Inadverently Administration Of Anti – D Immunoglobulin To Infant: A Case Report

Dr Subir Pal

MBBS, DCH, MD, Consultant Child Specialist & Neonatologist, Iris Hospital, Kolkata., Visiting Consultant Amri, Mukundapur, Kolkata.

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Abstract- A term girl baby was born by elective caesarean section was inadvertently injected with 300µg of ANTI – D Immunoglobulin 18hrs after birth. Mother’s blood group was O-ve. The baby was the second child. Her cord blood samples revealed that the blood group was A+ve, Hb 18gm%, DCT – Negative, Total Bilirubin-2.5mg/dl.

After realising the accidental injection of ANTI-D Immunoglobulin to infant, the Nursing Home authority called me urgently for immediate evaluation of the infant. There was no sign of anaphylactic reaction. Spo2 – 99% at room air HR – 138/min, Colour-Pink, CRT <3sec, Cry, Tone, Reflex ,Activity – Normal. Baby was constantly monitored for vitals and blood was sent for evidence of Haemolysis.

On Day 2. Blood Report was as follows:-

- SBR (Serum Bilirubin) – 8.1mg/dl.
- Conjugated – 0.71mg/dl.
- Unconjugated – 7.44mg/dl.

Peripheral Smear:

- RBC – Normocytic & Normochromic.
- WBC – Within normal limit.

Platelet Adequate:

- DCT (Direct Coombs Test) – Negative.

On Day 3:-

- G6PD – 16.6 U/gHb.
- SBR – 9.5mg/dl.
- Conjugated – 0.15mg/dl.
- Unconjugated – 9.05mg/dl.

Peripheral Smear: Within normal limit.

On subsequent day, there was no laboratory evidence of Haemolysis. There was no need of any intervention like Phototherapy. Baby was on exclusively breast feeding, urine, meconium passed as usual with complete recovery. She was discharged in stable condition and asked for follow up.

I. DECLARATION OF INTEREST

The Authors declared that there is no conflict of interest. The Authors declared that they did not receive any funding in the making of the present work.

II. RHESUS HAEMOLYTIC DISEASE OF NEWBORN:-

Rhesus negative mother having Rhesus positive fetus, the antigen of the fetal red blood cell may activate maternal immunologic system to produce anti body. The Anti – D anti bodies are IgG type. They can cross placenta and destroy D – positive fetal blood cells. [1]

There are not enough anti bodies during first pregnancy, in subsequent pregnancies with Rhesus+ fetus, the IgG memory b cells mounts an immune response. The RBC coated with anti body are haemolysed by anti bodies binding and complements. [2]

The haemolysis may leads to hydrops fetalis, fatal death, hyperbilirubinemia.

III. PREVENTION:-

If mother is RHD negative RHIG (Rho D Immunoglobulin) can prevent sensitization of the maternal immune system and Rhesus disease of fetus and newborn.

IV. TREATMENT OF RHESUS DISEASE OF NEWBORN:-

It may cause hyperbilirubinemia. In mild disease phototherapy for neonatal Jaundice or exchange transfusion if moderate or severe disease according to protocol. IVIG may shorten duration of phototherapy and reduce the need for the exchange transfusion. [3, 4]

Exchange transfusion was the first treatment of Rhesus disease. It was invented by Dr. Alexander S. Wiener. [5]

Ronald Finn of England said that the disease can be prevented by injecting anti body to mother against fetal RBC (anti RHD). [6]

Ms. Marianne Cummins was the first risk mother who received anti Rho (D) immunoglobulin (RHIG). [7]

V. CONCLUSION:-

Extensively literature search showed only two previous case reports and we report the third incidence. Anti – D Gamma globulin do not activate complement and causes mild anaemia to Rhesus positive babies due to immune mediated Haemolysis.

No intervention was required and Hospital – stay was uneventful.

REFERENCES


AUTHORS

First Author – Dr Subir Pal, MBBS, DCH, MD, Consultant Child Specialist & Neonatologist, Iris Hospital, Kolkata., Visiting Consultant Amri, Mukundapur, Kolkata.