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Research Article

Estimating the Risk of ABO Hemolytic Disease of the Newborn in Lagos

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Background. ABO hemolytic disease of the newborn is the most common hemolytic consequence of maternofetal blood group incompatibility restricted mostly to non-group-O babies of group O mothers with immune anti-A or anti-B antibodies. *Aim.* We estimated the risk of ABO HDN with view to determining need for routine screening for ABO incompatibility between mother and fetus. *Materials and Methods.* Prevalence of ABO blood group phenotypes in blood donors at the donor clinic of the Lagos University Teaching Hospital and arithmetic methods were used to determine population prevalence of ABO genes. We then estimated proportion of pregnancies of group O mothers carrying a non-group-O baby and the risk that maternofetal ABO incompatibility will cause clinical ABO HDN. *Results.* Blood from 9138 donors was ABO typed. 54.3%, 23%, 19.4%, and 3.3% were blood groups O, A, B, and AB, respectively. Calculated gene frequencies were 0.1416, 0.1209, and 0.7375 for A, B, and O genes, respectively. It was estimated that 14.3% of deliveries will result in a blood group O woman giving birth to a child who is non-group-O. Approximately 4.3% of deliveries are likely to suffer ABO HDN with 2.7% prone to suffer from moderately severe to severe hemolysis.

1. Introduction

ABO hemolytic disease of the newborn (ABO HDN) is the most common maternofetal blood group incompatibility. Unlike the rhesus disease, it is usually a problem of the neonate rather than the fetus. ABO HDN is restricted almost entirely to group A or B babies born to group O mothers with immune anti-A or anti-B antibodies.

ABO HDN is caused by IgG (immune) maternal antibodies which have the ability to cross the placental barrier. A high titre of these immune antibodies may not present with adverse effects in utero as A and B antigens are present on cells of all other tissues and body fluid and not only on red cells. The presence of these antigens helps to protect the incompatible fetal red cells by neutralizing the transferred maternal antibody with small amounts of antibody reacting directly with the fetal red cells [1]. The red cells which are sensitized by the antibodies are destroyed by macrophages in the fetal spleen with consequent hyperbilirubinaemia [2].

ABO-HDN in literature is described as a condition having a very low incidence in the population and characterized by a benign evolution because of a mild degree of hemolysis [3, 4]. Anaemia is rare with the main clinical problem being jaundice. Severe hemolysis and anaemia requiring exchange blood transfusion have however been reported [5]. Early detection and treatment of neonatal hyperbilirubinaemia is important in prevention of bilirubin-induced encephalopathy in the affected children [6].

The above statements, however, are not valid for all populations. Studies have revealed that statistically, mother and infant are ABO-incompatible in one of every five pregnancies among Caucasians [7, 8]. The incidence of ABO HDN in the United Kingdom is about 2% of all births, but severe hemolytic disease occurs in only 0.03% of births [9]. The incidence of ABO HDN in Blacks [10] is said to be higher than in Caucasians [11–13]. This is due to the higher prevalence and titres of immune anti-A and anti-B antibodies in the Black population [14–18].