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EVALUATION OF SERUM IRON PROFILE IN BETA THALASSEMIA PATIENTS

Original Research

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ABSTRACT

Background: Beta-thalassemia is an inherited hemoglobinopathy caused by mutations in the beta-globin gene, resulting in reduced or absent beta-globin chain synthesis. The imbalance in globin chains leads to ineffective erythropoiesis and severe anemia, especially in transfusion-dependent individuals. In regions like Pakistan, where the carrier frequency is high, regular blood transfusions are essential for survival in beta-thalassemia major patients. However, repeated transfusions contribute to progressive iron overload, making timely monitoring and management crucial. Serum ferritin serves as a simple, noninvasive, and cost-effective biomarker for assessing iron burden.

Objective: To evaluate the serum iron profile and transfusion frequency among beta-thalassemia major patients.

Methods: This cross-sectional study included 100 electrophoresis-confirmed beta-thalassemia major patients aged 2 to 19 years, admitted at Alkhidmat Thalassemia Care Centre, Lahore. Blood samples (2–3 mL) were collected using gel separator vials. Serum ferritin levels were analyzed using both manual ELISA and automated chemiluminescence immunoassays. Hemoglobin levels and the number of lifetime transfusions were also recorded. Data were analyzed using SPSS version 17.0.

Results: Among 100 patients, 55 were male (55%) and 45 female (45%), with a mean age of 9.68 ± 4.843 years. The average number of transfusions was 92.92 ± 34.677. Mean serum ferritin concentration was 2376.72 ± 749.03 ng/mL. Ferritin levels were <1500 ng/mL in 3 patients (3%), 1500–2500 ng/mL in 64 patients (64%), and >2500 ng/mL in 33 patients (33%). Mean hemoglobin concentration was 7.208 ± 0.7062 g/dL.

Conclusion: Serum ferritin remains an accessible and reliable indicator of iron overload in beta-thalassemia major patients. The findings underscore the importance of routine monitoring and effective chelation therapy to reduce iron-induced complications, improve quality of life, and guide evidence-based patient care.

Keywords: Beta-thalassemia, Chelation therapy, Hemoglobin, Iron overload, Pakistan, Serum ferritin, Transfusions.

INTRODUCTION

β-thalassemia is a genetically inherited blood disorder characterized by the partial (β^+) or complete (β^0) absence of β-globin chain synthesis, leading to an imbalance in hemoglobin tetramers and resulting in ineffective erythropoiesis and chronic hemolytic anemia (1,2). The condition arises due to mutations in the β-globin gene, affecting the production of structurally normal β-globin chains. These mutations, although diverse, uniformly impair the stability and functionality of red blood cells by promoting excess unpaired α-globin chains, which precipitate in erythroid precursors and contribute to cell death and peripheral hemolysis (3,4). Globally, approximately 1.5% of the population are carriers of the β-thalassemia gene, with an estimated 60,000 affected births annually, predominantly in underdeveloped regions (5). South Asia is particularly impacted, with countries like Pakistan reporting a carrier frequency of 5–7% and an annual birth rate of over 5,000 children with β-thalassemia major (6,7). Similarly, in India, the prevalence varies regionally from 1–15%, emphasizing the genetic heterogeneity and endemic nature of the disorder across the Indian subcontinent, the Middle East, tropical Africa, and parts of Southeast Asia (8-10). Clinically, β-thalassemia is categorized into three forms: major, intermedia, and minor. The most severe, β-thalassemia major, typically manifests within the first two years of life with symptoms including growth retardation, severe anemia, and skeletal deformities. These patients are transfusion-dependent for survival. In contrast, β-thalassemia intermedia presents with milder anemia requiring intermittent transfusions, while β-thalassemia minor is usually asymptomatic and discovered incidentally (11). At the cellular level, the pathology of β-thalassemia is driven by the accumulation of excess unmatched α-globin chains, leading to oxidative stress and apoptosis in red blood cell precursors. This imbalance not only compromises erythropoiesis but also results in the generation of reactive oxygen species (ROS) that damage multiple organs including the liver, heart, and endocrine system (11,12). Interestingly, co-inheritance of α-thalassemia can ameliorate the clinical phenotype by reducing the burden of excess α-globin (13).

A major complication in β-thalassemia management is iron overload, which arises from both increased gastrointestinal absorption—due to suppressed hepcidin—and repeated blood transfusions. Each transfused unit contributes approximately 250 mg of iron, and patients often accumulate up to 5 grams annually (5-7). The resulting non-transferrin bound iron catalyzes the formation of harmful ROS, contributing to organ dysfunction through lipid peroxidation and aldehyde production (8,9). Serum ferritin levels, though useful as a marker of iron burden, are influenced by age, inflammation, and liver status, necessitating regular monitoring for optimal management (10,12). Chelation therapy has become essential to mitigate iron toxicity in β-thalassemia patients. Early and consistent chelation improves survival and reduces the risk of cardiac, hepatic, and endocrine complications (12,13). Although deferoxamine was historically the standard treatment, it posed challenges in pediatric compliance. The advent of oral chelators has improved adherence and efficacy (14). Moreover, iron chelation is now being explored in related conditions such as sickle cell anemia, aplastic anemia, and post-hematopoietic stem cell transplantation scenarios, where iron overload remains a significant prognostic factor (15-17). Non-transfusion dependent thalassemia (NTDT) presents a unique challenge. Despite not receiving regular transfusions, these patients still accumulate iron due to increased intestinal absorption. Compared to β-thalassemia major, NTDT patients exhibit lower serum ferritin levels for equivalent liver iron concentrations (LIC), making reliance on serum ferritin alone misleading (18). Therefore, accurate assessment of LIC is crucial, although invasive procedures like liver biopsy are less preferred due to associated risks (13). Given these clinical complexities, a holistic approach to thalassemia management necessitates improved diagnostic modalities, early initiation of chelation therapy, and tailored monitoring protocols. This study aims to explore the relationship between serum ferritin levels and body iron burden in β-thalassemia patients, emphasizing the significance of timely interventions in minimizing transfusion-related complications and improving patient outcomes.

METHODS

This study was conducted following the approval of the Ethical Review Committee of Riphah International University and adhered to established research ethics, including the collection of informed verbal consent from all participating patients or their legal guardians prior to data collection. The study employed a cross-sectional design and was carried out over a period of six months. Data were collected from Alkhidmat Thalassemia Centre, while the electrophoretic confirmation of β-thalassemia in the recruited patients had already been performed at the diagnostic laboratory of Surraya Azeem Hospital. A total of 100 electrophoresis-confirmed patients with β-thalassemia,

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aged between 2 to 19 years, were enrolled through non-probability purposive sampling. Eligibility was limited to patients of either gender who were undergoing treatment at the thalassemia centers and had received at least two blood transfusions per month. Patients with any documented history of chronic infections or acute illness during transfusion-based therapy were excluded from the study to eliminate confounding variables related to inflammation or acute-phase responses, which may alter serum ferritin levels. Approximately 2–3 mL of venous blood was collected aseptically from each participant into gel separator tubes for biochemical analysis. The serum samples were processed and analyzed for serum ferritin concentration using both manual and automated methods to ensure accuracy and validation. The manual detection of ferritin was carried out through a strip-based enzyme-linked immunosorbent assay (ELISA) using the ANTEC Diagnostic Products UK kit and Readwell Strip ELISA reader. In parallel, the automated quantification of serum ferritin was performed on the Roche Cobas e411 immunoassay analyzer utilizing chemiluminescent technology, offering enhanced precision and efficiency.

In addition to ferritin, serum iron concentration was assessed using the Roche/Hitachi Cobas C system through a colorimetric assay. This method relies on the acidic release of iron from transferrin, followed by its reduction and reaction with FerroZine to form a colored complex, whose absorbance is directly proportional to the serum iron concentration. The reaction involves thiourea, citric acid, and sodium ascorbate as key reagents, ensuring standardized assay conditions. Interpretation of serum iron levels adhered to established reference ranges for adults (5.83–34.5 µmol/L or 33–193 µg/dL), though it is important to note that pediatric reference values may differ and were not specifically addressed in this protocol. Hemoglobin electrophoresis was confirmed using the Sebia Capillary Electrophoresis (OCTA 3) system, which separates hemoglobin fractions based on electrophoretic mobility within a narrow capillary under an electric field (6,7). This method allows rapid and precise separation of hemoglobin variants, utilizing a reference library of over 400 known variants. The system provides a graphical readout with standard interpretation ranges for HbA (96.8–97.8%), HbA2 (1.5–3.5%), and HbF (0.0–1.0%), aiding in diagnostic confirmation. Serum ferritin levels were further validated using an enhanced immunoturbidimetric assay on the Roche/Hitachi Cobas C platform. The technique involves the agglutination of latex particles coated with anti-human ferritin antibodies, measured turbidimetrically at dual wavelengths (570/800 nm). Reference ranges were interpreted as 30–400 µg/L for adult males and 15–150 µg/L for adult females; however, age-specific pediatric reference values were not delineated, which may limit precision in this age group. All laboratory analyses were performed in accordance with manufacturer protocols and standard biosafety guidelines. The use of multiple detection methods for serum ferritin adds methodological robustness.

RESULTS

A total of 100 patients diagnosed with beta thalassemia major were enrolled, comprising 55 males (55%) and 45 females (45%). The mean age of the participants was 9.68 ± 4.843 years, ranging from 2 to 19 years. The median age was 8.0 years, with the most frequently observed age being 7 years. Patients began transfusion therapy between 2 to 18 months of age. The number of lifetime blood transfusions ranged from 26 to 145, with a mean of 92.94 ± 34.677. The average annual blood transfusion requirement was recorded as 300.01 ml/kg/year, within a range of 250 to 350 ml/kg/year. The hemoglobin concentration at the time of data collection ranged from 5.8 g/dL to 8.9 g/dL, with a mean value of 7.2 ± 0.7062 g/dL. Notably, 85% of patients had hemoglobin levels below the recommended threshold of 9.6 g/dL, indicating suboptimal transfusion targets in the majority. All patients were undergoing iron chelation therapy with deferoxamine during the study period. Serum ferritin concentration, a key indicator of iron overload, ranged from 1409 to 3789 ng/mL with a mean value of 2376.72 ± 749.03 ng/mL. Ferritin levels below 1500 ng/mL were observed in 3% of patients, between 1500 and 2500 ng/mL in 64%, and above 2500 ng/mL in 33%. A greater proportion of patients aged above 10 years exhibited serum ferritin concentrations exceeding 2500 ng/mL. When stratified by gender, mean serum ferritin concentration in male patients was 2271.10 ± 656.58 ng/mL, while it was slightly higher in female patients at 2505.80 ± 838.05 ng/mL. However, the difference between male and female patients was not statistically significant ($p = 0.78$). Statistical analysis revealed a strong positive correlation between the number of transfusions and serum ferritin levels ($r = 0.717$, $p < 0.01$), indicating that an increased number of transfusions was associated with a higher iron burden. Furthermore, age also showed a very strong positive correlation with serum ferritin levels ($r = 0.910$, $p < 0.01$), suggesting progressive iron accumulation with increasing age in thalassemia patients.

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Table 1: Descriptive analysis of age, no. of transfusions, serum ferritin and Hb level

	N	Minimum	Maximum	Mean	Std. Deviation
Age	100	2	19	9.68	4.843
no.of transfusions	100	26	145	92.94	34.677
serum ferritin	100	1409	3789	2376.72	749.039
Hb level	100	5.8	8.9	7.208	.7062
Valid N (listwise)	100				

Table 2: Gender distribution of beta thalassemia patients

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Female	45	45	45	45
	Male	55	55	55	100.0
	Total	100	100.0	100.0	

Table 3: Age distribution of beta thalassemia patients

N	Valid	100
	Missing	0
Mean		9.68
Median		8.00
Mode		7
Std. Deviation		4.843
Minimum		2
Maximum		19

Table 4: Serum ferritin ranges and percentages

Serum Ferritin Range ng/ml	No. of Patients	Percentage
<1500	3	3%
1500-2500	64	64%
>2500	33	33%

Table 5: Correlations between serum ferritin and no. of transfusions

		Serum Ferritin	No. of Transfusions
Serum Ferritin	Pearson Correlation	1	.717**
	Sig. (2-tailed)		.000
	N	100	100
No. of Transfusions	Pearson Correlation	.717**	1
	Sig. (2-tailed)	.000	
	N	100	100

**. Correlation is significant at the 0.01 level (2-tailed).

Table 5(a): Correlation between Number of transfusions and serum ferritin level

	Value	Significance level 5% = P value <0.05
Pearson's correlation r=	.717	0.01

Table 5(b): Number of transfusion and age of patients

	Value	Significance level 5% = P value <0.05
Pearson's correlation r=	.910	0.01

Significance level 5% = P value <0.05

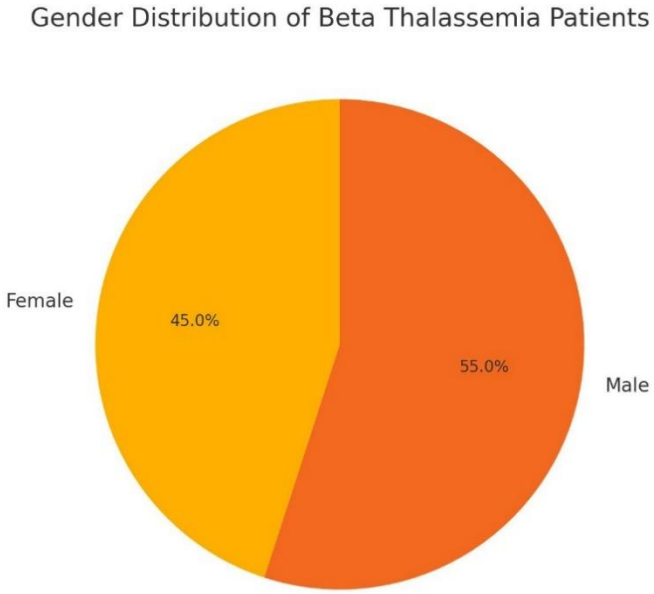

Figure 1 Gender Distribution of Beta Thalassemia Patients

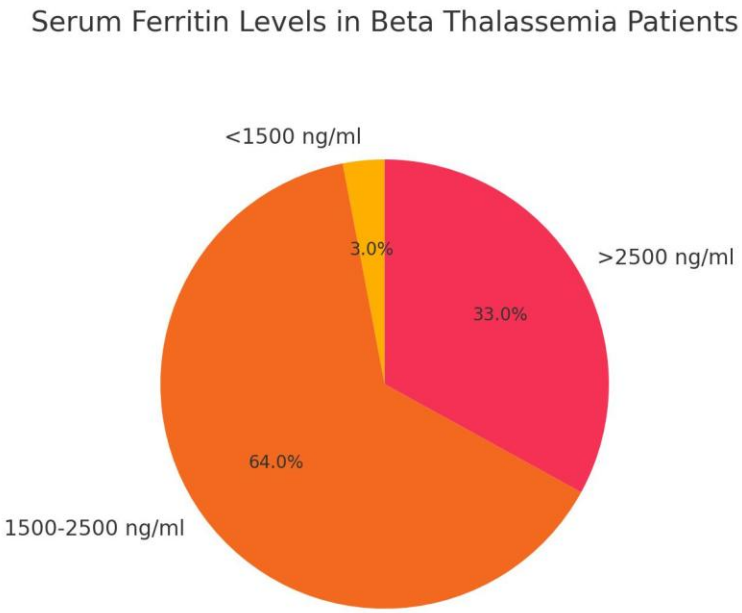

Figure 2 Serum Ferritin Levels in Beta Thalassemia Patients

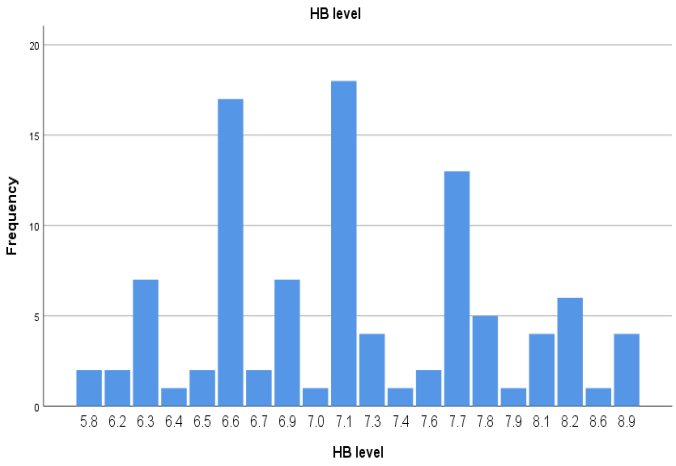

Figure: Hb Levels of Beta Thalassemia Patients

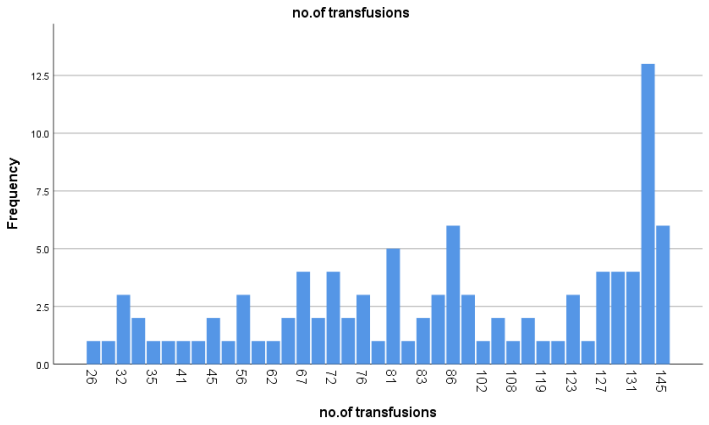

Figure: Number of transfusions of Beta Thalassemia Patients

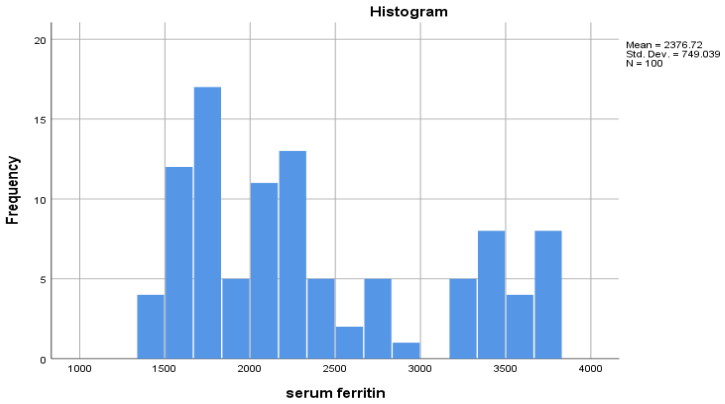

Figure: Serum Ferritin Levels of Beta Thalassemia Patients

DISCUSSION

Beta thalassemia major remains a significant public health concern in Pakistan, where it continues to represent one of the most common hereditary hemoglobinopathies. In patients undergoing regular blood transfusions, secondary complications such as iron overload and growth retardation frequently compromise treatment outcomes and quality of life. This study evaluated serum ferritin concentrations, transfusion frequency, and the overall iron overload status in a cohort of transfusion-dependent beta thalassemia major patients, all of whom were undergoing iron chelation therapy. The mean serum ferritin level in this study population was 2376.72 ± 749.03 ng/mL, which is markedly elevated compared to the standard reference range for children, typically cited between 12–122 ng/mL (10,14). A significant proportion of patients (33%) had serum ferritin concentrations exceeding 2500 ng/mL, indicating a high iron burden despite ongoing chelation therapy. Comparatively, similar studies reported a wide range of mean serum ferritin values, varying from 1696 ng/mL to over 5700 ng/mL in multi-transfusion beta thalassemia patients (15-18). These findings reinforce the critical need for early, regular, and adequately dosed iron chelation to prevent cumulative iron toxicity. The positive correlations found between serum ferritin levels, age, and number of transfusions strongly suggest that iron accumulation is progressive and closely tied to transfusion intensity. The observed mean of 92.92 ± 34.677 transfusions per patient aligns with reports indicating an increasing trend of iron overload with transfusional history.

In this context, chelation therapy should be individualized and closely monitored, particularly in older patients or those with prolonged transfusion histories. Serum ferritin levels above 1000 ng/mL generally indicate the need to initiate chelation therapy (19), making routine ferritin monitoring an essential tool in disease management. In terms of hemoglobin status, the mean hemoglobin concentration in this cohort was 7.208 ± 0.7062 g/dL. This is comparable to other studies, which have reported average levels around 7.1 g/dL in similar patient populations (20,21). However, these values are below the recommended pre-transfusion hemoglobin threshold of 9.6 g/dL for thalassemia major, indicating that transfusion adequacy remains a concern in clinical practice. Suboptimal transfusion regimens may not only compromise oxygen delivery but may also contribute to increased erythropoietic drive and subsequent gastrointestinal iron absorption, worsening the iron burden (22). Although all patients were receiving deferoxamine-based chelation therapy, the considerable variation in serum ferritin levels suggests variability in treatment compliance, dosage, or pharmacodynamic response. Moreover, the use of a single-point ferritin measurement is a recognized limitation, as it may not accurately reflect total body iron burden, particularly in the presence of inflammation or hepatic dysfunction. Ideally, serial measurements or more direct methods such as liver iron concentration (LIC) using MRI should be considered to provide a clearer picture of iron status.

Another limitation of the study is its single-center design and relatively small sample size, which limits generalizability to the broader population of beta thalassemia patients across Pakistan. The absence of a control group and the lack of growth comparison with age- and sex-matched healthy individuals further restrict the ability to conclusively determine associations between iron overload and growth failure, which has been reported as a common complication in thalassemia despite chelation therapy (23). Despite these limitations, the study offers valuable insights into the current status of iron overload and transfusion practices in a representative sample of thalassemia patients. Its strength lies in the comprehensive evaluation of key clinical markers and the use of standardized laboratory methods for serum ferritin estimation. The consistent association between iron load and transfusion history emphasizes the need for strict transfusion

protocols and enhanced chelation compliance. Future research should prioritize multicenter studies with larger sample sizes and longitudinal follow-up to assess the effectiveness of different chelation regimens. Incorporating molecular diagnostics and assessing liver iron concentration through non-invasive imaging could further refine clinical decision-making. Evaluating additional outcomes such as cardiac function, endocrine status, and growth metrics in comparison with healthy controls would also enrich understanding of disease burden and management efficacy. In conclusion, the findings highlight the continued challenge of managing iron overload in transfusion-dependent beta thalassemia major. While serum ferritin remains a useful and practical biomarker, comprehensive monitoring strategies and individualized treatment protocols are critical to improving long-term outcomes in this vulnerable population.

CONCLUSION

This study concluded that beta thalassemia major patients experience significant iron overload, as reflected by elevated serum ferritin levels, necessitating timely and effective iron chelation therapy. The association between frequent blood transfusions and rising iron burden emphasizes the need for careful transfusion management and regular monitoring. Hemoglobin levels remained below recommended thresholds, suggesting suboptimal transfusion support. These findings highlight the importance of comprehensive care strategies that include early diagnosis, appropriate chelation, and continuous follow-up to prevent iron-related complications. Raising awareness and implementing preventive measures can play a vital role in improving the quality of life and long-term outcomes for individuals living with beta thalassemia major.

AUTHOR CONTRIBUTION

Author	Contribution
Talha Ahmad	Substantial Contribution to study design, analysis, acquisition of Data Manuscript Writing Has given Final Approval of the version to be published
Asia Jahanzaib	Substantial Contribution to study design, acquisition and interpretation of Data Critical Review and Manuscript Writing Has given Final Approval of the version to be published
Tayyaba Baloch	Substantial Contribution to acquisition and interpretation of Data Has given Final Approval of the version to be published
Hamza Imtiaz	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Muhammad Iqbal	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Muhammad Nayyer Shaban	Substantial Contribution to study design and Data Analysis Has given Final Approval of the version to be published
Zarmeen Zahra Ali	Contributed to study concept and Data collection Has given Final Approval of the version to be published
Muhammad Zeshan	Writing - Review & Editing, Assistance with Data Curation
Anas Jahangir*	Writing - Review & Editing, Assistance with Data Curation
Fatima Ashraf Mughal	Writing - Review & Editing, Assistance with Data Curation

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