high proteinuria, immediate hospitalization and adequate medical or surgical treatment.

In any case wide use of the caesarean section does not improve the fetal prognosis. However very often it improves the maternal prognosis.

The main causes of maternal morbidity are cerebrovascular lesions (7.4% of the cases), acute pulmunary edema (1.4%), intravascular disseminated coagulation (5%), acute renal failure (4.8%), postpartum uterine atony (5.4%) and abruptio placentae (5.6%) ([1]).

The most common causes of maternal death are: cerebrovascular lesions (72.1%), coagulopathies (3.5%), postpartum hemorrhages (5.8 %) and respiratory failure of an outstanding entity ([1]).

In 22 patients there was only one case (4.54%) of acute pulmunary edema medically treated and one case of maternal death (4.54%) in a patient suffering from repeated eclamptic fits. The remaining patients were completely recovered on discharge. The immediate medical treatmeant considerably improved maternal prognosis, but owing to the seriousness of the illness and to its complications, prevention is the aim at present.

Therefore, it is fundamentally important for the patients to have an adequate sanitary education (the type of diet, periodic obstetric check-up, an accurate control of concomitant pathologies and hospitalization for the delivery) and an early diagnosis of hypertension in pregnancy.

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HEMOLYTIC DISEASE OF THE NEWBORN SECONDARY TO ANTI-FY^a AND ANTI-S

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Summary: One case of rare maternal-fetal immunization in a patient affected by Cooley's anemia, is reported. The opportunity for a complete characterization of the blood group and for a search for maternal antibodies in patients with a history of multiple blood transfusions is stressed.

INTRODUCTION

The maternal-fetal immunization due to antigens other than those of the Rh(D) and ABO systems achieved a new interest with the decrease in Rh(D) disease. It accounts for 2 to 5% of hemolytic disease of the newborn (Weinstein, 1982). In many cases exchange transfusions were performed

Table 1. — *Blood findings of the family members.*

	Blood group	Maternal serum	Direct Coombs	Cord serum	Cord cells eluate
Father	A Rh^+ S^+ Fy^{a+}				
Mother	O Rh^+ S^- Fy^{a-}				
First child	A Rh^+	Anti-A 1:256 anti-Fy^a 1:128	+ + − −	—	Anti-A
Second child	A Rh^+ S^+ Fy^{a+}	anti-Fy^a 1:1024 anti-S 1:128 anti-A 1:512 anti-B 1:128	+ + + −		Anti-Fy^a Anti-S

(Weinstein, 1976) and several perinatal deaths (16% for the Duffy system) have been reported (Lee et al., 1978).

The present work deals with a case of maternal-fetal immunization in a patient affected by Cooley's anemia, with a history of many blood transfusions.

CASE REPORT

The patient, aged 35, gravida 2, para 1, was admitted in labor at the 38th week of gestation. Her blood group was 0, CcDEe, $M^+N^+P^+S^-s^+$, K^-k^+, Fy^a. Her hushand blood groups was A, Cc De, $M^+N^-P^-S^+s^+$, K^-k^+, Fy^{a+}.

The patient, affected by thlassemia major, had received blood transfusions for several years. She suffered from viral hepatitis at the age of 22 years. At 28 years a splenectomy was done. In this circumstance an antibody determination revealed the presence of an anti-Duffy (Fy^a) titre of 1:512. At 30 years she developed a cardiopathy secondary to hemosiderosis, therefore no more transfusions were given. At 32 years, during her first pregnancy a titre of 1:128 for anti-Fy^a and of 1:256 for anti-A was registered at the 38th week of gestation. That pregnancy ended with a cesarean section at the 39th week. A 2650 g male fetus, Apgar 6/9, blood group A, CcDe, was born. The cord blood showed a positive direct Coombs (+ + − −). Immune antibodies were not found in the cord serum. Anti-A antibodies were present in an eluate of red cells. The presence of anti-Fy^a was not verified and it wasn't possible to determine Duffy antigens on cord erytrocytes. During the first day the newborn developed increasing jaundice. Bilirubin was 6,9 mg %ml, hemoglobin 20 g/dl, hematocrit 74%. An exchange transfusion was performed. Bilirubin reached the highest level of 7 mg %ml the next day, with decreasing values afterward.

In the present pregnancy an indirect Coombs test at the 35th week showed an anti-Fy^a titre of 1:512, and an anti-S titre of 1:256. At 38 weeks a 2500 g male fetus, Apgar 4/9, blood group A, CcDe, $M^+N^-S^+s^+$, $P^+K^-k^+$, Fy^{a+}. was delivered with cesarean section.

The direct Coombs was positive (+ + + −). There were no immune antibodies in the cord serum, while anti-S and anti-Fy^a were found in an eluate from cord cells.

The blood findings of the family members are summarized in tab. 1.

In the first day of life the bilirubin reached a level of 13.3 mg %ml, therefore an exchange transfusion was done. A value of 7.6 mg %ml of bilirubin was registered after two days, with slowly decreasing levels afterward. The infant presented tremors and spontaneous trepidations. An hypocalcemia (3.5 mEq/l) was corrected by means of calcium gluconate infusions. Signs of mild irritation with multiple focalizations were registered with electroencephalography.

DISCUSSION

The S antigen, one of 36 antigens of the MNSs system, is present in 59% of the white population, with a gene frequency of 0,337-0,344 (Mollison, 1979). The Fy^a antigen, and its allele Fy^b, belong to the Duffy system. An incidence of 66% for Fy^a, of 34% for Fy^{a-}, and of 0.03% for Fy^{a-b-}. has been reported in the white population (Weinstein & Taylor, 1975). Both S and Fy^a antigens should be responsible for weak antigenic stimuli (Mollison, 1979), however severe hemolitic diseases of the newborn have been reported (Lee, 1978; Griffith, 1980).

According to some Authors (Feldman et al., 1973), the maternal-fetal immunization to the S antigen should be a relatively frequent cause of mild erythroblastosis which can determinate a late anemia syndrome. In 3 out of 5 reported cases of fetal erythroblastosis due to the S antigen, the mother had previously been transfused: one case ended with the intrauterine death of the fetus, one with death after 60 hours from birth, one required an exchange transfusion, one developed jaundice and late anemia and one case was lost to follow-up (Feldman et al., 1973).

In 12 out of 19 reported cases of fetal erythroblastosis due to the Fy^a antigen, the mother had previously received blood transfusion. Six cases required exchange transfusion, while three other cases ended with death of the newborn (Greenwalt et al., 1959; Lee et al., 1978).

In our case the mother had shown immunization to the Fy^a antigen following multiple blood transfusions before her first pregnancy. Wether a possible Fy^a positivity, besides the ABO incompatibility, had contributed to the hemolytic disease of her first child is not known. Both anti-S and anti-Fy^a antibodies were found in the second pregnancy. Both seemed to originate from fetal antigenic stimuli, anti-Fy^a at an higher titre, probably due to the previous immunization. However the role that the two antigens played in the pathogenesis of the hemolysis in the newborn cannot be quantified, as it depends on the type of IgG that they produce. In fact an hemolytis property seems to belong only to IgG_1 and IgG_3 sub-classes, with different potential for hemolysis and different case to cross the placenta (Rote, 1982).

The increasing number of maternal-fetal isoimmunization due to rare antigens suggests the opportunity of characterizing completely the blood group and of searching for maternal antibodies, even in the first pregnancy, at least in the presence of a positive history for previous transfusions.

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