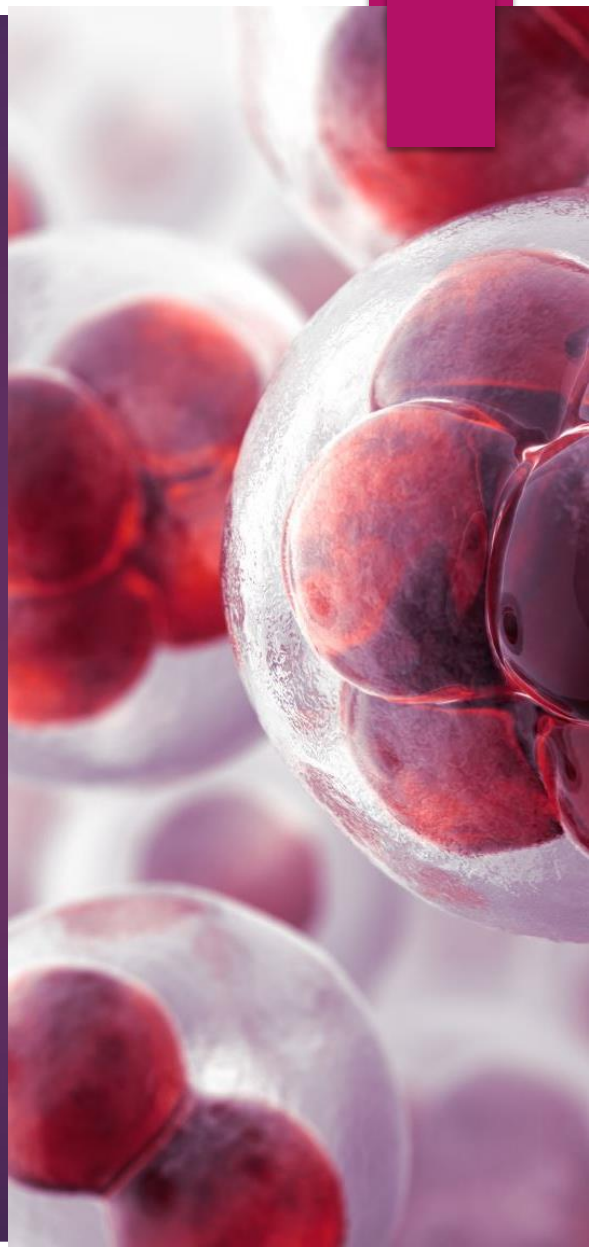
Erythroblastosis Fetalis, & Exchange Transfusion.

DR. AFSHEEN MAHMOOD NOOR



D antigen

- The type D antigen is widely prevalent in the population and considerably more antigenic than the other Rh antigens.
- Anyone who has this type of antigen is said to be *Rh positive*,
- A person who does not have type D antigen is said to be *Rh negative*.

Rh Immune Response

Formation of Anti-Rh Agglutinins

- In ABO system, the plasma agglutinins responsible for causing transfusion reactions develop spontaneously,
- whereas in the Rh system, spontaneous agglutinins almost never occur. Instead, the person must first be massively exposed to an Rh antigen, such as by transfusion of blood containing the Rh antigen, before enough agglutinins to cause a significant transfusion reaction.

- anti-Rh agglutinins develop slowly, reaching maximum concentration of agglutinins about 2 to 4 months later.

- With multiple exposures to the Rh factor, an Rh-negative person eventually becomes strongly "sensitized" to Rh factor

Characteristics of Rh Transfusion Reactions

- If an Rh negative person has never before been exposed to Rh positive blood, transfusion of Rh-positive blood into that person will cause no immediate reaction.
- However, anti-Rh antibodies can develop in sufficient quantities during the next 2 to 4 weeks to cause agglutination of those transfused cells that are still circulating in the blood. These cells are then hemolyzed by the tissue macrophage system.
- Thus, a *delayed transfusion reaction* occurs, although it is usually mild.

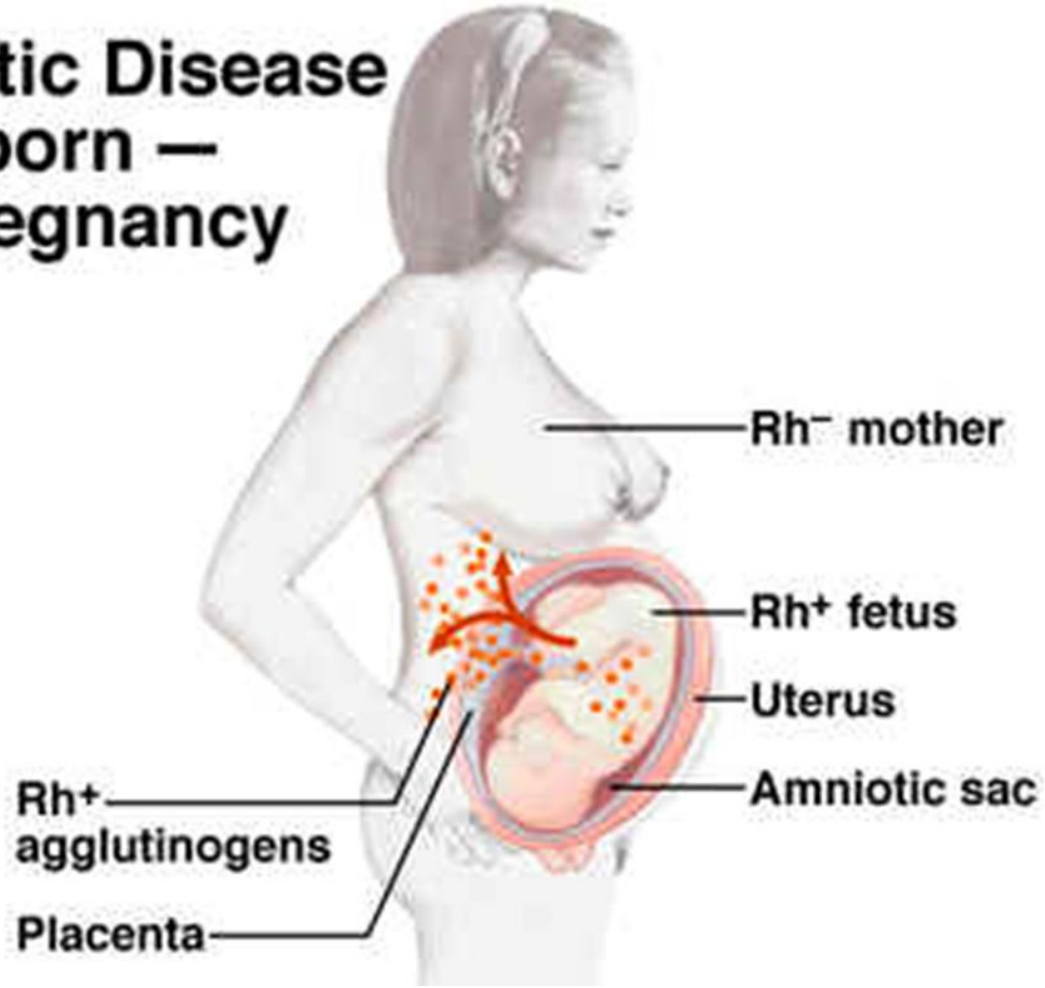
- On subsequent transfusion of Rh-positive blood into the Rh negative person, who is now already immunized against the Rh factor, the transfusion reaction is greatly enhanced
- can be immediate and as severe as transfusion reaction caused by mismatched type A or B blood.

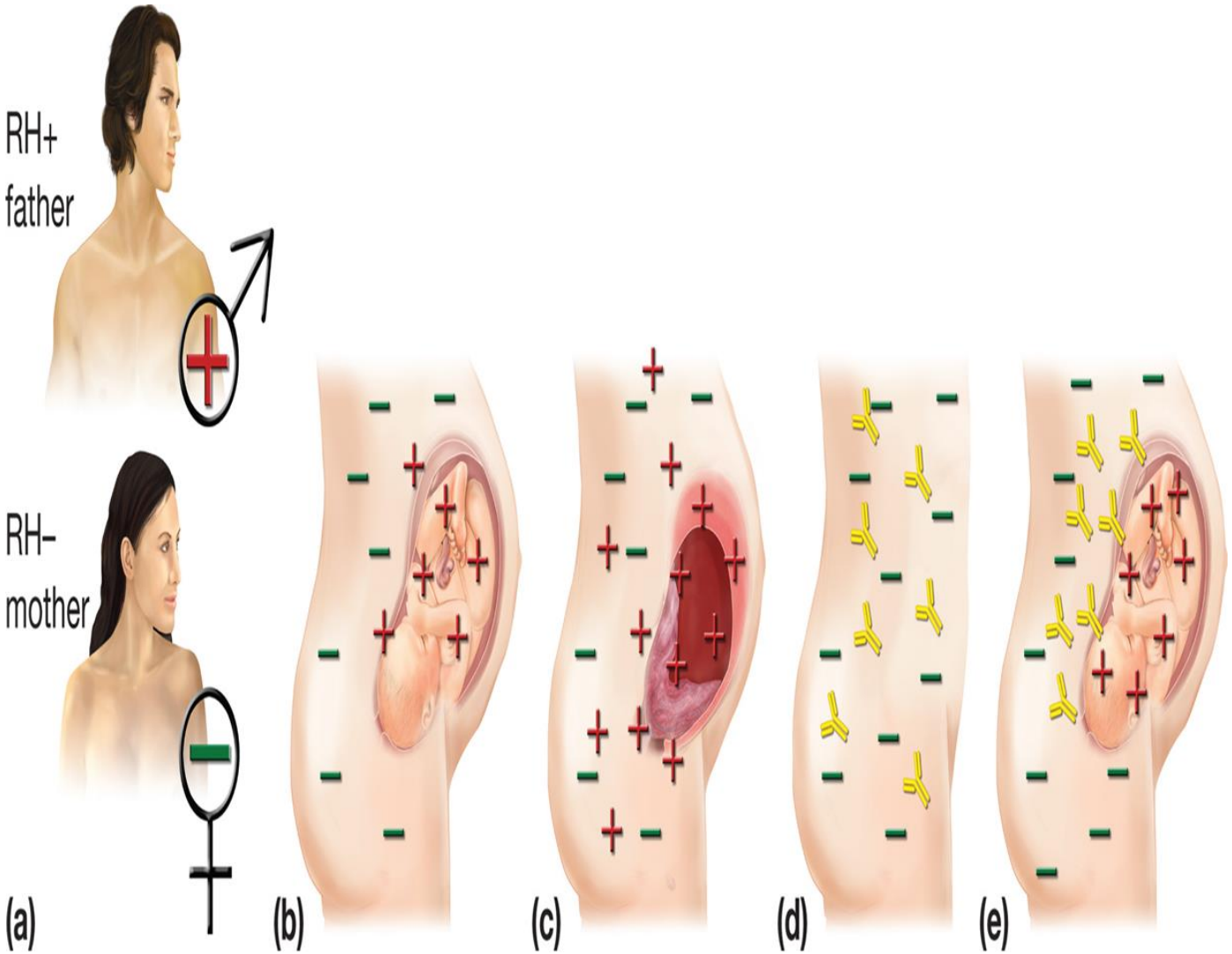
Erythroblastosis Fetalis "Hemolytic Disease of the Newborn"

- Erythroblastosis Fetalis is a disease of the fetus and newborn child characterized by agglutination and phagocytosis of the fetus's red blood cells.

- The mother is Rh negative and the father Rh positive. The baby has inherited the Rh-positive antigen from the father,
- the mother develops anti-Rh agglutinins IgG from exposure to the fetus's Rh antigen.
- In turn, the mother's agglutinins diffuse through the placenta into the fetus and cause red blood cell agglutination.

Hemolytic Disease of Newborn — First Pregnancy




RH+
father
RH–
mother
(a)
(b)
(c)
(d)
(e)

Incidence of the Disease

- A Rh-negative mother having her first Rh-positive child usually does not develop sufficient anti-Rh agglutinins to cause any harm.
- About 3 % of second Rh-positive babies exhibit some signs of Erythroblastosis Fetalis;
- About 10 % of third babies exhibit the disease;
- The incidence rises progressively with subsequent pregnancies.

Effect of the Mother's Antibodies on the Fetus

- Maternal antibodies cause agglutination of fetus's blood. The agglutinated red blood cells subsequently hemolyzed, releasing hemoglobin into the blood.
- The fetus's macrophages then convert the hemoglobin into bilirubin, which causes the baby's skin to become yellow (jaundiced).
- The antibodies can also attack and damage other cells of the body.

Clinical Picture of Erythroblastosis

- The newborn baby is usually anemic at birth,and the anti-Rh agglutinins from the mother usually circulate in the infant's blood for another 1 to 2 months after birth, destroying more and more red blood cells.
- The hematopoietic tissues of the infant attempt to replace the hemolyzed red blood cells.
- The liver and spleen become greatly enlarged and produce red blood cells in the same manner that they normally do during the middle of gestation.

- Because of the rapid production of red cells, many early forms of red blood cells, including many *nucleated blastic forms*, passed from the baby's bone marrow into the circulatory system.

- Severe anemia of erythroblastosis fetalis is usually the cause of death,many children who barely survive the anemia exhibit permanent mental impairment or damage to motor areas of the brain because of precipitation of bilirubin in the neuronal cells, causing destruction of many, a condition called *kernicterus*

Treatment of the HDN

- 1-Replace the neonate's blood with Rh-negative blood.

  EXCHANGE TRANSFUSION

- About 400 milliliters of Rh-negative blood is infused over a period of 1.5 or more hours while the neonate's own Rh-positive blood is being removed. This procedure may be repeated several times during the first few weeks of life, mainly to keep the bilirubin level low and thereby prevent kernicterus.
- By the time these transfused Rh-negative cells are replaced with the infant's own Rh-positive cells, a process that requires 6 or more weeks, the anti- Rh agglutinins that had come from the mother will have been destroyed.

Exchange Transfusion

Accomplishes the following:

1. Remove antibody coated RBCs: Not all but many.
2. Removal of maternal antibody. Remember this antibody is passively transferred so the more we remove the better.
3. Removal of bilirubin: reduce bilirubin in newborn.
4. Replacement of RBCs: Treating the anemia

Prevention of Erythroblastosis Fetalis

2-Rh immunoglobulinglobin, an anti-D antibody that is administered to the

1- expectant mother starting at 28 to 30 weeks of gestation.

2-Rh-negative women who deliver Rh-positive babies to prevent sensitization of the mothers to the D antigen.

This greatly reduces the risk of developing large amounts of D antibodies during the second pregnancy.

A- Effect of the anti-D antibody is to inhibit antigen-induced B lymphocyte antibody production in the expectant mother.

B- The administered anti-D antibody also attaches to D antigen sites on Rh-positive fetal red blood cells that may cross the placenta and enter the circulation of the expectant mother, thereby interfering with the immune response to the D antigen.

3-Plasmaphoresis of mother:

Rh antibodies from maternal blood are removed.

Tests done for transfusion of blood

- 1-ABO blood typing and Rh factor
- 2-Cross matching
- 3-Tests for infective agents
- A-Blood smear for malarial parasites
- B- Serological tests:
  - Hepatitis B surface antigen
  - Hepatitis C antigen
  - Human immune deficiency syndrome