PRIKAZ SLUČAJA

UDK BROJEVI:
616.155.194-053.31
DOI: https://www.doi.org/10.5937/zdravzast51-37028

HEMOLITIČKA BOLEST NOVOROĐENČETA

Mirjana Makević Đurić[1*], Milivoje Đurić[2]

[1] Dom zdravlja „Dr Darinka Lukić", Koceljeva, Republika Srbija
[2] Opšta bolnica „Dr Laza K. Lazarević", Šabac, Republika Srbija

[*] Korespondencija: mmakevicdjuric@gmail.com

SAŽETAK

Uvod/Cilj: Hemolitička bolest novorođenčeta predstavlja hemoliznu anemiju koja se javlja kao posledica nepodudarnosti krvi roditelja, odnosno nepodudarnosti krvnih elemenata majke i ploda u ABO ili Rh sistemu. Cilj ovog rada je da prikaže slučaj hemolitičke bolesti novorođenčeta nastale kao posledica ABO inkompatibilije kod majke.
Prikaz bolesnika: Ženskom novorođenčetu je u osamnaestom satu života registrovana indirektna hiperbilirubinemija. Krvna grupa novorođenčeta je bila A Rh (D) pozitivna, direktni Coombs test bio je pozitivan, a u krvi novorođenčeta su detektovana anti-A antitela. Ovaj slučaj je uspešno lečen fototerapijom i transfuzijom resuspendovanih eritrocita bez potrebe za eksangvinotransfuzijom.
Zaključak: ABO inkompatibilija može predstavljati veliki problem kako u prenatalnom, tako i u perinatalnom i neonatalnom periodu.Posebnu pažnju treba obratiti na trudnice čija su deca iz prethodnih trudnoća imala hiperbilirubinemiju ili su im se trudnoće završavale spontanim pobačajem.

Ključne reči: ABO inkompatibilija, hemolitička bolest, hiperbilirubinemija

Uvod

Hemolitička bolest novorođenčeta predstavlja hemoliznu anemiju koja se javlja kao posledica nepodudarnosti krvnih elemenata majke i ploda u ABO ili Rh sistemu.

ABO hemolitička bolest novorođenčeta je najčešći uzrok žutice koji se javlja u prvih 24 sata života, sa incidencijom 0,33–2,2% novorođenih (1). Pripada grupi patoloških nekonjugovanih hiperbilirubinemija ispunjavajući sledeće kriterijume: pojava žutice u prvih 24 sata života, porast bilirubina preko 8,5 µmol/h, snižena koncentracija hemoglobina ispod 100 g/l i pozitivan *Coombs*-ov test (2). Nastaje kao rezultat prolaska antitela imunoglobulina G (IgG) kroz placentu kod novorođenčadi žena krvne grupe O koje imaju IgG anti-A ili anti-B antitela (3). Prolazeći kroz posteljicu, antitela iz krvotoka majke oštećuju eritrocite u krvotoku ploda, zbog čega se javljaju znaci hemolize: anemija, žutica, a u težim slučajevima i fetalni hidrops (4). Fetalni hidrops, koji je i najteži oblik hemolitičke bolesti novorođenčeta, je redak i javlja se kod 0,13% novorođenih (5). Usled hemolize eritrocita dolazi do razgradnje hema i hiperprodukcije bilirubina koji je u plazmi delom vezan za albumin, a delom u vidu slobodnog (nevezanog) bilirubina. Nevezani bilirubin prelazi krvno-moždanu barijeru i deponuje se u mozgu novorođenčeta. Nezrela jetra novorođenčeta, a posebno prevremeno rođenog, nije u stanju da metaboliše povećanu količinu bilirubina koja nastaje usled hemolize eritrocita i razvija se nekonjugovana (indirektna) hiperbilirubinemija što rezultira pojavom žutice u prvom danu života (6).

Brzo prepoznavanje i lečenje hiperbilirubinemije i hemolitičke bolesti su od izuzetne važnosti za izbegavanje dugotrajne neurološke disfunkcije kod ove novorođenčadi.

Prikaz slučaja

Ženskom novorođenčetu je u osamnaestom satu života registrovana indirektna hiperbiliru-

CASE REPORT

UDK NUMBERS:
616.155.194-053.31
DOI: https://www.doi.org/10.5937/zdravzast51-37028

HEMOLYTIC DISEASE OF THE NEWBORN

Mirjana Makevic Djuric[1*], Milivoje Djuric[2]

[1] Health Center "Dr Darinka Lukic", Koceljeva, Republic of Serbia
[2] General Hospital "Dr Laza K. Lazarevic", Sabac, Republic of Serbia

[*] Correspondence: mmakevicdjuric@gmail.com

SUMMARY

Introduction/Aim: Haemolytic disease of the newborn is haemolytic anaemia that occurs as a consequence of mismatched blood of the parents, that is, the mismatch of blood elements between the mother and fetus in the ABO or Rh system. The aim of this study is to report a case of haemolytic disease of the newborn which occurred as a result of ABO incompatibility.
Case report: Indirect hyperbilirubinemia was registered in a female newborn in the 18th hour of life. The blood group of the newborn was A Rh (D) positive, the direct Coombs test was positive and anti-A antibodies were detected in baby's blood. This case was successfully managed with phototherapy and simple red blood cells transfusion without the need for exchange transfusion.
Conclusion: ABO incompatibility can be a major problem in the prenatal, perinatal or neonatal periods. Special attention should be paid to pregnant women whose children from previous pregnancies had hyperbilirubinemia or whose pregnancies were ended in miscarriage.

Keywords: ABO incompatibility, hemolytic disease, hyperbilirubinemia

Introduction

Hemolytic disease of a newborn is haemolytic anaemia that occurs as a consequence of the incompatibility of blood elements between a mother and child in the ABO or Rh system.

ABO hemolytic disease of a newborn is the most common cause of jaundice that appears during the first 24 hours of life, with the incidence of 0.33-2.2% in newborns (1). It belongs to the group of pathological unconjugated hyperbilirubinemias when it meets the following criteria: the occurrence of jaundice during the first 24 hours of life, the increase in bilirubin more than 8.5 µmol/h, lowered concentration of hemoglobin below 100 g/l and the positive Coombs test (2). It occurs when immunoglobulin G antibodies (IgG) pass through the placenta in newborns of women whose blood type is O and who have IgG anti-A or anti-B antibodies (3). When passing through the placenta, antibodies from mother's circulation damage erythrocytes in the circulatory system of a fetus, due to which signs of hemolysis occur: anemia, jaundice and in some cases hydrops fetalis (4). Hydrops fetalis, which is the most severe form of hemolytic disease in newborns, is rare and it occurs in 0.13% of newborns (5). Hemolysis of red blood cells causes the destruction of red blood cells and hyperproduction of bilirubin, which is partly bound to albumin in the plasma and partly it is free (non-albumin bound) bilirubin. Non-albumin bound bilirubin passes the blood-brain barrier and it is deposited in the brain of a newborn. The immature liver of a newborn and especially of prematurely born children cannot metabolize the increased amount of bilirubin resulting from hemolysis of red blood cells and therefore, unconjugated (indirect) hyperbilirubinemia develops, which causes the occurrence of jaundice in the first 24 hours of life (6).

Recognizing and treating hyperbilirubinemia and hemolytic disease on time are of great

binemija. Ukupan bilirubin u prvom danu života bio je 162 µmol/l. Novorođenče je drugo dete iz treće trudnoće sedamnaestogodišnje majke koja je krvne grupe O Rh (D) pozitivna. Porođaj je završen prirodnim putem u 36. gestacijskoj nedelji. Na rođenju, telesna masa novorođenčeta iznosila je 2380 g, telesna dužina 44 cm, Apgar skor 9. Iz anamnestičkih podataka smo saznali da je druga trudnoća majke završena spontanim pobačajem u 11. gestacijskoj nedelji.

Prilikom pregleda novorođenče je bilo budno, snažnog plača, hemodinamski stabilno, ikterične kože očuvanog turgora i elasticiteta. Grudni koš je bio simetrično respiratorno pokretan, sa 56 respiracija u minutu. Srčana radnja je bila ritmična, tonovi jasni, bez šumova, srčana frekvenca 137 otkucaja u minutu. Abdomen je bio iznad ravni grudnog koša, palpatorno bolno neosetljiv, obima 39 cm. Mišićni tonus je bio uredan, a primitivni refleksi su se uredno izazivali.

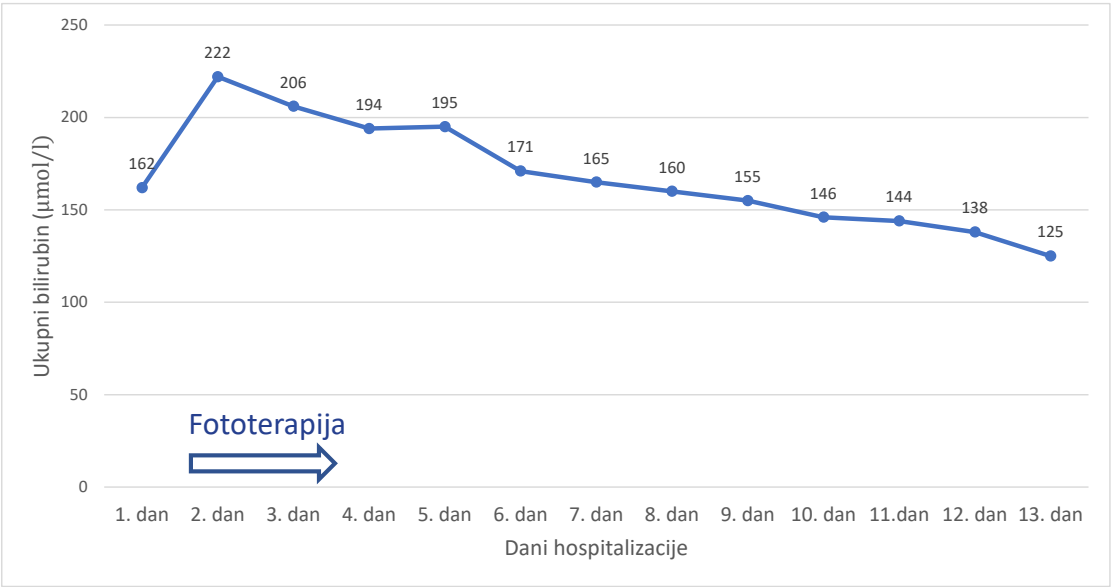
U drugom danu života došlo je do porasta ukupnog bilirubina na 222 µmol/l (direktni 8,1 µmol/l, indirektni 213,9 µmol/l). Hemoglobin je bio 137 g/l, procenat retikulocita 14,8%, haptoglobin < 0,1 g/l, laktat-dehidrogenaza (LDH) 739 U/l. Vrednosti gasnih analiza u krvi, pH i acidobazne ravnoteže kretale su se u normalnom opsegu. Protrombinsko vreme (PT) i aktivirano parcijalno tromboplastinsko vreme (apTT)su bili u granicama referentnih vrednosti čime je isključen poremećaj hemostaze kod novorođenčeta. Krvna grupa novorođenčeta bila je A Rh (D) pozitivna, direktni *Coombs* test bio je pozitivan. U plazmi novorođenčeta registrovano je prisustvo anti-A antitela. Ultrazvučnim pregledom CNS-a isključena je intrakranijalna hemoragija. Na ultrazvučnom pregledu trbuha, jetra, slezina, oba bubrega i nadbubrega, i mokraćna bešika su bili urednog položaja, oblika, veličine i ehostrukture i nije registrovano prisustvo tečnosti u trbušnoj duplji. Bakteriološkom obradom novorođenčeta isključena je sepsa.

Analiziranjem anamnestičkih, kliničkih i laboratorijskih rezultata zaključeno je da je uzrok patološke nekonjugovane žutice kod novorođenčeta bila imunska hemolizna anemija.

Prema protokolu za lečenje hemolizne bolesti novorođenčeta, zbog indirektne hiperbilirubinemije od drugog dana života, sprovođena je fototerapija u trajanju od 36 sati, nakon čega je zabeleženo opadanje vrednosti bilirubina u krvi (grafikon 1). Tokom hospitalizacije, usled hemolize eritrocita beležio se postepeni pad hemoglobina, zbog čega je novorođenčetu šestog dana života ordinirana transfuzija resuspendovanih eritrocita O krvne grupe (grafikon 2). Nije bilo potrebe za primenom eksangvinotransfuzije. Sve vreme hospitalizacije novorođenče je bilo stabilnog opšteg stanja i napredovalo je u telesnoj masi. Otpušteno je kući 13. dana života, telesne mase na otpustu 2440 g i urednog fizikalnog nalaza.

Diskusija

Rutinska upotreba profilakse Rh IgG rezultirala je značajnim padom incidencije RhD aloimuni-



Grafikon 1. Vrednosti ukupnog bilirubina po danima hospitalizacije

importance for the prevention of longstanding neurological dysfunction in these newborns.

Case report

Indirect hyperbilirubinemia was registered in a female newborn in the 18th hour of life. Total bilirubin in the first day of life was 162 µmol/l. The newborn was the second child from the third pregnancy of a seventeen-year old mother whose blood group was O Rh (D). The vaginal delivery was completed at a gestational age of 36 weeks. At birth, the newborn's body weight was 2380 g, body length was 44 cm, while the Apgar score was 9. We found in the medical history that the second pregnancy of this mother ended in miscarriage at 11 weeks of gestation.

During the examination, the newborn was awake, crying intensely, hemodynamically stable and had icteric skin with normal skin turgor and elasticity. The chest wall movement was symmetric with 56 breaths per minute. The heart rhythm was normal, sounds clear with no heart murmur, while heart rate was 137 beats per minute. The abdomen was above the chest line, abdominal palpation was insensitive, while circumference was 39 cm. The muscle tone was normal, and primitive reflexes were normal, as well.

In the second day of life, total bilirubin increased to 222 µmol/l (direct 8.1 µmol/l, indirect 213.9 µmol/l). Hemoglobin was 137 g/l, the percentage of reticulocytes was 14.8%, haptoglobin < 0.1 g/l, lactate-dehydrogenase (LDH) 739 U/l. The values of blood gas tests, the pH and acid-base balance were within the normal range. The prothrombin time (PT) and activated partial thromboplastin time (apt) were within the normal reference values, and therefore, hemostasis abnormality was excluded in the newborn. The newborn's blood group was A Rh positive, while the direct Coombs test was positive. Anti-A antibodies were registered in the plasma. The ultrasound examination of CNS excluded intracranial hemorrhage. The ultrasound examination of abdomen showed that liver, spleen, both kidneys and adrenal glands, and urinary bladder had normal position, shape, size and echostructure and no fluid was registered in the abdominal cavity. Sepsis was excluded by the bacteriological examination of the newborn.

The analysis of anamnestic, clinical and laboratory findings led to the conclusion that the cause of pathological unconjugated jaundice in the newborn was immune hemolytic anemia.

According to the protocol for the treatment of hemolytic disease of the newborn, due to indirect hyperbilirubinemia from the second day of life, phototherapy was applied lasting 36 hours, after which the value of bilirubin in blood decreased (Figure 1). During hospitalization, due to hemolysis of erythrocytes, gradual decrease in hemoglobin was noted and therefore, in the sixth day of life the transfusion of type O resuspended red cells was administered (Figure 2). There was no need for exchange transfusion. The newborn's general state was stable all the time during hospitalization

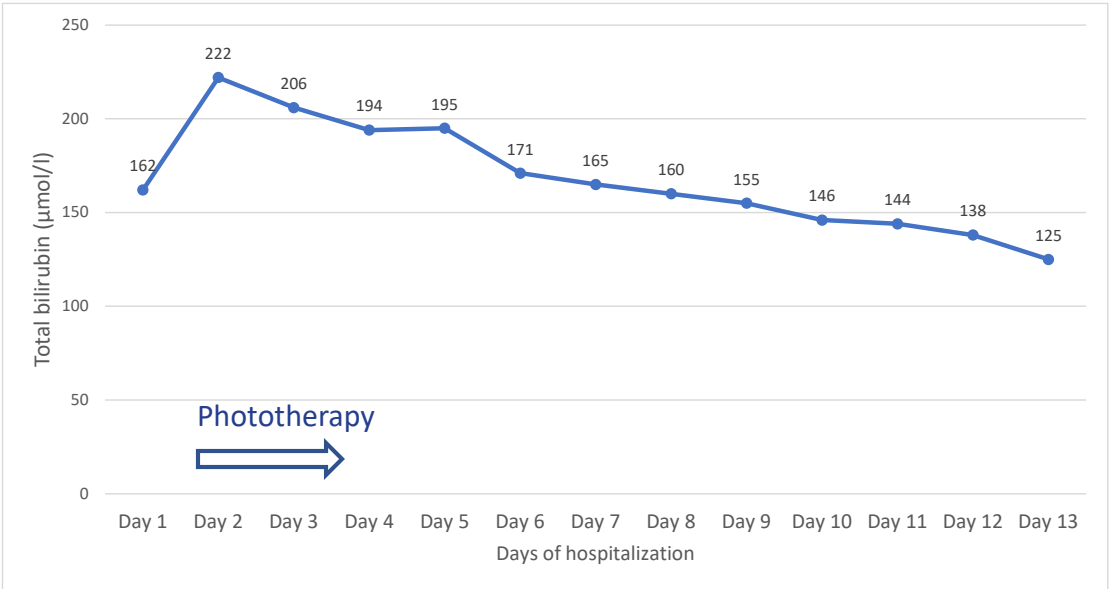
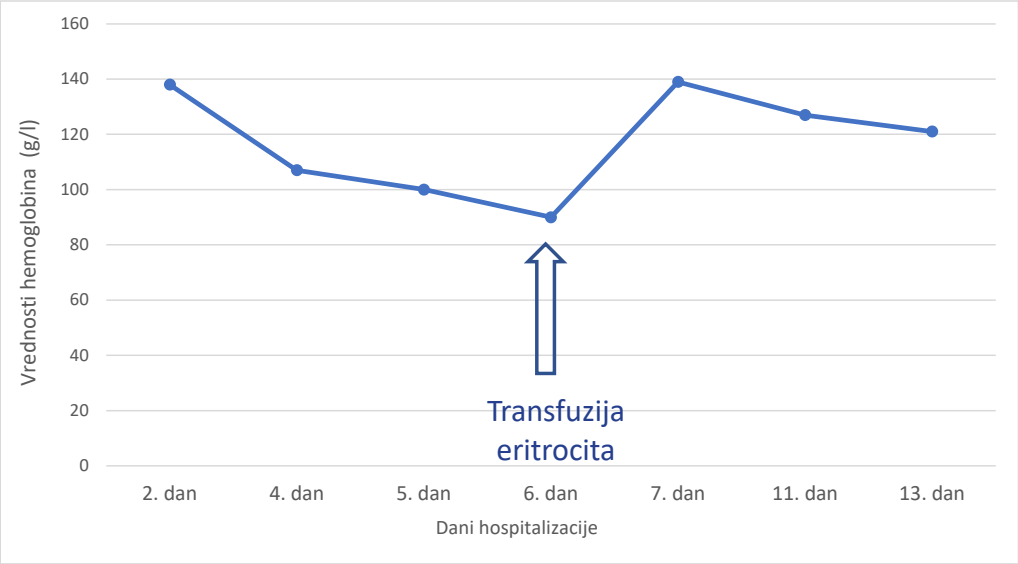


Figure 1. Total bilirubin values by days of hospitalization



Grafikon 2. Vrednosti hemoglobina po danima hospitalizacije

zacije (7). Zbog toga je ABO inkompatibilija postala najčešći uzrok izoimune hemolitičke bolesti novorođenčeta. Iako je hemolitička bolest zbog ABO inkompatibilije klinički blaža od one izazvane RhD inkompatibilijom, povremeno se može javiti teška hemoliza koja zahteva i eksangvinotransfuziju (8). U poslednje vreme ABO inkompatibilija posebno postaje značajna i aktuelna u prenatalnoj dijagnostici i lečenju ženske neplodnosti. Posle neobjašnjenih ili uzastopnih spontanih pobačaja sve više se pridaje značaj upravo imunološkim faktorima.

Literaturni podaci nam ukazuju na to da se ABO inkompatibilija češće javlja kod novorođenčadi koja pripadaju određenim etničkim grupama i imaju B krvnu grupu (9). *Adewuyi* i saradnici su otkrili da je serumska hemolitička aktivnost anti-A i anti-B antitela kod osoba crne rase veća od odgovarajuće aktivnosti nađene kod osoba bele rase. Njihova studija je pokazala da je hemolitička aktivnost anti-B antitela veća nego anti-A antitela kod obe rase (10). *McDonnell* je opisao dva slučaja fetalnog hidropsa kod osoba crne rase uzrokovana ABO-inkompatibilijom (11).

Nekoliko drugih studija je ispitivalo vezu između ABO inkompatibilije i reproduktivnog neuspeha. *Malekasgar* opisuje istraživanje sprovedeno u Britanskoj Kolumbiji u kome su trudnice sa O krvnom grupom i ABO inkompatibilijom imale značajno višu incidenciju spontanih pobačaja između 40. i 135. dana trudnoće od trudnica drugih krvnih grupa (12). *Stiller* i saradnici su u svom članku prikazali trudnoću komplikovanu anti-B izoimunizacijom koja je dovela do fetalnog ascitesa, anemije, hepatomegalije i polihidramniona (13).

S obzirom na to da hemolizna bolest novorođenčadi najčešće nastaje zbog ABO i Rh aloizoimunizacije, postoje jasni klinički vodiči za njihovo zbrinjavanje. Prema protokolima za lečenje, indikacija za fototerapiju kod preterminske novorođenčadi u 36. gestacijskoj nedelji sa hemoliznom bolešću i telesnom masom većom od 2000 g, su vrednosti bilirubina od 170-204 µmol/l u drugom danu života (14). Indikacije za eksangvinotransfuziju u hemoliznoj bolesti podrazumevaju tešku anemiju (Hb < 100 g/l) i porast bilirubina za više od 8,5 µmol/l/h. Prema kliničkim vodičima, manja gestacijska starost i manja telesna masa novorođenčeta sa hemoliznom bolešću podrazumevaju i niže vrednosti ukupnog bilirubina za započinjanje fototerapije i eksangvinotransfuzije.

U našem prikazu slučaja ABO hemolitička bolest novorođenčeta bila je srednje teške kliničke slike. Pošto je ukupni bilirubin u drugom danu života bio 222 µmol/l, novorođenčetu je, prema protokolu za gestacijsku starost i telesnu masu, primenjena fototerapija u trajanju od 36 sati koja je dovela do postepenog pada vrednosti ukupnog bilirubina. Pošto indikacije za eksangvinotrasfuziju podrazumevaju porast vrednosti ukupnog bilirubina za više od 8,5 µmol/l/h, što se kod našeg novorođenčeta nije događalo nego su vrednosti padale, fototerapija je bila dovoljna za lečenje i nije bilo potrebe za eksangvinotransfuzijom. Vrednosti hemoglobina usled hemolize eritrocita pale su ispod 100 g/l šestog dana života zbog čega je novorođenčetu ordinirana jedna transfuzija re-

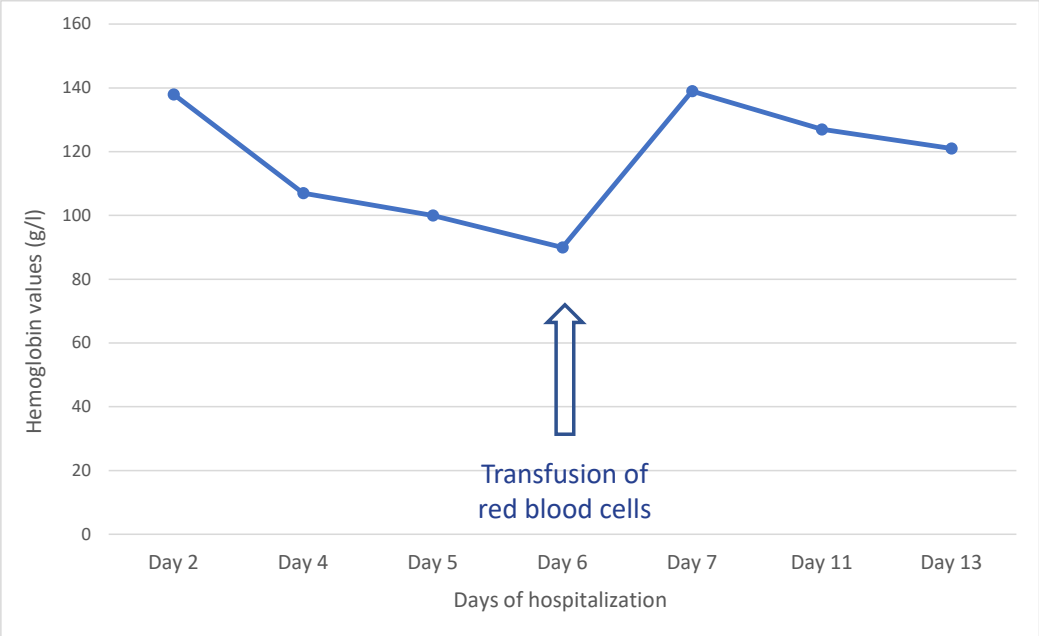


Figure 2. Hemoglobin values by days of hospitalization

and the newborn had healthy weight gain. The baby was discharged during the 13th day of life with body weight 2440 g and normal physical findings.

Discussion

Routine use of Rh IgG as prophylaxis resulted in the significant decrease in the incidence of RhD alloimmunization (7). Therefore, ABO incompatibility became the most frequent cause of isoimmune hemolytic disease of the newborn. Although hemolytic disease due to ABO incompatibility is clinically less severe than hemolytic disease caused by RhD incompatibility, severe hemolisis may occur occasionally, which demands exchange transfusion (8). ABO incompatibility has become particularly significant and actual lately in prenatal diagnostics and the treatment of female infertility. After unexplained or spontaneous recurrent miscarriages, immune factors are given more and more importance.

Literature data indicate that ABO incompatibility is more frequent in neonates that belong to certain ethnic groups and have B blood group (9). Adewuyi and associates found that serum hemolytic activity of anti-A antibodies and anti-B antibodies is greater in black persons than the corresponding activity found in white persons. Their study showed that hemolytic activity of anti-B antibodies is greater than of anti-A antibodies in both racial groups (10). McDonnell described two cases of hydrops fetalis caused by ABO incompatibility in persons belonging to black racial group (11).

Several other studies examined the connection between ABO incompatibility and reproductive failure. Malekasgar described a research that was conducted in British Columbia, in which pregnant women with O blood group and ABO incompatibility had significantly higher incidence of miscarriages between the 40th and 135th day of pregnancy in comparison to pregnant women with other blood groups (12). Stiller and associates reported in their article a pregnancy that was complicated by anti-B isoimmunization which resulted in fetal ascites, anemia, hepatomegaly and polyhydramnios (13).

Considering that hemolytic disease of newborns most frequently occurs due to ABO and Rh alloimmunization, there are clear clinical guidelines for their treatment. According to the treatment protocols, the indications for phototherapy in pre-term newborns with hemolytic disease and body weight higher than 2000 g at 36 weeks of gestation are values of bilirubin 170-204 µmol/l in the second day of life (14). Indications for exchange transfusion in hemolytic disease include severe anemia (Hb < 100 g/l) and the rate of rise of bilirubin that is more than 8.5 µmol/l/h. According to clinical guidelines, younger gestational age and smaller body weight of newborns with hemolytic disease include lower values of total bilirubin necessary to start phototherapy and exchange transfusion.

In our case study, ABO hemolytic disease of the newborn was moderately severe. Since total bilirubin in the second day of life was 222 µmol/l,

suspendovanih eritrocita O krvne grupe. Nasuprot literaturnim podacima koji govore da se ABO inkompatibilija češće javlja kod novorođenčadi koja pripadaju crnoj rasi i imaju B krvnu grupu, naše novorođenče pripada beloj rasi i imalo je A krvnu grupu. U krvi prikazanog novorođenčeta registrovana su anti-A antitela. Mladoj, sedamnaestogodišnjoj majci, je novorođenče dete iz treće trudnoće začete prirodnim putem u kratkom vremenskom intervalu, a druga trudnoća je završena spontanim pobačajem u 11. gestacijskoj nedelji. Na osnovu anamnestičkih podataka, kliničkog nalaza i rezultata učinjenih ispitivanja, prevremeni porođaj u našem slučaju mogao je nastati kao posledica ABO inkompatibilije.

Zaključak

Želeli smo da podsetimo da, iako retka i relativno lakše kliničke slike, sa već usvojenim protokolima za uspešno lečenje, ABO inkompatibilija može predstavljati veliki problem kako u prenatalnom, tako i u perinatalnom i neonatalnom periodu.Ginekolozi bi trebalo da obrate posebnu pažnju na trudnice sa O krvnom grupom, a koje imaju partnere A, B ili AB krvne grupe, da im prate titar anti-A i anti-B antitela i da na vreme obaveste i roditelje i pedijatre o mogućim posledicama po novorođenče. Posebnu pažnju treba obratiti na trudnice čija su deca iz prethodnih trudnoća imala hiperbilirubinemiju ili su im se trudnoće završavale spontanim pobačajem.

Konflikt interesa

Autori su izjavili da nema konflikta interesa.

Literatura

1. Dufour DR, Monoghan WP. ABO hemolytic disease of the newborn. A retrospective analysis of 254 cases. Am J Clin Pathol 1980;73(3):369-73. doi: 10.1093/ajcp/73.3.369. PMID: 7189092
2. Lazarus C, Avchen R. Neonatal hyperbilirubinemia management: a model for change. J Perinatol 2009; 29:S58–S60. doi: 10.1038/jp.2008.217
3. Kaur D, Sidana P, Mandhan G,Kanodia K. Oblivious of the Yellow World – A Newborn's Telltale: ABO Hemolytic Disease of Newborn. Glob J Transfus Med 2019; 4(1):87-89. doi: 10.4103/GJTM.GJTM_45_18
4. Hall V, Avulakunta ID. Hemolytic diseases of the newborn. [Updated 2021 Aug 31]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK557423/
5. Trainor B, Tubman R. The emerging pattern of hydrops fetalis--incidence, aetiology and management. Ulster Med J 2006;75(3):185-186.
6. American Academy of Pediatrics Subcommittee on Hyperbilirubinemia. Management of hyperbilirubinemia in the newborn infant 35 or more weeks of gestation. Pediatrics 2004;114(1):297-316. doi: 10.1542/peds.114.1.297
7. Fasano RM. Hemolytic disease of the fetus and newborn in the molecular era. Semin Fetal Neonatal Med 2016;21(1):28-34. doi: 10.1016/j.siny.2015.10.006
8. Chen JY, Ling UP. Prediction of the development of neonatal hyperbilirubinemia in ABO incompatibility. Zhonghua yi xue za zhi = Chinese Medical Journal; Free China ed. 1994;53(1):13-18. PMID: 8173995.
9. Murray NA, Roberts IA. Haemolytic disease of the newborn. Arch Dis Child Fetal Neonatal Ed 2007; 92(2):F83-8. doi: 10.1136/adc.2005.076794.
10. Adewuyi JO, Gwanzura C. Racial difference between white and black Zimbabweans in the haemolytic activity of A, B, O antibodies. Afr J Med Med Sci 2001; 30(1-2):71-74. PMID: 14510155.
11. McDonncell M, Hannam S, Devane SP. Hydrops fetalis due to ABO incompatibility. Arch Dis Child Fetal Neonatal Ed 1998; 78(3):F220-221. doi: 10.1136/fn.78.3.f220
12. Malekasgar AM. ABO blood group prevalence in spontaneously repeated abortion. Turk J Haematol 2004; 21(4):181-7. PMID: 27264282.
13. Stiller RJ, Herzlinger R, Siegel S, Whetham JC. Fetalascites associated with ABO incompatibility: case reportand review of the literature. Am J Obstet Gynecol 1996; 175: 1371-1372. doi: 10.1016/s0002-9378(96)70057-1
14. Hyperbilirubinemia: Unconjugated. In: Gomella T, Eyal FG, Bany-Mohammed F. eds. Gomella's Neonatology: Management, Procedures, On-Call Problems, Diseases, and Drugs, 8e. McGraw Hill; 2020. Accessed June 11, 2022. https://accesspediatrics.mhmedical.com/content.aspx?bookid=2762&sectionid=234453147

phototherapy lasting 36 hours was administered according to the protocol for gestational age and body weight and it resulted in the gradual decrease in values of total bilirubin. Since indications for exchange transfusion include the rise of values of total bilirubin for more than 8.5 µmol/l/h, which did not happen in case of our newborn, whose values of bilirubin decreased, phototherapy was sufficient for the treatment and there was no need for exchange transfusion. The values of hemoglobin due to hemolysis of erythrocytes decreased below 100 g/l, and therefore the transfusion of resuspended type O red blood cells was administered. Opposite to literature data, which indicate that ABO incompatibility is more frequent in newborns who belong to black racial group and have B blood group, our newborn belongs to white racial group and has A blood group. Anti-A antibodies were registered in the blood of the presented newborn. The newborn is the child of a young, seventeen-year old mother who got pregnant naturally at short interval and this was the third pregnancy, while the second pregnancy ended in spontaneous miscarriage at a gestational age of 11 weeks. According to the anamnestic data, clinical findings and results of examinations, pre-term delivery in our case could have been the result of ABO incompatibility.

Conclusion

Although ABO incompatibility is rare and has a mild clinical picture, with already adopted protocols for successful treatment, we wanted to remind that it can present a serious problem in the prenatal, perinatal and neonatal period. Gynecologists should pay special attention to pregnant women with O blood group who have partners with A, B or AB blood group, to observe the ani-A and anti-B antibodies titer and to inform parents and pediatricians about possible consequences for the newborn. Special attention should be paid to pregnant women whose children from previous pregnancies had hyperbilirubinemia or their pregnancies ended in spontaneous miscarriage.

Competing interests

The authors declare no competing interests.

Literature

1. Dufour DR, Monoghan WP. ABO hemolytic disease of the newborn. A retrospective analysis of 254 cases. Am J Clin Pathol 1980; 73(3):369-73. doi: 10.1093/ajcp/73.3.369. PMID: 7189092
2. Lazarus C, Avchen R. Neonatal hyperbilirubinemia management: a model for change. J Perinatol 2009; 29:S58–S60. doi: 10.1038/jp.2008.217
3. Kaur D, Sidana P, Mandhan G,Kanodia K. Oblivious of the Yellow World – A Newborn's Telltale: ABO Hemolytic Disease of Newborn. Glob J Transfus Med 2019; 4(1):87-89. doi: 10.4103/GJTM.GJTM_45_18
4. Hall V, Avulakunta ID. Hemolytic diseases of the newborn. [Updated 2021 Aug 31]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK557423/
5. Trainor B, Tubman R. The emerging pattern of hydrops fetalis--incidence, aetiology and management. Ulster Med J 2006;75(3):185-186.
6. American Academy of Pediatrics Subcommittee on Hyperbilirubinemia. Management of hyperbilirubinemia in the newborn infant 35 or more weeks of gestation. Pediatrics 2004;114(1):297-316. doi: 10.1542/peds.114.1.297
7. Fasano RM. Hemolytic disease of the fetus and newborn in the molecular era. Semin Fetal Neonatal Med 2016;21(1):28-34. doi: 10.1016/j.siny.2015.10.006
8. Chen JY, Ling UP. Prediction of the development of neonatal hyperbilirubinemia in ABO incompatibility. Zhonghua yi xue za zhi = Chinese Medical Journal; Free China ed. 1994; 53(1):13-18. PMID: 8173995.
9. Murray NA, Roberts IA. Haemolytic disease of the newborn. Arch Dis Child Fetal Neonatal Ed 2007; 92(2):F83-8. doi: 10.1136/adc.2005.076794.
10. Adewuyi JO, Gwanzura C. Racial difference between white and black Zimbabweans in the haemolytic activity of A, B, O antibodies. Afr J Med Med Sci 2001; 30(1-2):71-74. PMID: 14510155.
11. McDonncell M, Hannam S, Devane SP. Hydrops fetalis due to ABO incompatibility. Arch Dis Child Fetal Neonatal Ed 1998; 78(3):F220-221. doi: 10.1136/fn.78.3.f220
12. Malekasgar AM. ABO blood group prevalence in spontaneously repeated abortion. Turk J Haematol 2004; 21(4):181-7. PMID: 27264282.
13. Stiller RJ, Herzlinger R, Siegel S, Whetham JC. Fetalascites associated with ABO incompatibility: case reportand review of the literature. Am J Obstet Gynecol 1996; 175: 1371-1372. doi: 10.1016/s0002-9378(96)70057-1
14. Hyperbilirubinemia: Unconjugated. In: Gomella T, Eyal FG, Bany-Mohammed F. eds. Gomella's Neonatology: Management, Procedures, On-Call Problems, Diseases, and Drugs, 8e. McGraw Hill; 2020. Accessed June 11, 2022. https://accesspediatrics.mhmedical.com/content.aspx?bookid=2762&sectionid=234453147

Primljen: 17.03.2022. **Revizija:** 12.06.2022. **Prihvaćen:** 01.07.2022.

Received: 03/17/2022 **Revised:** 06/12/2022 **Accepted:** 07/01/2022