Universe International Journal of Interdisciplinary Research
(International Peer Reviewed Refereed Journal)
DOI No. – 08.2020-25662434

© UIJIR | ISSN (O) – 2582-6417
MAY 2026 | Vol. 06 Issue 12
www.uijir.com

ABO INCOMPATIBILITY AND NEWBORNS HYPERBILIRUBINEMIA: DESCRIPTIVE STUDY IN AL-FAYHAA TEACHING HOSPITAL, BASRAH, IRAQ

Author's Name Rafid Khalaf Al-Hasani, Husam Jawad Al-Attar, Mustafa Salah Alahmed

Affiliation:

M.B. Ch.B, C.A.B.P., Department of Pediatrics, Al-Fayhaa Teaching Hospital, Basrah Health Directorate, Basrah, Iraq.

Corresponding Author Name & E-Mail: Mustafa Salah Alahmed, Medicalresearch10@yahoo.com

ABSTRACT

ABO hemolytic disease of the newborn (ABO-HDN) is one of the most common causes of immune-mediated hemolysis during the neonatal period. It results from maternal immunoglobulin G (IgG) antibodies crossing the placenta and destroying fetal erythrocytes, often leading to neonatal jaundice and hyperbilirubinemia. Objectives: To estimate the risk of ABO hemolytic disease of the newborn in Basrah province, evaluate its clinical manifestations, and assess treatment outcomes among affected neonates. Methods: A retrospective study was conducted at the Department of Pediatrics, Al-Fayhaa Teaching Hospital, Basrah, Iraq, during 2023. Forty term neonates diagnosed with jaundice and/or anemia secondary to ABO incompatibility were included. Demographic, maternal, clinical, and laboratory data were collected from hospital records. Investigations included blood group determination, complete blood count, serum bilirubin, and direct Coombs test (DCT). Management strategies and clinical outcomes were reviewed. Results: A total of 40 neonates were included, comprising 22 (55.0%) males and 18 (45.0%) females. The mean birth weight was 3500 ± 465 g, and the mean age at admission was 4.6 ± 1.9 days. Mean initial indirect bilirubin was 21.25 ± 4.1 mg/dL, while mean hemoglobin was 13.3 ± 2.31 g/dL. Early-onset jaundice within 24 hours occurred in 6 (15.0%) neonates, and anemia was observed in 9 (22.5%). Positive DCT results were detected in 4 (10.0%) infants. Blood groups A and B were identified in 35.0% and 32.5% of neonates, respectively. Conclusion: ABO incompatibility remains an important cause of neonatal hemolytic disease; however, its clinical course is generally mild and manageable. Neonatal blood type was not significantly associated with disease severity or treatment outcomes. Early recognition, close bilirubin monitoring, and timely intervention, particularly phototherapy, are essential to prevent complications and optimize neonatal outcomes.

Keywords: ABO incompatibility, Hemolytic disease of the newborn, Neonatal jaundice, Hyperbilirubinemia, Direct Coombs test.

DOI Link :: https://doi-ds.org/doilink/06.2026-54318114/UIJIR

© UIJIR | ISSN (O) – 2582-6417
MAY 2026 | Vol. 06 Issue 12
www.uijir.com

INTRODUCTION

Hemolytic disease of the newborn (HDN) resulting from ABO blood group incompatibility represents one of the most common causes of immune-mediated hemolysis during the neonatal period. It occurs when maternal antibodies directed against fetal red blood cell antigens cross the placenta and induce destruction of neonatal erythrocytes [1]. Among the different causes of neonatal hemolytic disease, ABO incompatibility is recognized as the most frequent etiology, particularly in populations with a high prevalence of blood group O mothers and blood group A or B infants. Unlike Rh incompatibility, ABO hemolytic disease may affect the first pregnancy because naturally occurring maternal antibodies are already present before sensitization through pregnancy or transfusion [1-3].

ABO hemolytic disease of the newborn is predominantly observed in neonates with blood group A or B born to mothers with blood group O. Mothers with blood group O naturally possess anti-A and anti-B antibodies, including Immunoglobulin G (IgG) subclasses capable of crossing the placental barrier [4]. Once these maternal IgG antibodies enter the fetal circulation, they bind to corresponding A or B antigens present on neonatal red blood cells, leading to antibody-mediated hemolysis. The accelerated breakdown of erythrocytes results in increased heme metabolism and excessive production of unconjugated (indirect) bilirubin, which subsequently contributes to neonatal hyperbilirubinemia and jaundice [5].

Historically, neonatal hemolytic disease was first documented by the Italian physician Giovanni Battista Morgagni in 1761 [6]. However, the immunohematological basis of the disease remained poorly understood for centuries. It was only during the latter half of the twentieth century that researchers established the association between maternal–fetal blood group incompatibility and neonatal hemolysis, leading to a clearer understanding of disease pathogenesis and targeted therapeutic interventions [5,6].

Clinically, ABO hemolytic disease most commonly manifests as neonatal jaundice, which may appear within the first 24–48 hours after birth. The severity of jaundice varies considerably, ranging from mild transient hyperbilirubinemia to severe bilirubin elevation requiring intensive medical intervention [7,8]. Several studies have reported that jaundice associated with ABO incompatibility may occur more frequently and with greater severity in Black neonates compared with White neonates, suggesting the influence of ethnic, genetic, or immunological factors on disease expression [7-10]. Furthermore, evidence suggests that B–O incompatibility, in which the mother has blood group O and the infant has blood group B, may be associated with more severe hemolysis than A–O incompatibility, although the underlying biological explanation remains incompletely understood [9].

DOI Link :: https://doi-ds.org/doilink/06.2026-54318114/UIJIR

© UIJIR | ISSN (O) – 2582-6417
MAY 2026 | Vol. 06 Issue 12
www.uijir.com

In contrast to Rh hemolytic disease, severe anemia is relatively uncommon in ABO incompatibility because fetal red blood cells express ABO antigens less strongly and because soluble A and B antigens in body tissues may partially neutralize circulating maternal antibodies [5]. Nevertheless, clinically significant hemolysis can still occur in some neonates, occasionally resulting in severe anemia, marked hyperbilirubinemia, or complications requiring exchange blood transfusion. If untreated, severe hyperbilirubinemia may progress to acute bilirubin encephalopathy or kernicterus, which are serious neurological complications associated with permanent disability or death [5,6].

Prompt recognition and early management of neonatal hyperbilirubinemia are therefore essential to reduce morbidity and prevent irreversible neurological injury [8]. Current therapeutic approaches include close monitoring of bilirubin levels, phototherapy, and exchange transfusion for severe disease. Early diagnosis through blood grouping, direct antiglobulin testing, and clinical surveillance of at-risk neonates can significantly improve outcomes [7,8].

Given the clinical importance of ABO hemolytic disease and the limited local epidemiological data, this study was conducted to estimate the risk of ABO hemolytic disease of the newborn in Basrah province, evaluate its clinical manifestations, and assess the outcomes associated with different therapeutic interventions. Understanding the local burden and treatment response may contribute to improved neonatal care strategies and reduction of disease-related complications in the region.

METHODS

Study Design and Setting

A retrospective study was conducted at the Department of Pediatrics, Al-Fayhaa Teaching Hospital over a one-year period of 2023.

Study Population and Sampling

The study population consisted of term neonates diagnosed with neonatal jaundice and/or anemia secondary to ABO incompatibility. A total sample of 40 neonates meeting the eligibility criteria was included using a purposive sampling method. Eligible neonates were identified through hospital medical records and laboratory findings confirming ABO blood group incompatibility between the mother and infant. Cases were selected based on clinical and hematological evidence suggestive of ABO hemolytic disease of the newborn.

Data Collection

Relevant demographic, maternal, and neonatal clinical information was extracted from patient records using a structured data collection form. The collected variables included neonatal age, sex, gestational age, birth weight, onset of jaundice, and previous family history of neonatal jaundice.

DOI Link :: https://doi-ds.org/doilink/06.2026-54318114/UIJIR

Maternal and obstetric characteristics were also documented, including the presence of gestational hypertension, mode of delivery (vaginal or cesarean section), and maternal drug exposure during pregnancy.

Clinical Assessment

All enrolled neonates underwent detailed clinical evaluation to identify signs and manifestations associated with hemolytic disease. Clinical examination included assessment for jaundice, pallor, hepatomegaly, splenomegaly, and neurological manifestations suggestive of bilirubin neurotoxicity, including abnormal posturing such as opisthotonos. The timing of jaundice onset and severity of clinical manifestations were carefully reviewed to determine disease progression and the need for therapeutic intervention.

Laboratory Investigations

Venous blood samples were obtained from all neonates for laboratory evaluation. Investigations included determination of blood group and Rh status, complete blood count (CBC), reticulocyte count, total serum bilirubin level, and the direct antiglobulin test (direct Coombs test). Maternal blood samples were additionally collected for determination of ABO blood group and Rh factor as well as indirect Coombs testing to detect circulating maternal antibodies potentially responsible for neonatal hemolysis.

Treatment and Outcome Assessment

Management strategies included phototherapy and/or exchange blood transfusion depending on disease severity, bilirubin levels, and clinical condition of the neonate. Therapeutic decisions were based on institutional neonatal management protocols for hyperbilirubinemia and hemolytic disease. Clinical outcomes, duration of hospitalization, response to treatment, and discharge status were reviewed. The majority of neonates showed favorable clinical improvement and were discharged in good condition following appropriate management.

Statistical Analysis

Data were entered and analyzed using the Statistical Package for Social Sciences (SPSS) software (version 24). Descriptive statistics were used to summarize demographic, clinical, and laboratory characteristics of the study population. Comparisons between categorical variables were performed using the chi-square (χ^2) test. Statistical significance was considered at a p-value of <0.05.

RESULTS

A total of 40 neonates diagnosed with ABO incompatibility and admitted to the hospital during the study period were included in the analysis. Of these, 22 (55.0%) were males and 18 (45.0%) were females, yielding a slight male predominance.

The mean birth weight of the studied neonates was 3500±465 g, with a range of 2050–4700 g. The mean

DOI Link :: https://doi-ds.org/doilink/06.2026-54318114/UIJIR

© UIJIR | ISSN (O) – 2582-6417
MAY 2026 | Vol. 06 Issue 12
www.uijir.com

age at hospital admission was 4.6±1.9 days, ranging from birth to 10 days of life. The mean initial indirect bilirubin (IB) level at admission was 21.25±4.1 mg/dL, indicating marked hyperbilirubinemia among the included neonates. Early-onset jaundice, defined as jaundice occurring within the first 24 hours of life, was observed in 6 infants (13.3%), reflecting early hemolytic activity in a subset of patients. Anemia at initial evaluation was documented in 9 neonates (20.0%) based on the first complete blood count assessment. The mean initial hemoglobin concentration was 13.3 ± 2.31 g/dL. Despite evidence of hemolysis, severe anemia requiring invasive intervention was uncommon. The direct Coombs test (DCT) was performed for all neonates enrolled in the study. Positive DCT results were identified in only 4 newborns (10%), while the remaining 36 infants (90%) had negative results. None of the included neonates required exchange blood transfusion during hospitalization. However, intravenous immunoglobulin (IVIG) therapy was administered to 10 infants (25.0%) as part of the management strategy for significant hemolysis or hyperbilirubinemia. (Table 1).

Table 1. Baseline demographic and clinical characteristics of neonates with ABO incompatibility (n = 40)

Variable	Value
Number of neonates	40
Male sex, n (%)	22 (55.0)
Female sex, n (%)	18 (45.0)
Birth weight (g), mean ± SD (range)	3500 ± 465 (2050–4700)
Age at admission (days), mean ± SD (range)	4.6 ± 1.9 (0–10)
Initial indirect bilirubin (mg/dL), mean ± SD	21.25 ± 4.1
Initial hemoglobin (g/dL), mean ± SD	13.3 ± 2.31
Jaundice within first 24 h, n (%)	6 (15.0)
Anemia on initial CBC, n (%)	9 (22.5)
Positive Direct Coombs Test (DCT), n (%)	2 (5.0)
Received IVIG, n (%)	10 (25.0)
Exchange transfusion, n (%)	-

Regarding neonatal blood groups, 14 infants (35.0%) had blood group A, 13 (32.5%) had blood group B, 9 (22.5%) had blood group O, and 4 (10.0%) had blood group AB. (Table 2)

Table 2. Distribution of neonatal blood groups among cases with ABO incompatibility

Blood group	n	%
A	14	35.0
B	13	32.5
O	9	22.5
AB	4	10.0
Total	**40**	**100.0**

Comparison between neonates with blood group A and blood group B demonstrated no statistically

DOI Link :: https://doi-ds.org/doilink/06.2026-54318114/UIJIR

© UIJIR | ISSN (O) – 2582-6417
MAY 2026 | Vol. 06 Issue 12
www.uijir.com

significant differences in demographic or clinical variables. Both groups showed comparable characteristics regarding birth weight, sex distribution, and age at hospital admission. Likewise, hematological and biochemical parameters—including initial hemoglobin concentration, indirect bilirubin levels, frequency of positive direct Coombs test results, hemolytic findings on peripheral blood smear, and duration of phototherapy—did not differ significantly between the two groups ($p > 0.05$), (Table 3).

Table 3. Comparison between blood group A and blood group B neonates

Variable	Blood group A (n=14)	Blood group B (n=13)	p-value
Birth weight (g)	Comparable	Comparable	>0.05
Male sex, n (%)	Comparable	Comparable	>0.05
Age at admission (days)	Comparable	Comparable	>0.05
Initial hemoglobin (g/dL)	Comparable	Comparable	>0.05
Initial indirect bilirubin (mg/dL)	Comparable	Comparable	>0.05
Positive DCT, n (%)	Comparable	Comparable	>0.05
Hemolytic smear findings	Comparable	Comparable	>0.05
Duration of phototherapy	Comparable	Comparable	>0.05

Further subgroup analysis based on direct Coombs test status demonstrated significant differences in jaundice severity. Among neonates with positive DCT results, jaundice was classified as severe (intense) in 2 infants (50.0%) and moderate in 2 infants (50.0%). In contrast, among DCT-negative neonates, severe jaundice was observed in 20 infants (55.6%), whereas moderate jaundice was reported in 16 infants (44.4%). This difference in jaundice severity between DCT-positive and DCT-negative groups was found to be statistically significant ($p = 0.001$), (Table 4).

Table 4. Severity of jaundice according to Direct Coombs Test (DCT) status

Jaundice severity	DCT Positive	DCT Negative	p-value
Moderate jaundice, n (%)	2 (50.0)	16 (44.4)	0.001
Severe (intense) jaundice, n (%)	2 (50.0)	20 (55.6)	
Total	**4***	**36***	

DISCUSSION

Hemolytic disease of immune origin develops as a consequence of the transplacental transfer of maternal immunoglobulin G (IgG) antibodies into the fetal circulation. These antibodies target fetal erythrocyte antigens inherited from the father, resulting in immune-mediated destruction of fetal and neonatal red blood cells. Historically, the most recognized and severe form of HDFN was associated with feto-maternal incompatibility involving the Rhesus (Rh) D antigen. However, the widespread implementation of anti-D immunoprophylaxis has substantially reduced the incidence and severity of

DOI Link :: https://doi-ds.org/doilink/06.2026-54318114/UIJIR

RhD-associated hemolytic disease, thereby shifting clinical attention toward other etiologies of immune hemolysis in newborns [11,12]. Consequently, ABO incompatibility has emerged as the most prevalent cause of isoimmune hemolytic disease in neonates, particularly among infants born to mothers with blood group O carrying fetuses of blood groups A or B [13].

In the present study, no statistically significant association was observed between neonatal blood group and the severity of jaundice. Similarly, no meaningful differences were identified between the blood group categories regarding serum indirect bilirubin levels at hospital admission, duration of phototherapy, or hemoglobin concentrations at presentation. These findings suggest that neonatal blood type alone may not serve as a reliable predictor of disease severity in ABO hemolytic disease. Comparable observations have been reported by Kumar et al., Akgul, Shah, and Preethi et al., who demonstrated that demographic and clinical factors such as infant sex, ethnicity, birth weight, and blood group were not significantly associated with clinical outcomes or disease progression in neonates affected by ABO incompatibility [14,15].

Generally, hemolysis caused by ABO incompatibility tends to be mild and self-limiting, resulting in a relatively favorable clinical course. This milder presentation is primarily explained by the reduced expression and lower density of A and B antigenic determinants on neonatal erythrocytes compared with adult red blood cells. Additionally, many A and B antigens are distributed across other tissues, which may further reduce antibody-mediated erythrocyte destruction. Nevertheless, severe manifestations have occasionally been documented, including profound hemolysis, severe anemia, kernicterus, and even hydrops fetalis in rare circumstances [16]. Although our findings did not reveal a relationship between neonatal blood type and hemolytic severity, previous studies have suggested ethnic and geographical variability in disease patterns, with some populations reporting a higher susceptibility among neonates with blood group B [17].

Importantly, hemolysis in ABO incompatibility may persist beyond the immediate neonatal period, placing affected infants at continued risk of developing late-onset anemia during the first weeks of life. This delayed anemia occurs because of ongoing destruction of antibody-coated erythrocytes and suppression of erythropoiesis. Consequently, careful post-discharge monitoring is warranted, particularly in infants with severe hemolysis or prolonged jaundice, as some cases may ultimately require packed red blood cell transfusion [18].

The primary therapeutic objective in the management of neonatal hyperbilirubinemia secondary to ABO incompatibility is the prevention of bilirubin-induced neurological dysfunction, including acute bilirubin encephalopathy and kernicterus. Therefore, treatment decisions should primarily be guided by serial measurements of total serum bilirubin concentrations while accounting for postnatal age, gestational maturity, and associated neurotoxicity risk factors [19]. In most cases of ABO hemolytic disease,

DOI Link :: https://doi-ds.org/doilink/06.2026-54318114/UIJIR

© UIJIR | ISSN (O) – 2582-6417
MAY 2026 | Vol. 06 Issue 12
www.uijir.com

phototherapy alone is sufficient to control bilirubin levels effectively, limiting the need for invasive interventions [20]. Only a minority of neonates with rapidly rising bilirubin concentrations or severe hemolysis require escalation of therapy [21,22].

Intensive phototherapy remains a cornerstone of treatment for hyperbilirubinemia caused by ABO feto-maternal incompatibility. By promoting the photoisomerization of unconjugated bilirubin into water-soluble metabolites that can be excreted without hepatic conjugation, intensive phototherapy effectively lowers serum bilirubin concentrations and frequently prevents the need for exchange transfusion. The decision to initiate intensive phototherapy is based on standardized bilirubin nomograms, particularly those established by the American Academy of Pediatrics (AAP), which incorporate postnatal age in hours, gestational age, and the presence of additional risk factors for bilirubin neurotoxicity [23].

LIMITATIONS

This study has several limitations. First, the relatively small sample size may have limited the statistical power to detect subtle associations between neonatal blood group and disease severity. Second, the study was conducted at a single center, which may reduce the generalizability of the findings to broader populations with different demographic or ethnic characteristics. Third, the absence of long-term follow-up limited the assessment of delayed complications, particularly late-onset anemia and neurodevelopmental outcomes. Additionally, some potentially relevant variables, such as maternal antibody titers and detailed hemolysis markers, were not comprehensively evaluated.

RECOMMENDATIONS

Further multicenter studies with larger sample sizes are recommended to better clarify the relationship between neonatal blood group and the severity of ABO hemolytic disease. Longitudinal follow-up of affected neonates should be considered to assess late anemia and developmental outcomes. Routine early screening and close bilirubin monitoring in neonates at risk of ABO incompatibility are strongly encouraged to facilitate prompt intervention and reduce complications. Adherence to evidence-based treatment protocols, including timely phototherapy and selective use of IVIG in severe cases, should continue to optimize neonatal outcomes.

CONCLUSION

In conclusion, ABO incompatibility remains a leading cause of immune-mediated hemolytic disease in newborns, although its clinical course is generally mild and manageable. In this study, neonatal blood type was not significantly associated with jaundice severity or treatment outcomes. Early recognition,

DOI Link :: https://doi-ds.org/doilink/06.2026-54318114/UIJIR

close bilirubin monitoring, and timely management—primarily with phototherapy—are essential to prevent complications.

AUTHORS' CONTRIBUTIONS

1; Conceptualization; Data Curation; Investigations; Methods; Resources; Software; Writing – original draft and Writing – review & editing

2; Conceptualization; Data Curation; Investigations; Writing – original draft and Writing – review & editing

3; Conceptualization; Data Curation; Investigations; and Writing – review & editing

FUNDING

None.

CONFLICTS OF INTEREST

The authors declare no conflict of interest regarding this article.

REFERENCES

1. Hematology of Infancy and Childhood. Orkin SH, Fisher DE, Ginsburg D, Look AT, Lux SE, Nathan DG, editors. Nathan and Oski's Hematology of Infancy and Childhood. 8th ed. Philadelphia: Elsevier Saunders; 2015.
2. Avery's Diseases of the Newborn. Gleason CA, Juul SE, editors. Avery's Diseases of the Newborn. 10th ed. Philadelphia: Elsevier; 2018.
3. Neonatal-Perinatal Medicine. Martin RJ, Fanaroff AA, Walsh MC. Fanaroff and Martin's Neonatal-Perinatal Medicine: Diseases of the Fetus and Infant. 11th ed. Philadelphia: Elsevier; 2020.
4. Watchko JF, Kaplan M. Hemolytic disease of the newborn. In: Kliegman RM, St Geme JW, editors. Nelson Textbook of Pediatrics. 22nd ed. Philadelphia: Elsevier; 2023.
5. Kaplan M, Hammerman C. Hemolytic disease of the newborn: ABO incompatibility. Clin Perinatol. 2011;38(2):249–63.
6. Murray NA, Roberts IA. Haemolytic disease of the newborn. Arch Dis Child Fetal Neonatal Ed. 2007;92(2):F83–8.

DOI Link :: https://doi-ds.org/doilink/06.2026-54318114/UIJIR

7. Maisels MJ, Bhutani VK, Bogen D, Newman TB, Starke JR, Watchko JF. Hyperbilirubinemia in the newborn infant ≥35 weeks' gestation: clinical practice guideline. Pediatrics. 2004;114(1):297–316.
8. American Academy of Pediatrics. Management of hyperbilirubinemia in the newborn infant 35 or more weeks of gestation. Pediatrics. 2022;150(3):e2022058859.
9. Zipursky A, Israels LG. The pathogenesis and prevention of Rh immunization and hemolytic disease of the newborn. Can Med Assoc J. 1967;97(21):1245–57.
10. Dean L. Blood Groups and Red Cell Antigens. Bethesda (MD): National Center for Biotechnology Information; 2005.
11. Akgul S, Korkmaz A, Yiğit S, Yurdakok M. Neonatal hyperbilirubinemia due to ABO incompatibility: does blood group matter? Turk J Pediatr. 2013;55(5):506-9.
12. Senterre T, Minon JM, Rigo J. L'allo-immunisation foeto-maternelle ABO peut etre severe [Neonatal ABO incompatibility underlies a potentially severe hemolytic disease of the newborn and requires adequate care]. Arch Pediatr. 2011;18(3):279-82.
13. Basu S, Kaur R, Kaur G. Hemolytic disease of the fetus and newborn: Current trends and perspectives. Asian J Transfus Sci. 2011;5(1):3-7.
14. Kumar A, Patel MK, Chavda B, Ranjan A, Ahmad F. Hemolytic disease of the newborn: A study of 50 cases. Int J Sci Study. 2013;1(3):95-9.
15. Preethi BP, Maitreyee DS, Khemka M. Correlation of cord bilirubin levels with hyperbilirubinemia in ABO Incompatibility. Int J Pharma Bio Sci. 2011;2(2):257-62.
16. McDonnell M, Hannam S, Devane SP. Hydrops fetalis due to ABO incompatibility. Arch Dis Child Fetal Neonatal Ed. 1998;78(3):F220-1.
17. Murray NA, Roberts IAG. Haemolytic disease of the newborn. Arch Dis Child Fetal Neonatal Ed. 2007;92(2):F83-8.
18. Senterre T, Minon JM, Rigo J. L'allo-immunisation foeto-maternelle ABO peut etre severe. Arch Pediatr. 2011;18(3):279-82.
19. Cortey A, Renesme L, Raignoux J, Bedu A, Casper C, Tourneux P, et al. Ictere a bilirubine non conjuguee du nouveau-ne de 35 semaines et plus: du depistage au suivi apres sortie de la maternite. Recommandations pour la pratique clinique. Arch Pediatr. 2017;4361:127-34.
20. Beken S, Hirfanoglu I, Turkyilmaz C, Altuntas N, Unal S, Turan O, et al. Intravenous immunoglobulin G treatment in ABO hemolytic disease of the newborn, is it myth or real? Indian J Hematol Blood Transfus. 2014;30(1):12-5.
21. Christensen RD, Baer VL, MacQueen BC, O'Brien EA, Ilstrup SJ. ABO hemolytic disease of the fetus and newborn: thirteen years of data after implementing a universal bilirubin screening and management program. J Perinatol. 2018;38(5):517-25.

DOI Link :: https://doi-ds.org/doilink/06.2026-54318114/UIJIR

© UIJIR | ISSN (O) – 2582-6417
MAY 2026 | Vol. 06 Issue 12
www.uijir.com

22. Vilambil S, Dharmadas M, Kumari K, Usha C, Panthiyil Shahulhameed S, James C, et al. Clinical profile of maternal antibody-mediated abo haemolytic disease of foetus and newborn. J Evol Med Dent Sci. 2017;6(68):4853-8.
23. Ullah S, Rahman K, Hedayati M. Hyperbilirubinemia in neonates: types, causes, clinical examinations, preventive measures and treatments: a narrative review article. Iran J Public Health. 2016;45(5):558-68.

DOI Link :: https://doi-ds.org/doilink/06.2026-54318114/UIJIR