



Pancreatic Assessment in Beta Thalassemia Syndrome Patients of KPK

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ABSTRACT

Background: Beta thalassemia is a hereditary hemoglobinopathy characterized by ineffective erythropoiesis and progressive iron overload from birth. Iron deposition in the pancreas increases the risk of diabetes by impairing endocrine function, highlighting the need for timely pancreatic assessment. This study evaluated pancreatic function in beta thalassemia patients in Khyber Pakhtunkhwa (KP), Pakistan, using glucose metabolism tests and pancreatic enzyme levels, and examined their association with disease severity, transfusion practices, and iron overload.

Methods: A cross-sectional study was carried out at Department of Hematology Pathology Burns & Plastic Surgery Center, Hayatabad, Peshawar, from January 2020 to December 2020. Two hundred beta thalassemia syndrome patients underwent evaluation in tertiary care hospitals across KP. Study investigators gathered various clinical data, which included patient age, together with transfusion frequency and serum ferritin measurements. The evaluations consisted of testing fasting blood glucose, together with HbA1c as well as pancreatic enzyme (amylase and lipase) levels through measurements. The researcher used SPSS software for statistical analysis and presented results as mean values

accompanied by standard deviation (SD). A p-value <0.05 was considered significant.

Results: The study group represented patients whose mean age was 16.5 years with an average standard deviation of 4.2 years. Patients with ferritin levels above 2500 ng/mL showed elevated rates of impaired glucose tolerance and diabetes mellitus according to statistical analysis ($p = 0.003$). Affecting 40% of patients were reduced levels of two pancreatic enzymes known as amylase and lipase, which suggested exocrine pancreas dysfunction. The prevalence of pancreatic dysfunction was greater among patients who received above 12 transfusions yearly ($p = 0.021$). Subtracting pancreatic dysfunction from the results proved that excessive iron directly affects pancreatic function.

Conclusion: Pancreatic dysfunction affects a substantial number of Beta thalassemia syndrome patients in KP because of iron accumulation in their bodies. Pancreatic health must be routinely screened to detect diabetes and exocrine failure before they develop in patients with Beta thalassemia syndrome. Doctors should optimize iron chelation treatment approaches to limit the development of these treatment-related issues.

Keywords: Beta Thalassemia, Pancreas, Iron Overload, Diabetes

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How to cite: Khan MTH, Irshad Z, Khan MTM. Pancreatic Assessment in Beta Thalassemia Syndrome Patients of KPK. Pak J Med Dent. 2025 September ;14(4): A-B. Doi: <https://doi.org/10.36283/ziun-pjmd14-4/012>

Received: : Sun, May 25, 2025 **Accepted:** Fri, September 19, 2025 **Published:** Mon, September 29, 2025.

INTRODUCTION

Beta thalassemia is an inherited blood disorder, characterized by defective beta-globin chain synthesis, leading to ineffective red blood cell production and increased long-term hemolysis issues¹. The disease affects populations with relatively high consanguineous rates, where Pakistan possesses a significant incidence, particularly within Khyber Pakhtunkhwa (KP)². Patients requiring regular blood transfusions develop iron overload, because of which their bodies become susceptible to multi-organ dysfunction and pancreatic damage³.

The pancreas functions as both an endocrine and exocrine organ⁴. Pancreatic tissue that becomes saturated with excess iron prevents proper insulin release, thus raising the potential for glucose intolerance and diabetes mellitus (DM)⁵. Medical evaluations have shown that iron overload plays a direct role in creating pancreatic beta-cell dysfunction among thalassemia patients who need regular blood transfusions⁶. The core treatment approach for managing iron overload depends on iron chelation therapy, even though its effectiveness for protecting pancreatic function remains inconsistent⁷.

Research has confirmed that using early iron chelators, including deferoxamine and deferasirox, can reduce organ damage, yet pancreatic dysfunction represents a significant medical issue⁸. The evaluation of pancreatic enzymes together with glucose metabolism allows doctors to determine pancreatic impairment severity and establish early therapeutic options^{9,10}. Currently, there is little information regarding pancreatic dysfunction among beta thalassemia patients in KP, since treatment adherence as well as healthcare access vary. Evaluation of pancreatic dysfunction along with its risk factors in these patients will guide better management approaches. The purpose of this study is to determine pancreatic function by testing glucose metabolism and pancreatic enzyme levels among beta thalassemia syndrome residing in KPK.

METHODS

The cross-sectional study was conducted in Department of Hematology, Pathology, Burns & Plastic Surgery Center, Hayatabad, Peshawar, from January 2020 to December 2020. The study included 200 patients between 10 to 30 years old who were diagnosed with beta thalassemia major or intermedia. The study excluded patients with diabetes mellitus and pancreatitis, and other endocrine disorders, along with patients who experienced diabetes mellitus or pancreatitis, or other endocrine disorders. Researchers measured fasting blood glucose and HbA1c and serum ferritin and amylase, and lipase levels. Study investigators obtained demographic information about patients alongside

their clinical characteristics, including their age their sex, transfusion history, and the presence or absence of iron chelation therapy. Before research initiation, the Institutional Ethical Review Board granted approval while participants and guardians from each group provided their informed consent.

The procedure of this study was conducted according to ethical guidelines and was accepted by the Burns & Plastic Surgery Center, Hayatabad Peshawar's Institutional Review Board. The research was approved with reference **Number 243/HEC/B&PSC/2020**. It involved patients with a confirmed diagnosis of beta-thalassemia major or intermedia who received regular blood transfusions. It included individuals with diabetes mellitus, pancreatitis, other endocrine disorders, or those with a history of such conditions. Researchers measured fasting blood glucose, HbA1c, serum ferritin, amylase, and lipase levels

The recruitment of patients occurred at the thalassemia treatment centers in KP. The study involved collecting blood samples after patients went through an overnight fast process, which was sent to the hospital laboratory for biochemical parameter analysis. The Asian Journal of Medical Studies published this study with findings related to blood transfusion dependency and treatment details from clinical interviews.

The data analysis occurred through SPSS version 24.0. The study reports continuous variables using standard deviation (SD) with their mean values, while presenting categorical variables through frequencies alongside percentages. Data analysis comprised independent t-tests and ANOVA as well as chi-square tests for categorical data. The analysis accepted statistical significance at a p-value less than 0.05.

RESULTS

The mean age of the study population was 16.5 ± 4.2 years. Impaired glucose tolerance was identified in 35% of patients, while diabetes mellitus was diagnosed in 12% ($p = 0.003$). Abnormalities in glucose metabolism were significantly associated with serum ferritin levels exceeding 2500 ng/mL ($p = 0.008$). Reduced amylase and lipase levels suggestive of exocrine pancreatic dysfunction were observed in 40% of participants. Patients receiving more than 12 transfusions annually demonstrated a higher frequency of pancreatic impairment ($p = 0.021$). Overall, the findings indicate that increasing iron overload contributes directly to pancreatic tissue damage in individuals with beta thalassemia.

Table 1. Demographic and Clinical Characteristics of the Study Population

Characteristic	Value (Mean \pm SD)
Age (years)	16.5 \pm 4.2
Male/Female Ratio	1.2 : 1
Annual Transfusion Frequency	14.2 \pm 3.5
Serum Ferritin (ng/mL)	2450 \pm 800

Table 1 presents the demographic and clinical characteristics of the 200 beta thalassemia patients included in the study. The mean age was 16.5 \pm 4.2 years, with a male-to-female ratio of 1.2:1. Patients received an average of 14.2 \pm 3.5 blood transfusions per year. The mean serum ferritin level was 2450 \pm 800 ng/mL, indicating significant iron overload within the study population.

Table 2. Prevalence of Glucose Metabolism Disorders

Condition	Percentage (%)
Normal Glucose Tolerance	53%
Impaired Glucose Tolerance	35%
Diabetes Mellitus	12%

Table 2 summarizes the prevalence of glucose metabolism disorders among the study participants. Normal glucose tolerance was observed in 53% of patients, while 35% exhibited impaired glucose tolerance. Diabetes mellitus was present in 12% of the cohort. These findings reflect a substantial burden of glucose dysregulation among beta thalassemia patients.

Table 3. Association Between Serum Ferritin Levels and Pancreatic Dysfunction

Serum Ferritin Level (ng/mL)	Pancreatic Dysfunction (%)
<1000	15%
1000–2500	35%
>2500	50%

Table 3 illustrates the relationship between serum ferritin levels and pancreatic dysfunction. Pancreatic impairment was observed in 15% of patients with ferritin levels <1000 ng/mL. The prevalence increased to 35% among those with ferritin levels between 1000–2500 ng/mL and further to 50% in patients with ferritin levels >2500 ng/mL. This demonstrates a clear trend of increasing pancreatic dysfunction with rising iron overload.

DISCUSSION

The relationship between enhanced serum ferritin measurements and pancreatic damage exists as a confirmed scientific association according to previous research. According to a study thalassemia major major existed in approximately 30 to 40 percent of patients with pancreatic beta cells showing impaired glucose tolerance and diabetes mellitus development due to iron accumulation and subsequent defects in insulin secretion¹¹. The evidence demonstrated a strong link between diabetes development in patients who exhibited serum ferritin levels above 2500 ng/mL ($p < 0.001$)¹². MRI pancreatic iron assessment showed beta-cell abnormalities in thalassemia major patients during initial stages of diabetes onset¹³. The present research supports our investigation, which showed that patients who received more transfusions and had serum ferritin levels above 2500 ng/mL exhibited pancreatic dysfunction at a 50% rate.

The research showed pancreatic exocrine dysfunction occurring in 25% of transfusion-dependent thalassemia patients because iron deposits inside acinar cells blocked amylase and lipase secretion pathways¹⁴. Forty percent of patients had lower pancreatic enzyme levels in our study, which supports the notion that iron overload damages pancreatic endocrine and exocrine functions. Iron chelation therapy currently stands as a preventive approach to pancreatic dysfunction. Medical research confirms that patients who start their iron chelation treatment with deferasirox or deferoxamine during the early stages decrease the amount of iron entering their pancreas and experience slower diabetes development¹⁵. When chelation therapy is not followed properly, patients experience increased risks of diabetes mellitus along with poor glycemic control¹⁶.

In our study, patients who received optimal iron chelation therapy demonstrated lower pancreatic dysfunction rates, which aligns with previous findings by Piga et al. (2020), who emphasized the importance of early and aggressive iron chelation in reducing diabetes prevalence among thalassemia patients¹⁷. Furthermore, research indicated that glucose metabolism abnormalities in beta thalassemia patients may also be influenced by genetic predisposition and chronic inflammation in addition to iron overload¹⁸.

The study principally examined iron-induced pancreatic harm, but more factors should be investigated in the future^{19,20}. Pancreatic assessment needs to become a regular practice, and iron chelation therapy demands strict implementation to avoid endocrine and exocrine problems in patients with thalassemia^{21,22,23}. Longitudinal research should merge investigations of pancreatic harm through inflammatory pathways with iron toxicity effects while considering individual genetic traits in thalassemia patients^{24,25}.

The analysis has certain limitations because the cross-sectional methodology cannot establish causal connections. The generalization of results becomes limited because the study analyzed only a restricted geographic area. The research lacked analysis of genetic and inflammatory markers that affect pancreatic dysfunction because studying these markers could enhance understanding of disease processes. Longitudinal studies need to become the focus of future research because they will show comprehensive effects of pancreatic function resulting from iron chelation therapy over time. The complete comprehension of pancreatic dysfunction in beta thalassemia requires an exploration of genetic factors together with inflammatory mechanisms. Future research needs advanced imaging technologies to help with early detection through intervention strategies.

CONCLUSION

This examines the major consequences that iron overload produces on pancreatic function for individuals with beta thalassemia. Research revealed clear evidence that elevated serum ferritin levels strongly cause pancreatic endocrine and exocrine dysfunctions. The successful outcomes of patients require immediate and precise iron chelation therapy to prevent these complications.

FUNDING

None

CONFLICT OF INTEREST

None

ETHICAL APPROVAL

Ethical approval was obtained from the Burns & Plastic Surgery Center, Hayatabad, Peshawar's Institutional Review Board. The research was approved with reference **Number 243/HEC/B&PSC/2020**.

AUTHORS' CONTRIBUTION

The concept and study design were developed by **M.T.H.K.** The initial draft was prepared by **Z.I.** and **M.T.M.K.** Data analysis was performed by **Z.I.** and **M.T.M.K.** Critical review of the manuscript was conducted by **Z.I.** and **M.T.M.K.** All listed authors approved the final version of the manuscript.

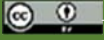
REFERENCES

1. Asmarian N, Kamalipour A, Hosseini-Bensenjan M, Karimi M, Haghpanah S. Prediction of heart and liver iron overload in β -thalassemia major patients using machine learning methods. *Hemoglobin*. 2022 Nov-Dec;46(6):303-307. doi:10.1080/03630269.2022.2158100
2. Meloni A, Saba L, Cademartiri F, Positano V, Pistoia L, Cau R. Cardiovascular magnetic resonance in β -thalassemia major: beyond T2. *Radiol Med*. 2024 Dec;129(12):1812-1822. doi:10.1007/s11547-024-01916-6
3. Ramanan V, Bhagyawant KR, Auti O, Gawande P. Assessment of cardiac, hepatic and pancreatic iron overload in transfusion-dependent thalassemia patients using T2* magnetic resonance imaging. *Indian J Hematol Blood Transfus*. 2024 Sep 17. doi:10.1007/s12288-024-01716-2
4. Mousa SO, Abd Alsamia EM, Moness HM, Mohamed OG. The effect of zinc deficiency and iron overload on endocrine and exocrine pancreatic function in children with transfusion-dependent thalassemia: a cross-sectional study. *BMC Pediatr*. 2021 Jan;21(1):1-9. doi:10.1186/s12887-021-02940-5
5. Musallam KM, Barella S, Origa R, Ferrero G, et al. Revisiting iron overload status and change thresholds as predictors of mortality in transfusion-dependent β -thalassemia: a 10-year cohort study. *Ann Hematol*. 2024 May;103(5):1234-1245. doi:10.1007/s00277-024-05000-1
6. Schwenzer NF, Machann J, Haap MM, Martirosian P, Schraml C, Liebig G, et al. T2* relaxometry in liver, pancreas, and spleen in a healthy cohort of one hundred twenty-nine subjects—correlation with age, gender, and serum ferritin. *Invest Radiol*. 2008 Dec;43(12):854-860. doi:10.1097/RLI.0b013e318185e9c5
7. Saggarr K, Sobti P. MRI assessment of iron overload in thalassemia: an overview. *Riv Ital Med Adolesc*. 2013 Jan;11(1):1-5.
8. Maggio A, Capra M, Vitabile S, Rigano P, Cassarà F, Midiri M. Procedures for the evaluation of body iron burden in thalassemia major. *Methods Mol Biol*. 2010;610:21-32. doi:10.1007/978-1-60327-029-8_2
(Month not provided)

9. Tziomalos K, Perifanis V. Liver iron content determination by magnetic resonance imaging. *World J Gastroenterol*. 2010 Apr;16(13):1587-1597. doi:10.3748/wjg.v16.i13.1587
10. Parale GP, Pawar SS, Tapare VS. Assessment of LV diastolic function in patients with β -thalassemia major with special reference to E/Eann ratio. *J Pediatr Hematol Oncol*. 2009 Jan;31(1):69-73. doi:10.1097/MPH.0b013e31818e0c2c
11. Kushner JP, Porter JP, Olivieri NF. Secondary iron overload. *Hematology Am Soc Hematol Educ Program*. 2001 Dec;2001(1):47-61. doi:10.1182/asheducation-2001.1.47
12. Tweed MJ. Pancreas, thyroid, parathyroid. In: *Exposing the Hidden Dangers of Iron*. 2004:67.
(Month not provided)
13. Sohail H, Fazal A, Lone KP, Kamran R, Ijaz F, Javed S, et al. Evaluation of bone density and leptin in thalassemic children. *Biomedica*. 2019 Jun;35(2):80-85.
14. De Sanctis V, Soliman AT, Daar S, Alansary N, Kattamis A, Skafida M, et al. A concise review on the frequency, major risk factors and surveillance of hepatocellular carcinoma in β -thalassemias. *Mediterr J Hematol Infect Dis*. 2020 Jan;12(1):e2020006. doi:10.4084/MJHID.2020.006
15. Drakonaki EE, Maris TG, Maragaki S, Klironomos V, Papadakis A, Karantanas AH. Deferoxamine versus combined therapy for chelating liver, spleen and bone marrow iron in β -thalassemic patients: a quantitative magnetic resonance imaging study. *Hemoglobin*. 2010 Jan-Feb;34(1):95-106. doi:10.3109/03630260903580320
16. Marsella M, Borgna-Pignatti C. Transfusional iron overload and iron chelation therapy in thalassemia major and sickle cell disease. *Hematol Oncol Clin North Am*. 2014 Aug;28(4):703-727. doi:10.1016/j.hoc.2014.04.005
17. Lee TA, von Riedemann S, Tricta F. Cost-utility of chelators in transfusion-dependent β -thalassemia major patients: a review of the pharmacoeconomic literature. *Expert Rev Pharmacoecon Outcomes Res*. 2014 Sep-Oct;14(5):651-660. doi:10.1586/14737167.2014.950229
18. Farmakis D, et al. A short guide for the management of TDT. Thalassaemia International Federation; 2023
19. Kumfu S, Chattipakorn SC, Chattipakorn N. Iron overload cardiomyopathy: Using the latest evidence to inform future applications. *Experimental Biology and Medicine*. 2022 Feb ;247(7):574-583. doi:10.1177/15353702221076397.
20. Marti-Aguado D, Ten-Esteve A, Baracaldo-Silva CM, Crespo A, Coello E, Merino-Murgui V, Fernandez-Paton M, Alfaro-Cervello C, Sánchez-Martín A, Bauza M, Jimenez-Pastor A.

- Pancreatic steatosis and iron overload increases cardiovascular risk in non-alcoholic fatty liver disease. *Frontiers in endocrinology*. 2023 Aug 3;14:1213441.
21. De Sanctis V, Soliman A, Tzoulis P, Daar S, Fiscina B, Kattamis C. The Pancreatic changes affecting glucose homeostasis in transfusion dependent β - thalassemia (TDT): a short review. *Acta Biomed*. 2021 Jul 1;92(3):e2021232. doi: 10.23750/abm.v92i3.11685. PMID: 34212898; PMCID: PMC8343719.
 22. Casale M, Forni GL, Cassinerio E, Pasquali D, Origa R, Serra M, Campisi S, Peluso A, Renni R, Cattoni A, De Michele E, Allò M, Poggi M, Ferrara F, Di Concilio R, Sportelli F, Quarta A, Putti MC, Notarangelo LD, Sau A, Ladogana S, Tartaglione I, Picariello S, Marcon A, Sturiale P, Roberti D, Lazzarino AI, Perrotta S. Risk factors for endocrine complications in transfusion-dependent thalassemia patients on chelation therapy with deferasirox: a risk assessment study from a multi-center nation-wide cohort. *Haematologica*. 2022 Feb 1;107(2):467-477. doi: 10.3324/haematol.2020.272419. PMID: 33406815; PMCID: PMC8804575.
 23. Casale M, Forni GL, Cassinerio E, Pasquali D, Origa R, Serra M, Campisi S, Peluso A, Renni R, Cattoni A, De Michele E, Allò M, Poggi M, Ferrara F, Di Concilio R, Sportelli F, Quarta A, Putti MC, Notarangelo LD, Sau A, Ladogana S, Tartaglione I, Picariello S, Marcon A, Sturiale P, Roberti D, Lazzarino AI, Perrotta S. Risk factors for endocrine complications in transfusion-dependent thalassemia patients on chelation therapy with deferasirox: a risk assessment study from a multi-center nation-wide cohort. *Haematologica*. 2022 Feb 1;107(2):467-477. doi: 10.3324/haematol.2020.272419. PMID: 33406815; PMCID: PMC8804575.
 24. Taher A, et al. Guidelines for the management of non-transfusion-dependent thalassemias: updated version. *Thalassaemia International Federation*; 2023.
 25. Musallam KM, Cappellini MD, Taher AT. Iron overload in non-transfusion-dependent thalassemia: a clinical perspective. *Blood Rev*. 2012;26(Suppl 1):S16-S19. doi:10.1016/S0268-960X(12)70006-5

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