# Correlation of liver enzymes and haematological profile in thalassemia major patients

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#### **Abstract**

Introduction: Although red cell transfusions are lifesavers for patients with thalassemia, they are responsible for a series of complications and expose the patients to a variety of risks. Thalassemia major is the most common genetic disease caused by defective gene which produces abnormal haemoglobin. Material and Methods: We have investigated 100 patients of Thalassemia coming to blood bank for regular blood transfusion. Level of hemoglobin, TLC,DLC and Liver enzymes i.e. SGOT, SGPT, Alkaline Phosphatase along with S. Bilirubin were also estimated. The age group of children suffering from Thalassemia major was between one year to fifteen years. Results: Serum Ferritin Level was 2251 ± 315 ng/ml(mean±S.D), Haemoglobin level was 7.82 ± 0.83 gm/dl,TLC was 7200 ± 1000 cells/cmm,DLC was 68 ± 12 Neutrophils,29 ± 08 Lymphocytes,02 ± 01 Eosinophils,01 ± 00Monocytes,00 ± 0.1 Basophils, SGOT (AST) value was 117 ± 35.5 IU/L and SGPT(ALT) concentration 151 ± 47.2 IU/L and Alkaline Phosphatase level was found to be 288 ± 20.80 IU/L with mean ± S.D. Bilirubin was 2.55 ±1.12 mg/dl (mean + S.D). Conclusion: In Present study Liver Enzymes increased and haemoglobin level was reduced but no changes were found in TLC and DLC.

Key words: Thalassemia Major, Liver Enzymes, Hemoglobin, Ferritin

#### Introduction

Hepatic dysfunction is a frequent manifestation in patients with Thalassemia and sickle cell anemia [1,2]. The thalassemias are the most common genetic disorder on a worldwide. The beta form of Thalassemia is particularly prevalent among Mediterranean people and this geographical association is responsible for its naming: Thalassa is the Greek word for sea and Haema, the Greek word for blood. The prevalence of Thalassemia carriers in the Indian population is 3-4%. Some ethnic groups like Sindhis, Kutchis, Lohanas, Punjabis, few Muslim groups and few tribal populations have higher prevalence (5-17%) [3, 4]. Thalassemia major is a genetic disorder characterized by reduction in the ability to produce adult hemoglobin. The incidence of Thalassemia is very high and approximately 30 million people affected worldwide with carrying

Manuscript received: 20<sup>th</sup> Oct 2015 Reviewed: 4<sup>th</sup> Nov 2015 Author Corrected: 14<sup>th</sup> Nov 2015 Accepted for Publication: 27<sup>th</sup> Nov 2015 defective gene which is responsible for abnormal production of hemoglobin continuous hemolysis due to splenomegaly, decreased level of hemoglobin [5]. Iron overload is the most important cause of mortality in patients with Thalassemia major. However total leucocyte cell count fall within normal limits. Differential leucocyte count also remain in normal limits. Serum Ferritin level is high. Multiple transfusion of blood induces hepatotoxicity due to iron-overloadcaused by hemoglobin destruction [6]. Liver enzyme SGOT (AST), SGPT(ALT), Alkaline phosphatase (ALP) increased manifolds [7,8]. In present study with view of these finding our aim was to investigate correlation between hematological parameters (Serum ferritin, hemoglobin,TLC and DLC) and hepatic enzymes (SGOT, SGPT, Alkaline phosphatase) along with bilirubin level.

Hepatic dysfunction is a multifactorial process. Posttransfusion viral hepatitis, cholelithiasis and massive

hemolysis are common etiologies causing hyperbilirubinemias.

The aim of the present work was to study the correlation of liver enzymes among thalassemia major patients in blood bank, S.R.G.Hospital, Jhalawar, Rajasthan.

## **Material and Methods**

The study was conducted in the department of pathology of S.R.G.Hospital and Jhalawar Medical College, Jhalawar, Rajasthan. One hundred patients (63 males and 37 females) between age group of 10 months

to 15 years coming to blood bank at S.R.G.Hospital for regular blood transfusion between period of March 2011 to April 2013 were taken for study. Patient's history (Clinical and Pathological) were recorded. Level of hemoglobin, Serum ferritin, TLC, DLC and SGOT, SGPT, ALP and bilirubin were evaluated by commercial kit [9] method and instrument used for serum ferritin [10] was Immuno-assay analyzer maglumi 1000 and hemoglobin by sahli's method, TLC-DLC by automatic cell counters. Liver enzymes (SGOT, SGPT, Alkaline phosphatase and bilirubin were estimated using Fully autoanalyzer MIUR500 ). Stastical analysis was done by using SPSS version 16.

#### Results

In our study mean serum ferritin value was increased up to 2251 ng/ml with S.D 315 ng/ml and level of hemoglobin was in the range of  $7.82 \pm 0.83$  gm/dl, TLC mean + S.D was  $7200\pm1000$  and DLC was Neutrophils  $68 \pm 12$  %, Lymphocytes  $29 \pm 08$  %, Eosinophils  $02 \pm 01$ , monocytes  $01 \pm 00$ , basophils  $00 \pm 01$ . The mean + S.D level of liver enzymes SGOT (AST) was  $117 \pm 35.5$  IU/L, SGPT (ALT) was  $151 \pm 47.2$  IU/L and Alkaline phosphatase value was found to be  $288 \pm 20.80$  IU/L. The concentration of bilirubin was  $2.55 \pm 1.20$  mg/dl (mean + S.D)

Table 1: Level of serum ferritin, Haemoglobin, TLC, DLC, SGPT, SGOT and ALP in Thalassemia major patients.

Parameters	Mean±S.D.
Haemoglobin	7.82 <u>+</u> 0.83 gm/dl
S.Ferritin	2251 <u>+</u> 315 ng/dL
TLC	7200 <u>+</u> 1000 cells/mm
DLC	N-68%,L-29,E-02%,M-01%,B-00%
S.Bilirubin	2.55 <u>+</u> 1.12 mg/dl
SGOT	117 <u>+</u> 35.5 IU/L
SGPT	151 <u>+</u> 47.2 IU/L
Alkaline Phosphatase	288 <u>+</u> 20.80 IU/L

## **Discussion**

Hemoglobin disorders present a significant health problem. The basic defect is reduced rate of globin chain synthesis. Thalassemia is classified according to globin chain that is deficient. Two major forms involve impaired production or stability of either alpha or beta peptide chains, causing alpha or beta Thalassemia.

The present study was designed to correlate the decrease of hemoglobin and rise of liver enzymes in thalassemia major patients coming regularly for blood transfusion at blood bank, S.R.G.Hospital, Jhalawar, Rajasthan.

Thalassemia is a genetic disorder of blood and it is also known as beta-thalassemia, cooley's anemia, meditteranean anemia and about 6.5 % of the world population are carrier of different inherited disorders of haemoglobin [11]. Changes in the Liver enzymes (SGPT, SGOT and Alkaline Phosphatase) along with serum bilirubin due to excessive destruction of RBCs and it is abnormal globin chain which is unable to protect RBC in oxidative stress and results in hepatic cell damage.

The value of Haemoglobin, TLC and DLC were examined by studying peripheral blood film examination and CBC count also was helpful in

diagnosis of thalassemia major. Peripheral blood film examination revealed marked hypochromic macrocytes and target cells. Polychromasia, nucleated RBCs, Basophil stippling and immature leucocytes were found. Haemoglobin level was reduced in our study in Thalassemia patients and serum ferritin level was increased due to iron overload. However an assessment of serum ferritin may underestimate the iron concentration in the liver independent of patients with thalassemia which is similar to reported to literature [12]. Serum ferritin was mainly increased due to iron overload by repeated blood transfusions. Every patient was transfused once every 2-3 months due lack of hemoglobin level. This increase of ferritin may cause pituitary dwarfism or diabetes later on and decrease life span of patients. Average life of beta thalassemic child was 12-14 yrs in our study. Total leucocyte count (TLC) in our study was within normal limit and differential leucocyte count (DLC) i.e. Polymorphs, Lymphocytes, Monocytes, Eosinophils and Basophils were also in normal limits which findings were similar to study done by other workers (Eastman et al 1996)

Hepatic dysfunction is a multifactorial process. Posttransfusional viral hepatitis, cholelithiasis and massive transfusion practises can cause hyperbilirubinemia [13].

# Conclusion

Majority of children suffering from Thalassemia major were having decreased haemoglobin level and raised Serum Ferritin, Serum Bilirubin, SGPT, SGOT & Serum Alkaline Phosphatase level and no effect on Total or differential Leucocyte Count.

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