BLOOD TRANSFUSION PRACTICE IN PAEDIATRICS

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BY
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1993

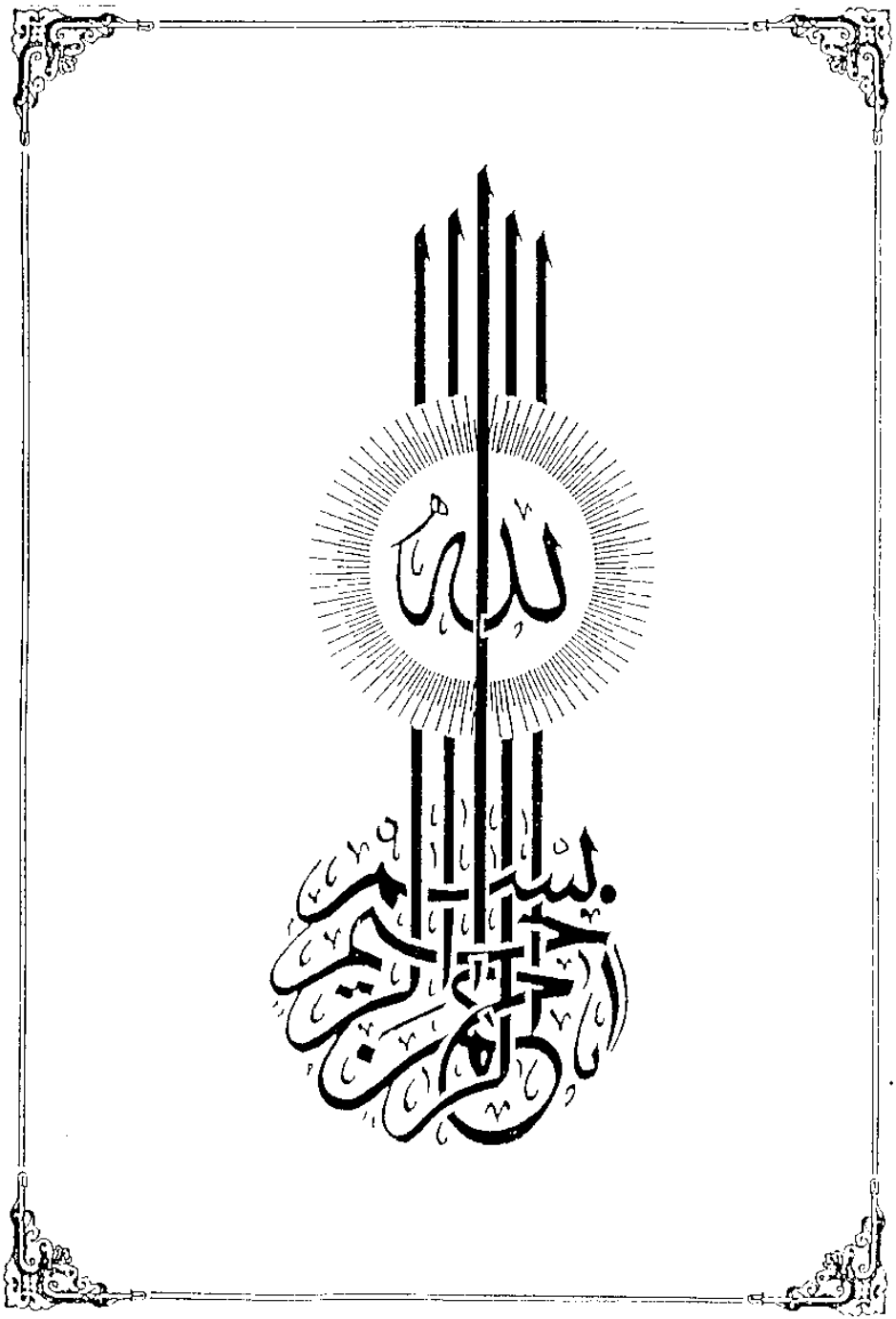
DEDICATED TO

MY HUSBAND

AND

MY SON

SEIF EL-DIEN



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INTRODUCTION

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Transfusion medicine continues to grow as a medical discipline. The fact that transfusion medicine polices and procedures are usually generated with inadequate consideration of neonatal and pediatric recipients is now recognized. Therefore, the transfusion literature is now addressing more issues of concern in Pediatric hematology (**Strauss, 1986**).

The main indication for transfusion of blood and blood products are well-established. Moreover, the principles of transfusion in the new-born are essentially similar to those in adult, but great care has to be taken because of small size of the baby particularly if it is premature (**King, 1991**).

Transfusion practice has evolved from the non-specific use of the whole blood to the highly selective use of individual components and plasma fractions such as coagulation factors (**King, 1991**).

Improvement of blood storage continue to be documented (**Ness, 1988**). On the other hand, the risks of blood transfusion transmitting bacterial, protozoal and viral infections are well recognized. The major concerns remain

hepatitis B and C, Human immuno-deficiency virus (HIV) and Cytomegalovirus (CMV) infection in certain groups such as recipients of bone marrow transplants. Recently, the risk of transmission of human T-cell leukemia virus type I (HTLV-1) is recognized (**Weber, 1990**).

It seems that the transfusion medicine has finally become recognized, but many factors seem to be attacking its foundation. In the coming years, the use of recombinant hematopoietic growth factors may replace the need for volunteer blood for many anaemic recipients (**King, 1991**).

AIM OF THE WORK:

The aim of the present work is to review transfusion in pediatrics as regard indications, complications and recent advances in neonates, infants and children.

INDICATIONS OF TRANSFUSION OF BLOOD AND ITS PRODUCTS IN PEDIATRICS

Indications:

I- **Anaemia:** It is the main indication for blood transfusion in pediatrics.

Types of anaemia:

[A] Hemolytic anaemia:

1- Extrinsic (extra-corpuscular) abnormalities of RBC's:

* Immunologic:
  - Hemolytic disease of the new born, e.g. Rh, ABO incompatibility.
  - Active antibody formation, e.g., idiopathic auto-immune hemolytic anaemia.
* Non immunologic: e.g. infections and toxins.

2- Intrinsic abnormalities of RBC's:

* Hemoglobinopathies:
  - Thalassemia (Alpha, Beta and others).
  - Abnormal hemoglobin (Sickle cell anaemia).
* Enzymatic defect: G-6-P-D deficiency.
* Cell wall defect: Hereditary spherocytosis.

[B] Decreased production of RBC's: Aplastic anaemia.

[C] Blood loss: e.g. hemorrhage and parasites.

The commonest causes of anaemia in neonates are hemolysis, hemorrhage and infections. The commonest causes in infancy are nutritional and prematurity. The commonest causes in childhood are blood loss, infections, nutritional and hemolysis.

II- Neonatal coagulation defect.

III- Neonatal hyperbilirubinemia.

IV- Direct blood transfusion in the complex treatment of children with suppurative surgical infection.

V- Premature infant who weight less than 1.5 Kg.

VI- Infant in congestive heart failure.

VII- Neonates.

VIII- Neonatal alloimmune thrombocytopenic purpura.

[I] Hemolytic disease of the new born (HDN):

Definition:

Hemolytic disease of the fetus and new born is a condition in which the life span of the baby's red cells is shortened by the action of specific antibodies derived from the mother by placental transfer. The disease begins in intrauterine life and may result in death in utero.

Causes:

It is caused by a blood group difference between fetus and the mother. Most blood groups can be involved.

(A) The most common and severe cases are caused by differences in the rhesus blood group system, the mother being rhesus negative (dd) while the fetus is rhesus positive carrying the D-antigen (DD or Dd) (**Urbaniak, 1985**).

(B) Hemolytic disease of the new born not due to anti-D, i.e. non-D antibodies (C-E).

(C) Hemolytic disease of new born due to ABO incompatibility.

(D) K in the Kell system (Other antibodies) (**Clarke et al., 1987**).

Pathophysiology:

Maternal serum IgG antibodies cross the placenta by binding to an Fc receptor on the plasma membrane of the placenta lead to hemolysis of fetal red blood cells resulting in fetal anaemia. This stimulates the production of erythropoietin and erythropoiesis in extra-medullary sites in the liver which may distort the hepatic circulation, may cause portal hypertension and decreased albumin production with subsequent manifestation of fetal hydrops (i.e., subcutaneous oedema, ascites and pleural and pericardial effusions) (**Van der Meulen et al., 1980**). Chronic anaemia leads to cardiac failure which leads to tissue hypoxia which increase hydrops

by causing endothelial defect causing leakage of protein from the intra-vascular compartment (**Nicolaides et al., 1985a**).

Hydropic fetuses have hematocrit of < 15%, total protein concentration < 30 gm/L and albumin level < 20 gm/L (**Grannum and Copel, 1988**). According to **Nicolaides et al. (1987)**, hydropic and non hydropic fetus have similar blood volume [Figure 1]. Most hydropic infants are not hyper-volemic or in heart failure at birth but do have low plasma colloid osmotic and hypoalbuminemia (**Phibbs et al., 1984**). The measurement of cardiac output by pulsed doppler studies in fetus before and after intravascular transfusion and reversal of hydrops were within normal (**Barrs et al., 1987**).

Diagnosis and assessment of severity of Rh disease in fetus:

[A] Indirect assessment by prenatal diagnosis:

(1) **History of previous pregnancies:** The history is important in Rh disease. If there have been no previous pregnancies and no transfusion, the likelihood of serious erythroblastosis in the current fetus is slight. If there have been previous pregnancies but no neonatal jaundice, the probability of involvement in the current pregnancies is greater, but still low. However, if previous babies have had hemolytic disease, the present fetus if Rh positive will be affected. If the last baby was stillborn, the chances are

about 2 out of 3 that the present one will be stillborn if the pregnancy is allowed to go to term (Mollison, 1983).

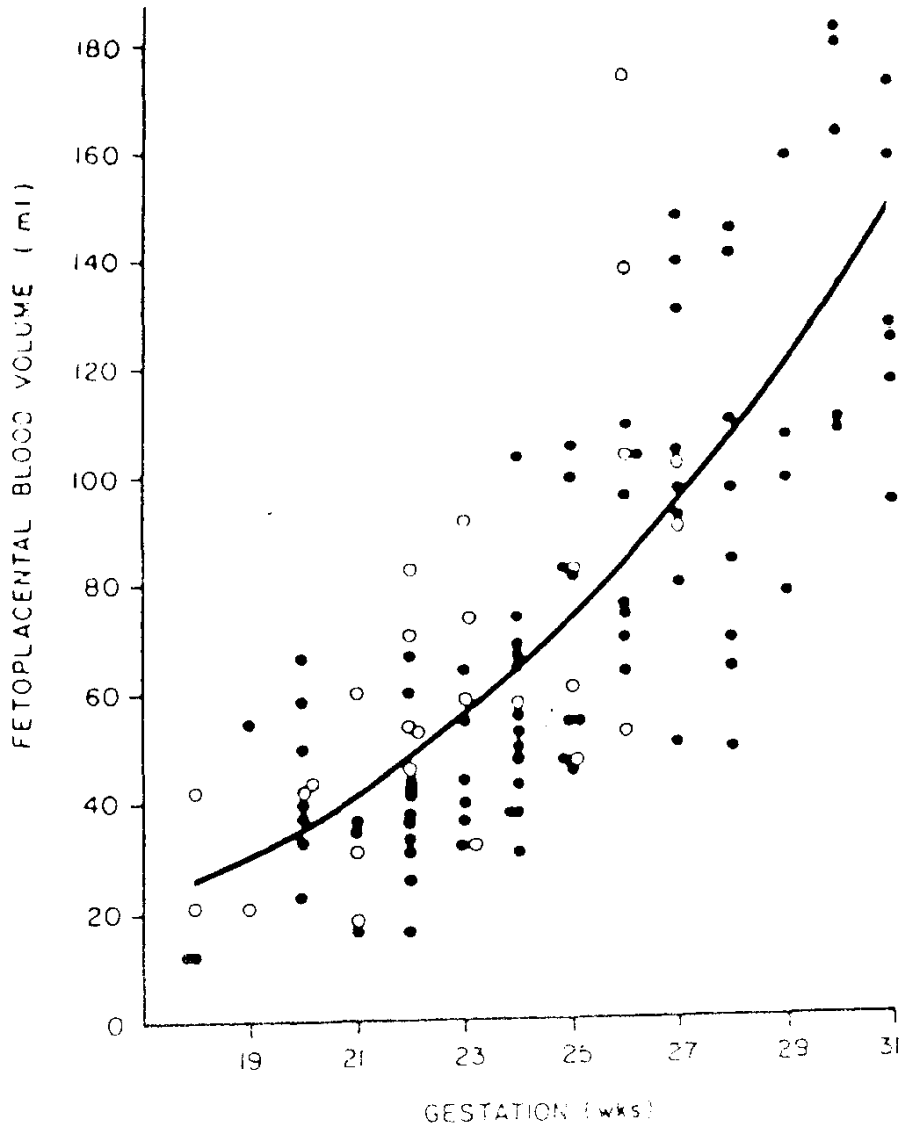

Figure [1]: Estimated feto-placental blood volume. ● = non-hydropic fetus, O = hydropic fetus. (Cited from **Nicolaides et al., 1987**).

(2) **Blood typing:** In each pregnancy, every patient should have a blood sample drawn for ABO and Rh typing and a test for unexpected blood group. It is important to test apparently Rh-negative mother for future reference at the time of delivery.

(3) **Rh-antibodies:**

i) Detection of Rh-antibodies: A test for unexpected antibodies should be done, even if the patient is Rh positive, because some people have had transfusions and do not know about it. Any transfusion may be immunizing, and pregnancy with a fetus possessing the corresponding antigen may result in erythroblastosis fetalis. Finally, if the mother herself requires transfusions, prior knowledge of an antibody will save a great deal of hurried laboratory work and prevent delay in providing compatible blood (**Bowman et al., 1980**).

If the mother is Rh-negative, it is helpful to know whether the father is Rh-positive because of unimmunized Rh-negative mother with Rh-negative husband requires no additional serologic testing. On the other hand, if the father is Rh-positive, genotype of the father is also important to know if he is Dd, then the fetus has 50% chance of being Rh-negative.

When no antibody can be detected, but a strong erythroblastosis detected by finding of fetal ascites by ultrasound, the disease may be due to a 'private antigen' carried by father. In this situation, the diagnosis can be made by reacting the father's red cells with the maternal serum (after absorption of complicating naturally occurring maternal antibodies if indicated). Once an antibody has been discovered, it must be identified (**Bowman, 1989**).

ii) **Antibody identification:** Once an unexpected antibody is detected, the antibody specificity must be determined. Like those of the Kell and Duffy system, may be important if the father has the corresponding antigen (**Yllen and Rodeck, 1990**).

iii) **Maternal Rh antibody concentration:** The titre of maternal antibody by indirect anti-globulin technique (indirect Coombs' titre) cannot be used alone to determine the severity of the disease. This is because of, it is possible to have a fetus that is severely affected with a low titre, or a fetus that is mildly affected with a much higher titre. However, quantitation of maternal antibody in the auto-analyzer using an international standard, is a better predictor of severity than the antibody titre and is used in United Kingdom as a guide to further evaluation of the