

Renal Dysfunction in Iraqi Pediatric Patients with β -Thalassemia Major and Intermedia

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Abstract

Background: With optimum transfusion and chelation therapy, the survival of β -thalassemia patients and incidence of various complications, including renal complications, have improved.

Objectives: To investigate renal involvement in β -thalassemia patients using serum and urinary biochemical markers of glomerular and tubular dysfunction.

Methods: This case-control study included 60 β -thalassemia major (β -TM) patients, 25 β -thalassemia intermedia (β -TI) patients and 100 healthy controls, all ranging from 1–16 years in age. Blood urea nitrogen, serum ferritin, and serum and urinary levels of creatinine, uric acid, calcium, phosphorus, magnesium, sodium and potassium and the urinary albumin/creatinine ratio were evaluated.

Results: The blood urea nitrogen level and the urinary sodium/creatinine, potassium/creatinine, calcium/creatinine, magnesium/creatinine, phosphorus/creatinine, albumin/creatinine and uric acid/creatinine ratios were significantly higher in the β -thalassemia patients than the controls, while the serum sodium, potassium, calcium and magnesium levels were significantly lower in the patients ($P<0.05$). An elevated urinary uric acid/creatinine ratio was found in 61.9% of β -thalassemia patients, an elevated urinary calcium/creatinine and urinary albumin/creatinine ratio was found in 53.2%, and an elevated sodium/creatinine ratio was found in 41.3%. The serum and urinary renal markers showed no significant differences between patients with β -TM and β -TI, except for microscopic hematuria, which was significantly higher in β -TI patients (34.8%) than in β -TM patients (13%), $P=0.02$. An older age, high serum ferritin level and deferasirox therapy were associated with significant tubular and glomerular dysfunction in β -thalassemia patients.

Conclusions: Pediatric patients with β -thalassemia have significantly abnormal tubular and glomerular function, necessitating early detection and monitoring to prevent/reverse renal function deterioration.

Categories: Pediatrics, Nephrology, Hematology

Keywords: urine ratio, children, iron overload, renal function, β -thalassemia

Introduction

Beta-thalassemia is a group of hereditary blood disorders characterized by the reduction or absence in the synthesis of the β chain of hemoglobin, resulting in variable phenotypes ranging from no clinical symptoms to severe anemia [1].

Patients with a severe form of β -thalassemia, such as β -thalassemia major (β -TM), present early in infancy with debilitating anemia that requires life-long regular transfusions for survival. However, patients with β -thalassemia intermedia (β -TI) present later in life with less severe anemia and remain transfusion independent except within specific clinical settings [2].

Although the survival of patients with β -thalassemia has improved because of blood transfusion, iron chelation therapy and advances in knowledge of the disease, many patients develop various complications, including cardiopulmonary complications, endocrine diseases, liver impairment and thromboembolism [3,4].

Among patients with β -TM, a shortened red blood cell (RBC) life span, hypoxia, rapid iron turnover, excess iron deposition in tissue, and the use of specific iron chelators may adversely affect the kidneys [5–7]. Splenectomy has also been found to be associated with renal complications, including both tubular and glomerular dysfunction, in these patients [8,9]. In β -TI, iron overload (IO) occurs in nontransfused patients because of increased intestinal iron absorption due to ineffective erythropoiesis, which can eventually cause complications similar to those observed in β -TM patients [5].

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