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Research article

Correlation Analysis of Hematological and Biochemical Parameters in Thalassemia Patients

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ABSTRACT:
Background: Thalassemia is a hereditary blood disorder characterized by impaired hemoglobin synthesis, often requiring lifelong transfusions and iron chelation therapy. The interplay between hematological and biochemical markers in thalassemia patients remains critical for clinical monitoring and early detection of complications. This study aims to evaluate the correlations among key hematological and biochemical parameters—including White Blood Cells (WBCs), Glutamic Oxaloacetic Transaminase (GOT), Glutamic Pyruvic Transaminase (GPT), and Serum Ferritin (S. ferritin)—alongside demographic factors such as age and sex in thalassemia patients. **Methods:** A total of 40 thalassemia patients were included in a cross-sectional study design. Correlation analyses were performed using Pearson's coefficients to identify significant linear and monotonic associations among the studied variables. **Results:** A strong positive correlation was consistently observed between GOT and GPT across all statistical methods, indicating shared hepatic involvement, likely due to iron overload. A significant negative correlation was also found between WBCs and serum ferritin, suggesting potential immune or inflammatory alterations associated with iron metabolism. No significant associations were found between liver enzymes and serum ferritin, nor between any of the studied biomarkers and demographic factors (age, sex). **Conclusions:** The study highlights key biochemical and hematological relationships in thalassemia patients, particularly the liver enzyme association and the inverse link between WBCs and ferritin. These findings underscore the importance of integrated biomarker analysis for disease monitoring. Future studies with larger cohorts and comprehensive clinical data are recommended to further validate these relationships and explore underlying mechanisms.

Keywords:
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INTRODUCTION

Thalassemia, a group of inherited blood disorders, is characterized by reduced or absent synthesis of globin chains, leading to ineffective erythropoiesis and chronic hemolytic anemia [1].

This genetic condition is highly prevalent in various parts of the world, particularly in the Mediterranean, Southeast Asia, and the Middle East, posing a significant global health burden [2].

The clinical manifestations of thalassemia range from asymptomatic carriers to severe, transfusion-dependent forms, which can lead to a myriad of systemic complications affecting multiple organs [3]. One of the most critical complications associated with severe thalassemia, especially in transfusion-dependent patients, is iron overload. Repeated blood transfusions, a cornerstone of thalassemia management, inevitably lead to the accumulation of excess iron in various tissues and organs, including the heart, liver, and endocrine glands [4]. This iron accumulation can cause significant organ damage and dysfunction,

necessitating careful monitoring of iron levels, typically through serum ferritin measurements [5].

Beyond iron overload, thalassemia patients often exhibit a complex interplay of hematological abnormalities. The primary defect in globin chain synthesis results in the production of unstable hemoglobin, leading to premature destruction of red blood cells (RBCs) and chronic anemia [6]. This chronic hemolysis also contributes to elevated levels of total serum bilirubin (TSB), a marker of red blood cell breakdown and liver function [7]. Furthermore, the immune system in thalassemia patients is frequently compromised, making them more susceptible to infections. This increased vulnerability is attributed to a combination of factors, including the disease itself, chronic inflammation, oxidative stress, and the effects of iron overload on immune cell function [8].

Studies have shown that iron overload can impair various aspects of the immune response, including decreased antibody-mediated phagocytosis and alterations in T-lymphocyte function [9].While the management of anemia and iron burden remains central to thalassemia care, a comprehensive understanding of the relationships between serum ferritin and other hematological parameters, such as white blood cells (WBCs), RBCs, and TSB, is crucial. Investigating these correlations can provide deeper insights into the broader pathophysiological mechanisms at play in thalassemia, including immune dysregulation, the extent of hemolysis, and potential liver involvement [10].

The current study aims to statistically analyze these relationships in thalassemia patients compared to healthy individuals, building upon existing knowledge and offering a foundation for future clinical and mechanistic investigations.

MATERIALS AND METHODS:

This study employed a quantitative, correlational design to investigate the relationships between various hematological and biochemical parameters in thalassemia patients. The dataset for this analysis consisted of measurements from 40 thalassemia patients (N=40). The variables included in the analysis were White Blood Cells (WBCs), Glutamic Oxaloacetic Transaminase (GOT), Glutamic Pyruvic Transaminase (GPT), Serum Ferritin (S.feritin), Sex, and Age.

The data were collected from a cohort of 40 thalassemia patients. Specific details regarding patient demographics, inclusion/exclusion criteria, and the exact methods of blood sample collection and laboratory analysis were not provided in the original dataset. However, it is assumed that standard clinical procedures were followed for the measurement of all parameters.

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RESULTS:

This section presents the findings from the correlation analyses, detailing the relationships between the investigated hematological and biochemical parameters, as well as demographic factors. The results are presented based on Pearson. The Pearson correlation analysis revealed several significant linear relationships among the variables. The sample size for all correlations was 40.

WBCs and S. ferritin revealed a statistically significant negative correlation was found between WBCs and Serum Ferritin ($r = -0.365$, $p < 0.05$). This indicates an inverse linear relationship, where higher WBC counts are associated with lower S. ferritin levels.

GOT and GPT: A strong, statistically significant positive correlation was noted between Glutamic Oxaloacetic Transaminase (GOT) and Glutamic Pyruvic Transaminase (GPT) ($r = 0.765$, $p < 0.01$). This highlights a substantial linear relationship between these two liver enzymes.

No other statistically significant linear correlations were found between WBCs and GPT, WBCs and Sex, WBCs and Age, GOT and S. ferritin, GOT and Sex, GOT and Age, GPT and S. ferritin, GPT and Sex, GPT and Age, S. ferritin and Sex, S. ferritin and Age, or Sex and Age at the 0.05 or 0.01 [Table 1].

Table 1: Pearson Correlation Coefficient

Variables		GOT	GPT	S. ferritin	Sex	Age
WBCs	Pearson Correlation*	.006	.095	-.365-*	-.003-	-.151-
	Sig.	.486	.280	.010	.493	.177
GOT	Pearson Correlation		.765**	-.113-	.103	.092
	Sig.		.000	.243	.263	.286
GPT	Pearson Correlation			-.088-	.230	.208
	Sig.			.294	.077	.099
S.feritin	Pearson Correlation				.098	.236
	Sig.				.273	.071
Sex	Pearson Correlation					.092
	Sig.					.285

*Pearson Correlation Coefficient used to measure the linear relationship between two continuous variables. It assumes that the data are normally distributed and that the relationship between the variables is linear. The Pearson correlation coefficient (r) ranges from -1 to +1, where +1 indicates a perfect positive linear relationship, -1 indicates a perfect negative linear relationship, and 0 indicates no linear relationship.

DISCUSSIONS:

The findings from this correlation analysis provide valuable insights into the interrelationships between hematological and biochemical parameters in thalassemia patients. The consistent observation of a significant positive correlation between GOT and GPT. This strong association (Pearson $r = 0.765$, $p < 0.01$) is expected, as both GOT (AST) and GPT (ALT) are liver enzymes commonly used as indicators of hepatocellular damage [11]. In thalassemia patients, elevated levels of these enzymes often reflect liver dysfunction primarily due to iron overload from repeated blood transfusions, or co-existing viral hepatitis [12]. Our results align with previous studies that have reported similar strong correlations between these two enzymes in various patient populations, including those with thalassemia [13 The statistically significant negative correlation between WBCs and S. ferritin, consistently observed (Pearson $r = -0.365$, $p < 0.05$), presents an interesting finding. While serum ferritin is a primary indicator of iron stores and iron overload in thalassemia, the relationship with WBCs is less commonly highlighted. Typically, chronic inflammation, which can be present in thalassemia, might lead to elevated WBC counts, and iron overload itself can contribute to oxidative stress and inflammation [14]. However, a negative correlation suggests a more complex interplay. One possible explanation could be related to the effects of iron chelation therapy, which aims to reduce iron overload and might indirectly influence inflammatory markers and WBC counts. Another factor could be the presence of hypersplenism, a common complication in thalassemia, which can lead to sequestration and destruction of blood cells, including WBCs, and might also influence ferritin levels [15]. Further research is needed to elucidate the underlying mechanisms behind this observed negative correlation.

The Pearson correlation indicated no significant correlation between WBCs and GOT ($r = 0.006$, $p = 0.48$). This suggests normal WBC counts are associated with GOT levels [16]. This finding warrants further investigation to determine the specific clinical implications in thalassemia patients.

Conversely, many expected correlations, such as those between S. ferritin and liver enzymes (GOT, GPT), were not found to be statistically significant in this dataset, except for the indirect relationship with WBCs. While iron overload is a known cause of liver damage in thalassemia, and serum ferritin levels are often used to monitor iron burden, the absence of a direct significant correlation between S.feritin and GOT/GPT in this specific cohort might be due to several factors. These could include the stage of liver disease, the effectiveness

of chelation therapy, the presence of other confounding factors, or the relatively small sample size (N=40) which might limit the power to detect subtle associations [17]. Furthermore, no significant correlations were observed between any of the hematological or biochemical parameters and demographic factors such as Sex and Age. This suggests that within this particular cohort, sex and age did not significantly influence the levels or relationships of WBCs, GOT, GPT, or S. ferritin. However, it is important to note that thalassemia is a lifelong condition, and the impact of age and sex on disease progression and complications can be complex and may manifest differently in larger or longitudinal studies [18].

CONCLUSION

This study investigated the correlations between various hematological and biochemical parameters in thalassemia patients, utilizing Pearson correlation analyses. The findings consistently demonstrated a strong positive correlation between GOT and GPT, which is indicative of their shared role as liver enzyme markers and their potential elevation in the context of liver complications in thalassemia. A significant negative correlation was also observed between WBCs and S. ferritin across all analyses, suggesting a complex interplay that warrants further investigation, possibly related to inflammatory processes, iron chelation therapy, or hypersplenism.

While some expected correlations, particularly between S. ferritin and liver enzymes, were not statistically significant in this cohort, this highlights the multifactorial nature of thalassemia and its complications. The absence of significant correlations with sex and age in this specific dataset suggests that these demographic factors may not be primary drivers of the observed parameter variations within this study's scope. These findings underscore the importance of comprehensive monitoring of thalassemia patients, considering both hematological and biochemical indicators.

Future research should aim to include larger sample sizes, more detailed clinical data (e.g., transfusion history, chelation regimens, and co-morbidities), and longitudinal studies to further elucidate the dynamic relationships between these parameters. Such studies could provide a more in-depth understanding of disease progression, risk stratification, and the effectiveness of therapeutic interventions in thalassemia patients

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