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Hematological and Plasma Minerals Examination of Iron Load in Thalassemia Patients

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Abstract

Thalassemia is a common genetic disorder in which defected and decreased the production of hemoglobin (Hb) occur. In thalassemia, people are anemic because they have reduced the number of RBCs due to destruction and ineffective production of RBCs. In Pakistan β-thalassemia is the most common genetic disorder. The study was aimed to estimate the hematological parameters (RBC, WBC, Platelets, Hb, Neutrophils, Eosinophils, Basophils, Lymphocytes, Monocytes, PCV, MCV, MCH, MCHC) and the outcome of iron load on plasma minerals such as sodium (Na), potassium(K), calcium (Ca), magnesium (Mg), chloride (cl) and phosphorus (P) in β–thalassemic patients. The results were found propose that alterations in electrolytes concentrations then reference values, particularly increased phosphorus concentration (2.59 ± 0.50mmol/l) and decreased concentration of calcium (1.65 ± 0.33mmol/l) and magnesium (0.58 ± 0.61), but no significant difference was found in case of sodium (136 ± 2.70 mmol/l), potassium (5.0 ± 0.40mmol/l) and chloride (131 ± 14.1). There is a strong need to create awareness among patients about the consequences of iron overload in their body. Proper chelation of iron overload could improve the quality of life of these patients.

Introduction

The most common monogenic disorder in this world is thalassemia. About 270 million carriers of mutant globin alleles present worldwide, and they can cause different forms of hemoglobinopathy and thalassemia [1]. In developing countries, Thalassemia is a major health and economic burden, infants' patients of thalassemia died from malnutrition and infection. Hence thalassemia is a huge and increasing hematologic health issue in both developing and developed countries [2].

Different forms of thalassemia have different incidence rates which vary from region to region. HbE-thal and α- thalassemia is more common in Southeast Asian countries, whereas β-thal is more common in Africa and Mediterranean region [3]. While worldwide α-thal is very common, and it fluctuates among different populations. The incidence of deletion of one α-globin gene is getting above 75% in Southwest pacific, Nepal and India [4]. In Pakistan β–thal frequently inherited Hb disorder [5]. Approximately 125 million, in Northern regions, 80% of the children are anemic due to β-thal [6]. The carrier incidence is about 5.3% [7] while the occurrence of β-thal differs from 1.5% [8] to 8% [7]. While the incidence of α- thalassemia is about 2.4% [9]. These statistics showed that α and β thalassemia existing in Pakistan. A Major cause of thalassemia in Pakistan is consanguineous marriages and the preference to marry within ethnic groups, due to these factors thalassemia rate increased in Pakistan. [10].

The high frequency of thalassemia is a health issue in Pakistan. This study aimed to determine the relationship and compare between growth and hemoglobin level, serum ferritin level/iron overload parameters, and other clinical factors.

Materials and methods

This study was conducted on 88 male children suffering thalassemia, with the age-group of 3-12 years, who were already enrolled in Peoples Medical College (PMC) Hospital, Nawabshah, Sindh, Pakistan. These thalassemic children were treated with frequent transfusions and long-term iron chelating therapy (**Figure 1**).

Samples of blood were collected without anticoagulant into labeled sample bottles and these blood sample bottles were kept in a refrigerator at 4 °C. After that centrifuge it for 10 minutes at 3000 rpm and the serum stored at -10°C for further examination. Serum used to analyze the serum minerals (Sodium, Potassium, Phosphorus, Calcium, Chloride and magnesium). Calcium magnesium, phosphorous and chloride were analyzed by ELI TECH, BIO MERIEUX, DIALAB and DIASYS Gmbh respectively, while flame photometer (Jenway-Clinical PFP7) was used for the estimation of Potassium and Sodium.

The whole blood samples were collected into well-labeled set of sample bottles with anticoagulant (K2EDTA,1mg/ml of blood) was used for the determination of hematological parameters (Hb, RBC's Count, WBCs Count, Platelets, Neutrophils, Eosinophils, Basophils, Lymphocytes, Monocytes, PCV, MCV, MCH and MCHC) hematological analyzer was used for determination of Hb, total leukocyte count, RBC's count, differential leukocytes count, platelets count and absolute indices (PCV, MCV, MCH, and MCHC). Micro lab 200 (Germany) and Flame Photometer technique were used for the estimation of electrolytes during this study.

All Equipment was calibrated on a daily basis with quality control reagents, and instrumentation standards were maintained within 2 standard deviations (SD) of those QC values. The research work was carried out in the pathological laboratory Peoples University of Medical & Health Sciences for Women (PUMHSW) Nawabshah, Sindh, Pakistan.

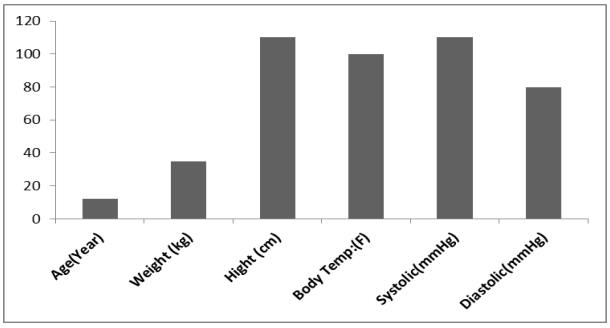

Figure 1: Demographic data of thalassemia patients (n=88)

Results

To compare the difference between reference or normal value and thalassemia patients the results were analyzed statistically. **Table 1** shows that the mean and standard deviation of plasma minerals (Sodium, Potassium, Phosphorus, Calcium, Chloride, and Magnesium) in thalassemia patients. Our result shows that thalassemia patients have increased a concentration of phosphorus (2.59 ± 0.50mmol/l) while decreased concentration of calcium (1.65 ± 0.33mmol/l) when compared with controls. Sodium (136 ± 2.70 mmol/l), Potassium (5.0 ± 0.40mmol/l),

Magnesium (0.58 ± 0.61), and chloride (131 ± 14.1) concentration was within the normal range.

Table 1: Difference between electrolytes values of reference and thalassemia patients

Parameters (n=88)	Normal Values	Mean ± SD
Phosphorus	0.75 – 1.45 mmol/l	2.59 ± 0.50
Calcium	1.9 – 2.5 mmol/l	1.65 ± 0.33
Magnesium	0.7 -1.0 mmol/l	0.58 ± 0.61
Chloride	80 – 110 mmol/l	131 ± 14.1
Sodium	135 – 145 mmol/l	136 ± 2.70
Potassium	3.5 – 5.5 mmol/l	5.0 ± 0.40

Table 2 shows that the mean and standard deviation of hematological parameters (Red blood cells count, platelets count, white blood cells count, and hemoglobin) in thalassemia patients. Our data suggested platelets and white blood cells concentration was found with in normal range but that Hb and RBCs decreases in Thalassemia in comparison of control.

Table 2: Difference between Hb, RBCs, WBCs and PLTs values of reference and thalassemia patients

Hematological Parameters	Normal Values	Mean ± SD
Hemoglobin	13.5 – 16.5g/dL	7.2 ± 1.62
Red Blood Cells Count	4.5 – 6.5m/cmm	2.6 ± 0.55
White Blood Cells Count	4000 – 10000/cmm	8.10 ± 4.10
Platelets Count	150,000–450,000/cmm	1.60 ± 101.90

Table 3 shows the mean and standard deviation of Differential Leucocytes Count (Neutrophils, Eosinophils, Basophils, Monocytes and Lymphocytes) of thalassemia patients. Our results showed that there is increased concentration of Lymphocytes and decreased a concentration of Neutrophils, Eosinophils and Monocytes while a concentration of Basophils is found within the normal range.

Table 3: Difference between neutrophils, eosinophil's, basophils, lymphocytes and Monocytes values of reference and thalassemia patients

Hematological Parameters	Normal Values	Mean ± SD
Neutrophils	40 – 75 %	26.9 ± 11.60
Eosinophils	1 – 6 %	0.86 ± 0.16
Basophils	0 – 1 %	0.32 ± 0.51
Lymphocytes	20 – 45 %	75.4 ± 12.22
Monocytes	2 – 10 %	2.35 ± 4.42

Table 4 shows the mean and standard deviation of Absolute Indices parameters (Packed Cell Volume, Mean Corpuscular Hemoglobin, Mean Corpuscular Volume, and Mean Corpuscular Hemoglobin Concentration) in thalassemia patients. Our results showed that there is decreased a concentration of MCV (71.5 ± 4.90cμ), MCHC (30.1 ± 1.45%), PCV (22.0 ± 4.01%), and MCH (20.9 ± 2.10μg) in thalassemia patients.

Table 4: Difference between PCV, MCV, MCH and MCHC values of reference and thalassemia patients

Hematological Parameters	Normal Values	Mean ± SD
PCV	42 – 49 %	22.0 ± 4.01
MCV	76 – 96 cμ	71.5 ± 4.90
MCH	26 – 32 μg	20.9 ± 2.10
MCHC	32 – 36 %	30.1 ± 1.45

Note: PCV (Packed Cell Volume), MCV (Mean Corpuscular Volume), MCH (Mean Corpuscular Hemoglobin), and MCHC (Mean Corpuscular Hemoglobin Concentration).

Discussion

The goal of the present study was to understand the effects of iron load on plasma minerals (Ca, P, Mg, Cl, Na and K) and hematological parameters (Hb, RBCs, WBCs, Platelets, Neutrophils, Eosinophils, Basophils, Lymphocytes, Monocytes, PCV, MCV, MCH, MCHC) in thalassemia Patients.

The results of this study suggest that alteration in plasma mineral mainly phosphorus increased wile calcium decreased in thalassemia patients. Hematological parameters are affected by many factors, including age, sex, diet, recent nutritional status, consumption of medications or illicit drugs and the chronic disorders [13-16].

Calcium was significantly decreased in ß-thalassemiac patients if we compare it with a control group. Other studies also reported that in thalassemia calcium decreased [5, 6,17] saka et al also reported the same result for decreased calcium level in thalassemia. [18]. Due to transfusion of RBC's iron overload occur which increased the absorption of iron in the digestive tract. All of these happened in thalassemia. The Pituitary gland also damaged by this iron overload. Hypothyroidism, endocrine complication, hypogonadism and hypoparathyroidism also occur due to iron overload [19]. Parathyroid gland secreted parathyroid hormone; it mobilizes calcium from bone [19, 20].

Rapid lysis of RBC's in thalassemia increased the concentration of Phosphorus. Plasma membrane is made up of phospholipid when the RBCs plasma membrane damaged in thalassemia phosphorus concentration increased.

Homozygous β-thalassemia is a severe hereditary disorder characterized by decreased synthesis of the β-polypeptide chain of hemoglobin, which leads to the development of a hypochromic microcytic anemia [10, 21, 22]. Alternatively, frequent blood transfusion, in turn, can lead to iron overload [23-25].

As iron loading progresses, the capacity of transferrin in blood, may bind and detoxify iron may be exceeded

and a non–transferrin-bound fraction of iron in blood may promote the generation of free hydroxyl radicals, propagators of oxygen-related damage [26, 29].

Conclusion

There is a strong need to create awareness among patients about the consequences of iron overload in their body. Proper chelation of iron overload could improve the quality of life of these patients. The problems of poverty, low education level and inadequate provision of health care are the main stumbling blocks in effective treatment of iron overload in thalassemic patients which is the main cause of morbidity and mortality in thalassemia major.

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