REVIEW ARTICLE

The Role of Ob/Gyn Ultrasound in Medical Student Education: Immunological Disorders of Pregnancy Assessed by Ultrasound

Janet F Piskurich

Associate Professor of Immunology and Microbiology, Department of Medical Education, TTUHSC-Paul L Foster School of Medicine, 5001 El Paso Drive, El Paso, Texas 79905, USA

Correspondence: Janet F Piskurich, Associate Professor of Immunology and Microbiology, Department of Medical Education TTUHSC-Paul L Foster School of Medicine, 5001 El Paso Drive, El Paso, Texas 79905, USA, Phone: (915) 783-1700 Ext. 236 Fax: (915) 783-1715, e-mail: janet.piskurich@ttuhsc.edu

Abstract

To learn immunology in the absence of visual images would be an onerous and tedious chore. Most immunology textbooks use a large number of illustrations and diagrams to assist students as they endeavor to grasp the semantics and fundamentals of immunology, which are exacting and can be confusing to the uninitiated. Likewise, ultrasound imaging can be used to elucidate the consequences of an immune incompatibility between the mother and child during pregnancy in a manner that brings both clarity and clinical relevance to the medical students' understanding of this basic concept. Here we illustrate in the context of medical student education the use of ultrasound in the assessment of immunological disorders of pregnancy.

Keywords: Immune hydrops fetalis, alloimmunized pregnancy, ultrasonography, medical education.

INTRODUCTION

Immune hydrops fetalis is an accumulation of edema fluid that results from hemolytic disease in the fetus during intrauterine growth.[1] This occurs as the result of a blood-group incompatibility between the mother and the child. When the fetus inherits paternal red blood cell antigens that are foreign to the mother, the mother can develop an immune reaction that leads to this hemolytic disease. Theoretically, any of the red blood cell antigens can produce this effect. However, those that result in disease are certain Rhesus (Rh) blood group antigens, especially the D antigen, and the ABO antigens.

IMMUNOPATHOGENESIS

The basis for immune hydrops fetalis is immunization of the mother by paternal blood group antigens on fetal red blood cells and the unrestricted passage of maternal antibodies through the placenta to the fetus. When the cytotrophoblast barrier is lost during the last trimester of pregnancy or during childbirth, the mother is exposed to foreign paternal blood group antigens and becomes sensitized. A previous miscarriage, amniocentesis or blood transfusion may also result in sensitization.

When an Rh D-negative woman is pregnant with an Rh D-positive fetus, Rh D-positive fetal red blood cells cross into the maternal circulation causing the mother to produce anti-Rh D antibody.[2] Sensitization is even more likely if the mother and fetus are ABO compatible, since concurrent ABO incompatibility allows the fetal red blood cells to be coated with anti-A or anti-B and quickly removed from the maternal circulation. The dose of the antigen is also important. The mother must be exposed to at least 1 mL of Rh-positive red blood cells for sensitization to occur. Likewise the isotype of the antibody is important. Initial exposure of the mother results in formation of anti-Rh D IgM antibodies which do not cross the placenta. Therefore, Rh hemolytic disease is uncommon for a first pregnancy, while exposure during subsequent pregnancies results in a vigorous anti-Rh D IgG response which is capable of crossing the placenta.

Due to the prophylactic use of anti-Rh D IgG, administered at 28 weeks and within 72 hours of delivery, incidence of hemolytic disease due to this anti-D has been lowered dramatically such that a significant proportion of Rh hemolytic disease cases are now caused by anti-C or anti-E. The most frequent cause of hemolytic disease is now ABO blood group antigens, in particular anti-A produced by a mother who is group O against a fetus who is

group A. Most anti-A and anti-B antibodies are of the IgM isotype. However, some group O women produce IgG antibodies directed against group A and B antigens. In this case, the disease can occur even during the first pregnancy. Neonatal red blood cells express only small amounts of A and B antigens, and maternal IgG antibodies are partially neutralized by binding to A and B antigens in plasma and tissue fluid, and on other cells. Therefore, this form of the disease is relatively mild.

The excessive destruction of red blood cells results in anemia and accumulation of bilirubin in the neonate. In particular, this anemia can lead to hypoxic injury to the heart and liver. If severe, circulatory and hepatic failure can occur. Cardiac failure results in an increase in venous capillary pressure while hepatic failure causes a drop in plasma protein levels and a decrease in oncotic pressure. It is this combination of cardiac and hepatic failure that leads to the generalized edema that is called hydrops fetalis.

DIAGNOSIS AND TREATMENT OF ALLOIMMUNIZED PREGNANCY

If one pregnancy has been complicated by immune hydrops fetalis due to Rh incompatibility, the chance that an Rh-incompatible fetus in a subsequent pregnancy will be affected is very high (as high as 80-90%). Maternal serum anti-Rh antibodies are quantified at regular intervals. Historically severity of hemolysis in the at-risk fetus has been assessed by measuring bilirubin levels of amniotic fluid obtaining by amniocentesis, but ultrasonography is a much less invasive method that can be used to monitor the amount of fluid in the amniotic cavity, placental thickening, fetal liver enlargement, pericardial effusion and ascites.[3] Conventional ultrasound can detect ascites and soft tissue edema, definite signs of severe fetal involvement (Fig. 1). Doppler sonography measuring the middle cerebral artery peak systolic velocity (MCA-PSV) can be performed weekly to monitor anemia in the fetus (Fig. 2). When fetal anemia is severe, peak systolic velocity is markedly increased because the blood viscosity is markedly decreased. Cases of severe hemolytic disease can be treated by performing intrauterine transfusions and early delivery.

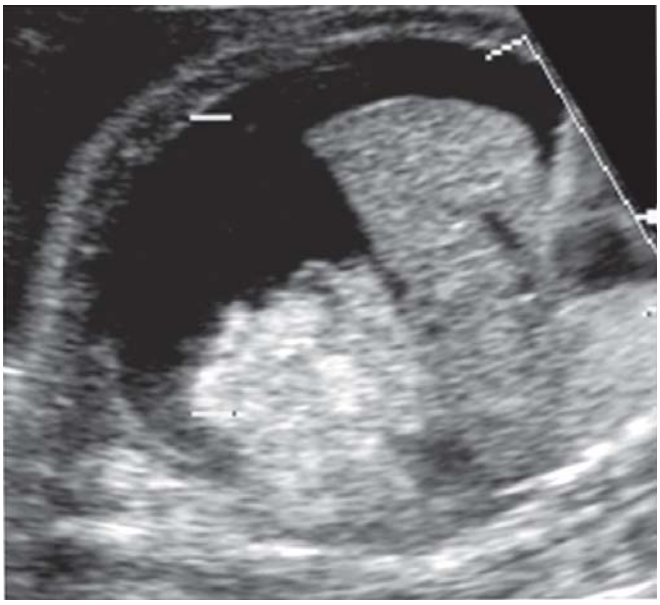

Fig. 1: Severe ascites and enlarged liver in a hydropic fetus at 27 weeks gestation

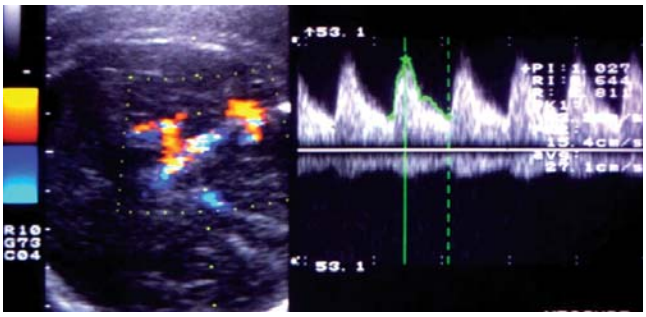

Fig. 2: Pulsed Doppler waveform analysis of middle cerebral artery in a fetus with nonimmune hydrops. Increased peak systolic velocity (63.2 cm/s) is an indicator of severe anemia and decreased blood viscosity

REFERENCES

1. Fetal hydrops. In: Kumar V, Abbas AK, Fausto N, Aster JC (Eds): Robbins and Cotran Pathologic Basis of Disease, Philadelphia: Saunders 2010;449-61.
2. Pregnancy and neonatal haematology. In: Hoffbrand AV, Moss PA H, Pettit JE (Eds): Essential Hematology, Oxford: Blackwell Publishing 2007;356-59.
3. Skupsky D. Ultrasound in the management of the alloimmunized pregnancy. In: Kurjak A, Chervenak FA (Eds): Donald School Textbook of Ultrasound in Obstetrics and Gynecology, New Delhi: Jaypee Brothers Medical Publishers 2008;501-06.