THE IMPORTANCE IN DETERMINING THE LEVEL OF ALBUMINURIA IN SICKLE CELL PATIENTS

Sickle cell anemia is a genetic disease in which the red blood cells produce a mutated form of hemoglobin. This causes the red blood cells to form a sickle cell, which can clog blood vessels and lead to diseases and damage to organs. This is especially true in the kidneys, where this build up can cause kidney malfunction and often lead to death. Originally it was believed that only a very small percentage of sickle cell patients, 4 percent, were in danger of developing kidney damage. However, a recent method has shown that the older method of using serum creatinine levels is very insensitive and does not give a very accurate reading. We hypothesized that more than 4 percent of sickle cell patients have kidney problems. We tested the albuminuria levels, which is an accurate indicator of the damage to the kidneys, of all sickle cell patients who visited the Grady Memorial Hospital clinic. We also investigated the type of factors that attribute to a sickle cell patient’s deteriorating kidneys. We included such factors as the patient’s weight, age, current medications, and other factors to determine if there was any correlation between the patients with the more severe kidney damage. The overall goal of our research was to discover what factors contribute the most to albuminuria, a condition that allows the build-up of albumin in the kidneys. Generally, it is normal to excrete a small amount of albumin in the urine, but when it exceeds about 30 mg/d, samples should be monitored. However, albumin cannot be tested alone because the amount of fluids someone drinks affects the amount of albumin. To counteract this, the excreted albumin was tested with another protein called creatinine. The two amounts were then calculated into an equation, which provides for an accurate account of the amount of albumin leaving the body. Our results have shown that annual testing will lead to early detection of complications.

Data Analysis

Patients with albuminuria were divided into 3 groups based on severity: 1) normoalbuminuria (<30mg/g creatinine); 2) microalbuminuria (30–300mg/g creatinine); and 3) macroalbuminuria (>300mg/g creatinine).

Of the 54 urine samples collected, 32 had hemoglobin sickle cell (HB SS) and 22 had other forms of sickle cell anemia. These were tested and added to a group of 106 urine samples that were collected a few months earlier, creating a total of 160 urine samples tested in a four-month period. Of these, 95 had HB SS while the remaining 65 had other forms. Each patient was assigned to one of the three categories depending on data results.

CONCLUSION

Our results proved our hypothesis: abnormal albuminuria levels occurred in 68% of patients with hemoglobin SS disease and occurred in 42% of patients with other Hemoglobin S variants. In HB SS patients, the percentage is 17 times higher than the creatinine serum level (CSL) suggests, and 10.5 times higher in non-HB SS patients than CSL suggests. The measurement of albuminuria may help in the management of patients with sickle cell anemia by detecting early kidney damage.

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INTRODUCTION

Typically, kidney damage is assessed by abnormal blood test results (serum creatinine levels). Based on abnormal serum creatinine levels (SCL), kidney damage was estimated to occur in about 4% of sickle cell patients. However, serum creatinine is now known to be an insensitive marker of kidney damage, and we hypothesized that kidney damage is relatively much higher than 4% of sickle cell anemia patients. When albumin excreted into the urine exceeds the rate of 30 mg of albumin per gram of creatinine, it is a clear indication that the kidneys are not functioning properly. The kidneys have a large vascular network which puts the organ at a much larger risk of injury than the other organs. Because of this risk, we expected a higher rate of sickle cell anemia patients with kidney damage than the accepted 4%.

METHODS

After receiving patient consent and authority to review medical records, we took urine samples, froze them, and transferred them to the laboratory for testing. We took samples only from patients who were feeling healthy at the time. A creatinine analyzer was used to test the amount of creatinine within each sample and through radioimmunoassay (RIA) the albumin was measured.

Student Researcher: Andy Zorrilla
Mentor: Antonio Guasch, MD

From Emory University and Grady Memorial Hospital; Atlanta, Georgia.