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Neonatal Jaundice: A Study of the Incidence in Children of Rh (D) Negative and 0 Rh (D) Positive Mothers

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ABSTRACT

Despite advances in neonatal care, neonatal jaundice remains a common problem in maternity wards. The present retrospective epidemiological study collected data on a sample of 710 newborns and compared the incidence of neonatal jaundice in infants born to Rh (D) negative and 0 Rh (D) positive mothers. The primary aim was to determine whether the higher incidence of maternal alloimmunisation in newborns was causally related to a potentially higher incidence of neonatal jaundice in newborns of 0 Rh (D) positive mothers. To the end, we investigated a possible association between the incidence of neonatal jaundice in 0 Rh (D) positive mothers and the neonatal blood group. The incidence of neonatal jaundice was not found to differ between maternal blood groups. We discuss new preventive measures that may reduce the incidence of neonatal jaundice and thereby reduce the length of hospital stay.

KEYWORDS

neonatal jaundice; AB0 and Rh (D) alloimmunisation; haemolytic disease of the foetus and newborn

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SUMMARY

Neonatal jaundice is a common diagnosis in the neonatal period, occurring in more than half of all newborns. Most cases represent the physiological postnatal jaundice that does not threaten the life or development of the child and resolves spontaneously. Less commonly, early onset of jaundice is a symptom of a condition that can threaten the newborn's health. A typical example is haemolytic disease of the foetus and newborn. In this article we report a retrospective epidemiological study of over seven hundred newborn babies. We compared the incidence of neonatal jaundice between maternal blood groups with negative Rh (D) factor and mothers with blood group 0 Rh (D) positive and the incidence of maternal alloimmunisation. This study didn't confirm statistically significant differences in the incidence of neonatal jaundice between newborns of Rh (D) negative and 0 Rh (D) positive mothers (P = 0.292). The incidence of alloimmunisation was also comparable in both groups of newborns. These results confirm the need to monitor 0 Rh (D) positive mothers during pregnancy and their newborns after delivery to prevent the development of severe neonatal jaundice. This practice is already common and standard in most neonatal units.

INTRODUCTION

Neonatal jaundice affects almost half of all newborns and is one of the most common problems in all neonatal units (1). Jaundice becomes visible at a serum bilirubin level of 60–80 μmol/L and above. Typical symptoms include yellow skin and mucous membranes, scleral discolouration, poor feeding and excessive sleepiness (1–3). Bilirubin is the product of haemoglobin metabolism from the accelerated breakdown of fetal red blood cells (RBCs). Unconjugated (free, indirectly reactive) bilirubin is a hydrophobic molecule that is normally bound to plasma albumin and transported to the liver. In hepatocytes, bilirubin is bound to glucuronic acid by glucuronosyltransferase. Conjugated (directly reactive) bilirubin is excreted in the bile and passed into the small intestine (see Figure 1) (4). Based on these findings, a distinction can be made between unconjugated and conjugated hyperbilirubinemia. The study reported in this article deals only with unconjugated hyperbilirubinemia.

The most common cause of hyperbilirubinemia in newborns is physiological postnatal jaundice. Its onset usually peaks on the second or third day after birth. It is influenced by several factors, most importantly by an increased number of fetal RBCs and therefore a higher haematocrit. Fetal RBCs are developmentally adapted to a lower partial pressure of oxygen in the blood. After birth, the partial pressure of oxygen is increased, causing the RBCs to degrade more rapidly and the serum bilirubin level to rise. The hepatobiliary system is also less mature in neonates than in adults (1). Several studies have examined the effect of early initiation of breastfeeding in reducing the risk of neonatal jaundice, suggesting that it may reduce neonatal weight loss after birth (5).

The onset of jaundice in the first hours after birth is always pathological. It may be an early symptom of several

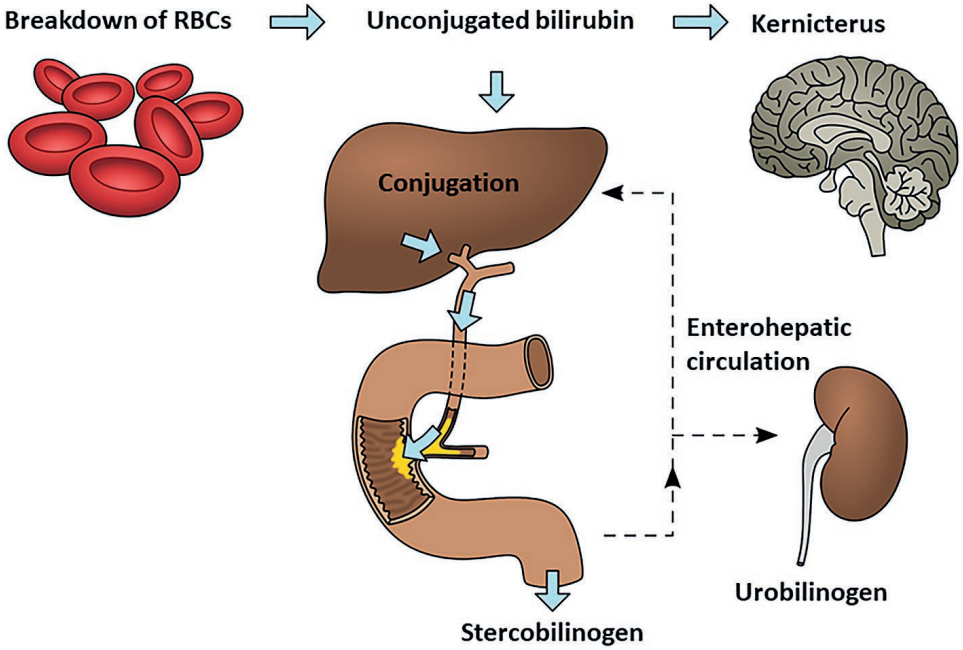

Fig. 1 Bilirubin metabolism and excretion. Haemoglobin and other haem proteins are released from the breakdown fetal red blood cells (RBCs). Unconjugated bilirubin is bound to albumin and transported to the liver. When the albumin binding capacity is saturated, unconjugated bilirubin can cross the blood-brain barrier and can form deposits (core icterus). In hepatocytes the bilirubin is conjugated with glucuronic acid. Conjugated bilirubin is water-soluble and excreted into the bile and passed into the small intestine. Bacterial oxygenation converts bilirubin into urobilinogen and stercobilinogen, which stains the stool. Part of conjugated bilirubin or urobilinogen are reabsorbed by the enterohepatic circulation. Scheme adopted and freely modified from Lissauer T, Clayden G. Neonatal medicine. In: Illustrated Textbook of Paeditrics. 4th edition. Mosby, Elsevier; 2011: 168–172.

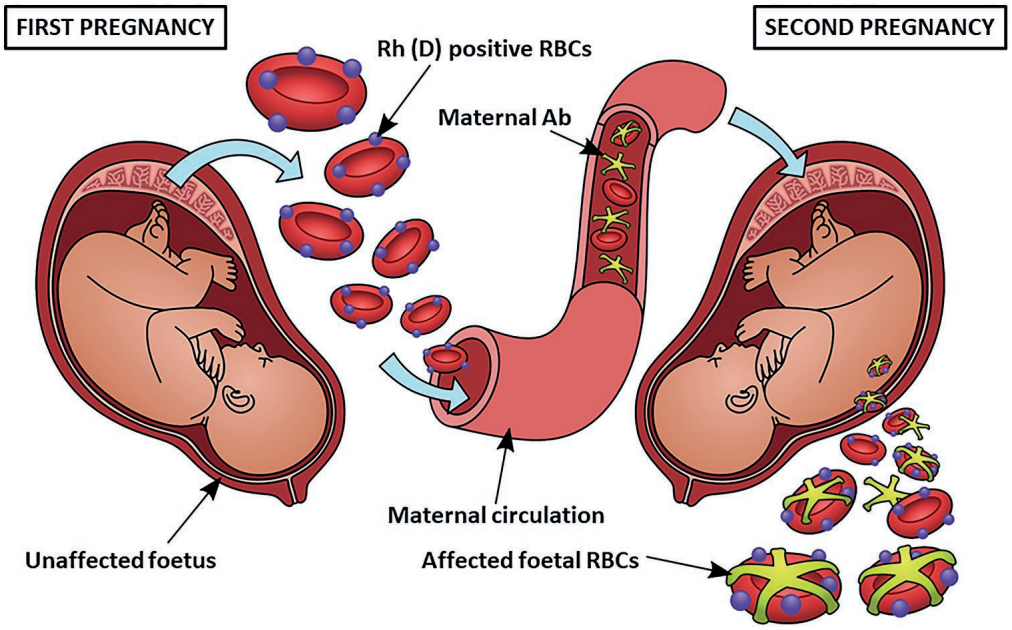

Fig. 2 Scheme of the maternal alloimmunisation and transplacental transfer of antibodies. Typically, the immune system of a Rh (D) negative mother, or less commonly a 0 Rh (D) positive mother, is stimulated by a previous fetomaternal haemorrhage and can produce antibodies. The IgG immunoglobulins cross the placental barrier, enter the fetal circulation during the next pregnancy and bind to the fetal red blood cells (RBCs), causing severe haemolysis. RBCs with bound antibodies are destroyed in the reticuloendothelial system. Scheme adapted and freely modified from MedlinePlus [Internet]. Bethesda (MD): National Library of Medicine (US); Hemolytic disease of the newborn [reviewed 2023 Dec 31]. Available from: https://medlineplus.gov/ency/article/001298.htm.

diseases (e.g. early infection, inborn errors of metabolism, spherocytosis). In this article, we focus on the epidemiology of haemolytic disease of the foetus and newborn (HDFN), excluding other causes of hyperbilirubinemia. HDFN was described in 1941 by the immunohaematologist Phillip Levin and his colleagues on the basis of maternal alloimmunisation (6). The key factor in the pathophysiology of the alloimmune response is the Rhesus (Rh) factor. The Rh factor is an inherited protein of RBCs that is determined by several antigens. The most important is antigen D. Its presence is called Rh (D) positive and its absence Rh (D) negative. Antigen D is a powerful immunological stimulant (7). The immune system of the Rh (D) negative mother is stimulated after a previous pregnancy or abortion of the Rh (D) positive newborn/foetus (when even minimal fetal haemorrhage occurs) and produces antibodies. These immunoglobulins cross the placental barrier and enter the fetal circulation during the next pregnancy and bind to Rh (D) positive fetal RBCs (see Figure 2). In rare cases, maternal alloimmunisation can be caused by an incorrectly administered transfusion or invasive diagnostic procedures – amniocentesis, chorionic villus sampling (8, 9). RBCs with bound antibodies are increasingly taken up and destroyed in the reticuloendothelial system. The resulting fetal anaemia can lead to tachycardia, fetal hydrops and even miscarriage if not treated appropriately (see Figure 3) (6). Classical HDFN is mostly described in Rh (D) negative mothers, but up to 25% of severe cases of HDFN occur in Rh (D) positive mothers. In newborns of 0 Rh (D) positive mothers, it has been suggested that different neonatal blood groups may result in different levels of antigenic stimulation. This stimulation could lead to increased antibody production. A higher incidence of maternal alloimmunisation can be expected, especially in neonates with blood groups A and B (10, 11). The high incidence can also be attributed to other types of Rh antibodies, such as anti-c. As mentioned above, the incidence of severe HDFN caused by incompatibility in the ABO blood system is comparable to the incidence of HDFN caused by Rh incompatibility. In the case of fetal-maternal blood group incompatibility, IgM antibodies are produced which cannot cross the placental barrier due to their pentameric molecular structure. However, some women produce IgG antibodies (namely IgG anti-A haemolysin antibodies in blood group A children and IgG anti-B haemolysin antibodies in blood group B children) which can cross the placental barrier and cause immune haemolysis of RBCs. Taking this mechanism into account, a higher incidence of maternal alloimmunisation or HDFN can be expected in children born to 0 Rh (D) positive mothers (3).

In HDFN, unconjugated hyperbilirubinemia can rise to levels that significantly exceed the binding capacity of plasma proteins. Bilirubin crosses the blood-brain barrier in excess, and bilirubin encephalopathy may develop, with deposits mainly in the basal ganglia and midbrain. If treatment is not started (phototherapy, intravenous immunoglobulins or, in extreme cases, exchange transfusion), the brain damage may become permanent. This is also known as kernicterus (8). The main treatment method used today is phototherapy. This involves using a lamp that emits the blue-green spectrum of visible light (wavelengths 430–490 nm). The light disrupts the structure of bilirubin, facilitating its metabolism and removal. For the first-line treatment of mild hyperbilirubinemia, a lamp or bili-bed is used. This is called general or conventional phototherapy. When the bilirubin level rises to the threshold

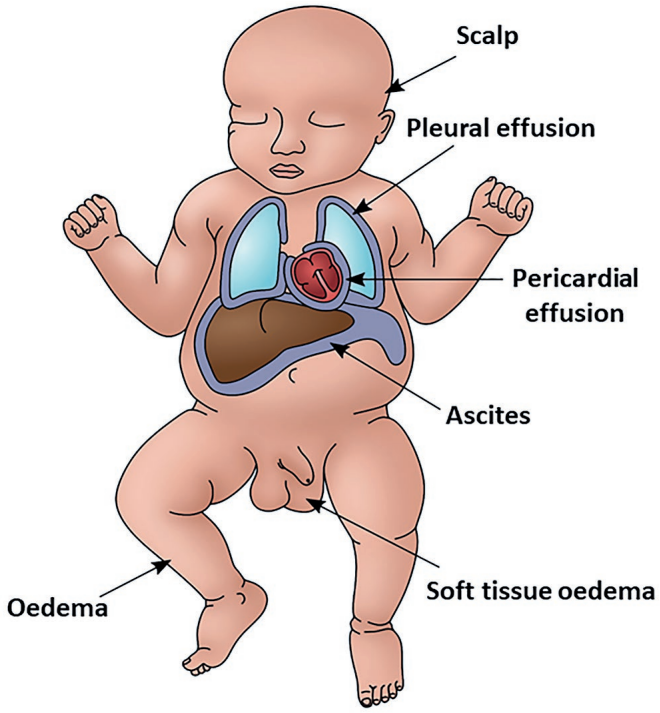

Fig. 3 Hydrops foetalis. Increased destruction of fetal RBCs may result in anaemia. Severe anaemia can cause tachycardia and even heart failure associated with pericardial and pleural effusion, ascites, and skin oedema. All these symptoms are part of hydrops foetalis. If the anaemia is not adequately treated, it may lead to miscarriage.

for exchange transfusion, second-line treatment is indicated, which is intensive phototherapy. This method uses two lamps with the same light characteristics. The irradiance of the light measured on the baby's skin below the centre of the phototherapy lamps is stronger compared to conventional phototherapy.

NEWBORNS COHORT AND METHODS

This paper presents a retrospective epidemiological study of newborns. Clinical and laboratory data were collected at the neonatal department of the Havlíčkův Brod Hospital between 12/2020 and 6/2022. These data were then statistically processed, i.e. the incidence in different groups was evaluated with the χ^2 test. The study group included 710 newborns born to mothers with blood groups A, B, AB, 0 Rh (D) negative and mothers with 0 Rh (D) positive. This selection of newborns is based on the screening of Rh (D) negative mothers to prevent the development of HDFN in subsequent pregnancies. The introduction of blood group testing of newborns of 0 Rh (D) positive mothers was prompted by the increased incidence of HDFN. Blood groups are not routinely tested in newborns of A and B Rh (D) positive mothers. Although HDFN can also occur in these infants, maternal alloimmunisation is less common. Therefore, investigation would be costly and, with current medical knowledge, would not allow intervention in the planning of the next pregnancy.

Inclusion criteria for the participants were: birth in the 36th week of gestation and later, no severe peripartum hypoxia (Apgar score in the first minute 5 points and above), no medication except the usual prophylaxis with vitamin K (1). Laboratory investigations of each participant after delivery from umbilical cord blood sample consisted a blood group, total umbilical bilirubin (μmol/L) and direct antiglobulin test (DAT). These tests should be standard in neonatal care. The diagnostic criteria for HDFN in this study were a positive umbilical PAT, anaemia in the first postnatal blood sample (haemoglobin 140g/L and below), and jaundice requiring phototherapy treatment. We did not monitor the occurrence of concomitant neonatal injuries such as cephalohaematoma, which can increase hyperbilirubinemia levels and cause severe neonatal jaundice. These injuries are common in newborns with delayed adaptation after birth, which were not included in this study.

The incidence of neonatal hyperbilirubinemia was assessed using a transcutaneous bilirubinometer, usually twice a day until discharge. More frequent transcutaneous monitoring was indicated by the paediatrician, especially in newborns with elevated umbilical bilirubin or positive DAT. Elevated umbilical or transcutaneous bilirubin levels above the cut-off value prompted subsequent serum bilirubin testing. Hodr-Poláček charts were used to assess hyperbilirubinemia. These charts were historically used in this department. Nowadays they are replaced by charts of the American Academy of Pediatrics (AAP) recommended by the Czech Society of Neonatology. As mentioned above, children born to A and B Rh (D) positive mothers were not included because the blood group of these children is not routinely checked. Their blood groups are only tested if there is a suspicion of HDFN. This could distort the incidence of HDFN.

RESULTS

710 healthy newborns were included in this study. The mean gestational age at birth was $39^{3/7}$ weeks (from 36 to 42 weeks of gestation). The mean birth weight was 3425 grams (minimum 2300 g, maximum 4630 g), the mean birth length was 50 centimetres (minimum 44 cm, maximum 55 cm). The average Apgar score was 9 points in the first minute and 10 points in the fifth and tenth minutes. As shown in the bottom three rows of Table 1, out of a total of 710 newborns, 137 were children of Rh (D) negative mothers (19.3%) and 573 were children of 0 Rh (D) positive mothers (80.7%). Both groups of newborns had similar characteristics. There were no statistically significant differences in the characteristics of neonates born to Rh (D) negative and 0 Rh (D) positive mothers.

Serum bilirubin was elevated above the physiological limit according to the charts in 85 infants for whom phototherapy was indicated. 20 of these newborns had Rh (D) negative mothers and the remaining 65 newborns had 0 Rh (D) positive mothers (see Table 1). The mean duration of phototherapy was almost the same in both groups, 47 and 47.4 hours, respectively. The incidence of alloimmunisation (positive umbilical DAT) was comparable in both groups of neonates (see Table 2). The incidence of hyperbilirubinemia in the newborns of Rh (D) negative and 0 Rh (D) positive mothers was compared using the

Tab. 1 Incidence of neonatal jaundice (indexed by indication for phototherapy) in the sample of 710 newborns, split by maternal Rh (D): Rh (D) negative pooled across maternal blood groups 0, A, B, AB, and Rh (D) positive pooled across maternal blood group 0.

Phototherapy		Maternal Rh (D) factor		Total
		N	P	
Yes	Observed	20	65	85
	% within row	23.5%	76.5%	100.0%
	% within column	14.6%	11.3%	12.0%
Nop	Observed	117	508	625
	% within row	18.7%	81.3%	100.0%
	% within column	85.4%	88.7%	88.0%
Total	Observed	137	573	710
	% within row	19.3%	80.7%	100.0%
	% within column	100.0%	100.0%	100.0%

Tab. 2 Incidence of neonatal jaundice (indexed by indication for phototherapy) in the sample of 710 newborns, split by neonatal blood group and maternal Rh (D).

Neonatal blood group	Rh (D) mother	Phototherapy indicated		Total
		No	Yes	
0	N	48	7	55
	P	296	25	321
	Total	344	32	376
A	N	50	5	55
	P	144	28	172
	Total	194	33	227
AB	N	3	3	6
	P	0	0	0
	Total	3	3	6
B	N	16	5	21
	P	68	12	80
	Total	84	17	101
Total	N	117	20	137
	P	508	65	573
	Total	625	85	710

χ^2 test. The test showed no statistically significant difference in the incidence of neonatal jaundice between Rh (D) negative and 0 Rh (D) positive mothers (χ^2 = 1.111, df = 1, P= 0.292).

In a more detailed analysis of the 65 children of 0 Rh (D) positive mothers, maternal alloimmunisation was detected in 22 newborns. 7 newborns had bilirubin levels above the threshold for intensive phototherapy. Maternal antibodies (positive umbilical DAT) were confirmed in 5 of these 7 newborns. For comparison, 2 infants of Rh (D) negative mothers were indicated for intensive phototherapy and both infants were DAT positive (see Table 3). This therapy was successful in all cases. There was only one newborn of a 0 Rh (D) positive mother who met the

Tab. 3 Incidence of common and intensive phototherapy compared with maternal alloimmunisation in neonates born to 0 Rh (D) positive and all Rh (D) negative mothers. Positive maternal alloimmunisation is similar in both groups.

Maternal blood group	Phototherapy indicated in newborns		
	Common	Intensive	Total
0 Rh (D) positive	58	7	65
	Maternal alloimmunisation (DAT) positive		
	17	5 (71%)	22 (33.8%)
All Rh (D) negative	18	2	20
	Maternal alloimmunisation (DAT) positive		
	4	2 (100%)	6 (30%)

criteria for HDFN (ABO incompatibility). The incidence of HDFN in this study was 0.14% of all newborns and 1.17% of newborns with neonatal jaundice for whom phototherapy was indicated, respectively. Despite proven alloimmunisation, no neonates with severe haemolytic anaemia requiring transfusion, administration of IVIG or exchange transfusion were reported.

The sample was also characterised according to neonatal blood group, based on our assumption that different neonatal blood groups could lead to differences in the strength of antigenic stimulation (10, 11). Table 4 shows the incidence of neonatal jaundice responding to phototherapy according to neonatal blood group and maternal blood group.

The incidence of jaundice in newborns of Rh (D) negative versus 0 Rh (D) positive mothers, by neonatal blood

Tab. 4 Incidence of neonatal jaundice (indexed by indication for phototherapy) in the sample of 710 newborns, subdivided by neonatal blood group and maternal Rh (D). The percentages show the incidence of jaundice for each subgroup compared to the total number of children.

Neonatal blood group	Maternal Rh (D)	Phototherapy indicated		Total
		No	Yes	
0	N	48	7 (12.7%)	55
	P	296	25 (7.8%)	321
	Total	344	32	376
A	N	50	5 (9.0%)	55
	P	144	28 (16.2%)	172
	Total	194	33	227
AB	N	3	3 (50.0%)	6
	P	0	0 (0.0%)	0
	Total	3	3	6
B	N	16	5 (23.8%)	21
	P	68	12 (15.0%)	80
	Total	84	17	101
Total	N	117	20 (14.6)	137
	P	508	65 (11.3)	573
	Total	625	85	710

Tab. 5 Incidence of neonatal jaundice (indexed by indication for phototherapy) in the sample of 710 newborns, split by neonatal blood group and maternal Rh (D). The abbreviation NaN (Not a Number) stands for no number.

Neonatal blood group		Value	df	p
0	χ^2	1.47	1	0.2252
	N	376		
A	χ^2	1.73	1	0.1880
	N	227		
AB	χ^2	NaN	1	NaN
	N	6		
B	χ^2	0.92	1	0.3369
	N	101		
Total	χ^2	1.11	1	0.2918
	N	710		

group, was tested using separate χ^2 tests. For none of the newborn blood groups did the tests detect a statistically significant difference between the incidence in newborns of Rh (D) negative versus 0 Rh (D) positive mothers (see Table 5). Therefore, we cannot conclude that there is an association between the development of neonatal jaundice and the blood groups of mothers and newborns. This null result should be interpreted with caution, as the lack of effect could be due to the small number of children represented especially for newborns blood type AB (only 3 newborns in this subgroup). However, given the caveats about interpreting null effects (where failure to detect an effect does not mean that the effect does not exist), further research with larger samples is needed to better understand the relationship between maternal Rh (D), neonatal blood group and the incidence of neonatal jaundice.

DISCUSSION

Before the introduction of prophylaxis in Rh (D) negative mothers with anti-D antibodies (introduced in the former Czechoslovakia in the 1980s), the worldwide incidence of HDFN was about 1% of all newborns and the mortality rate reached the level of 50% (12, 13). With the introduction of prophylaxis, the incidence of HDFN in newborns of Rh (D) negative mothers has decreased to about 0.5%. Nowadays, the incidence of Rh (D) and ABO alloimmunisation is similar (14). In our study, the incidence of ABO alloimmunisation was present in approximately 1% of ABO mismatched pregnancies. The results of our study correlate with other reviewed studies – the incidence of maternal alloimmunisation was not found to differ between the studied groups of newborns (10). These results don't support the initial hypothesis of a higher incidence of neonatal jaundice in newborns of 0 Rh (D) positive mothers. It's still necessary to continue with the established prophylaxis mentioned above. However, the incidence of neonatal jaundice and ABO alloimmunisation might be higher with extended screening of newborns of all Rh (D) positive mothers.

Because of this incidence, prenatal screening for maternal antibodies to the minor antigens of the Rh factor and other RBC antigens (e.g. anti-C and anti-K) is essential (ref 7, 8, 15–17). Combined first and third trimester screening for these antibodies has already been introduced in several countries (e.g. the Netherlands) to detect alloimmunisation not captured at first trimester screening ("late" alloimmunisation) and subsequent HDFN (8). The results of this screening showed that Rh antibodies were found in 1 in 300 pregnant women. Since 2011, the Czech Republic has introduced first and third trimester screening (at 12 and 28 weeks' of gestation) for numerous maternal antibodies (C, Cw, c, D, E, e, K, k, Fya, Fyb, Jka, Jkb, S, s, M, N, Lea) (18). These antibodies are more common in Rh (D) negative women, but further research may shed new light on the alloimmunisation of newborns of Rh (D) positive mothers. This knowledge would be important as the onset of jaundice caused by alloimmunisation in newborns of 0 Rh (D) positive mothers could be as rapid as in newborns of Rh (D) negative mothers (19). Pathological elevation of umbilical cord bilirubin is also observed, and serum bilirubin may reach higher levels. Possible prevention of new antigens may lead to a reduction in the incidence of HDFN, similar to the previously established anti-D prophylaxis (20). New interventions would reduce neonatal morbidity associated with multiple neonatal jaundice and have also economic benefits. The authors suggest that further research into alloimmunisation in children born to 0 Rh (D) positive mothers would improve the quality of care for neonates born to A and B Rh (D) positive mothers.

CONCLUSION

This study compared the incidence of pathological neonatal jaundice in neonates born to Rh (D) negative and 0 Rh (D) positive mothers. Contrary to our expectations, based on screening practice and universal HDFN prophylaxis in all Rh (D) negative mothers, the predicted higher incidence of jaundice due to maternal alloimmunisation in 0 Rh (D) positive mothers wasn't confirmed. There are no reports in the reviewed literature of similar data prior to the introduction of the now widespread HDFN prophylaxis in Rh (D) negative mothers. In line with the present results, which didn't show a higher incidence of neonatal jaundice in Rh (D) negative compared to Rh (D) positive mothers, we confirm the need to monitor Rh (D) positive mothers and their newborns. However, this practice was introduced in our neonatal units only a few years ago. Its initiation was also the impetus for the conduct of this study.

CONFLICT OF INTEREST STATEMENT

All authors declare no conflicts of interest.

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