Robbing Peter to Pay Paul?

Balancing Maternal and Fetal Risks in a Jr^a-immunised Pregnancy

O Firth[1], N Wilkes[2], C Booth[3], J Wong[4], Z Achercouk[4], C Ellis[4], C Rees[4], J Gill[5], S Grimsley[5], D Lam[6]

1 – NHSBT Cambridge
2 – NHSBT Birmingham
3 – NHSBT Colindale
4 – Frimley Park NHS Trust
5 – IBGRL, NHSBT
6 – NHSBT Tooting

Introduction

Alloantibodies to red cell antigens occur in 1.2% of pregnancies[1] and can lead to haemolytic disease of the fetus and newborn (HDFN). Rare antibodies pose a challenge as there is often a paucity of data about their risks. Anti-Jr^a is a rare antibody that has been reported in four severe or fatal cases of HDFN[2,3]. There is little evidence that anti-Jr^a causes haemolytic transfusion reactions and therefore antigen negative red cells are not usually required for transfusion[4]. However, in pregnancy, transfusion of antigen positive blood might stimulate increasing antibody titres and exacerbate the already recognised risk of HDFN. As the Jr^a-negative phenotype is rare within UK donors, balancing the potential risk of anti-Jr^a-associated HDFN against the scarcity of Jr^a-negative units requires involvement from a broad MDT.

A patient presented to NHSBT at 14/40 gestation with a pan-reactive panel, later confirmed as an anti-Jr^a. She has β-thalassaemia intermedia and three previous pregnancies requiring transfusion support of 2-8 units, increasing with each subsequent pregnancy. Only one ABO Rh and K compatible, Jr^a-negative unit, was available in the National Frozen Blood Bank, with no wet units available. MDT discussion led to the decision to use Jr^a-unselected units throughout her pregnancy with careful monitoring; her case is presented below.

Results

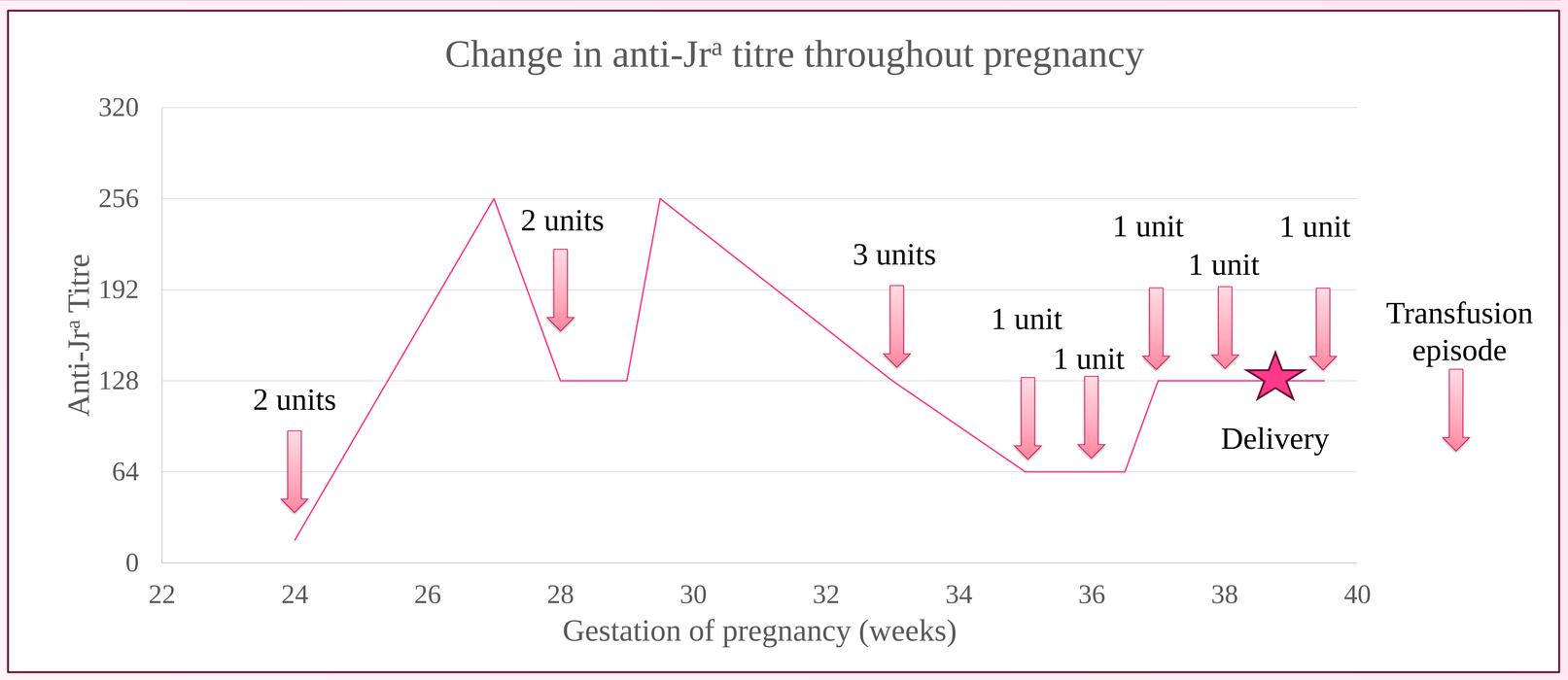
12 Jr^a-unselected red cell units were transfused during the pregnancy with no reactions or evidence of maternal haemolysis.

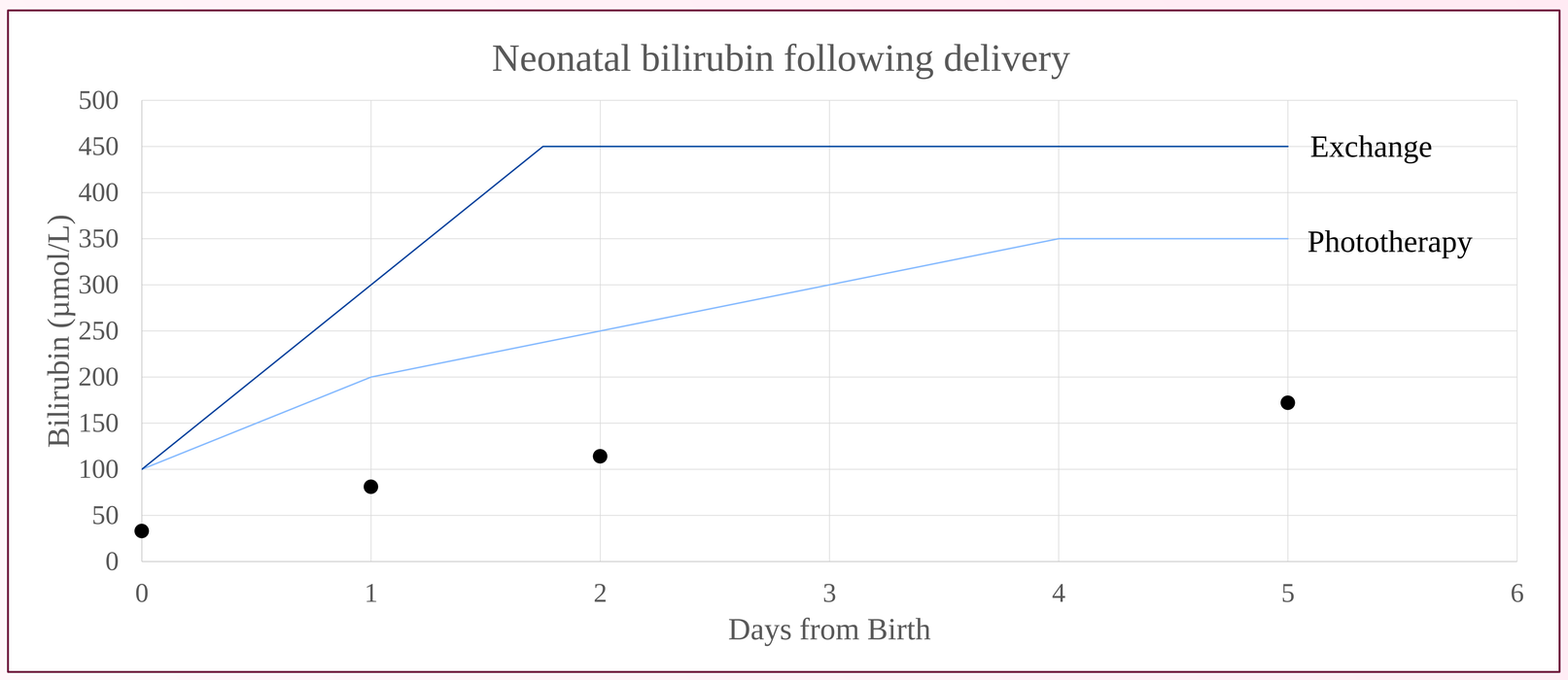
Anti-Jr^a titres rose to a peak around 28-30 weeks gestation and fell following this despite ongoing transfusion.

Fetal scans conducted fortnightly showed no signs of anaemia, with normal middle cerebral artery peak systolic velocities throughout.

Delivery was by planned C-section and cord bloods showed a normal haemoglobin and bilirubin.

Subsequent bilirubin levels remained below the phototherapy and exchange transfusion treatment lines without intervention.





Conclusion

There is a tendency to publish case studies where atypical antibodies have led to poor outcomes, and this could easily skew our view on the relative clinical significance of rare antibodies. This case highlights that despite reported cases of severe HDFN due to anti-Jr^a, and a high maternal anti-Jr^a titre in this patient, that the risk of stimulating antibody formation and HDFN through transfusion of Jr^a-unselected units can be successfully managed. Additionally, positive fetal and maternal outcomes can be achieved through close collaborative management across Trusts and different teams.

Acknowledgements

IBGRL, NHSBT
RCI, NHSBT
Frimley Park NHS Trust

References

1 – Koelewijn JM, et al., Effect of screening for red cell antibodies, other than anti-D, to detect hemolytic disease of the fetus and newborn: a population study in the Netherlands. *Transfusion* 2008;48:941-952
2 – Kim MS, et al., Fatal hemolytic disease of the fetus and newborn caused by anti-Jr^a antibody: A case report and literature review. *Transfusion and Apheresis Science* 2020 Feb;59(1):102605
3 – Tran A, et al., Severe fetal anaemia caused by anti-Jr^a: Burst forming unit-erythroid colony formation inhibition assay suggesting possible erythroid suppression mechanism. *Transfusion* 2023;63(4):877-882
4 – SPN214/5 – The Clinical Significance of Blood Group Alloantibodies and the Supply of Blood for Transfusion. *NHSBT*