PREVALENCE OF WOMEN AT RISK FOR Rh ISOIMMUNIZATION IN DIFFERENT RACES

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By

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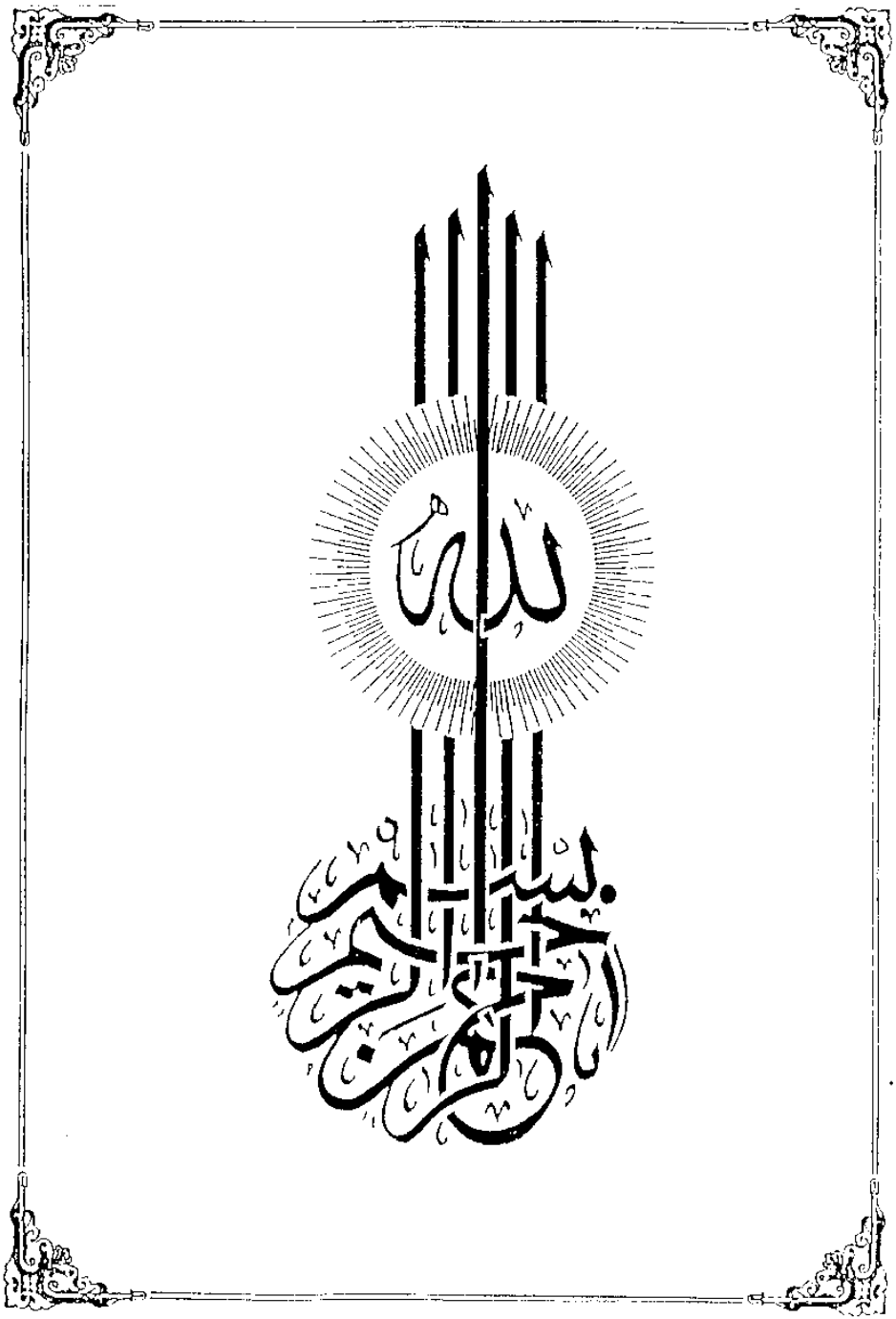
بِسْمِ اللَّـهِ الرَّحْمَنِ الرَّحِيمْ

أَلَمْ تَرَ أَنَّ اللَّـهَ أَنْزَلَ مِنْ اَلسَّمَاءِ مَـاءً فَأَخْرَجْنَـا بِـهِ ثَمَـرَاتٍ مُخْتَـلِفاً أَلْوَانُهَـا وَمِـنْ اَلْجِبَـالِ جُـدَدُ بِيـضُ وَحُمْـرُ مُّخْتَـلِفُ أَلْوَانُهَـا وَغَرابِيبُ سُـودُ * وَمِـنَ النَّاسِ وَالدوَّابّ وَالأَنْعَـامِ مُخْتَـلِفُ أَلْوَانُـه كَذَلِـكَ إِنَّمَـا يَخْشَـى اَللَّـهَ مِـنْ عِبَـادِهِ الْعُلَمَـؤُاْ إِنَّ اللَّـهَ عَزِيـزُ غَفُورُ .....(٢٧ ، ٢٨)

صَدَقَ اللَّهُ الْعَظِيمْ ....

" سورة فاطر "



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INTRODUCTION

Erythroblastosis fetalis is a disease of the fetus and newborn caused by an incompatibility of fetal and maternal blood. The Rh negative mother becomes immunized by exposure to Rh positive fetal erythrocytes during pregnancy or delivery (or occasionally as the result of an incompatible blood transfusion).

The antibodies formed by the mother pass through the placenta to the fetal circulation where they react with the Rh positive fetal erythrocytes, causing hemolytic anemia, *(Cunningham etal., 1989).*

Although the first description of hemolytic disease of the newborn dates back to 1609, no rational treatment was possible until the 20th century, when the ABO blood groups and Rh factor were discovered.

In the ensuring years, advances in the therapy dramatically reduced perinatal mortality, and Rh immune-globulin (Rh IgG) was developed to prevent the condition.

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The Rh antigens are inherited independent of other blood group antigens and are located on the short arm of the chromosome 1. There is apparently no difference in the distribution of the various Rh antigens with regard to sex; however, there are important racial difference. American Indians and Chinese and other Asiatic people are almost all Rh0 (D) positive (99%).

Among black Americans there is a lesser incidence of Rh0 negative individuals (7 to 8 percent) than among white Americans (13 percent).

Of all racial and ethnic groups studied far, the Basques show the highest incidence of Rh0 negativity (34 percent), ***(Cunningham etal., 1989).***

The incidence of hemolytic disease of the newborn has gradually decreased but not disappeared since the introduction of Rh IgG. Paradoxically, as the disorder becomes less frequent, it is increasingly difficult to keep abreast of the new and sometimes controversial recommendation for prophylaxis as well as sophisticated advances in treatment.

AIM OF THE WORK

1. To compare between different races as regards prevalence of Rh factor.

2. To estimate the prevalence of women at risk for Rh isoimmunization (married to Rh +ve husbands).

3. To estimate the percentage of women covered by postpartum anti-D prophylaxis.

HISTORICAL REVIEW

Brill and Platt (1983) reported that, erythroblastosis fetalis was probably first recorded by Hippocrates in 400 B.C. However, its etiology remained without convincing elucidation until the discovery of Rh blood group system in 1940 by *Landsteiner* and *Colleagues*..

Shortly thereafter, *Levine* and co-workers (1941) demonstrated the central role of Rh-antigen incompatibility in a reported pregnancy complicated by maternal transfusion reaction that ended in hydropic stillbirth. A modern concept of maternal immunologic intolerance for a foreign fetal blood cell antigen then emerged. Despite conceptual insight, however, perinatal mortality remained at 40-50%.

In 1946, *Wallerstein,* introduced the use of exchange transfusion. This nearly halved the neonatal mortality. His original use of the sagittal sinus for transfusion was later modified by Diamond (1947) who successfully used umbilical vesseles for cannulation.

in 1950, *Allen,* demonstrated the actual relationship between hyperbilirubinemia from fetal hemolysis and subsequent newborn kernicterus, this helped explain how exchange transfusion improved neonatal outcome.

In 1954, induction of labor as early as 32 weeks' gestation was instituted, based on either high maternal Rh-antibody titres or the poor outcome of previously affected pregnancies. Mortality rates were further reduced to less than 20%.

In 1956, ***Bevis,*** used spectrophotometric analysis of fluids obtained by amniocentesis, established the inseparable relationship between amniotic fluid bilirubin concentration and the severity of disease in utero. This provided the first reliable tool for antenatal assessment of potentially hydropic fetus.

In 1957, ***Kleihauer*** and ***Betke*** developed a differential staining technique to identify and quantitate fetal red blood cells in the maternal circulation, this technique served to cement the thesis of transplacental passage of fetal red blood cells.

In 1960, ***Liley,*** reported the first successful intrauterine transfusion, with which the mortality dropped to nearly 10%.

Finn, and associated (1961) in England, and ***Freda*** and ***Gorman*** (1962) in the United States established that Rh-isoimmunization could be prevented by administration of an anti-Rh antibody preparation following delivery.

Since 1968, the use of Rh-immunoglobulin for prevention under the trade name RhoGAM became a wide public health program.

Bowman and Co-workers in Canada demonstrated further reduction of maternal sensitization by prophylactic administration of RhoGAM in the late second and early third trimester of pregnancy.

Thus, after a relatively short period of distinguished efforts, the enigma of erythroblastosis fetalis was unraveled. Its clinical description has long been well known and with the introduction of RhoGAM, along with a heightened clinical consciousness, the sensitized women fortunately becoming an infrequent clinical problem.

Nevertheless, every obstetrician, midwife and general practioner participating in antenatal care must be alert to the importance of identifying the few new Rh(D) immunization cases that will still occur. They also should detect as early as possible, during pregnancy, the presence of any other irregular antibodies of the Rh system.

PHYSIOLOGY OF BLOOD GROUPS

BLOOD TYPES:

Human blood is classified into 4 groups depending on the presence or absence of 2 mucopolysaccharide substances : (A) and (B) in the membrane of RBC. They are called "Agglutinogen", (*Ganong 1979).*

Agglutinogens start to appear in the sixth week of fetal life. Their concentration gradually rise and at birth they reach one-forth of adult level. The adult level is reached at about puperty, (*Cheraskin* and *Langley.,* 1978).

In the plasma there are other 2 globulin (antibodies) called "Agglutinin" alpha (α) and (B). These antibodies may occur naturally (i.e. inherited) or may be produced by exposure to the red cells of another individual via a blood transfusion, or during pregnancy, when fetal red cells cross the placenta and enter the maternal circulation, (*Ganong* 1979).

Antibodies to blood group antigens that not possessed by the individual are termed alloantibodies and these are capable of reacting with the red cells of people of a different blood group, (*Walter* and *Isreal.,* 1979).

A. ABO-BLOOD GROUP SYSTEM

The A and B agglutiongens are inherited as mendelian allelomorphs, (*Cheraskin* and *Langley.*, 1978).

A and B being dominant, there are 4 major ABO blood groups. These groups are called O,A,AB,B.

Group A is subdivided into A_1 and A_2 groups AB is also divisible into A_1 B and A_2 B. Approximately 75-80% of group A belong to the subgroup A_1 while 60% of group AB belong to the subgroup A_1 B. The remainder belong to subgroup A_2 and A_2 B.

Splitting group A blood into the subtype is usually unnecessary from the practical point of view because transfusion reaction between bloods of these subgroups have rarely occurred (*Guyton.*, 1967).

B. Rh-BLOOD GROUP SYSTEM

The CDE antigens are inherited independent of other blood group antigens, and are located on the short arm of chromosome 1, (*Cunninghams* et al., 1989).

In the Rh system, the Rho (D) antigen is by far the most important, and its presence or absence is used to categorize a patient as Rh-positive or Rh-negative respectively.

Antibodies to other Rh antigens { rh'(C), rh''(E), hr'(c), hr''(e) } may develop, but the most commonly formed Rh antibody is anti Rho (anti-D)., (***Whitfield.,*** 1988).

EPIDEMIOLOGY

A-RACIAL DISTRIBUTION OF BLOOD GROUPS:

There is a racial difference in the distribution of ABO blood group system as well as the Rh blood group system.

In 1970, ***Farrell,*** reported that the B gene is more prevalent among blacks than among whites.

In 1978, ***Cheraskin*** and ***Langley,*** reported that group O is more common among Indian Tribes of America, a section of Australians, Africans, north and Western Europeans.

Group A is more common in people of America, West Europeans and West Asians, while group B is more common in central and southeast Asians.

In 1985, ***Pritchard et al.,*** reported that American, Indian, Chinese and Asiatic people are almost all Rho (D) positive (99%). Among black Americans there is lesser incidence of Rho (D) negative individuals (7 to 8%) than among white Americans (13%). Of all racial and ethnic groups studied thus far, the Basques show the highest incidence of Rho (D) negative (34%), ***(Cunninghams et al.,*** 1989).