

# SCIENTIFIC ARTICLES

## TRANSFUSION OF THE DANGEROUS UNIVERSAL DONOR BLOOD LEADING TO MATERNAL MORTALITY: A Case Report

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### KEYWORDS

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### ABSTRACT

#### BACKGROUND

In a health-care setting in which group-identical donor blood is not always available for transfusion, group O whole blood, in the obsolete concept of its being a universal donor, is sometimes given to group A and B recipients without necessary precautions.

#### OBJECTIVES

The objective is to draw attention to the danger of transfusing group A or B recipients with group O blood.

### MATERIALS AND METHODS

The case is presented of a multiparous blood group A pregnant woman who was transfused with whole blood group O. The woman developed a haemolytic blood transfusion reaction, which led to intravascular haemolysis, disseminated intravascular coagulation, multiple organ failure and death.

### CONCLUSION AND RECOMMENDATION

Whenever group-identical compatible blood is not available, and group O blood has to be given to a group A or B recipient, haemolysin test must be included in the pre-transfusion tests, and blood should be given preferably as red cell concentrate.

### INTRODUCTION

Blood transfusion has for a long time been a modality for the treatment of severe anaemia in pregnancy with consequent reduction of maternal morbidity and mortality.<sup>1,2</sup> This benefit may, however turn harmful when incorrect blood is transfused.<sup>2</sup> In a sub-Saharan African country like Nigeria, where there is perennial shortage of donor blood, the concept of group O donors being universal donors is still being applied, and group O blood is sometimes given to group A or B recipients.<sup>3</sup> However, the presence, sometimes, of haemolysins A and/or B in group O blood makes this practice very dangerous.<sup>3</sup> Haemolysins are the potent immune IgG anti-A and anti-B which occur in the ABO blood group system.<sup>4</sup> The occurrence, distribution and potency of haemolysins vary with ABO blood group, ethnicity, geographical location, previous exposure through transfusion and obstetric and other incidents.<sup>4</sup> In Zaria, group O individuals constitute between 46.9% and 49.2% of blood donors, of whom 32.3% have very strong anti-A and anti-B haemolysins.<sup>5,4</sup> Okafor and Enebe from

Enugu, reported a blood group O haemolysin rate of 53.6%, 62.7%, and 47.8% for anti-A, anti-B and anti-A,B respectively among blood donors.<sup>6</sup> Haemolysins were also detected in non-group O persons with anti-B haemolysins occurring in 35.7% of group A individuals while anti-A haemolysins were found in 8.8% of group B individuals.<sup>6</sup> Anyanwu *et al* in Calabar reported an occurrence of anti-A (15.2%), anti-B (12.1%) and anti- A,B (30.3%) haemolysins among group O individuals with Haemoglobin SS (HbSS).<sup>7</sup> Sometimes, health-care providers in rural areas may be caught in the dilemma between saving life in emergency situations by transfusing so-called universal donor blood on the one hand, and on the other, waiting for the correct group-identical blood which may not be immediately available.<sup>8</sup> Where such blood is not routinely screened for haemolysins, a significant proportion of group A or B recipients of group O blood may be at risk of haemolytic transfusion reactions.

What happens in such cases is that the IgG anti-A and/or anti-B antibodies in the donor plasma cause intravascular lysis of recipient A and B cells, and lead to haemoglobinaemia, haemoglobinuria, disseminated intravascular coagulation, (DIC), and acute renal, and other organ failure, with a high fatality index. We describe a case of fatal haemolytic transfusion reaction following transfusion of group O whole blood to a group A recipient.

## CASE REPORT

HA was a pregnant 29 year old housewife in a rural area in Northern Nigeria. She was G6P4+1 with 3 living children. She presented at a secondary health care facility, where a diagnosis of placenta praevia was made. She was also found to be severely anaemic with a packed cell volume of 18%. She was transfused with two units of group O RhD positive whole blood, even though she herself was blood group A Rh D positive. Six days after blood transfusion, she developed bruising at injection and venepuncture sites, and was passing dark-coloured urine. Nine days post-transfusion, there was loss of foetal movement, which prompted an induction of labour with oxytocin. She was later delivered of a macerated foetus weighing 2.7 kg and was referred to the teaching hospital for further management. On presentation, the patient was found to be severely pale, jaundiced, and febrile to touch with a body temperature of 38.5 °C. She had ecchymoses on the trunk and generalised exfoliation of the skin. There was and mild ankle oedema, Bilateral fine basal crepitations were heard in the lung fields, The heart rate was 160 beats per minute with a moderate-volume but regular peripheral pulse. Her blood pressure was 100/70 mmHg. The abdomen was distended, but abdominal organs were not palpably enlarged. The patient was disorientated in place and time. The bladder catheter was draining dark-coloured urine, while vaginal examination revealed a lochial flow with clots, from an empty uterus. Results of investigations on admission revealed a haematocrit of 0.19, total leucocyte count of  $11.2 \times 10^9/L$ , and platelet count of  $45 \times 10^9/L$ . The blood film showed marked hypochromic microcytosis, with some macrocytosis, few target cells and fragmented red cells. The reticulocyte count was 6%. Her blood group was confirmed to be A RhD positive, and she had a positive direct anti-human globulin test, and anti-A haemolysin titre of 2. The donors of the blood with which she was earlier transfused were traced, and their blood groups rechecked. Both were blood group O Rh D positive, but one had an anti-A haemolysin titre of 128. The thrombin, prothrombin, and activated partial thromboplastin times were indefinitely prolonged. The hepatic enzyme levels were 126 IU/L and 314IU/L for alanine transaminase, and aspartate transaminase respectively. The urea level was 63.5mmol/L while serum creatinine was 874umol/L and serum bicarbonate 20mmol/L The urine was positive for protein and urobilinogen, and the urine sediment was positive for haemosiderin by Pearls method. The chest x-ray was essentially normal, and urine, stool and blood cultures yielded no pathogens. A diagnosis of intravascular haemolysis with disseminated intravascular coagulopathy and renal failure was made. Therapeutic management included transfusion with 2 units of compatible blood group A fresh whole blood, intravascular injection of frusemide at a dose of 40mg, and hydrocortisone 100mg stat. intravascular course of amoxicillin-clavulanate at a dose of 375mg 8 - hourly was commenced. She had one session of haemodialysis. Oedema worsened, with development of anasarca, along features of uraemic encephalopathy. In spite of the management, the patient's condition deteriorated, and she died 5 days later.

## DISCUSSION

The decision of the referring hospital to transfuse the patient was dictated by severe anaemia in late pregnancy, probably due to the combination of multiparity, micronutrient deficiency and chronic blood loss, termed "maternal depletion syndrome".<sup>9</sup> The choice of group O donor blood for transfusion to a group A patient was probably made in the absence of compatible donor blood group A, and in the notion that group O blood could be given to a group A person under the circumstances. However, there was failure to check the group O donor blood for haemolysin A. in spite of the reported high frequency of haemolysins in the blood of Nigerians, and black Africans in general.<sup>4,6,7,10,11</sup> Treatment of severe anaemia in late pregnancy with whole blood transfusion was ill-advised, but may have been done for lack of plasma reduction facility at the primary and secondary levels of health establishments in Nigeria. The consequence was the development of a delayed haemolytic transfusion reaction, in which the antibodies in the donor plasma destroyed the recipient's own red cells. This led to intravascular haemolysis, disseminated intravascular coagulation, multiple organ failure and ultimately death. The unavailability of appropriate blood components to treat DIC, even at the tertiary health facility, probably contributed to the rapid clinical deterioration.

## CONCLUSION AND RECOMMENDATIONS

The concept that blood group O can serve as a universal donor is dangerous, and is now obsolete (refs). There is need for greater awareness of this fact among all levels of healthcare facilities in Africa, where blood transfusion is practiced. Screening of group O donor blood for haemolysins should always be done, and more particularly when group O blood is to be transfused to group A or B recipients. Such group-compatible, but unidentical transfusion should preferably be in form of red cell concentrate, to reduce recipient exposure to ABO haemolysins. Finally, blood services in Africa should be improved to make safe and adequate blood available always for transfusion, and appropriate blood components available for treatment of special cases.

## REFERENCES

1. Malcolm D. Black, Blood transfusion in obstetrics, *BMJ* 1937; 903-6
2. Bates I, Chapotera GK, McKew S, van den Broek N Maternal mortality in sub-Saharan Africa: the contribution of ineffective blood transfusion services. *BJOG* 2008; 115 (11): 1331-39.
3. Donald M. Ervin, Lawrence E. Young, Dangerous Universal Donors Blood, 1950; 5(1): 61-73
4. Kulkarni AG, Ibazebe R, Fleming AF: High frequency of anti-A and anti-B-haemolysins in certain ethnic groups in Nigeria. *Vox sang* 1985; 48(1): 39-41.
5. Hassan A, Babadoko AA, Ahmed AJ, Ahmed AJ Isa HA, Suleiman AM, The pattern of of distribution of ABO blood groups in North Western Nigeria, *Annals of Nigerian Medicine*, 2005; 1(2): 17-8.
6. Okafor LA Enebe S, Anti-A and anti-B haemolysins, dangerous universal blood donors and the risk of ABO antagonism in a Nigerian community, *Trop Geogr Med* 1985; 37(3): 270-2.
7. Anyanwu R. A., Emeribe A O, Igwe C U, Ajayi I, Akpotuzor J, Lele KC Emelike FO, Occurrence of haemolysin antibodies among sickle cell anaemia patients within Calabar metropolis of Nigeria. *African Journal of Biotechnology* 2007; 6 (10):1217-1220.
8. Olawumi HO, Olatunji PO. Prevalence and titre of alpha and beta Haemolysins in blood group 'O' donors in Ilorin, *Afr .J.Med.Sci* 2001; 30: 319-321.
9. Ugwuja EI, Akubugwo EI, Ibiam UA, Obodoa Onyechi, Impact of Maternal Iron Deficiency and Anaemia on Pregnancy and its outcomes in a Nigerian Population. *The Internet Journal of Nutrition and Wellness*, 2010; Vol 10(1).
10. Adewuyi JO, Gwanzura Christine and Mvere D. Characteristics of anti-A and anti-B in Black Zimbabweans. *Vox Sanguinis* 1994, 67 :307-309
11. Adewuyi JO and Gwanzura Christine. Racial difference between White and Black Zimbabweans in the haemolytic activity of ABO antibodies. *Afr J Med Med Sci* 2001; 30;71-74