SAWA UNIVERSITY

اسم المادة و الكورس

Rh blood group system

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Overview

-RH group system.

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-CHARECTERISTIC OF TRANSFUSION

-Incidence of the Disease

Clinical Picture of Erythroblastosis

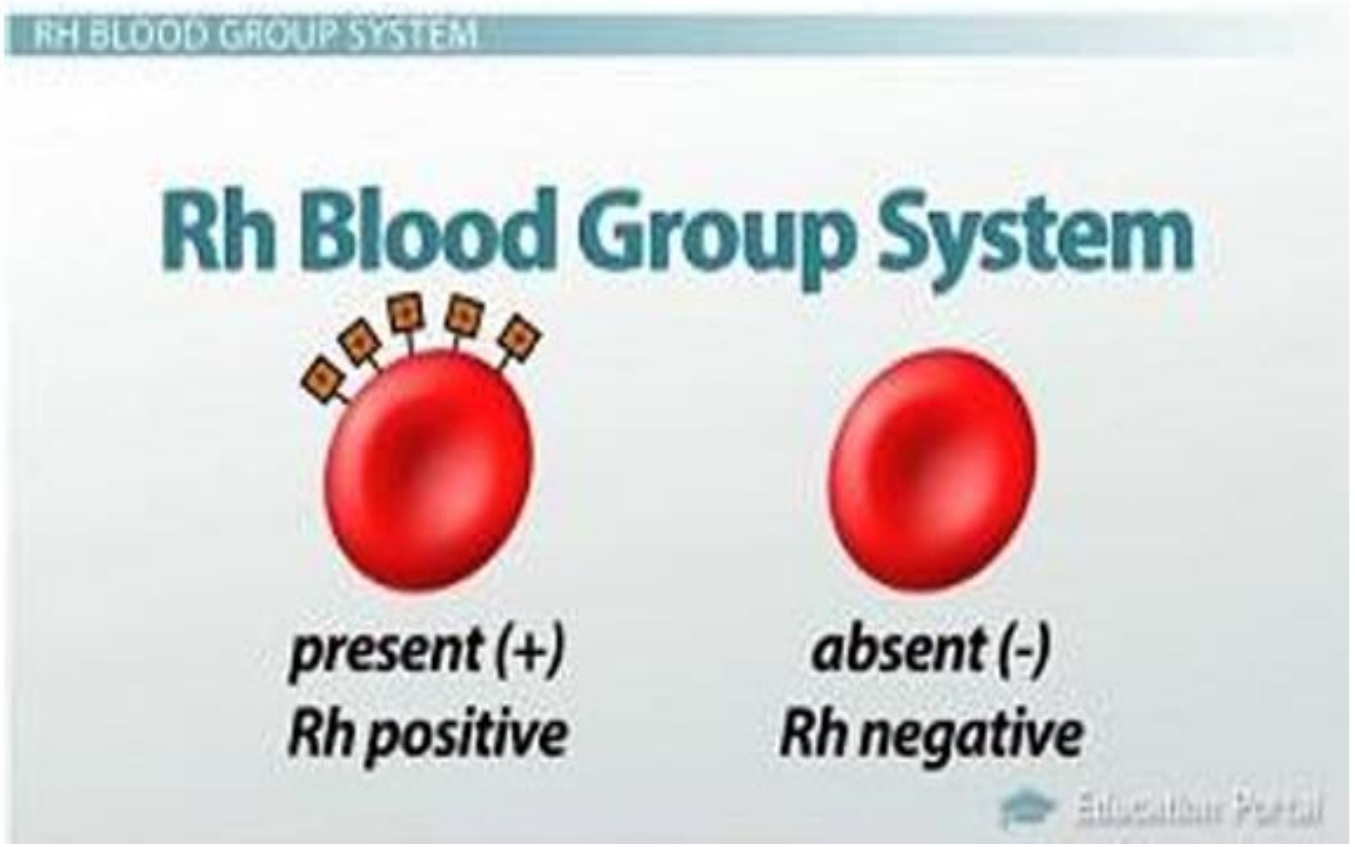
Rh blood group system

Rh blood group system is a system for classifying blood groups according to the presence or absence of the Rh antigen on the cell membranes of RBC, it is also important when transfusing blood. The designation Rh is derived from the use of the blood of **rhesus monkeys** in the basic test for determining the presence of the Rh antigen in human blood. **The major difference between the O-A-B system and the Rh system is the following**:

In the O-A-B system, the plasma agglutinins responsible for causing transfusion reactions develop spontaneously, whereas in the Rh system, spontaneous agglutinins almost never occur

RH ANTIGENS—RH-POSITIVE" AND RH-NEGATIVE PEOPLE.

There are **6** common types of Rh antigens, each of which is called an Rh factor. These types are designated C, D, E, c, d, and e. A **person who has a C antigen does not have the c antigen, but the person missing the C antigen always has the c antigen. The same is true for the D-d and E-e antigens. Also, because of the manner of inheritance of these factors, each person has one of each of the three pairs of antigens.** 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, whereas a person who does not have type D antigen is said to be Rh negative.



About 85% of all white people are Rh positive and 15%, Rh negative.

Rh Immune Response

Formation of Anti-Rh Antibody (Agglutinins)

When RBCs containing Rh factor are injected into a person whose blood does not contain the Rh factor—an Rh-negative person—anti-Rh agglutinins develop slowly, reaching maximum concentration of agglutinins about 2-4 months later. This immune response occurs to a much greater extent in some people than in others. With multiple exposures to the Rh factor, an Rh negative person eventually **becomes strongly** "sensitized" to Rh factor. Rh antibodies stimulated as a result of transfusion or pregnancy, they are immune. Associated with **Haemolytic Transfusion Reaction (HTR) and Haemolytic Disease of the Fetus Newborn (HDFN).**

Characteristics of Rh Transfusion Reactions

1. **If an Rh-negative person** has never before been exposed to Rh positive blood, transfusion of Rh-positive blood into that person will likely cause no immediate reaction. However, anti-Rh antibodies can develop in sufficient quantities during the next 2-4 weeks to cause agglutination of the 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.

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

What happen during pregnancy for the Rh-positive offspring of Rh-incompatible parents?

During pregnancy, there are a similar danger exists for the Rh positive offspring of Rh-incompatible parents, when the mother is Rh negative and the father is Rh-positive.

1. The first child of such parents is usually in no danger, but the mother has acquired anti-Rh antibodies by virtue of incompatible blood transfusion.

2-During labour a small amount of the fetus's blood may enter the mother's blood stream.

3-The mother will then produce anti-Rh antibodies, which will attack any Rh-incompatible fetus in subsequent pregnancies.

4-. This process produces erythroblastosis fetalis, or hemolytic disease

1. of the newborn, which can be fatal to the fetus or to the infant shortly after birth.

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 RBCs. In most instances of erythroblastosis fetalis, the **mother is Rh negative and the father is Rh positive**. The baby has inherited the Rh-positive antigen from the father, and the mother develops anti-Rh agglutinins from exposure to the fetus’s Rh .

antigen. In turn, the mother’s agglutinins diffuse through the placenta into the fetus and cause RBC agglutination.

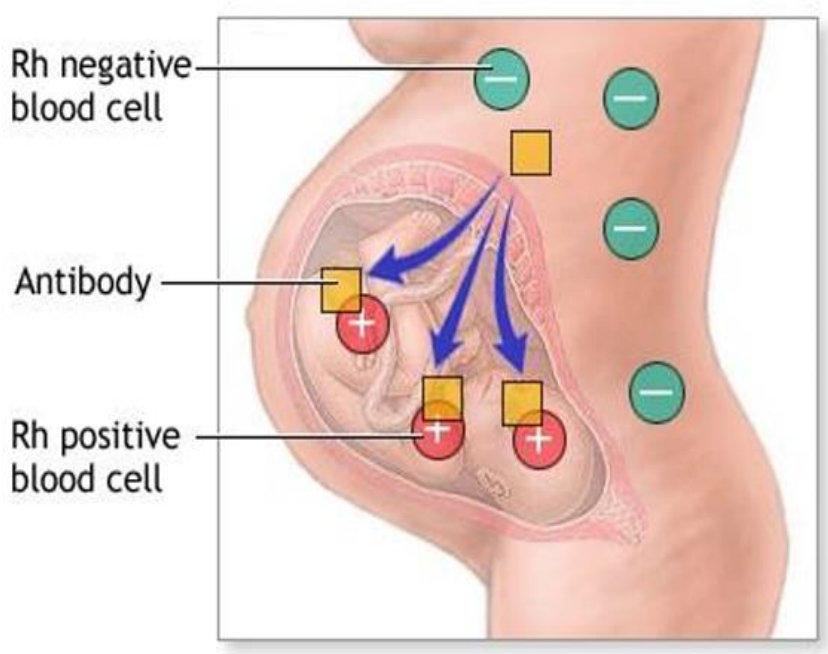
Incidence of the Disease

1. An Rh-negative mother having her first Rh-positive child usually does not develop sufficient anti-Rh agglutinins to cause any harm.
2. However, about 3% of second Rh-positive babies exhibit some signs of erythroblastosis fetalis
3. About 10% of third babies exhibit the disease
4. The incidence rises progressively with subsequent pregnancies.

Effect of the Mother's Antibodies on the Fetus

After anti-Rh antibodies have formed in the mother, they diffuse slowly through the placental membrane into the fetus's blood, as shown in figure below.

There they cause agglutination of the fetus's blood. The agglutinated RBCs subsequently **hemolyze, 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

1. **The jaundiced, erythroblastotic** newborn baby is usually anemic at birth, and the anti-Rh agglutinins from the mother usually circulate in the infant's blood for **another 1-2 months** after birth, destroying more and more RBCs.

2-. **The hematopoietic tissues** of the infant attempt to replace the hemolyzed RBCs.

3. **The liver and spleen become greatly enlarged** and produce RBCs in the same manner that they normally do during the middle of gestation.

4. Because of the rapid production of RBCs including many nucleated **blastic forms** are passed from the baby's bone marrow into the circulatory system, and it is because of the presence of these nucleated blastic RBCs that the disease is called **erythroblastosis fetalis.**

5. Although the 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 Neonates with Erythroblastosis Fetalis.

One treatment for erythroblastosis fetalis is **to replace the neonate's blood with Rh-negative blood.** About 400 milliliters of Rh-negative blood are 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.**

Prevention of Erythroblastosis Fetalis

The disease can be avoided **by vaccinating the mother with Rh immunoglobulin after delivery of her firstborn if there is Rh incompatibility**. The Rh vaccine destroys any fetal blood cells in the mother's body before the mother's immune system can develop antibodies.

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

Any questions?