

The role of thalassemia on some immunological and hematological parameters in thalassemia patients in holy Karbala province

Muhammad Riyadh Alkenany

Received on: 20-05-2026

Published on: 30-08-2026

ABSTRACT

Background: Thalassemia is a hereditary red blood cell disorder characterized by defective synthesis of hemoglobin globin chains, leading to microcytic, hypochromic anemia.

Methods: This research was conducted at the Thalassemia Test Unit, Gynecology and Obstetrics Teaching Hospital, Holy Karbala Province, Iraq, between December 1, 2024, and February 28, 2025. One hundred fifty diseased individuals were used in this study; their ages were ranged between 2-48 years old from male and female affected with thalassemia. The exclusion from the study about 2 individuals, that samples were excluded because participants had chronic illnesses or genetic disorders. In addition, cases whose health conditions were deemed unsuitable according to the study criteria were excluded. The number of samples 87 males and 61 females for testing, The study involved people in a, specified age group ranging from 2 and 28 years old that have a good health and free from any chronic or genetic diseases. The study included both genders to ensure representation of both sexes. The study Participants had a normal body weight, and people with obesity or severe underweight were excluded.

Results: The study showed that there is relation between thalassemia and ABO blood groups, it revealed that B+ is the highest affected blood group among all especially major thalassemia, whereas AB+ showed the lowest results. On the other hand, the study showed the relation of RBC, WBC and Platelets counts to thalassemia, revealing that RBC count is equal in male and female in minor thalassemia but female was higher in major thalassemia. At the meantime, Platelet count was higher in female than male in minor thalassemia but quite the opposite for major thalassemia. Lastly, the study showed the relation between WBC count and thalassemia, it was higher in female than male in both types.

Conclusion: This study led to the recording of some effects and relationships of thalassemia disease with some immunological and hematological parameters in patients of Holy Karbala Province as a sample of Iraqi population.

KEYWORDS: ABO, PLT, RBC, THALASSEMIA, WBC.

Era's Journal of Medical Research. 13(2);2026 [doi: 10.24041/ejmr.2026.##]

INTRODUCTION

Thalassemias are a family of autosomal recessive hemoglobinopathies. They are among the most common monogenic disorders worldwide¹ and are caused by different molecular defects. Alpha-thalassemia is caused by mutations that reduce production of alpha-globin peptides, and beta-thalassemia is caused by mutations that stop production of beta-globin peptides.² These conditions tend to occur in clusters in the Mediterranean basin, the Middle East, the Indian subcontinent and Southeast Asia.³

In a number of populations in southern Asia, alpha-thalassemia can be almost genetically fixed with carrier rates ranging from 80% to 90%.⁴ This epidemiological footprint is similar to that seen in areas of the world where Plasmodium falciparum malaria is endemic, and is a hallmark of a balanced polymorphism; heterozygous carriers of the infection have a survival advantage against severe infection.⁵ While no single figure can be given for the total global costs, the socioeconomic and clinical impact of thalassemia is steadily increasing, especially because of clinical improvements and increased life expectancy in the endemic countries.⁶

Department of Medical Physics, Faculty of Medical Applied Sciences, University of Kerbala, Karbala, Iraq.

Corresponding Author: Muhammad Riyadh Alkenany

Email: muhammad.r@uokerbala.edu.iq

How to cite: Alkenany MR. The role of thalassemia on some immunological and hematological parameters in thalassemia patients in holy Karbala province. Era J Med Res. 2026;13(2):##-##.

The genetics of alpha-thalassemia are similar to those of a duplicated-gene architecture. Usually an individual will have two copies of the alpha-globin gene (HBA1 and HBA2) on each of the 16 chromosomes for a total of 4 working copies of the gene. Offspring can inherit alpha-thalassemia trait, Hemoglobin H (HbH) disease or fatal Hydrops fetalis syndrome caused by the presence of alpha-thalassemia deletions or inactivating point mutations in the parental alleles. The severity of the phenotype depends on the number of the disabled loci, the locus of the disabled gene (HBA2 versus HBA1) and cis versus trans configuration. For example, a person with

a cis-deletion ($\alpha\alpha$), also known as α -thalassaemia) has a high risk of passing on hydrops fetalis to their child than does a person with a trans-deletion ($\alpha\alpha$), also known as homozygous α -plus-thalassemia). Above all, a definitive molecular typing for α -plus and α -0 variants is a vital tool in the prenatal screening and genetic counseling of patients.⁷

In contrast with the duplicated α cluster, only one HBB gene on chromosome 11 is responsible for the synthesis of beta-globin. Beta-thalassemia is a clinically highly heterogeneous disorder:

- As for the milder heterozygous mutations, the carrier state (beta-thalassemia minor), is usually asymptomatic, with only slight red cell count variations, mild hypochromia and borderline microcytosis. This is the commonest clinical presentation in clinical screening.
- Beta-thalassemia intermedia: Classifies a state of moderate anemia in which beta-globin production is sufficiently reduced, but where a lifelong need for regular transfusion is not present [8].

In terms of cells, quantitative reduction (beta-plus) or complete loss (beta-zero) of beta-globin will cause a stoichiometric imbalance as cells will not be coordinated. When α -globin is free (not paired with beta-globin), it cannot make functional tetramers, but instead precipitates as hemichromes, which are insoluble, inside developing erythroid precursors. These inclusions cause oxidative damage to the membranes, apoptosis, severe ineffective erythropoiesis in the bone marrow and peripheral extravascular hemolysis. This cellular destruction is

proportional to the type of molecular lesion found in the HBB locus on the 11th chromosome [9].

Materials and Methods

Experimental Design:

This experiment was carried out at Thalassemia test Unit/ Gynecology and Obstetrics Teaching Hospital, Iraq. 150 healthy individual were used in this study; their ages were ranged between 2- 48 years old. blood samples were obtained as follow Figure (3-1) study design :

Blood sample collection:

Samples were collected from Thalassemia test Unit laboratories in the form of samples in an EDTA tube with the results of their cellular analysis. They were separated by centrifuge at 3000 rpm for 10 minutes. Plasma samples were collected in an Eppendorf 1.5 ml container and stored at -40 degrees.

Hematological parameters

Immunological and hematological parameters were calculated by using a complete blood count device or auto-hemolyzer supported by Abbott®.

Statistical Analysis

The Statistical Packages of Social Sciences -SPSS (2019) program was used to detect the effect of difference groups/ factors in study parameters. T-test was used to significant compare between means. Chi-square test was used to significant compare between percentage (0.05 and 0.01 probability) in this study.

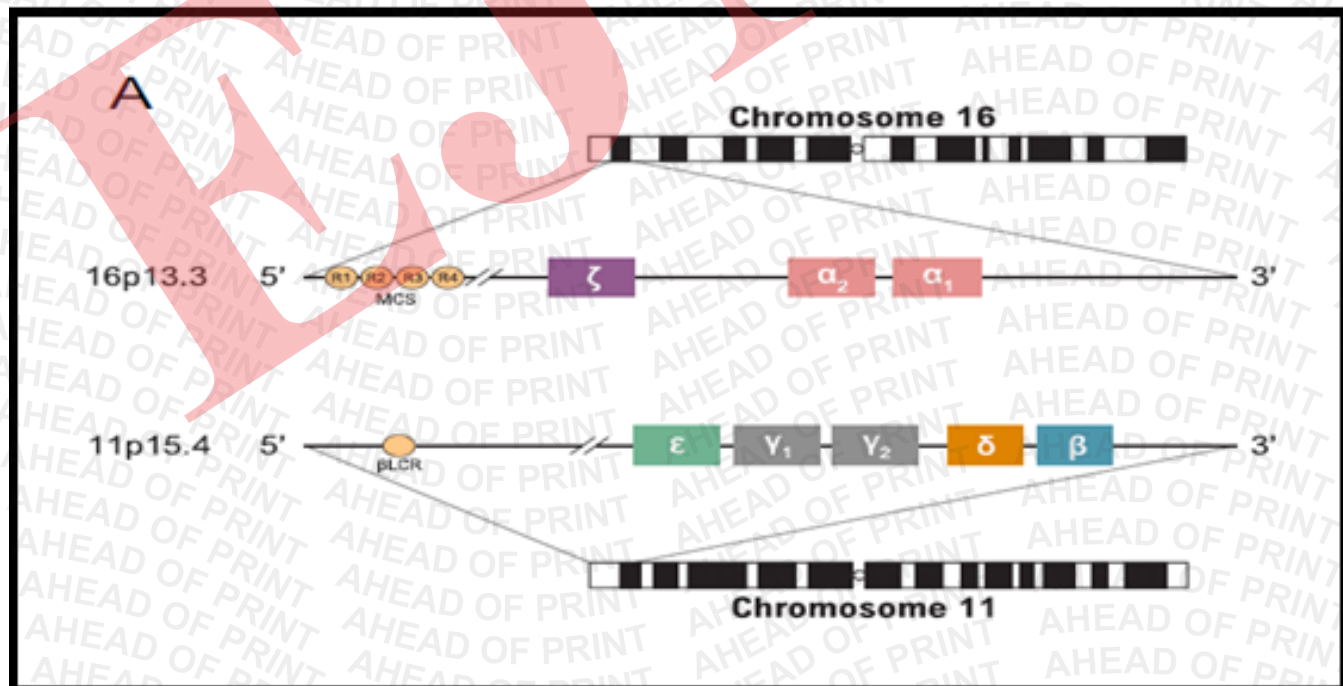


Figure (1-1): Chromosomal location and organization of the α - and β -globin gene clusters.

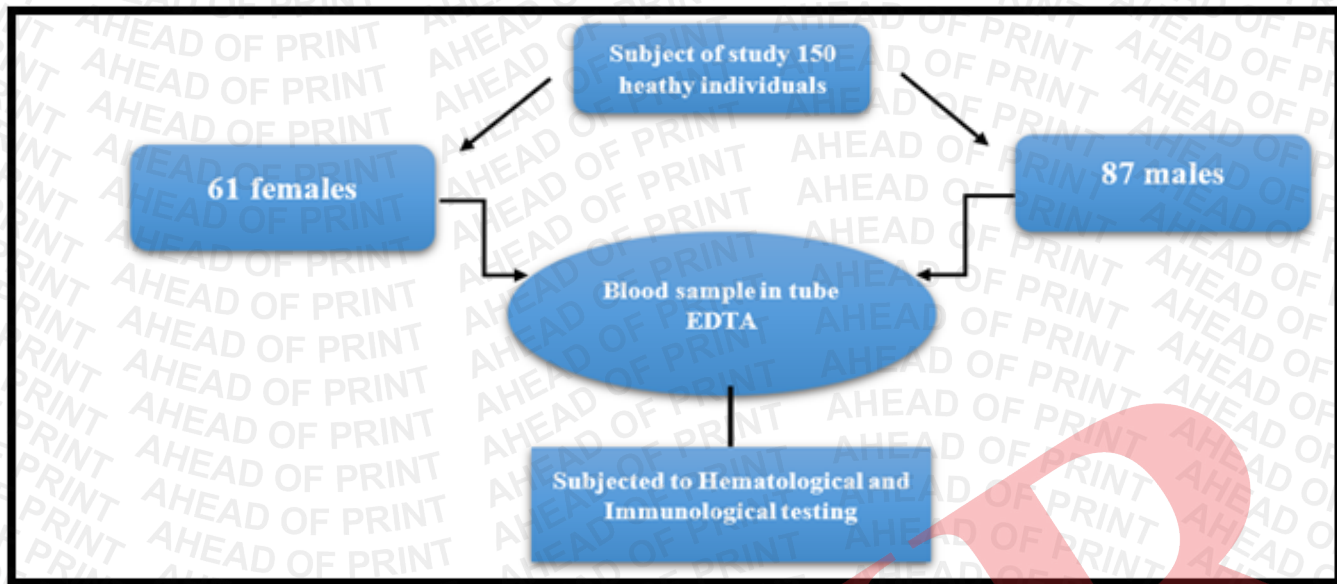


Figure (2-1): Study design

Table (3-1): Distribution of the sample study according to types of Thalassemia and age in Male individuals

Type Age (y)	Minor	Major	Total	P-value
2-15	14 (34.15%)	27 (65.85%)	41	0.0423 *
16-38	4 (8.70%)	42 (91.30%)	46	0.0001 **
Total	18 (20.69%)	69 (79.31%)	87	0.0001 **
P-value	0.0392 *	0.0388 *	--	--

* (P≤0.05), ** (P≤0.01).

Table (3-2): Distribution of the sample study according to types of Thalassemia and age in Female individuals

Type Age (y)	Minor	Major	Total	P-value
2-9	7 (38.89%)	11 (61.11%)	18	0.346 NS
10-30	10 (23.26%)	33 (76.84%)	43	0.0005 **
Total	17 (27.87%)	44 (72.13 %)	61	0.0005 **
P-value	0.179 NS	0.266 NS	--	--

** (P≤0.01), NS; Non-Significant.

Table (3-3): Distribution of the sample study according to types of Thalassemia and Gender

Type Gender	Minor	Major	Total	P-value
Male	18 (20.69%)	69 (79.31%)	87	0.0001 **
Female	17 (27.87%)	44 (72.13%)	61	0.0005 **
Total	35 (23.65%)	113 (76.35%)	148	0.0001 **
P-value	0.562 NS	0.519 NS	--	--

* (P≤0.05), ** (P≤0.01).

Table (3-4): Distribution of sample study according to types of Thalassemia and Blood group

Type Blood-group	Minor	Major	Total	P-value
A+	12 (22.22%)	42 (77.78%)	54	0.0001 **
B+	6 (16.67%)	30 (83.33%)	36	0.0001 **
AB+	3 (37.50%)	5 (62.50%)	8	0.4795 NS
O+	14 (28.00%)	36 (72.00%)	50	0.0019 **
Total	35	113	148	--
P-value	0.0072 **	0.0077 **	--	--

** (P≤0.01), NS; Non-Significant.

Table (3-5): Relationship of Thalassemia type in the Hematological

Parameters	Thalassemia types (Mean ±SE)		P-value
	Minor	Major	
RBC (N x10 ⁶ cell/mm ³)	3.11 ±0.09	2.98 ±0.06	0.227 NS
Hb (g/dL)	8.14 ±0.19	7.98 ±0.12	2.315 NS
HCT (%)	22.78 ±0.61	22.70 ±0.63	0.463 NS
MCV (fL)	73.94 ±1.14	75.05 ±0.63	2.554 NS
MCH (pg/cell)	26.73 ±0.52	27.18 ±0.29	1.169 NS
PLT (x 10 ³)	360.11 ±33.31	373.12 ±23.79	91.55 NS
MPV	9.18 ±0.19	9.38 ±.14	0.535 NS

Table (3-6): Relationship of Thalassemia type with Immunity

Parameters	Thalassemia Types (Mean ±SE)		P-value
	Minor	Major	
WBC (N x10 ³ cell/mm ³)	10.21 ±1.82	11.58 ±1.34	5.157 NS
Lymphocyte (%)	45.51 ±1.49	45.23 ±1.14	4.331 NS
GRA (%)	46.78 ±1.54	46.26 ±1.23	4.652 NS

RESULTS

Distribution of sample study according to types of Thalassemia and age in Male individuals

Table (3-1) shows the highly significant (P< 0.05) distribution of samples in major thalassemia than minor thalassemia in all ages, also shows the highly significant (P< 0.05) distribution of thalassemia in the age group between (16-38) more than the age group (2-15) in male patients.

Distribution of sample study according to types of Thalassemia and age in Females individuals

The result in table (3-2) shows the highly significant (P< 0.05) in distribution of samples in major thalassemia than minor thalassemia in all ages, also indicated highly significant (P< 0.05) the distribution of thalassemia in the age range between (10-30) more than age (2-9) in female.

Distribution of the sample study according to types of Thalassemia and Gender

The result of table 3-3 indicated the highly significant (P< 0.05) in distribution of samples in major thalassemia than minor thalassemia in male group and female group, also show highly significant (P< 0.05) in total number in major thalassemia than minor thalassemia, but no significant (P< 0.05) between minor thalassemia and major thalassemia in both gender groups.

Distribution of sample study according to types of Thalassemia and Blood group

The result in Table (3-4) indicated a highly significant (P< 0.05) difference in the distribution of samples in major thalassemia than minor thalassemia in all blood groups except the AB+, where there was a non-significant difference between minor and major thalassemia. Also, the A+ blood group is found more frequently than in other groups.

Relationship of Thalassemia type in Hematological and Immunological parameters

The results of in table (3-5) shown non- significant ($P < 0.05$) compared in RBC, Hb, HCT, MCV, MCH, PLT and MPV between minor and major thalassemia, shown figure (3-5). On the other hand, shown the non-significant ($P < 0.05$) comparison of WBC, lymphocyte and granulocyte between minor and major thalassemia, table (3-6), figure (3-6).

Effect the Types of Thalassemia and Gender on Hematological and Immunological parameters

Looking at the core erythrocyte profiles in Table 3-7, neither the specific type of thalassemia nor the patient's gender produced

any statistically significant effect on overall RBC, hemoglobin, or HCT levels. The red cell morphological indices in Table 3-8 follow the exact same pattern: MCV and MCH values show no meaningful variance across sex or clinical diagnosis.

The leukocyte data in Table 3-9, however, breaks this uniform trend. Gender exerts a clear, significant effect on total WBC counts ($p < 0.05$), with male patients consistently recording higher baseline levels than females. Interestingly, the clinical severity of the disease (major versus minor) does not drive total WBC numbers at all. This sex-based divergence is also strictly limited to the total count—when we break down the white cells into specific lymphocyte and granulocyte fractions, both gender and thalassemia type

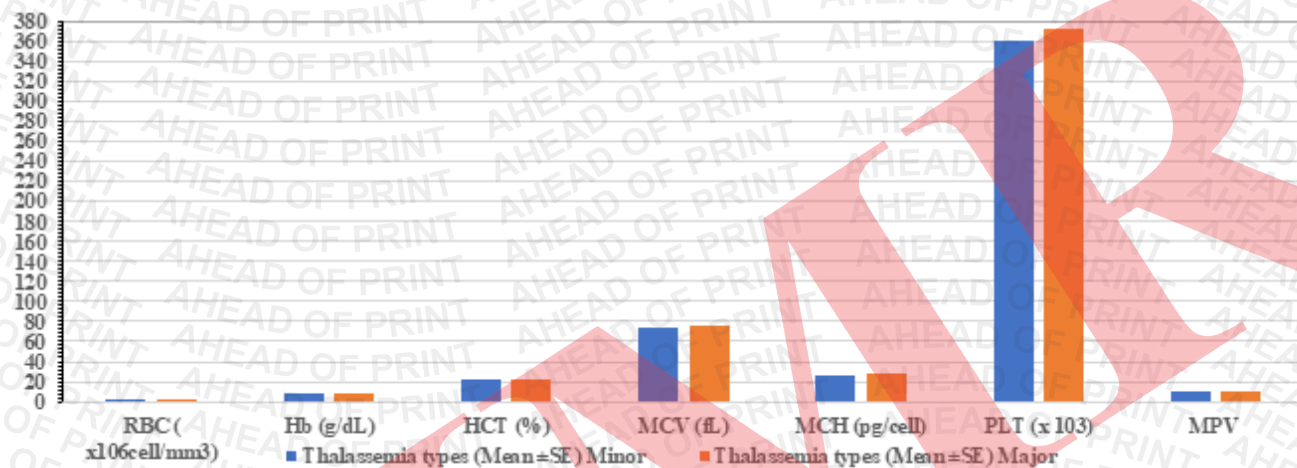


Figure (3-5): Relationship of Thalassemia type in the Hematological

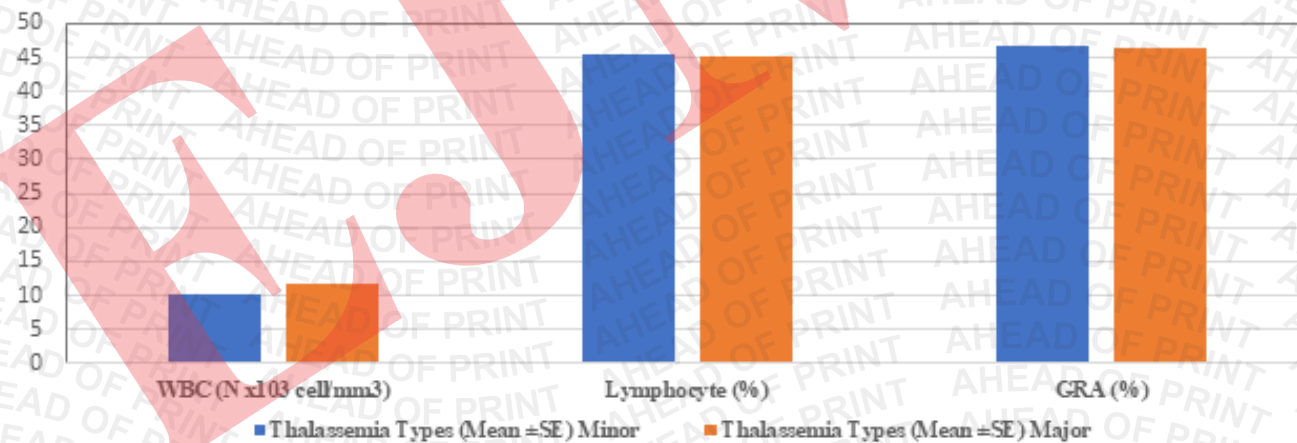


Figure (3-6): Relationship of Thalassemia type in Immunity

Table (3-7): Effect of types of Thalassemia and Gender in RBC, HCT and HGB

Parameters Gender	RBCs		HCT		HGB	
	Minor (Mean)	Major (Mean)	Minor (Mean)	Major (Mean)	Minor (Mean)	Major (Mean)
Male	3.16 ± 0.17	2.95 ± 0.07	22.85 ± 1.04	22.88 ± 0.58	8.12 ± 0.32	7.96 ± 0.15
Female	3.07 ± 0.11	3.03 ± 0.08	22.71 ± 0.66	22.41 ± 0.58	8.16 ± 0.24	8.03 ± 0.18
T-test	0.407 NS	0.229 NS	2.032 NS	2.559 NS	0.809 NS	0.472 NS

NS: Non-Significant.

Table (3-10): Effect of types of Thalassemia and Gender in PLTs, MPV (fl)

Parameters Gender	MCV		MCH	
	Minor (Mean)	Major (Mean)	Minor (Mean)	Major (Mean)
Male	73.44 ±2.04	75.31 ±0.85	26.50 ±0.92	27.39 ±0.34
Female	74.43 ±1.09	74.64 ±0.92	26.97 ±0.49	26.84 ±0.51
T-test	4.705 NS	2.563 NS	2.117 NS	1.176 NS

NS: Non-Significant.

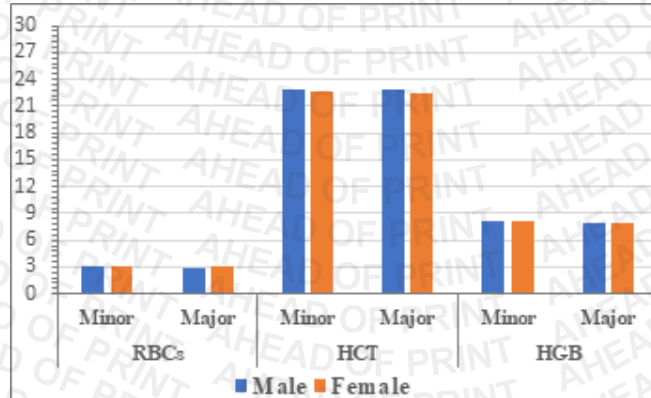


Figure (3-7): Effect of types of Thalassemia and Gender in RBC, HCT and HGB

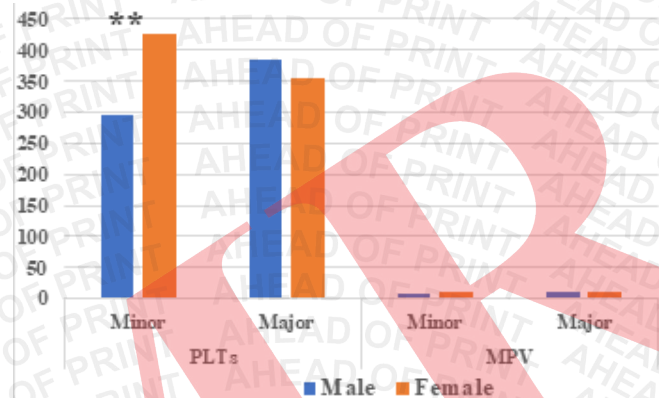


Figure (3-10): Effect of types of Thalassemia and Gender in PLTs, MPV (fl)

lose all statistical significance.

We see a parallel dynamic in the thrombocyte data outlined in Table 3-10. Total platelet counts are significantly higher in males than in females ($p < 0.05$), yet the actual type of thalassemia has zero statistical bearing on these numbers. Finally, mean platelet volume (MPV) stays entirely flat across the board, completely unaffected by either the patient's sex or their specific disease classification.

DISCUSSION

Major thalassemia was more prevalent than minor thalassemia in all age groups analyzed for males as revealed by our findings (Table 3-1 and 3-2) with a statistically significant difference ($p < 0.05$). This finding is in keeping with the literature, which indicates that the diagnosis of beta-thalassemia major is made much more reliably, because of the acute nature of its onset. Pallor, failure to thrive and progressive hepatosplenomegaly are common features and are usually a clinical emergency in the infant or early childhood period [6]. On the other hand, people with thalassemia minor are usually not symptomatic or only have minor microcytic indices. As a result, they often go undiagnosed or are misdiagnosed, especially in health jurisdictions not implementing population wide screening for hemoglobinopathies (Camaschella, 2015).¹¹

It was found that there was a clustering of the ages within the adolescent and young adult age groups (10–38 years) compared to the early pediatric age groups (2–15 years) by stratification. This distribution is due to several clinical and demographic factors. First, the disease has become a manageable chronic condition rather than a deadly pediatric illness, as a result

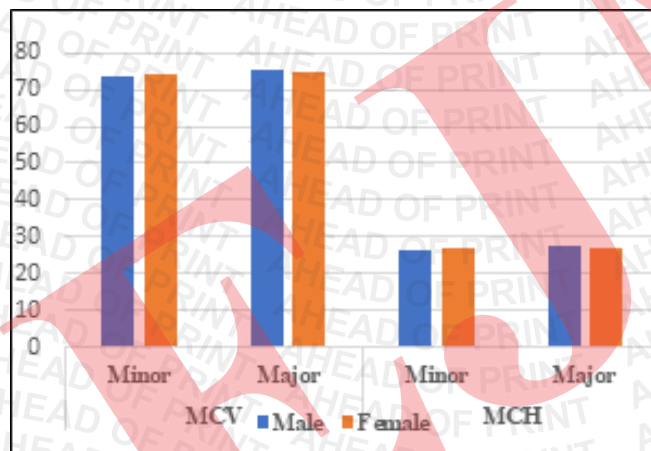


Figure (3-8): Effect of types of Thalassemia and Gender in MC and MCH

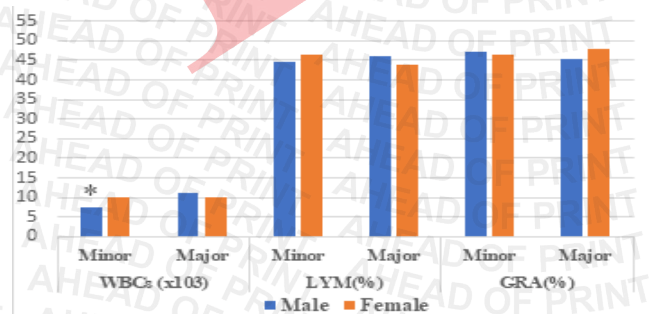


Figure (3-9): Effect of types of Thalassemia and Gender in WBCs, Lymphocyte (%) and Granulocytes

of systematic hypertransfusion protocols used in modern clinical care in addition to the use of contemporary iron chelation therapy, which has resulted in improved survival into the adult decades.¹¹ Second, the widespread adoption of pre-marital carrier screening programs in high prevalence areas such as South Asian and Middle East countries with areas of customarily consanguineous marriages has significantly enhanced diagnostic ascertainment.¹² Routine primary care was not used to identify these mildly symptomatic people or latent carriers, which these public health programs systematically capture.

A comparison by sex also showed a significant differential distribution ($p < 0.05$) between major and minor presentations in both males and females, and the numbers of cases were heavily skewed towards major presentations. This is an intrinsic ascertainment bias: minor variants with silent or borderline anemia are not systematically screened in the family and are therefore not reported in the registers of major centers, while severe cases end up in major hospital registers.⁹

This is due to the increase in clinical contact rates, periodic transfusion dependency and the active clinical record-keeping of severely symptomatic individuals with thalassemia major as reported here.¹³ On the other hand, asymptomatic individuals with a single copy of the gene do not normally request a specialist hematological evaluation unless they are identified during a complete blood count performed as a chance examination or as part of a family cascade testing.¹⁴

Blood group analysis showed high frequency differences between major and minor cohorts for most of the blood group phenotypes ($p < 0.05$); with the highest frequency of A+ blood group. The only exception was seen in the AB+ group where no statistically significant difference was seen between clinical variants. However, the aggregate phenotypic distribution of our sample was $A > O > B > AB$. Published regional reports show slight differences in the distribution of the alleles of the ABO system, as the distribution of alleles varies significantly from one region to another, and also from one race to another.¹⁵ In fact, results of the region showed that B+ phenotype was the most common and O- was the rarest one in the beta-thalassemia major patients of the region. Similarly, Waheed et al. (2023)¹⁶ and Solanki et al. (2023)¹⁷ found that the O+ blood group was more common and the AB- was less common among patients of beta-thalassemia and no significant difference was seen in ABO distribution among males and females.

Our data showed that all thalassemic patients had a significant decrease in total RBC count, hemoglobin (Hb) and hematocrit (HCT), and no statistically significant difference was observed between major and minor patients. The results are consistent with those of AL-MASAOODI et al. (2020)¹⁸ in which patients with thalassemia showed significant decrease in Hb levels when compared with healthy controls ($p < 0.05$).

This is in line with Shanthi, et al. (2013)¹⁹ who attributed the simultaneous decrease in Hb, erythrocytes and HCT to accelerated uncompensated peripheral hemolysis and ineffective erythropoiesis. Likewise, Bhuiyan et al. 2020 (20) investigated 40 patients and determined that only 2.5% of the patients maintained target levels, whereas 97.5%

maintained Hb levels below 10 g/dL. But appropriate use of structured transfusion protocols is still essential to maintain an adequate level of hemoglobin in the trough, which helps to preserve physiological somatic development, avoid compensatory extramedullary erythroid expansion and reduce erratic secondary iron loading.

The indices of the erythrocytes were also depressed without any significant difference between the major and minor phenotypes in our thalassemia group. The mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH) are the first-line diagnostic discriminators, in keeping with the recommendations of the Thalassaemia International Federation (TIF) for screening. A combination of markedly low MCV and MCH indices coupled with characteristic peripheral smear morphology, which exhibits microcytosis, hypochromia and obvious anisopoikilocytosis are highly suggestive of a carrier state or latent variant.¹¹

The hematological parameters of thalassemics were found to be significantly different from the reference values (Table 2). In structural chain imbalances, membrane instability leads to early destruction of erythrocyte precursor cells, which results in moderate to severe chronic anemia (mean Hb 8 ± 1 g/dL). This hypoxic stress leads to downstream effects such as growth retardation, delayed bone maturation and systemic endocrine stress.²¹

In thalassemic patients there was a rise in total leukocyte count, absolute lymphocytes and granulocytes levels when compared to reference values. Absolute differential counts (WBC%) did not differ by any significant amount among the clinical classifications, but the total WBC levels were significantly different between the major and minor groups. Developing leukocytosis as a result of chronic baseline immune activation was also seen by Munir et al.²¹ as in the case of immunostimulatory effects seen under Lentinan.²²

There was no significant difference between major and minor presentations in platelet counts or mean platelet volume (MPV) but absolute platelet counts were mostly elevated compared to normal reference ranges. Thromboembolic events have been shown to be significantly more common in thalassemic patients compared to age matched normal controls, especially those with beta-thalassemia intermedia, and those patients who have undergone splenectomy. This is a multi-factorial hypercoagulable state that is responsible for this thrombotic vulnerability. Increased blood platelets and reactive thrombocytosis are known indicators of vascular occlusion risk in splenectomized persons and intermediate phenotypes.²⁴ In this particular context, MPV offers a valuable morphological marker for baseline platelet activation, aggregation potential, and turnover rate.²⁵

CONCLUSION

This study shows a distinct clinical distribution and the AB+ blood group was the least and major thalassemia cases were the most prevalent. Thalassemic patients showed uniform reduction in the mature erythrocyte indices while there was a corresponding increase in circulating leukocytes and platelets. Sex-stratified analyses revealed comparable erythrocyte counts between male and female carriers in

minor thalassemia, whereas females retained slightly higher RBC counts in major cases. Platelet numbers trended higher among females in minor thalassemia, yet skewed higher in males presenting with major phenotypes. Total leukocyte counts remained consistently elevated in female cohorts across both disease categories.

ETHICAL CLEARANCE

Ethics was As per the World Medical Association's Declaration of Helsinki, which has been created by independent and experimental processes, (ethics committees approval/ Faculty of Medical Applied Sciences, University of Kerbala, Iraq.

REFERENCES

- Jaing TH, Chang TY, Chen SH, Lin CW, Wen YC, Chiu CC. Molecular genetics of β -thalassemia: A narrative review. *Medicine*. 2021 Nov 12;100(45):e27522.
- Kattamis A, Kwiatkowski JL, Aydinok Y. Thalassaemia. *The Lancet*. 2022 Jun 18;399(10343):2310-24.
- Wong P, Fuller PJ, Gillespie MT, Milat F. Bone disease in thalassemia: a molecular and clinical overview. *Endocrine reviews*. 2016 Aug 1;37(4):320-46.
- Hockham C, Ekwattanakit S, Bhatt S, Penman BS, Gupta S, Viprakasit V, Piel FB. Estimating the burden of α -thalassaemia in Thailand using a comprehensive prevalence database for Southeast Asia. *Elife*. 2019 May 23;8:e40580.
- Ndila CM, Uyoga S, Macharia AW, Nyutu G, Peshu N, Ojal J, Shebe M, Awuondo KO, Mturi N, Tsofa B, Sepúlveda N. Human candidate gene polymorphisms and risk of severe malaria in children in Kilifi, Kenya: a case-control association study. *The Lancet Haematology*. 2018 Aug 1;5(8):e333-45.
- Weatherall DJ, Clegg JB. Inherited haemoglobin disorders: an increasing global health problem. *Bulletin of the World Health Organization*. 2001;79(8):704-12.
- Tesio N, Bauer DE. Molecular basis and genetic modifiers of thalassemia. *Hematology/oncology clinics of North America*. 2023 Apr 1;37(2):273-99.
- Putri IM, Gultom FP, Auerkari EI. Genetics and Epigenetics Aspects of Thalassemia. In *Proceedings of the 4th International Conference on Life Sciences and Biotechnology (ICOLIB 2021)* 2023 Feb 10 (Vol. 27, p. 288). Springer Nature.
- Galanello R, Origa R. Beta-thalassemia. *Orphanet journal of rare diseases*. 2010 May 21;5(1):11.
- Longo DL, Camaschella C. Iron-deficiency anemia. *N Engl J Med*. 2015 May 7;372(19):1832-43.
- Taher AT, Musallam KM, Cappellini MD. β -Thalassemias. *New England Journal of Medicine*. 2021 Feb 25;384(8):727-43.
- Ansari, S. H., Shamsi, T. S., Siddiqui, S., et al. (2012). Consanguinity, maternal education, and birth order in children with β -thalassemia major. *Pediatric Hematology and Oncology*, 29(2), 95–100.
- Sayani FA, Kwiatkowski JL. Increasing prevalence of thalassemia in America: implications for primary care. *Annals of medicine*. 2015 Oct 3;47(7):592-604.
- Cappellini MD, Cohen A, Porter J, Taher A, Viprakasit V, editors. Guidelines for the management of transfusion dependent thalassaemia (TDT). 2014.
- Khan MI, Rabnawaz M, Ullah I, Ullah I, Ahmad M. Frequency of beta thalassemia major ABO blood groups in different foundation of district Peshawar. *National Journal of Life and Health Sciences*. 2024 Mar 30;3(1):30-4.
- Waheed K, Rizvi SM, Mubarak B. Association of β -Thalassemia and its types with ABO and Rh blood groups in Lahore, Pakistan. *BioScientific Review*. 2023 Jun 27;5(2):10-7.
- Solanki SD, Amin R, Mehta NC. A study to find the distribution of ABO blood group in beta thalassemia. *Int J Sci Res*. 2020;9(9).
- Al-Masaoodi Ra, Alwaid Sh, Al-Shemery Mk. Determination The Effect Of Iron Overload On Vitamin D And The Percentage Of Each Blood Groups In Patients With Thalassemia. *Periódico Tchê Química*. 2020 Sep 1;17(36).
- Shanthi G, Balasubramanyam D, Srinivasan R. Studies on the Haematological Aspects of Beta (β)-Thalassemia in Tamilnadu. *J. Pharm. Bio. Chem Sci*. 2013;4(3):784.
- Bhuiyan MN, Giti S, Akhter M, et al. Haematological and Biochemical status of Adolescent and Young Adults with Transfusion dependent thalassaemia-A study of 40 cases. *Bioresearch Communications-(BRC)*. 2020 Jan 1;6(1):782-90.
- Munir B, Iqbal T, Jamil A, et al. Effect of β -Thalassemia on Hematological and Biochemical Profiles of female Patients. *Pakistan Journal of life and social Sciences*. 2013 Mar 1;11(1):25-8.
- Alkenany MR, Khalil LW. The effect of Shiitake mushroom derived Lentinan (β -glucan) administration on some hematological and biochemical parameters in intact rabbits. *Int. J. Health Sci*:5845-53.
- Cappellini MD, Motta I. New therapeutic targets in transfusion-dependent and-independent thalassemia. *Hematology 2014, the American Society of Hematology Education Program Book*. 2017 Dec 8;2017(1):278-83.
- Taher AT, Musallam KM. Coagulation and thrombotic risk in thalassemia intermedia. *Thalassemia Reports*. 2011 Dec;1(52):e15.
- Jabbar,A. Hassan, , Alsailawi,H. Ali, , Mudhafar,M. , , Alkenany,M. Riyadh,, Hussny,H. Askar, ,Ibrahim,I.H., Mahdi,H. Mahmood, , Almosawey,H. Ali Salman, , Rashid Ghareeb,A. Kareem, and Mohamed,S. Hameed. "Antibiotic Resistance of Non-Lactose-Fermenting Gram-Negative Bacteria in Urinary Tract Infections", *Asian Journal of Green Chemistry*, 9, 4, 2025, 404-414. doi: 10.48309/AJGC.2025.506240.1691
- Ming L, Jiang Z, Ma J, Wang Q, Wu F, Ping J. Platelet-to-lymphocyte ratio, neutrophil-to-lymphocyte ratio, and platelet indices in patients with acute deep vein thrombosis. *Vasa*. 2018 Jan 12.

Orcid ID:

Muhammad Riyadh Alkenany - <https://orcid.org/0000-0001-8292-5629>