HDN DUE TO ANTI-D AND ANTI-C

Case Study by Jim Perkins, M.D. (©2009)

M.C. was a 42 year old white woman who was found to be group B, Rh negative with a positive antibody screen on her initial prenatal testing. Antibody identification demonstrated anti-D at a titer of 16 and anti-C at a titer of 8, and she was referred here for management of her high risk pregnancy.

Her past obstetric history was significant for an uncomplicated full term LTCS 11 years earlier; caesarean section was indicated for active HSV infection. RhIG was administered after the delivery. She also had 2 elective abortions, both followed by RhIG, and one first trimester spontaneous abortion and D&C in 2 years earlier, again with RhIG prophylaxis.

A paternal blood sample was requested for use in calculating the likelihood of having an Rh positive fetus:

Paternal Rh phenotype

ANTI-	D	C	E	c	e
TEST	+	+	+	+	+

Serial antibody titers were performed, leading to serial intrauterine transfusion. The mother was CMV-seropositive and was therefore not offered directed donation of maternal blood for the fetus for the initial transfusions.

Antibody titers and IUT data

Week	Maternal		Fetal			
	Anti-D	Anti-C	Anti-D	Anti-C	DAT	Hct*
15	128	8				
21	128	8	16	2	3+	21.1 → 44.8
23	2040	64	256	8	2+	31.0 → 55.7
26	5120	128	512	32	vw+	23.4 → 37.8
29	2560	64	512	16	neg	15.6 → 31.5
31	5120	64	1024	32	neg	11.1 → 28.9
34 (Delivery)	5120	64	1024	16	neg	16.9

*Hematocrit was determined after-the-fact in the laboratory, but treatment decisions were guided by a bedside hemoglobinometer (results not available).

Prior to scheduling the last IUT or possibly delivery, the mother was again evaluated for possible directed donation for the fetus/neonate. Donor screening revealed a history of IV drug use by the parents including needle sharing between them.

Amniocentesis at 34 weeks revealed fetal lung maturity (LBND 108,000, L/S ratio 3:1). The mother underwent an uncomplicated cesarian delivery (LTCS/BTL) of a 1950 gm male infant with Apgars of $9^1/9^5$.

On exam the baby had hepatospenomegaley (liver down 5 cm, spleen down 4 cm), and looked pale and jaundiced. There was mild respiratory distress treated with low flow oxygen.

The infant typed as group O, Rh negative at birth. The DAT was negative and the antibody screen was positive (see last line of table above). His subsequent hospital course is summarized in the following table:

Day of life	Time	Bili, T/D	Hgb/Hct	Plts	Retic
1	1051	6.4/1.5	5.8/16.9	66,000	2.8
	1259	Transfused			
	2208	14.9/3.5			
2	0437	18.7/5.1			
	0814	Exchange transfusion			
	1227	16.3/4.8	13.7/40.5	26,000	0.4
	1343	16.2/4.8			
	1449	Exchange transfusion			
	1849	16.8/5.2			
	2002	Platelet transfusion			
	2106	13.4/3.4			
3	0109		14.4/43.0	77,000	
	0608	13.6/3.7			
	2103	19.7/7.7			
	2157	Exchange transfusion			
4	0300	14.8/5.9	13.6/40.3	44,000	0.4
	0847	11.7/4.9			
	1514	13.7/5.4			
	2228	13.0/5.6			
5	1041	12.7/6.2			
6	0655	12.3/6.6	13.9/41.6	38,000	
7	0747	10.9/6.2	14.5/42.7	56,000	0.1
	1840	11.3/6.4			
8	0739	10.1/5.9			
10	0652	8.6/4.9			
11	1015		13.9/42.1	198,000	<0.1

DISCUSSION QUESTIONS:

1. Calculate the likelihood of paternal transmission of an Rh positive gene.

2. What considerations would be appropriate in deciding whether to use of maternal blood? How would you process it?

3. Why did this mother become immunized in spite of receiving RhIG?

4. Why did the antibody titer increase so dramatically after the transfusion in week 21.

5. How would you predict when the next in a series of IUT's is required?

6. How do you explain the infant’s Rh typing results and DAT at birth?

7. Why might the baby have needed so many exchange transfusions?