



The Influence Of Parathyroid Gland On Beta-Thalassaemia Patients in Kirkuk City

Demet F. Saber, Mossa M. Marbut , Sami A. Zbaar

Collage of Medicine , University of Tikrit , Tikrit , Iraq

DOI: <http://dx.doi.org/10.25130/tjps.24.2019.023>

ARTICLE INFO.

Article history:

-Received: 8 / 10 / 2018

-Accepted: 3 / 12 / 2018

-Available online: / / 2019

Keywords: Thalassaemia, PTH, Calcium, S.ferritin , ALP.

Corresponding Author:

Name: Demet F. Saber

E-mail:

demetfekert1990@gmail.com

Tel:

Introduction

become a hypoparathyroidism; an irreversible and preventable disorder caused by iron overload[4].

The aim of the study

The aim of this study was to show the role of parathyroid gland in beta-thalassaemia patients in kirkuk city.

Material and Methods

The study was conducted in thalassaemia center at Azadi Teaching Hospital in Kirkuk city from the 1st of November 2017 to the end of February 2018 .the study included (70) male thalassaemic patients and (30) normal healthy male, non thalassaemic, with no family history of thalassaemia,. The thalassaemic patients were interviewed and general information was taken from them to fill the questionnaire which including: Serial number, date, name, age, gender, consanguinity of parents age at diagnosis of thalassaemia,a number of blood transfusions per year, type of chelating therapy, All subjects were aged between (4-18) years. Blood was drawn through antecubital vein puncture between 8.00am and 11.00am by trained personnel. About 5 ml of blood was drawn from every patient. All these tubes were kept frozen up to (- 20 c°) until the time of estimation 1 ml of blood used at once for (PCV) and (Hb) measurements. The remaining 4 ml of blood placed in tube for serum separation for measurement of S. Calcium, ferritin, ALP and parathyroid hormone (PTH). Parathyroid hormone was measured by ST

ABSTRACT

Thalassaemia is an inherited blood disorder in which the body makes an abnormal form of hemoglobin. The symptoms of thalassaemia appear in the first two years of life and include paleness of the skin, anemia, bone abnormalities, weakness and growth retardations. (70) male thalassaemic patients are diagnosed in thalassaemia center at Azadi Teaching Hospital in Kirkuk city and(30) normal male healthy Controls were included in this study Anthropometric measures include, Age, BMI, were done for all participants. there is significant elevation, (P> 0.01) in PTH in P2 age group (9-13) years as compare with control healthy subjectsand as compare with P1(4-8) and P3(14-18) age group.

Beta Thalassaemia major (thalassaemia) is a heterogeneous inherited disorder of haemoglobin synthesis. There is ineffective erythropoiesis and anemia. Regular blood transfusions and chelation therapy has noticeably prolonged survival in thalassaemic patients, However they suffer growth, multiple endocrine and metabolic abnormalities[1]. Thalassaemia is usually treated by blood transfusion and iron chelation therapy to provide the patients with healthy red blood cells containing normal hemoglobin. However, repeated blood transfusions can lead to iron overload, where by excess iron accumulates in the body and is deposited in body organs such as the heart, liver and endocrine glands causing organ damage. the Patient also needs some complement medicines such as folic acid, calcium, vitamin K and many other medicines which depend on patient condition[2].

The parathyroid glands produce parathyroid hormone (PTH), PTH is a polypeptide hormone which is hidden by the chief cells of parathyroid glands. PTH is secret in response to low blood serum calcium (Ca²⁺) levels, Its main function is to increase the concentration of calcium in the blood plasma[3].

Repeated blood transfusion results in citrate toxicity and leads to iron deposition in the parathyroid gland, which in turn may cause hypoparathyroidism. Patients with beta-thalassaemia major are prone to

Patients with thalassemia are exposed to many situation abnormalities that contribute to stunted growth which may include mainly persistent of chronic anemia, iron overload due to multiple blood transfusion, splenomegaly and toxicity of chelating therapy [5]. Growth retardation may be also commonly reported in children and adolescents with Thalassaemia Major (TM) patients, the child with TM has a growth pattern, which was relatively normal until the age of (9-10) Years, after this age a slowing down of growth velocity and absent or reduction in the pubertal growth [6,7]. other study explained that frequent blood transfusions normally reestablish the normal growth spurt[8]. It has been also reported that growth delay sets in after the age of 4 years in boys and 3years in girls [9]. These studies show that patients of major thalassemia who are treated with frequent transfusions and chelation therapy, grow normally up to the age of (8-11) years, but there after show growth retardation most often coupled with delay in sexual maturation. Although the cause of short stature in children with thalassemia major is Still not well understood until now, it is believed to be multifactorial [10]. Many different factors including iron overload also are intensive use of iron chelators, gonadal damage may interact, making it difficult to understand each factor's relative contribution [11,12].

Calcium is a mineral that has an important role in any physiological processes of the body, which causes the contraction of striated muscles, smooth muscle and heart, blood clots and nerve impulse transmission. There is a disagreement between the current study and other studies is that the Ca level in TM patients is lower than that of healthy controls, and these results differ significantly (P=0.001) [13].

The Comparison of Serum Parathyroid hormone with Ferritin between male thalassaemia patients and male control subjects according to age groups

There is significant elevation in PTH with P2 age group (9-13) years, as compared with control healthy subjects (23.6 ± 11.6 pg/mL), and as compare with P1 and P3 age group, (P>0.05).

There is a significant increase (P≤0.01) in serum ferritin in p2 (2635.3 ± 782 ng/ml), and p3, (2817.6 ± 1072 ng/ml), groups as compared with P1 group, (1485.7 ± 651 ng/ml).

Table(1.3) The variation in Serum Parathyroid hormone and Ferritin between male thalassaemia patients and male control subjects according to age groups.

Parameters	PTH (pg/mL)	Ferritin (ng/ml)
TMpatients		
P1 (4 – 8)	25.69 ±14.32a	1485.7 ± 651a
P2 (9 – 13)	33.13±21.32b	2635.3 ± 782b
P3 (14 – 18)	24.87 ± 14.33a	2817.6 ± 1072b
Control subjects	23.6 ± 11.6	57.4 ± 9.2
P-Value	0.373	0.01

Parathyroid dysfunctions are thought to be a rare consequence of iron overload that show in beta-

AIA-PACK Intact PTH. serum Calcium concentration was measured by Spectrophotometer and use VIDAS for Serum Ferritin estimation by using the ELFA technique (Enzyme Linked Fluorescent Assay). Serum concentrations of calcium, alkaline phosphatase were measured by routine laboratory methods.

Statistical Analysis

All data were presented as a mean & standard deviation (S.D). F-test was used to compare between mean of variables (One way, ANOVA) and unpaired student T test was used to compare between means of different variables. P value less than 0.05 or 0.01 was used as significant value. P -value less than 0.05 was accepted as a significant value.

Results and Discussion

The distribution of patients according to parathyroid hormone results in thalassemia male patients; 9 thalassemia patients had hyperparathyroidism, (12.9%), while 18 patients had hypoparathyroidism, (25.7%), and the majority of patients had normal parathyroid hormone levels, (61.4%).

The Table (1.1) show the distribution of patients according to parathyroid hormone

PTH levels	Number of patients	Percent
Above	9	12.9%
Below	18	25.7
Normal	43	61.4
Total	70	100%

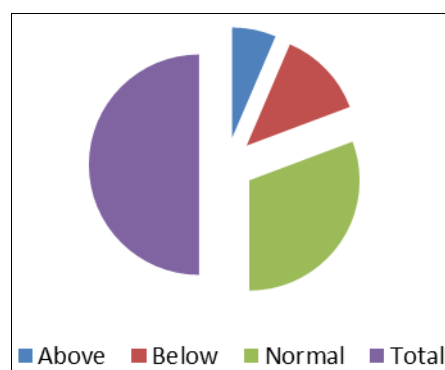


Figure 1 The distribution of patients according to parathyroid hormone

The Comparison of Age, BMI, Serum Parathyroid hormone and S. Calcium between male controls and male thalassemia patients .

Results of the present study revealed no-significant differences of age and PTH.Regarding BMI: There is a highly significant reduction (P≤0.01) in the body mass index between male controls (21.20±3.35) and thalassaemia male patients (16.57±2.11) .

Table (1.2) The Variation of age, BMI, PTH and Calcium between Control male subjects and male thalassaemic Patients.

Parameters	Control	Patients	P value
Age (years)	12.8 ± 2.6	13.7 ± 3.4	NS
BMI	21.20 ± 3.35	16.57 ± 2.11	0.01
PTH (pg/mL)	23.6 ± 11.6	26.6 ± 14.7	NS
Calcium (mg/dL)	8.76 ± 0.42	8.65 ± 0.36	NS

NS- non significant

parathyroid gland which affects its normal functioning[16].

Ferritin is the principal iron storage protein found in the liver, spleen, bone marrow and to a small extent in the blood. The reasons for the rise in serum ferritin (SF) trends indicated the increase in iron burden of the body. Chronic transfusion would lead to iron overload and excessive iron in the body which is toxic to many tissues of heart, liver and endocrine organ Also, serum ferritin(SF) is a good marker for monitor the iron overload[17]. Also The other studies show in their studies of 43 patients in age group of 3–36 (mean 13.4 ± 7.5) did not demonstrate any correlation with age. Similarly other studies did not find any correlation of serum ferritin with age[18].

References

patients. Proceedings ISHG 2002 XXVII Annual Conference of Indian Society of Human Genetics. "Recent Trends in Genomics",89: 14-16.

[11] **Palamidou, F. et al. (1998)**. Growth and management of short stature in thalassaemia major. *J Pediatr Endocrinol Metab*,11: 835-44.

[12] **Skordis, N. and Kyriakou, A. (2011)**. The multifactorial origin of growth failure in thalassaemia. *Pediatr Endocrinol Rev* ,8(2):271-277.

[13] **Soliman, A.T. et al.(2015)**. Insulin-like growth factor and factors affecting it in thalassemia major. *Indian J Endocrinol Metab* ,19(2):251-245.

[14] **Shapiro, R. and Heaney, R.P. (2003)**.Co-dependence of calcium and phosphorus for growth and bone development under conditions of varying deficiency. *Bone* ,32: 532-540.

[15] **Gabriele, O. (1971)**. Hypoparathyroidism associated with thalassemia. *South Med J* ,64:115-6.

[16] **Chern, J.P. and Lin, K.H. (2002)**. Hypoparathyroidism in transfusion dependent patients with B thalassemia. *Pediatr Hematol Oncol* ,24(4): 291-3.

[17] **Zelly, D. R. et al. (2017)**. Vitamin D and Parathyroid Hormone Levels and Their Relation to Serum Ferritin Levels in Children with Thalassemia Major: One-Center Study in Western Indonesia. *Journal of Advances in Medical and Pharmaceutical Sciences* ,15(1): 1-5.

[18] **Vichinsky. E. et al. (2007)**. Serum ferritin underestimates liver iron concentration in transfusion independent thalassemia patients as compared to regularly transfused thalassemia and Sick cellpatients. *Pediatr Blood Cancer*,49:329–332.

thalassemia and also basically observed as hypoparathyroidism, which accompanied by other endocrinopathies. Serum PTH levels were not significantly different between cases (26.6 ± 14.7 pg/ml) and controls (23.6 ± 11.6 pg/ml). In different previous studies the incidence of hypoparathyroid varies from 0% to 22.5% of patients[14].Although overt hypoparathyroidism is very rare and even subtle abnormalities are not well established in these cases.The majority of cases of hypoparathyroidism in Beta Thalassemic children were seen in second decade of life[15]. Parathyroid hormone levels are chiefly regulated by alteration in calcium levels in the body along with Calcitonin. Repeated blood transfusion results in the iron deposition in the

[1] **Basha, K.P.; Shetty, B. and Shenoy, U.V. (2014)**. Prevalence of Hypoparathyroidism (HPT) in Beta Thalassemia Major. *Journal of Clinical and Diagnostic Research* ,8 (2):24-26.

[2] **Eman A. Askar.et al. (2013)**. Growth Parameters and Vitamin D status in Children with Thalassemia Major in Upper Egypt. *I JHOSCR* ,7(4):10-14.

[3] **Hadi, H. et al. (2013)**. The dverse effects of thalassemia treatments including bloodtransfusion and main pharmacological therapies. *J Pharm Sci* ,8(4):199–204.

[4] **Hamann, K.L. & Lane, N.E. (2006)**. Parathyroid hormone. *Rheumatic Diseases Clinics of North America* ,32: 703–719.

[5] **Hamidieh, A.A. et al. (2009)**. Hypoparathroidism in patients with beta thalassemia major. *IJHOSCR* ,3:17-20.

[6] **Khalafallah. H.;and Soliman, A.T, Ashour,R. (2009)**.Growth and factors affecting it in thalassemia major. *Hemoglobin* ,33(1):S116-S126.

[7] **Mehta, Vikram. et al. (2010)**. Splenectomy In Management Of Thalassemia Major – A Boon For The Little Angel. *The Internet Journal of Surgery* ,24(1): 1-11.

[8] **Prestcott E.et al. (2000)**. Hepatic iron concentration combined with long-term monitoring of serum ferritin to predict complications of iron overload in thalassaemia major. *B J Haematol*,110: 971-977.

[9] **Pung-Amritt P. et al. (2001)**. Linear growth in homozygous beta-thalassemia and beta thalassemia hemoglobin E patients under different treatment regimens *J Med Assoc Thai*,84: 929-4.

[10] **Phadke, S.R. and Agarwal, S.S. (2002)**. Growth faltering in non-compliant thalassemia major

تأثير الغدة جار الدرقية على مرضى بيتا ثلاثيميا في مدينة كركوك

دمت فكرت صابر ، موسى محمود مربوط ، سامي اكريم زيار

كلية الطب ، جامعة تكريت ، تكريت ، العراق

الملخص

الثلاسيما هو اضطراب دموي موروث يصنع فيه الجسم شكلاً غير طبيعي من الهيموجلوبين. تظهر أعراض الثلاسيما في السنتين الأوليين من العمر وتشمل شحوب الجلد وفقر الدم والتشوهات العظمية والضعف وتأخر النمو ولا يوجد حتى الآن أكثر الطرق نجاحاً للشفاء التام لمرض الثلاسيما. تضمنت الدراسة الحالية (70) سبعين عينة دم لذكور مصابين بالثلاسيما في مركز الثلاسيما بمستشفى ازادي التعليمي في مدينة كركوك وفي هذه الدراسة تم تسجيل القياسات الانثروبومترية والعمر ومؤشر محتوى كتلة الجسم لجميع المرضى ومجموعة السيطرة وقد اظهرت نتائج الدراسة الحالية ارتفاع ملحوظ ($P > 0.0$) في المجموعة العمرية الثانية لمستوى هرمون الغدة جار الدرقية (9-13) مقارنة مع المجموعة العمرية الاولى (4-8) والمجموعة العمرية الثالثة (14-18).