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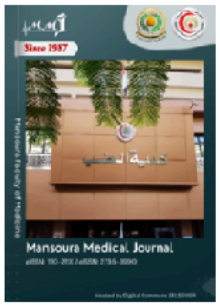

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INCIDENCE OF HDNB AND FOETOMATERNAL BLOOD GROUP INCOMPATIBILITY IN MANSOURA UNIVERSITY HOSPITALS

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ABSTRACT

The study included 731 full term babies aged 1-6 days, presented with hyperbilirubinaemia (S.bilirubin > 12 mg/dl) in the period from 1985 to 1989. The babies and their mothers were subjected to I) full history taking and thorough clinical examination 2) determination of S. bi lirubin, Hb% and reticulocyte count in infants 3) blood group typing ABO, Rho and antibody screening for mothers and babies. In some instances they were typed for other blood group systems. 4) IgG antibodies (anti-A and anti-B) was done in ABO incompatible pregnancies.

It was found that out of the 731 cases with hyperbilirubinaemia, 73 babies (9.99%) had HDNB. In the later group, anti-D was detected in 24 infants (32.88%), anti-D and anti-C in 4 cases (5.48%), anti-c in 8 cases (10.96%), ABO incompatibility in 34 cases (46.58%) and each of anti Fy^a, anti-Jk^a, & anti-K in one case (1.37%)

The results of the work showed that HDNB due to foetomaternal blood group incompatibility still a major problem which needs concern.

Abbreviation (Fy^a : Duffy a; JK^a : Kidd a; K : Kell; D. C : direct coombs, E. T : exchange transfusion).

INTRODUCTI ON

Red blood cells contain blood group antigens inherited from the father that may be lacking in the mother. Due to foetomaternal haemorrhage, some mothers make blood group antibodies against these paternally derived antigen before and after birth. Maternal IgG antibodies are transported across the placenta and can cause haemolysis of foetal red blood cells.

Haemolytic disease of newborn (HDNB) can vary in severity from hydrops foetalis in utero to only IgG sensitization of foetal red blood cells without haemolysis, (Synder et al., 1983, Quinn et al., 1988 and Ichikawa et al., 1989).

This work was intended to study the incidence of HDNB in Mansoura University Hospitals due to foetomaternal blood group incompatibility in the period from 1985 to 1989. This may throw a light on the importance of each blood group system incompatibility and help in selection of the suitable donor for exchange transfusion free from the accused antigen when needed. More over, we try to outline the approach to diagnosis in foetomaternal blood group incompatibility.

MATERIAL AND METHODS

This work was carried out on 731 full term babies aged 1-6 days presented to the neonatal intensive care unit with icterus and total serum bilirubin > 12 mg/dl. Each newborn was subjected to :

1- Full history taking and thorough clinical examination.

2- Laboratory investigations which include blood picture, reticulocyte count and serum bilirubin determination.

3- Determination of blood group ABO, Rh and minor groups when required.

4- Direct antiglobulin test, heat elution of sensitized RBCs, then the eluted antibodies were tested using the commercial screening cells (Diamed cells).

Each mother of each selected newborn was subjected to :

1- Full history taking and details of delivery events.

2- Blood grouping, Rh typing and antibody screening (A. S) and in some instances they were typed for other systems. A. S was made by testing mother serum against two or three reagent red cell preparations "Diamed cell" of known Phenotype using antiglobulin technique. The test procedures were selected to provide optimum conditions for detection of antibodies in the Rh, Kell, duffY, Kidd, MNSs, Lewis, lutheran & Xg systems. When an antibody had been de-

tected, we identify it's specificity using (Diamed -Cell identification).

3-Indirect coombs test.

4- In ABO-incompatible pregnancies, antenatal tests for IgG antibodies (anti-A & anti-B) were introduced in vaccinated women and in those who experienced ABO-HDNB following prior delivery, IgM is neutralized with neutrab, or by agglutination with a panel of known group A and B and then separation of supernatant serum which were used in detection of IgG antibodies (Simmons, 1980).

RESULTS

The results of this work are shown in tables (1-6) and Fig. 1.

Table (I) shows the incidence of red cell antibodies causing the foetomaternal incompatibility among 731 cases of icteric newborn at Mansoura University Hospitals in the period from 1985 to 1989. From this table it could be observed that 73 cases (9.99%) are due to foetomaternal blood group incompatibility. The most predominant cause of HDNB is ABO incompatibility (46.58%), then anti-D and anti-D plus anti-C accounting for about 38.36% of cases of HDNB, followed by anti-c (10.96%).

Tables (2-6) show the detailed results of foetomaternal incompatibility due to ABO blood grouping system, anti-D in d-women, anti-D in D and D^u-women, anti-D plus anti-C in ddcc and D^u cc-women and anti-c in CC -women respectively.

DISCUSSION

This work demonstrated that the most common cause of HDNB is ABO incompatibility with an incidence of 4.65% in relation to total icteric babies and 46.58% relative to type specificities. These data are moderate between the finding of Maisels and Giffords (1986) "20%" and that of Valentine (1958) "60%". Fourteen cases out of 34 were first born infants, this is in agreement with Mollison, (1979), Grundbacher (1980) and Haestis et al. (1988). Gupte and Bhatia (1980) explained the affection of first born infant by the formation of IgG antibodies by mothers on exposure to various agents e.g. food stuffs, infective agents and vaccines which contain A or B like substances. As regard the blood grouping, we found O-

A combination in 20 situations and O-B combination in 14 (Table 2). This go with the finding of Bengtason and Virneholt 1974. DCT was found to be negative in 35.25% of cases with ABO incompatibility, which go with the finding of Oski (1984) and Huestis et al., (1988).

However, antibody detection in elute in DCT negative babies was found to be positive in 10 cases out of 12 and IDC test for mothers was found to be positive. So in suspected ABO-HDNB with negative DCT we must do IDC test for mothers and antibody detection for the elute of red cells of the infant. Moreover, IgG (anti-A and anti-B) was found to be more than 64 mg/dl in all mothers with ABO incompatibility.

Although the Rh immunoglobulin was introduced since many years, anti-D and anti-D Plus anti-C mediated HDNB was found to account for 38.36% of cases with HDNB. This go with the figures given by Clarke et al. (1983) and Wilke (1988), whereas Hardy and Napier (1981) reported a reduced incidence. On the other hand, our finding demonstrated the importance of D^u (with weak positive Rho antigen) which may demonstrate an allo-anti-D causing HDNB (Table 4). A phenomenon, well recognized by White et al (1982) and Lacey et al. (1983). Moreover, we found anti-D causing HDNB in 2 Rho-positive mothers. This may be explained by missing a portion of the mosaic structure of Rh-D antigen (Ostgard et al., 1985) or this antibody is anti-Lw and not anti-D (Tippett, 1977 and Ostgard et al., 1986).

The Lw gene is regulated by the Rh gene, it's absence lead to absence of Lw antigen inspite of presence of Rh-gene. The majority of Rho positive cells are Lw-positive, so anti-LW reacts with allmost all Rho Positive cells, in this case anti-LW as anti-D is suggested (Tippett, 1977).

HDNB caused by anti-c was found in 10.96% of cases (Tables 1 and 5). an incidence agree with that recorded by Smith et al. (1967) and Hardy and Napier (1981). This finding denote the importance of phenotyping for complete Rhesus blood grouping of young girls and women before blood transfusion to avoid immunization by supplying blood lacking c-antigen for those negative for this antigen. Phenotyping of red cells of babies is also essential for confirming the allo immunization.

Moreover, HDNB could be caused by other blood group systems than Rh and ABO as anti-K. anti-FY^a and anti-JK^a (Soleta et al., 1983).

Caine and Heubach (1986) reported that anti-K is the most potent among the irregular antibodies; after those of Rh system, which can cause severe HDNB and still-birth. So we advocate the use of only K-negative blood for female recipients.

Antibodies of the Duffy system come next to those of Rh and kell systems among the irregular antibodies causing HDNB (Shah & Gilyas, 1983). Duffy sensitization must be managed in a manner similar to Rh sensitization, with aminocentesis being performed at the appropriate time as it is diagnostic and is of higher predictability (Sturket et al., 1988). The blood bank should be notified prior to delivery of a possible affected foetus, so that FY(a-b-) or Fy (a-B+) blood can be obtained for exchange transfusion if indicated (Contreras et al., 1983).

HDNB caused by anti-JK^a is mentioned by many investigators (Simpson et al., 1978, and Harrison and Popper, 1981).

In this work, each of anti-K, anti-Fy^a and anti JK^a was found in one case (1.37%), and this go with the Previous reports.

CONCLUSION

- ABO incompatibility is the most common cause of HDNB in our locality (46.58%) and the best Predictor for ABO-HDNB is the titre of maternal IgG anti-A or anti-B, and the results of direct antiglobulin test on RBCs of cord blood fortified by elute examination for antibodies in cases with negative D.C.

- Rh D-mediated HDNB still significant (38.36%), though Rh-immunoglobulin prophylaxis was introduced since many years, so it needs reevaluation. Also, the use of anti-D immunoprophylaxis in D^u variants is suggested.

- HDNB can occur secondary to foetomaternal incomPatibility in minor blood group systems so it is important to select the donor lacking the corresponding antigen for ET of the affected infant.

Table (1) : Incidence of red cell anti - bodies causing haemolytic disease among 731 cases of icteric newborn in Mansoura University Hospital.

Ceuse	NO . of Ceuse	Frequency In relat lon to total specimens (%)	Frequency In relation to HDNB (%)
- Anti - D	24	3.28	32.88
- Anti - D + Anti - C	4	0.55	5.48
- Anti - c	8	1.09	10.96
- ABO incompa - tibility	24	4.65	46.58
- Anti - FY^a	1	0.14	1.37
- Anti - JK^a	1	0.14	1.37
- Anbi - K	1	0.14	1.37
Total	73	9.99	

Table (2) : HDNB due to ABO incompatibility .

	NO . of cases	% relat . to all icteric infants	% re lat. to HDNB	% relat . to ABO incompa - tibility	Remarks
Total NO .	34	4.65	46.58	100%	- 2 infants recover withou t treatment
Blood g roup Combinations					
O - A	20	2.74	27.4	58.82	- 11 treated with pho -
O - B	14	1.92	19.9	41.18	totherapy
DCT of cord blood					- 21 treated wi th pho - therapy & exchange transfusion
- Positive	22	3.02	30.14	64.75	
- Negative	12	1.63	16.44	35.25	
E lute in negative DC	10	1.37	13.7	29.41	
NO . of mothers with positive IDC (Ig G antibody > 64)	32	4.38	43.12	43.12	

Table (3) : pregnancy outcome in d - women with D - husband and significant Anti - D .

Ceuse NO .	Maternal group & pheno type	Anticedent risk Fs				Cord blood type and group	D.C.	Remarks
		parity	HDNB	Abortion	Bl, trasf			
1	A 1 B - ccddee	2nd	-	-	-	A 1 B - ccDee	++	2 ET
2	A 1 ccddee	2nd	-	-	-	O - ccDEE	++	1 ET
3	A 1 ccddee	2nd	Ist bady	one	-	B - CcDee	+++	1 ET & died
4	O - ccddee	2nd	-	-	-	O - CcDee	++	1 ET
5	AB - ccddee	4th	2nd & 3rd	-	-	A 1 - CcDee	+++	2 ET
6	O - ccddee	4th	2nd	-	-	O - ccDee	+++	2 ET
7	A 1 B - ccddee	3rd	2nd	-	-	A 1 - ccDee	++	1 ET
8	B - ccddee	3rd	-	-	-	O - ccDee	++	1 ET
9	O - ccddee	4th	2nd & 3rd	-	-	O - ccDee	++	2 ET
10	O - ccddee	3rd	2nd	-	+	O - ccDee	+++	1 ET & died
11	A 1 B - ccddee	3rd	2nd	-	-	A 1 ccDee	+++	1 ET
12	B - ccddee	3rd	-	-	-	O- ccDee	++	1 ET
13	O - ccddee	4th	2nd	-	-	A 1 - CcDee	-	2 ET
14	A 1 B - ccddee	3rd	-	-	-	B - ccDee	+	1 ET
15	B 1 ccddee	4th	-	-	-	B , ccDee	++	1 ET
16	B , ccddee	2nd	-	one	-	O , ccDee	+	1 ET
17	A 1 B , ccddee	4th	2nd & 3rd	-	-	A 1 , ccDee	++	1 ET

ET : Exchange trans fusion

Table (4) : pregnancy outcome in D and Duwouen with significant anti - D and D - husband .

case No .	Maternal blood group and pheno type	Antecedent risk factors	Cord blood group	D . C .	Re marks
1	A 1B - ccD^{U} Ee	6 th para - from 2 nd to 5 th infant had HDNB & died .	B - ccDEe	+++	Died
2	A 1 - ccD^{U} ee	3 rd para	perinatal death	-	
3	O - ccD^{U} ee	2 nd para	O - Cc Dee	++	ET
4	A 2 B - ccD^{U} ee	3 rd gravida , 2 nd 1 abour with HDNB	A2 - ccDee	+	ET
5	A 1 - ccD^{U} ee	2 nd pare	A 1- ccDee	+	ET
6	A 1 - ccDee	6 th para - previous 5 infants with good conditions .	O - CcDee	+++	ET
7	B - ccDee	5 th para - previous still birth	B - ccDee	++	ET

Table (5) : pregnancy outcome with anti - D + anti C

case No .	Maternal blood group and pheno type	Antecedent risk factors	Cord blood group	D . C .	Re marks
1	A 1 B - ccddee	8 th para, 2 nd to 7 th infants died with HDNB	perinatal death	- -	
2	B - ccD^{u} Ee	6 th para 5 th died with HDNB	O - Cc DEe	+++	one ET & died
3	A1 B - ccD^{u} ee	5 th gravida with history of previous HDNB	B - CcDee	++	one ET & died
4	B - ccD^{u} Ee	7 th gravida with history of mu ltiple HDNB	B - CcDee	++	two ET & died

Table (6) : pregnancy outcome in patients with anti - c

case No .	Maternal blood group and pheno type	Antecedent risk factors	Cord blood group	D . C .	Re marks
1	A 1 CCDee	2 nd para	B - ccDee	++	1 ET
2	B - CCDee	3 rd para	O - CcDee	+++	2 ET
3	B - CCDee	4 th para with parinatal death of 2nd infant	B - CcDee	+++	2 ET
4	O - CCDEe	3 rd para	perinatal death		
5	O - CCDee	4 th para , 2 nd & 3 rd child died with HDNB	O - CcDee	++	
6	O - CCDee	6 th para	B - Cc DEe	++	1 ET
7	O - CCDEe	2 nd para	B - CcDEe	+++	1 ET
8	A 1 - CCDEe	2 nd para	A I - CcDEe	++	1 ET

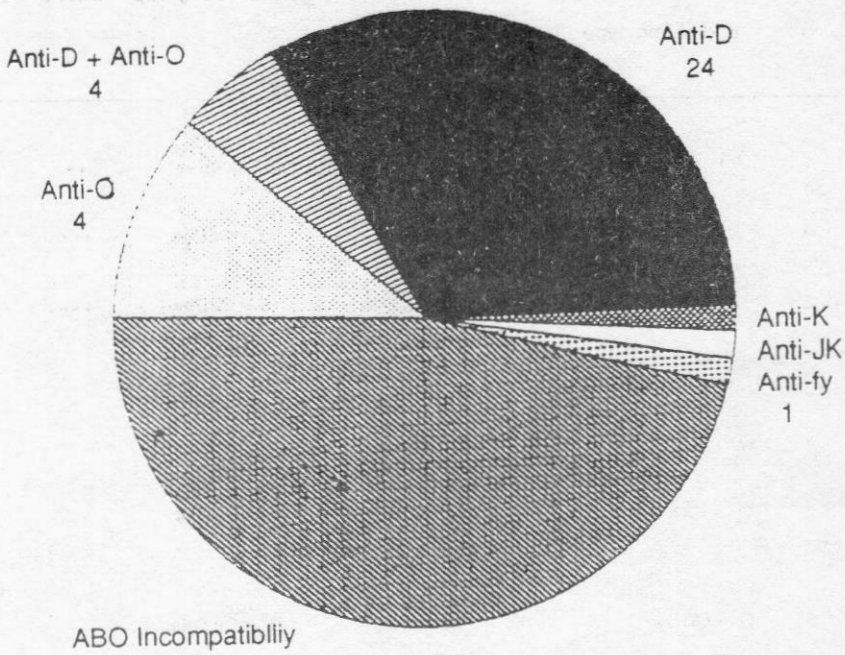

Fig. I Incidence of red cell anti-bodies causing HDNB among 731 jaundiced newborns

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الملخص العربى

معدل حدوث مرض تكسير الدم عند الأطفال حديثى الولادة وعدم توافق فصائل الدم بين الطفل والأم فى مستشفيات جامعة المنصورة

أجرى هذا البحث فى الفترة من ١٩٨٥-١٩٨٩ وشمل ٧٣١ طفلاً كامل النمو يتراوح عمره بين ١-٦ أيام ويشكو من اليرقان، وكانت نسبة البيلروبين أكثر من ١٢ مجم لكل ١٠٠سم٣ من مصل الدم.

وقد تم عمل الآتى لكل من الأم والطفل :

١) التاريخ المرضى والفحص الاكلينيكى الدقيق.

٢) تحديد نسبة البيلروبين فى مصل الدم ونسبة الهيموجلوبين وعد الخلايا الشبكية فى دم الطفل.

٣) تحديد فصائل الدم (ABO)، (Rh) وفحص الدم بحثاً عن الأجسام المضاده عند الأطفال والأمهات وتحديد فصائل الدم الأخرى فى الحالات الايجابية.

٤) تحديد كمية الأجسام المضاده (جـ) المضاد لنوع الفصيلة ،

وقد خلص البحث لما يأتى :

١) عدد ٧٣ طفلاً كانوا مصابين بمرض تكسير الدم عند الأطفال حديثى الولادة نتيجة عدم توافق دم الطفل مع الأم بنسبة ٩٩ر٩٪ من حالات اليرقان المبكر.

٢) الأجسام المضاده لـ (Rh D) كانت موجوده فى ٢٤ طفلاً من الأطفال حديثى الولادة المصابين بتكسير فى الدم بنسبة ٨٨ر٣٢٪ من هؤلاء الأطفال، والأجسام المضاده لـ Rh C Rh D + فى ٤ حالات ٤٨ر٥٪ والأجسام المضادة لـ Rh C فى ٨ حالات (٩٨ر١٠٪).

٣) مرض تكسير الدم بسبب عدم التوافق فى فصيلة الدم (ABO) كان موجوداً فى ٣٤ حالة بنسبة ٥٨ر٤٦٪.

٤) الأجسام المضاده لكل من K, JK^a, Fy^a كل منهما كان موجوداً فى حالة واحدة بنسبة ٣٧ر١٪من حالات تكسير الدم فى الأطفال حديثى الولادة.

ومن هذا يتضح أن مرض تكسير الدم عند الأطفال حديثى الولادة بسبب عدم توافق دم الطفل مع الأم مازال يمثل مشكلة تحتاج لمزيد من الاهتمام.