri B. Oxidative status and plasma lipid profile in β-thalassemia patients. Hemoglobin.2015;39(1):36-41.