Original Article

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# Microalbuminuria among children and adolescents with sickle cell disease

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Abstract:

**BACKGROUND:** Sickle cell nephropathy, a heterogeneous group of renal abnormalities resulting from complex interactions of sickle cell disease (SCD)-related factors and non-SCD phenotype characteristics, is associated with an increased risk for morbidity and mortality.

**AIMS:** The aims of this study were to determine the frequency of microalbuminuria (MA) among pediatric patients with SCD and to determine risk factors for MA among those patients.

**SUBJECTS AND METHODS:** A case–control study was carried out on 120 patients with SCD, 2–18 years old, registered at Basrah Center for Hereditary Blood Diseases, and 132 age- and sex-matched healthy children were included as a control group. Investigations included complete blood panel, blood urea, serum creatinine (Cr), urinalysis, and urinary albumin-to-Cr ratio (ACR). Logistic regression analysis was used to assess the predictors of MA.

**RESULTS:** Among SCD patients, 39 (32.5%) had MA compared to 6 (4.5%) in the control group. The mean levels of blood urea, serum Cr, and ACR were significantly higher, and the urine-specific gravity was significantly lower in SCD patients than in the control group ($P < 0.05$). Logistic regression analysis revealed that frequent painful crisis (odds ratio [OR]: 12.146, confidence interval [CI]: 3.439–42.952), high serum ferritin (OR: 8.146, CI: 1.802–36.827), deferoxamine therapy (OR: 23.423, CI: 3.961–60.509), and female sex (OR: 4.590, CI: 1.225–17.202) are independent risk factors for MA ($P < 0.05$).

**CONCLUSION:** The frequency of MA was high among our pediatric SCD patients. Risk factors for MA include female sex, nutritional factors, painful episodes, and iron overload. This is important for planning for future follow-up and management of this common disease in our locality.

Keywords:
Children, microalbuminuria, predictors, sickle cell disease

## Introduction

Sickle cell disease (SCD) is the most common monogenic disorder with remarkable phenotypic diversity.[1] SCD results from a single point mutation (Glu6Val) that causes polymerization of the mutant hemoglobin (HbS), resulting in sickling of red blood cells (RBCs). Inflammation, hemolysis, microvascular obstruction, and organ damage characterize the clinical course of the disease.[1]

SCD results from either homozygosity for HbS (HbSS), also known as sickle cell anemia (SCA), or compound heterozygosity with ß-thalassemia mutations and other ß-globin structural variants such as Hb C.[1] Although SCD has a high prevalence in Africa, the Middle East, the Mediterranean Basin, and India, SCD now has a worldwide distribution because of population migration, and a substantial number of children are born with the condition in Europe and North and South America.[1]

SCD is a multisystem disease associated with episodes of acute illness and progressive organ damage. The clinical course is

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