Original Article

Evaluation of Serum Transaminases Levels In Transfused β-Thalssaemia Major Patients

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Summary:

Background: β-thalassemia major is a genetic disorder characterized by reduced rate of β-globin chain production. Clinically, β-thalassemia major is a severe, transfusion-dependant disorder; repeated blood transfusion will lead eventually to chronic liver disease.

Patients and Methods: One hundred patients ; 56 males and 44 females who were known cases with β-thalassemia major on regular blood transfusion, aged between 6 months and 18 years, were studied in a private pathology laboratory, between January 2002-January 2006.Blood was drawn to estimate serum glutamic pyruvate transaminase (SGPT) and serum glutamic oxaloacetic transaminase (SGOT) levels.

Results: Sixty-six patients (66%) had elevated SGPT and SGOT levels ranging between two and more than five fold increase than normal.Thirty-four patients (34%) had normal SGPT and SGOT values of less than 40 i.u. /L. The levels of SGPT and SGOT were significantly higher in splenectomised patients than nonsplenectomised.

Conclusion: Serum transaminases were elevated in (66%) of transfused patients with B-thalassemia major.

Fac Med Baghdad 2010; Vol. 52, No. 1
Received Aug. 2009
Accepted Oct. 2009

Keywords: Serum Transminases, Thalassemia Major, Transfusion-Dependant.

Introduction:

The thalassemias are a heterogeneous group of genetic disorders of hemoglobin synthesis, all of which result from a reduced rate of production of one or more of the globin chains of hemoglobin. Most thalassemias are inherited in a Mendelian recessive fashion (1). The thalassemia is considered the most common genetic disorder worldwide. It occurs with a particularly high frequency in a broad belt extending from the Mediterranean basin through the Middle East to parts of North and West Africa (2). In B-thalassemias, the B-globin chains are produced in reduced amount, and they are the most important types of thalassemia because they are so common and usually produce severe anemia in their homozygous states (1). Clinically, thalassemia major is a severe, transfusion-dependant disorder (1). Repeated transfusions over many years, in the absence of blood loss, will lead eventually to damage of various organs of the body, particularly the liver, mainly due to iron deposition and post-transfusion hepatitis(3).

Materials and Methods:

One hundred patients, 56 males and 44 females, who were known cases with homozygous β-thalassemia major on regular blood transfusions, aged between 6 months and 18 years with a mean age of 7.3 years, were studied in a private laboratory in Baghdad during their attendance to the laboratory for their various routine laboratory investigations, for the Period between January 2002 to January 2006. A control group of 50 healthy individuals; 29 males and 21 females (age and sex-matched) were included in this study.Every patient included in this study, accompanied by one or both of his parents, has been interrogated about the history of illness, the frequency of blood transfusions and about splenectomy. Four mls of venous blood was collected into sterile, plain plastic tubes to obtain serum for estimation of SGPT and SGOT levels.

Biochemical tests:Biochemical tests were done by using commercial kits (bioMeriux), and the readings were measured by spectrophotometer Optima SP-300(China). Estimation of SGPT and SGOT were done according to the Reitman and Frankel method as follows; Measurement of SGPT.

1-Mix 0.2 ml serum+1ml GPT substrate,and incubate at 37C for 30 minutes

2-Add 1ml of color reagent, mix and let stand for 20 minutes. At room temperature.

3-Add 10ml NaOH 0.4N mix and wait 5 minutes.

4-Read the results at wavelength 505 nm.

Measurement of GOT follows the same procedure except we added 1ml of GOT substrate, and incubate for one hour. Statistical analysis: statistical analysis was done using the statistical package for social science (SPSS) version 10 software. Regarding t-test, P-value <0.05 was considered to indicate statistically significant difference.

Results:

Fifty healthy individuals; 29 males and 21 females, were investigated to serve as a control group. All the individuals had normal SGPT and SGOT levels (<45 i.u. /L for SGPT and < 40 i.u. /L for SGOT).

Forty-three patients had been splenectomised and 57 patients were not. Serum transaminases levels concerned in this study are summarized in table (1). The significance of differences in serum transaminases levels concerned between splenectomised and non-splenectomised patients are

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shown in table (2). Sixty-six patients (66%) had abnormally elevated SGPT and SGOT levels ranging between two and more than five fold increase than normal. Thirty-four patients (34%) had normal SGPT and SGOT values of less than 40 i.u. /L. The effect of splenectomy on serum transasminases levels: A significant difference was found in the mean SGPT and SGOT levels between splenectomised and non-splenectomised patients. All of which were significantly increased among splenectomised patients.

Table (1): Summary of serum transaminases levels in transfused thalassemic patients

	Mean	S.D. ±	Range
SGPT (i.u./L)	78.0	64.0	10.0->300
SGOT (i.u./L)	93.0	79.0	6.0-300

Table (2):The significance of differences in serum transaminases levels between splenectomised and nonsplenectomised transfused thalassemia major patients

	Splenectomised patients		Non-splenectomised patients			
	Mean	S.D. ±	Mean	S.D. ±	t	p
SGPT (i.u./L)	92.0	65.0	66.0	61.0	2.23	< 0.05
SGOT (i.u./L)	121.0	95.0	70.0	55.0	3.7	<0.005

Discussion:

Sixty-six patients (66%) had elevated SGPT and SGOT levels ranging between two and more than five fold increase. These results most probably reflect chronic liver disease These findings were in agreement with previous studies on liver disease in multiple transfused thlassemic patients (4) (5). Since liver biopsy was not done in our study, we used increased transaminases values as a screening test, and this may have underestimated the frequency of liver abnormalities in our patients, because in one study it was shown that 3 out of 21 patients with normal liver chemistry were found to have chronic hepatitis proved by liver biopsy(5). The liver disease in our patients may be due to various causes, hemosiderin overload may provoke hepatocyte necrosis and fibrosis(6), multiple transfusion may have caused repeated episodes of hepatitis that tend to become chronic(4)(7). A significant higher value for SGPT and SGOT were found in the splenectomised patients than the nonsplenectomised ones. This may represent more liver damage in the splenectomised patients due to more iron deposition in hepatocytes with consequent necrosis and fibrosis. These findings had been documented by other workers who showed that after splenectomy, more iron derived from transfusions will be stored in the Kupffer cells in the liver(8). These cells will, therefore, become more rapidly overloaded, so that any additional iron will be added to the serum iron compartment, and larger amounts of iron will now be deposited in the hepatocytes leading to liver cell damage and eventually cirrhosis(9).

Conclusion:

Serum transaminases were elevated in (66%) of transfused patients with B- thalassemia major, these results most probably reflect chronic liver disease caused by post transfusion hepatitis and hemosidrin deposition in the liver. A significant higher value of serum transaminases were found in the splenectomised patients than the nonsplenectomised ones, this may represent more liver damage in the splenectomised patients due to more iron deposition in hepatocytes with consequent necrosis and fibrosis.

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