

# Anti-U Antibody - An Obstacle for Blood Transfusions in Patients with Sickle Cell Disease: Case Report and Review of Literature

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## Citation

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## Abstract

We are reporting a case of a 75-year-old African-American male with a history of sickle cell disease who presented to our hospital with loss of consciousness. Laboratory work revealed severe anemia.

During pre-blood transfusion testing the patient was found to be positive for anti-Kell, anti-s, anti-S, and anti-U antibodies. Anemia improved after transfusion with matching blood.

Anti U antibodies testing is not a routine testing before blood transfusion. The U-negative phenotype occurs almost exclusively in the African population and more in sickle cell disease patients. In these patients, it is important to identify and classify the alloantibodies.

## INTRODUCTION

Blood transfusion is the cornerstone of treatment of anemia in sickle cell disease (SCD). After the first exposure to U antigen, a class of antigens on all red blood cells, SCD patients with negative U antigen will produce anti- U antibodies, making the subsequent transfused red blood cells at risk for a delayed hemolytic reaction by alloantibodies.

## CASE PRESENTATION

A 75-year-old African-American male with sickle cell disease presented with loss of consciousness. The patient was intubated and mechanically ventilated, to secure his airway, and was started on IV fluids. Laboratory work revealed severe anemia (hemoglobin 5.2 g/dl, hematocrit 17%, MCV 89 FL, RDW 27.1 %) and acute renal failure (BUN 88 mg/dl, creatinine 1.7 mg/dl). Urine analysis did not show any erythrocytes. Fecal occult blood testing was negative on multiple different occasions. Peripheral blood smear, bilirubin, reticulocyte count/index, LDH, direct Coombs, hemoglobinemia, haptoglobin, PT/INR, PTT, liver function tests were within normal limits. Further testing of the patient's blood revealed the presence of anti-Kell, anti-s, anti-S, and anti-U antibodies. Anti-U positive packed red

blood cells (PRBCs) units were obtained from the Red Cross. Transfusion with four matched PRBCs after two days restored the hemoglobin level to 7.9 g/dl and the hematocrit to 24%. Subsequently, the patient was extubated and discharged home in a stable condition.

## DISCUSSION

In 1953, Wiener et al. discovered anti- U antibodies in an African American patient who had a fatal hemolytic transfusion reaction. U antigen exists in 99% of black population and nearly 100% in Caucasians [1]. In a study involving 2,462 Brazilian blood donors, all Caucasian Brazilians were antigen U-positive, and only 0.87% of black Brazilians were antigen U-negative. All U-negative blood donors were also both antigen S-negative and antigen s-negative simultaneously [2]. Because the majority of the population in the United States is Caucasian, most RBC units used in transfusions do not possess antibodies against the U antigen (anti-U).

U antigen is part of the MNS system, a non-ABO blood subgroup. M and N glycoproteins are located on glycophorin A, and S antigens are located on glycophorin B [3]. Individuals who lack glycophorin B lack the S and s

antigens, and also lack the U antigen. Therefore, the matching process in the blood banks assumes that Antigen U is negative in patients with negative S and s antigens. The routine blood testing includes RhHr, Kell, Duffy, Kidd, Lewis, P group, MNS, Lutheran and X testing. This combination does make antigen U indirectly evaluated.

Patients with negative U antigen will produce anti-U antibodies after the first exposure, placing them at risk of delayed hemolytic reaction (DHTRs) by alloantibody in next red blood cell transfusions. Typically most DHTRs are caused by a single alloantibody of Rh, Kidd or Kell blood group specificities [4-7]. Only few cases have reported anti-U antibodies as a cause of DHTRs. Anti-U are generally non-complement binding IgG antibodies containing an IgG-1 component [8, 9], which are an uncommon but recognized cause of hemolytic disease in fetus and newborn in black women [10, 11].

In SCD patients, where blood transfusions are still the cornerstones of prevention and treatment, it is important to identify and classify the alloantibodies. More studies are needed for further assessment of whether routine screening of all SCD patients for anti-U antibodies will be beneficial.

## **CONCLUSION**

It is important to investigate high risk patients for alloantibodies and to inform these patients of these rare antibodies. Because of the challenge of finding matched RBC units during critical times, establishing appropriate communication with previous facilities where patients have

received transfusions is paramount in case future transfusions are required.

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