Al Mustaqbal University
Department of Medical Laboratory Techniques
Lab: Practical Blood Transfusion

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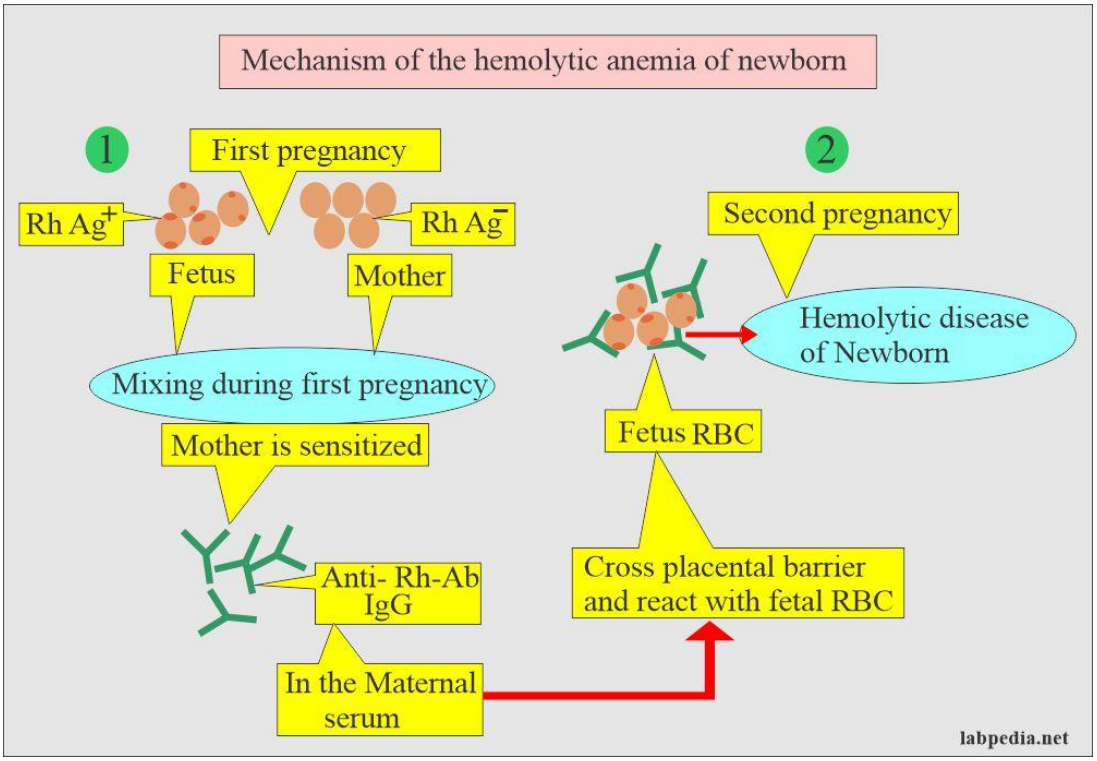
LAB 5

Haemolytic disease of the newborn (HDN)

HDN is caused by blood group difference between the fetus and the mother. Most blood groups can be involved, but the most common and sever 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).

Mechanism:

In most normal pregnancies, small numbers of fetal red cells enter the maternal circulation late in pregnancy or at delivery. During delivery, sufficient fetal red cells cross the placenta and enter the maternal circulation and lead to immunization the mother, and production of antibodies to any blood group differences between the fetus and the mother. Any IgG antibodies produced will pass across the placenta in a subsequent pregnancy, coating the red cells of the fetus. Later such coated cells will be hemolyzed in the fetus causing anemia.



Formation of maternal antibodies

The formation of antibodies in the mother depends on the dose of the fetal cells. When one pint of blood (Rh+ve) is given by transfusion to Rh-ve individual, 70% will produce anti-D (responder) while 30% will not produce anti-D (non responder). There is some variation in the antigenicity of D according to the genotype of the fetus, and DU is a less potent antigen. A significant factor which affects the number of fetuses sensitized is the ABO compatibility of the fetal blood and that of the mother.

Clinical effects:

In utero, the only effect of antibody coating is hemolysis. The earlier this starts, the more severely affected the fetus becomes.

When the degree of anemia gets more severe, the fetus suffer from cardiac failure and becomes edematous with ascites, namely hydrops fetalis, and most severely affected fetuses die in utero.

Before birth the placenta clears any bilirubin from the fetal blood and jaundice at birth usually only mild. After birth, the hemolysis of the baby's cells continues until any material antibody has been used up, and the anemia often becomes severe. More importantly, the unconjugated bilirubin level can rise to levels that can cause kernicterus (brain damage).

Severity of the disease in the fetus

Before birth, the severity of the disease is assessed by the amount or titer of antibody in the maternal blood, the history of previous pregnancies, the bile pigment (bilirubin) level in the amniotic fluid, and findings on ultrasound examination.

Antenatal treatment of HDN

1- Early induction of lab our (between 32 week and term) depending on the severity of the disease.
2- Intrauterine transfusion can be carried out by two techniques; intraperitoneally or intravascularly using fetoscope. For both, group O, rhesus negative (cde/cde) packed cells, WBC depleted are used. The donor cells should be cross matched to the mother's serum
3- Plasmapheresis to remove maternal antibody, is used forcases which have high antibody levels early in pregnancy, particularly in women with previously severely affected infants.

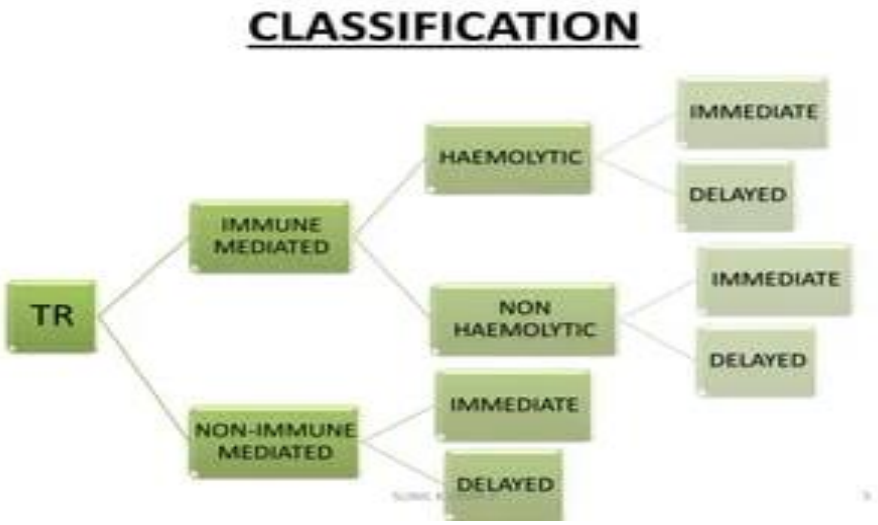
LAB.6

Transfusion reactions

They are any unfavorable transfusion-related event occurring in a patient during or after transfusion of blood components.

- ❖ Each blood product transfused carries a small risk of an acute or late adverse effect.
- ❖ The most common immediate adverse reactions to transfusion are fever, chills and urticarial
- ❖ The most potentially significant reactions include acute and delayed hemolytic transfusion reactions and bacterial contamination of blood products.

❖ During the early stages of a reaction it may be difficult to ascertain the cause most cases of transfusion reactions occur as the result of an **error – mistakes** made in the labeling or typing of the blood or recipient or failure to check that the two blood types match .



Immune transfusion reaction

Rapid destruction of donor RBC by recipient Ab ……Ab bind donor RBCs Complement activation RBC lysis.

- ❖ Acute (within 24 hours)
- ❖
- ❖ or delayed (within several days)

1- Acute hemolytic transfusion reaction

Causes of Acute hemolytic transfusion reaction

- Inadequate, incorrect labeling
- Confusion in the identity of the patient
- Improper identification of patients sample by the blood bank technician

Technical errors

- Grouping and cross matching errors
- Incorrect interpretation of the results

Signs and symptoms

- Fever chills, rigors
- Pain at the infusion site, chest, flank
- Tachycardia
- Hypotension
- Respiratory distress – dyspnea, tachypnea, hypoxemia
- Hemoglobinuria
- Shock

1-Acute Haemolytic Reactions Management:

A. Immediately stop transfusion.

B. Notify hospital blood bank urgently (another patient may also have been given the wrong blood!)

C. Send all tubing and a patient sample to the blood bank.

Investigation of suspected AHTRs, Send the following lab investigations:

A-Immediate post transfusion blood samples (clotted and EDTA) for:

- **Repeat ABO & Rh (D) grouping**
- **Repeat antibody screen and crossmatch**
- **Direct antiglobulin test**
- **Complete blood count (CBC)**
- **Plasma hemoglobin**

- **Coagulation screen**
- **Renal function test (urea, creatinine and electrolytes)**
- **Liver function tests (bilirubin, ALT and AST)**

B. Blood culture in special blood culture bottles

C. Blood unit along with BT set

D. Specimen of patient's first urine following reaction

2- Delayed Haemolytic reaction:

It is delayed due to weak antibody in the recipient the Hb fall within the 3-10 days of post transfusion.

- The patient has been primarily immunized by previous transfusion or pregnancy.
- Due to secondary immune responses following re-exposure to a given red cell antigen:

A. The antibody is too weak to be detected in routine cross-match, but becomes detectable 3 to 7 days after transfusion, e.g. Antibodies of the **involved - Rh , Kidd, Duffy and Kell** system

B. No clinical signs of red cell destruction but positive DAT

C. Rarely fatal

Sign and symptoms

Most delayed hemolytic reactions produce **few symptoms** and may go unrecognized, however there are reports of serious consequences in critically ill patients.

- Fever
- Fall in Hb concentration
- Jaundice and hemoglobinuria
- Hypotension & renal failure – may require expert medical advice

Diagnosis & Management

A. Routine examination

B. Monitor vital signs, urine out put

C. Verify identification of the patient

D. Evaluate for evidence of HTR, septic shock, anaphylaxis.

Blood Bank:

- Recheck the records for clerical error
- check for identification error
- Visual check for hemolysis, appearance of returned unit
- Evidence of blood group incomparability
- Gram stain, culture

Treatment:

- **Antipyretics**
  - **acetaminophen ;** 325-650mg orally (adult) 10-15mg/kg (children)
- **Meperiedine**
  - **severe chills -** 25-50mg IV
  - **contraindication:** renal failure