

## Reply

To the Editor,

We thank Dr. Sarıcı for his comments and critique of our manuscript entitled “Neonatal hyperbilirubinemia due to ABO incompatibility: does blood group matter?”, as published in the Turkish Journal of Pediatrics [2013; 55: 506-509].

In this study, our *primary aim* was to evaluate and compare the *clinical severity of neonatal jaundice*, which was mainly reflected by “serum indirect bilirubin level and need for therapeutic approaches such as phototherapy or exchange transfusion” in newborn infants with maternal-fetal A-O or B-O blood group incompatibility. In newborn infants with maternal-fetal blood group incompatibility, the severity of the hemolysis is not always proportional to the severity of the clinical picture. There are many newborn infants with severe fetal and neonatal hemolysis and anemia but without significant hyperbilirubinemia. For that reason, we do not think that the title of our manuscript is incompatible with the aim and content of the manuscript.

In our hospital, reticulocyte count is not a routine laboratory examination for newborn infants with neonatal hyperbilirubinemia, and it is usually performed in newborn infants with severe and long-lasting hemolytic diseases. Peripheral blood smear, which includes most of the qualitative markers of hemolysis such as normoblasts and morphological erythrocyte abnormalities such as spherocytosis, is also important in detecting the presence of hemolysis. Normal reticulocyte value is 4-5% for term and 6-10% for preterm newborn infants of 30-36 weeks' gestational age. In “ABO hemolytic disease of the newborn”, values are much higher and usually range between 10-30%. In your manuscript entitled “An early (sixth-hour) serum bilirubin measurement is useful in predicting the development of significant hyperbilirubinemia and severe ABO hemolytic disease in a selective high-risk population of newborns with ABO incompatibility”<sup>1</sup>, although the mean reticulocyte value found in newborns with significant hyperbilirubinemia was higher than the mean reticulocyte value of the newborns without

significant hyperbilirubinemia ( $4.39 \pm 3.46\%$  vs  $2.95 \pm 1.63\%$ ), it was still within the normal reticulocyte values mentioned above<sup>1</sup>. Thus, the mean reticulocyte count in newborns with significant hyperbilirubinemia does not seem to be actually correlated with the severity of hyperbilirubinemia in the study mentioned above.

We agree with the sentence that isoimmunization is unlikely to be the cause of hemolysis in all ABO-incompatible newborn infants. In the literature, although risk factors for ABO incompatibility are present in 12-15% of pregnancies, evidence of fetal sensitization (positive direct Coombs test) occurs in only 3-4%. In addition, although direct Coombs test positivity is a strong marker of “real ABO incompatibility”, it is not an ideal marker of hemolysis and does not always correlate with the severity of neonatal hyperbilirubinemia<sup>2</sup>. In our study, we excluded the newborn infants who had other hemolytic diseases, such as Rh incompatibility, glucose-6-phosphate dehydrogenase (G6PD) deficiency or other known causes of neonatal hyperbilirubinemia, such as endocrine and metabolic disorders. Thus, we think that we minimized the risk of “contamination” of other causes of neonatal hyperbilirubinemia in our study population.

In addition, our study was not a prospective study based on the development of predictive factors for significant hyperbilirubinemia in newborn infants with ABO incompatibility, but rather was a retrospective study attempting to define the clinical severity of neonatal hyperbilirubinemia that was already developed in newborn infants with ABO incompatibility.

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## REFERENCES

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2. Murray NA, Roberts IA. Haemolytic disease of the newborn. *Arch Dis Child Fetal Neonatal Ed* 2007; 92: F83-88.