International Journal of Advanced Biotechnology and Research (IJBR)
ISSN 0976-2612, Online ISSN 2278–599X,
Vol-9, Issue-2, 2018, pp955-958
http://www.bipublication.com

Research Article

Study of IGT in children of β-thalassemia major

[1]Muhammad Javeed Ali, [2]Sara Sardar

and [3]Muhammad Yasiriqbal

[1]Medical OfficerDHQ Hospital Muzaffargarh
[2]Woman Medical OfficerRural Health Centre Dullewala, Bhakkar
[3]Medical OfficerBHU RasoolpurTehsile $hujabad

ABSTRACT

Objective: To determine thefrequency of impaired glucose tolerance in children with beta thalassemia major on regular blood transfusion.

Material and methods: Thisstudy wasconducted at DHQ Hospital Muzaffargarh from January 2017 to June 2017. Total 161 children having age from 3 years to 20 years with thalassemia major were selected for this study.

Results: Total, 53.42%(n=86) patients were between 3-10 years and 46.58%(n=75) were between 11-20 years, with mean age 10.55±4.90 years, 60.25%(n=97) were male and 39.75%(n=64) were females and frequency of impaired glucose tolerance reveals in 10.56%(n=17) patients.

Conclusion: We concluded that the frequency of impaired glucose tolerance in children with thalassemia major on regular blood transfusion is higher, however, some other studies are required to authenticate our findings.

Keywords: Children, Thalassemia major, blood transfusion, impaired glucose tolerance, frequency

INTRODUCTION:

The thalassemias are autosomal recessive conditions which affecting quantity of hemoglobin molecules within the red blood cells. Beta thalassemia arises out of the mutations in ß globin genes on chromosome 11.[1] The β-thalassemia is very common in Pakistan, India, Mediterranean region and middle east.[2]About 1.5 percent of world population is carrier.[3]

In Greece its carrier rate is 6% to 19%, in Cyprus 15% to 17%, Iran 4% to 5%, in Saudi Arabia 1% to 2% and in Pakistan 1% to 8%.[4-6]Children with thalassemia major need regular lifelong red blood cell (RBC) transfusions for their survival. By these patients, most of the problems encountered are actually the complications of the transfusion rather than disease complication itself and iron over load is one of them. Iron overload may lead to development of endocrine complications especially impaired glucose tolerance.[1] The frequency of impaired glucose tolerance (IGT)varies from 7.1-28.04% in children with thalassemia on regular blood transfusion.[7-9]

MATERIAL AND METHODS:

This was a cross sectional study conducted at DHQ Hospital Muzaffargarh from January 2017 to June 2017.Total 161 children older than 3 years and less than 20 years having thalassemia major and receiving more than 5 blood transfusions (75ml packed cells /kg) per year as an average and minimum 20 blood transfusions were recruited for this study. Children with hemolytic anemias other than beta thalassemia major were excluded from the study.

Thalassemia major: children having severe microcytic hypochromic anemia (Hb< 5gm %) with reduced adult hemoglobin and fetal hemoglobin greater than 50% of total hemoglobin

at the time of diagnosis. An approval was taken from institutional review committee and written informed consent was taken from attendant of every child. The oral glucose tolerance test (OGTT) was performed in morning after an overnight fast and at least 2 weeks after the last transfusion. The dosage of glucose for OGTT was 1.75g/kg up to the maximum 75g dissolved in 250ml of water. Blood samples were obtained at 0 and 120 minutes post glucose administration and send to laboratory for plasma glucose, serum ferritin and hepatitis B and C investigations. Impaired glucose tolerance: fasting plasma glucose <126mg/dl while 2–h plasma glucose ≥140mg/dl but < 200mg/dl. All the findings with demographic profile of the patients was entered in pre-designed proforma.

The collected data was analyzed by using SPSS version16. The frequency and percentage of impaired glucose tolerance was calculated. Stratification was performed to control effect modifier like age, gender, number of blood transfusion, serum ferritin level and hepatitis B and C status. The mean±sd was calculated for age of the patients, number of transfusions, height and weight.

Table No. 1 Frequency Of Impaired Glucose Tolerance (n=161)

Impaired Glucose Tolerance	No. of patients	%
Yes	17	10.56
No	144	89.44
Total	161	100

Table No. 2 Stratification For Age (n=17)

Age(in years)	No. of patients	%
3-10	11	64.71
11-20	6	35.29
Total	17	100

Table No. 3 Stratification For Gender (n=17)

Gender	No. of patients	%
Male	7	41.18
Female	10	58.82
Total	17	100

Table No. 4 Stratification For Hepatitis B & C (n=17)

Hepatitis	No. of patients	%
B	3	17.65
C	1	5.88

RESULTS:

Total 161 cases fulfilling the inclusion/exclusion criteria were enrolled. Mean age of children was 10.55±4.90 andmean weight & height was calculated which shows 41.5 ± 10.78 kg and mean height was calculated as 132.8 ± 16.34 cm.

Frequency of impaired glucose tolerance reveals in 10.56%(n=17) while 89.44%(n=144) had no findings of the morbidity. (Table No. 1)

Stratification for frequency of impaired glucose tolerance in relation to age was done.Out of 17 cases of impaired glucose tolerance 64.71%(n=11) were between 3-10 years and 35.29%(n=6) were between 11-20 years. (Table No. 2)

Stratification for frequency of impaired glucose tolerance in relation to gender was done. Out of 17 cases of impaired glucose tolerance 41.18%(n=7) children were male and 58.82%(n=10) children were females. (Table No. 3)

Stratification for frequency of impaired glucose tolerance with regards to Hepatitis B&C was recorded which shows that out of 17 cases of impaired glucose tolerance 17.65%(n=3) had Hepatitis B, 5.88%(n=1) had Hepatitis C while 76.47%(n=13) had no hepatitis. (Table No. 4)

No hepatitis	13	76.47
Total	17	100

DISCUSSION:

β-thalassemia major is a pack red cell transfusion-dependent condition requiring lifelong blood transfusions for afflicted patients to stay alive. Patients suffering from this disease facesmany problems if the transfusion is inadequate, but at same time repeated blood transfusions are associated with hazards. One of the most frequent endocrine complication in thalassemia major patients is diabetic so that 25-50 % these patient have IGT test or diabetes.[10-11]

In our study, 53.42%(n=86) were between 3-10 years and 46.58%(n=75) were between 11-20 years, mean±sd was calculated 10.55±4.90 years, 60.25%(n=97) were male and 39.75%(n=64) were females and frequency of IGT reveals in

10.56%(n=17) while 89.44%(n=144) had no findings of the morbidity.

One study documented the frequency of IGT as 7.1% in children with thalassemia children on regular blood transfusion.[7] These findings are in favor of our study. Another study by Chern JPet al[9]reported the frequency of IGT as 8.5% which is also in agreement with our study.

Khalifa AS and others[12] concluded that abnormal glucose tolerance is very common in b thalassemic children receiving multiple blood transfusion children, which could be contributory factor to progressive and early loss of beta-cell mass, along with persistent insulin resistance. Viral infection like chronic hepatitis C may play a role inprogression of abnormal glucose tolerance.

Jaruratanasirikul S and co-workers[13] in a study concluded that the frequency of IGT in thalassemicchildren receiving hypertransfusions with suboptimal iron chelating therapy was 12.5%. The clinical characteristics of children with thalassemia who developed IGT were stunting, wasting, higher ferritin levels and lower AUC insulin.

The mechanism of abnormal glucose homeostasis with β-thalassemia major is still not clear but is attributed mainly to insulin deficiency because of iron deposition in pancreas[14-16] and secondly from insulin resistance[17]due to iron deposition in both liver and muscle. Glucose intolerance and overt diabetes[18]may be the final result ofpersistent insulin resistance along with a progressive decrease level of circulating insulin.

CONCLUSION:

We concluded that the frequency of impaired glucose tolerance in children with thalassemia major on regular blood transfusion is higher, however, some other studies are required to authenticate our findings.

REFERENCES:

1. Langlois S, Ford JC, Chitayat D, Désilets VA, Farrell SA, Geraghty M. Carrier screening for thalassemia and hemoglobinopathies in Canada. J ObstetGynaecol Can 2008;30(10):950-71.
2. Cousens NE, Gaff CF, Metcalfe SA, Delatycki MB. Carrier screening for Beta-thalassaemia: a review of international practice. Eur J Hum Genet 2010;18(10):1077–83.
3. Colah R, Gorakshakar A, Nadkarni A. Global burden, distribution and prevention of β-thalassemias and hemoglobin E disorders. Expert Rev Hematol 2010;3(1):103-17.
4. Weatherall DJ, Clegg JB. Inherited haemoglobin disorders: an increasing global health problem. Bull World Health Organ 2001;79(8):704–12.
5. Khattak MF, Saleem M. Prevalence of heterozygous beta-thalassemia in northern areas of Pakistan. J Pak Med Assoc 1992;42(2):32-4.
6. Khan F. Thalassemia: Still a challenge [editorial]. Gomal J Med Sci 2006;4(2):53-56.
7. Najafipour F, Aliasgarzadeh A, Aghamohamadzadeh N, Bahrami A, Mobasri M, Niafar M. A cross-sectional study of metabolic and endocrine complications in beta-thalassemia major. Ann Saudi Med. 2008;28(5):361-6.
8. Jaruratanasirikul S, Chareonmuang R, Wongcharnchailert M, Laosombat V, Sangsupavanich P, Leetanaporn K. Prevalence of impaired glucose metabolism in beta-thalassemic children receiving hypertransfusions with a suboptimal dosage of iron-chelating therapy. . Eur J Pediatr 2008;167(8):873-6.
9. Chern JP, Lin KH, Lu MY, Lin DT, Lin KS, Chen JD. Abnormal glucose tolerance in transfusion-dependent beta-thalassemic patients. Diabetes Care 2001;24(5):850-4.
10. Quirolo K, Vichinsky E. Hemoglobin disorder In: Behrman RE, Kliegman RM, Jenson HB. Nelson Text Book of pediatrics. 17P th P ed. Philadelphia: W.B. Saunders; 2004:1630- 4.
11. Weatheral DJ, Clegg JB. The thalassemia syndromes. 4PthP ed. London: Blackwell Science; 2001:302-5.

12. Khalifa AS, Salem M, Mounir E, El-Tawil MM, El-Sawy M, Abd Al-Aziz MM. Abnormal glucose tolerance in Egyptian beta-thalassemic patients: possible association with genotyping. Pediatr Diabetes. 2004 Sep;5(3):126-32.
13. Jaruratanasirikul S, Chareonmuang R, Wongcharnchailert M, Laosombat V, Sangsupavanich P, Leetanaporn K. Prevalence of impaired glucose metabolism in beta-thalassemic children receiving hypertransfusions with a suboptimal dosage of iron-chelating therapy. Eur J Pediatr. 2008;167(8):873-6.
14. Lassman MN, Genel M, Wise JK, Hendler R, Felig P. Carbohydrate homeostasis and pancreatic islet cell function in thalassemia. Ann Intern Med 1974;80:65–9.
15. Saudek CD, Hemm RM, Peterson CM. Abnormal glucose tolerance in β-thalassemia major. Metabolism 1977;26:43–52
16. Rahier JR, Loozen S, Goebbels RM, Abrahem M. The haemochromatic human pancreas: a quantitative immunohistochemical and ultrastructural study. Diabetologia 1987;30:5–12.
17. Kowdley KV, Kaplan MM. Iron-chelation therapy with oral deferiprone: toxicity or lack of efficacy? N Engl J Med 1988;339:468–9.
18. Cavallo-Perin P, Pacini G, Cerutti F, Bessone A, Condo C, Sacchetti L, Pica A, Pagano G. Insulin resistance and hyperinsulinemia in homozygous β-thalassemia. Metabolism 1985;44:281–6.