



Obstetric Practice Related Severe Neonatal Jaundice

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Case-I

A 2300 gm neonate born at 38 weeks to an O negative woman by vaginal route was referred from outside hospital for increasing jaundice despite phototherapy. Father was O positive. Mother received 300 microgram of Anti D immunoglobulin within 24 hours of birth of her first baby whose blood group was O positive. Antenatal period of current pregnancy was uneventful. Natal events were normal following vaginal delivery. Icterus was noticed at 14 hours of life. Phototherapy was started. As bilirubin was raising baby was referred to us at 64 hours of life. At admission baby had icterus involving palms and soles. He was active with normal cry and vitals. There were no features of encephalopathy. There was pallor and mild hepatomegaly.

Investigations revealed blood group of O positive, total serum bilirubin of 27.5mg/dl (at 69 hours) with direct of 0.5mg/dl, positive DCT, PCV of 23.3% and reticulocyte count of 10.5%. Peripheral smear showed evidence of hemolysis. Sepsis screen, electrolytes and ABG were normal.

Rh isoimmunisation was considered. Double volume exchange transfusion was done and blue light phototherapy was continued. Post exchange bilirubin was 13.4mg/dl. Baby required 4 more days of phototherapy. Initially we thought this was a case of anti D failure. Further, we reviewed the maternal history. Later mother reported that one year after the birth of her first child she did conceive and underwent MTP at 3 months of pregnancy. However, she did not receive Anti D following MTP. This has led to sensitization and disease in the next child. At six months follow up child was normal without any sequelae.

Case-II

A 3100 gm term neonate born to an Rh negative mother by LSCS was referred at 80 hours of life in view of hyperbilirubinemia. Mother's blood group was A negative. Father was O positive. Mother received Anti D immunoglobulin within 24 hours of delivery of first born O positive male neonate. Antenatal period was uneventful in the current pregnancy. Baby was born by LSCS and cried soon after birth. Noticed to have jaundice within 24 hours and started on phototherapy. As the bilirubin was increasing baby was referred to our centre. At admission baby was active with normal vitals. Icterus was involving palms and soles. There was splenomegaly of 1 cm. CNS examination was normal.

Investigations revealed blood group of O positive, total serum bilirubin of 28.7 mg/dl (at 82hours), with direct of 2.1 mg/dl. DCT was strongly positive with reticulocyte count of 9.2%, PCV of 32.5 and serum albumin of 3g/dL. Peripheral smear showed hemolysis.

Considering Rh isoimmunisation exchange transfusion was done. Post exchange bilirubin was 15.5 mg/dl. Baby required 5 more days of phototherapy and discharged. On first follow up at 6 months of life baby was doing well. There was a written document regarding the anti D prophylaxis following first child birth. So possibility of a case of anti D failure was thought of. Maternal history was reviewed. It was noted that mother had a pregnancy in between the first and the current pregnancies. However, she had spontaneous abortion at one and half month of gestation. Dilatation and curettage was done. She did not receive anti D immunoglobulin following the procedure. This resulted

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in the severe Rh disease in the subsequent newborn.

Both the above cases of severe neonatal jaundice were particularly related to the obstetric practice. Hence needs recognition and sensitization(1). Howard(2) et al raised a concern regarding the adequacy of practice of established guidelines. Before the introduction of anti D immunoglobulin, hemolytic disease of the fetus and newborn affected 9-10% of the pregnancies. Seventeen percent of negative women who do not receive anti D immunoglobulin prophylaxis become alloimmunised. Among the affected pregnancy, mild to moderate hemolytic anemia and hyperbilirubinemia occur in 25-30% of fetuses and neonates. Hydrops fetalis occurs in 25 % of such cases. Introduction of anti D immunoglobulin dramatically reduced alloimmunisation. Among fetomaternal transfusion 90 % occurs at delivery and 10 % antenatally in third trimester. Less than 0.1 ml of fetal blood is enough to cause alloimmunisation. Therapeutic and spontaneous abortions are associated with 4-5 % and 1.5-2% risk of alloimmunisation in nonimmunised women respectively. In threatened abortion there is 10 % risk of fetomaternal hemorrhage(1,3,4,5).

Postnatal prophylaxis decreases the risk of alloimmunisation by 90 %. This practice has been considered by many as an acceptable and beneficial routine intervention for the last thirty years. Further a dose at 28-29 weeks reduces risk from 2% to 0.1%. Antenatal dose remained little controversial because of potential shortage, cost effectiveness and possible effects on the fetus. Appropriateness of routine antenatal anti D administration has been hotly debated and argued in favor on the basis of existence of silent' fetomaternal transfusion causing sensitization(4,5). There was a concern for some anemia in the unborn baby as ten per cent of the anti-D will cross the placenta with effect on babies. In 1997 consensus conference in Britain decided to recommend routine antenatal administration as the

best way forward in moving closer to one hundred per cent protection from iso- immunization.

Fetal RBC mass is small in first trimester. Hence 50 microgram of anti D immunoglobulin is enough for 1st trimester events to protect against isoimmunisation (1,5,6).

Current scientific evidence recommends anti D prophylaxis after first trimester pregnancy loss. In both the above cases established protocols are not followed resulting in severe Rh isoimmunisation requiring exchange transfusion. Severe neonatal jaundice would have been prevented from occurring. Fortunately early referral and timely exchange transfusion resulted in normal outcome in babies. This probably represents only tip of the iceberg and we presume that there may be many such cases occurring and going unnoticed. Hence there is a need to increase awareness of the necessity for anti-D administration after potentially immunising events during pregnancy. In this regard we would like to stress again for sensitization of the concerned practitioners.

References

1. ACOG practice bulletin. Prevention of Rh D alloimmunization. Number 4, May 1999 (replaces educational bulletin Number 147, October 1990). Clinical management guidelines for obstetrician-gynecologists. American College of Obstetrics and Gynecology. *Int J Gynaecol Obstet* 1999; 66: 63-70.
2. Howard HL, Martlew VJ, McFadyin IR and Clarke CA. Preventing Rhesus D hemolytic disease of the newborn by giving anti-D immunoglobulin: are the guidelines being adequately followed? *Br J Obstet Gynaecol* 1997; 194: 37-41.
3. Herman M, Kjellman H, Ljunggren C. Antenatal prophylaxis of Rh isoimmunisation with 250microg anti-D immunoglobulin. *Acta Obstet Gynecol Scand* 1984; 124: 1-15.
4. Ghosh S, Murphy WG. Implementation of the rhesus prevention programme: a prospective study. *Scott Med J* 1994; 39: 147- 49.
5. Bowman JM. Controversies in Rh prophylaxis. Who needs Rh immunoglobulin and when should it be given? *Am J Obstet Gynecol* 1985; 151: 289-294.
6. Stewart FH, Burnhill MS, Bozorgi N. Reduced dose of Rh immunoglobulin following 1st trimester pregnancy termination. *Obstet Gynecol* 1978; 51:318-322.