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The Kidney in Beta-Thalassaemia major

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Beta-thalassaemia is the most common chronic haemolytic anaemia in Egypt [1]. Children with beta-thalassaemia major may be kept alive by repeated blood transfusions. Every 100 ml of blood provide the body with 50 mg of iron. The daily excretion, however, is only about 1 mg. There is also increased intestinal iron absorption irrespective of the level of the body iron stores due to ineffective erythropoiesis. These mechanisms lead to siderosis with deposition of iron in various organs [2]. In the present work, a study of the kidney in patients with beta-thalassaemia major under regular blood transfusions is presented. Urine was screened for haematuria in 45 patients 18 months-16 years of age with a median of 7 years. 24 were males and 21 females. Blood urea, serum creatinine, and beta2-microglobulin (B2M) levels in serum and urine were estimated. Ultrasonography and renal biopsy were done.

Haematuria was detected in 67% of the patients. Blood urea, serum creatinine and urinary B2M did not differ significantly from normal controls. On the contrary, serum B2M levels were significantly higher than normal, especially in patients with haematuria. Ultra-sonographic examination revealed positive findings in 15.5% of patients, all of whom had haematuria. These findings consisted of variable degrees of ill differentiation of the renal parenchyma and pelvicalyceal system as well as multiple cystic changes of different sizes in one or both kidneys. Renal biopsy was done in 5 patients and revealed moderate and diffuse thickening of capillary walls with segmental and focal cellular proliferation in the glomeruli. The tubules were atrophied and decreased in number and size with desquamation of their lining epithelium. There was round cell infiltration of the interstitial tissue. The increased serum levels of B2M, encountered in the present work, were probably the result of a glomerular filtration defect since urinary excretion was not increased despite the moderate degree of tubular changes detected by renal biopsy. If these tubular

changes were not accompanied by defective filtration, urinary B2M would have been increased, a finding not observed in the present study. However, increased synthesis of B2M due to reticuloendothelial hyperplasia, known to occur in patients with beta-thalassaemia major, cannot be ruled out [3, 4]. Pathological changes of the kidney were demonstrated both by

ultrasonography and biopsy. However, it is possible that they were not severe enough to cause an increase in blood urea and serum creatinine. Serum B₂m estimation is recommended as a screening test for renal lesions in these patients, since it seems to be more sensitive than other routine tests used.

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