Case Discussion

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- A 31 year old, gravida 3 with 38 weeks 5days of pregnancy presented with decreased fetal movement since morning of the same day. There was no h/o pain,any trauma or vaginal bleeding. Previous 2 pregnancies were uneventful. Current pregnancy was also uneventful until the patient presented with decreased fetal movements.

- Blood group O positive, HIV/HBsAg/HCV negative, Rubella immune

- CTG showed fair variability with intermittent heart rate spikes

- Ultrasound done (2 PM on the same day) revealed no fetal movement for 25 mins with middle cerebral artery showing normal flow velocities

- Emergency Caesarean was performed at around 6 pm on the same day

- A severely pale, limp male infant with no respiratory effort delivered with a birth weight of 2400 gm(10th centile) .

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- Started on PPV after initial steps in the OT

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- shifted to endotracheal intubation & 100% oxygen

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- chest compression for 60 sec and a normal saline intravenous bolus 10 ml/kg given (with first spontaneous breath recorded at 3 minutes)

- At 6 min of life: HR 120/min with gasping respirations, poorly palpable peripheral pulses and marked pallor

- 2 more saline boluses were repeated

- Baby was shifted to NICU and was put on Mechanical Ventilation

- SIMV PEEP-5, PIP-16 and FiO2 50%.

- UVC put in and samples for a CBC & reticulocyte counts, Blood culture, bilirubin, DCT and blood grouping was taken

- O negative whole blood was arranged

- Cord blood pH 7.03,HCO3-12, Hb-2.8mg/dl, Base Deficit of 20

- At 20 min of life: Whole blood bolus 25 ml over 15 min given,

- Peripheral pulses palpable now with regular breathing efforts and improving muscle tone

- Iv fluids started @ 60 ml/K/day & put on Dobutamine @10 mcg/k/min for borderline perfusion

- APGAR -1, 4 &6 at 1, 5 and 10 minutes respectively.

O/E:

- HR- 166/min
- RR- on SIMV with spontaneous efforts of 44/min
- SaO2>92%
- No facial dysmorphism or any congenital malformation
- No visible signs of blood collection/cephalhematomma/subgaleal bleed

RESP-air entry was bilateral equal and clear

CVS- normal heart sounds

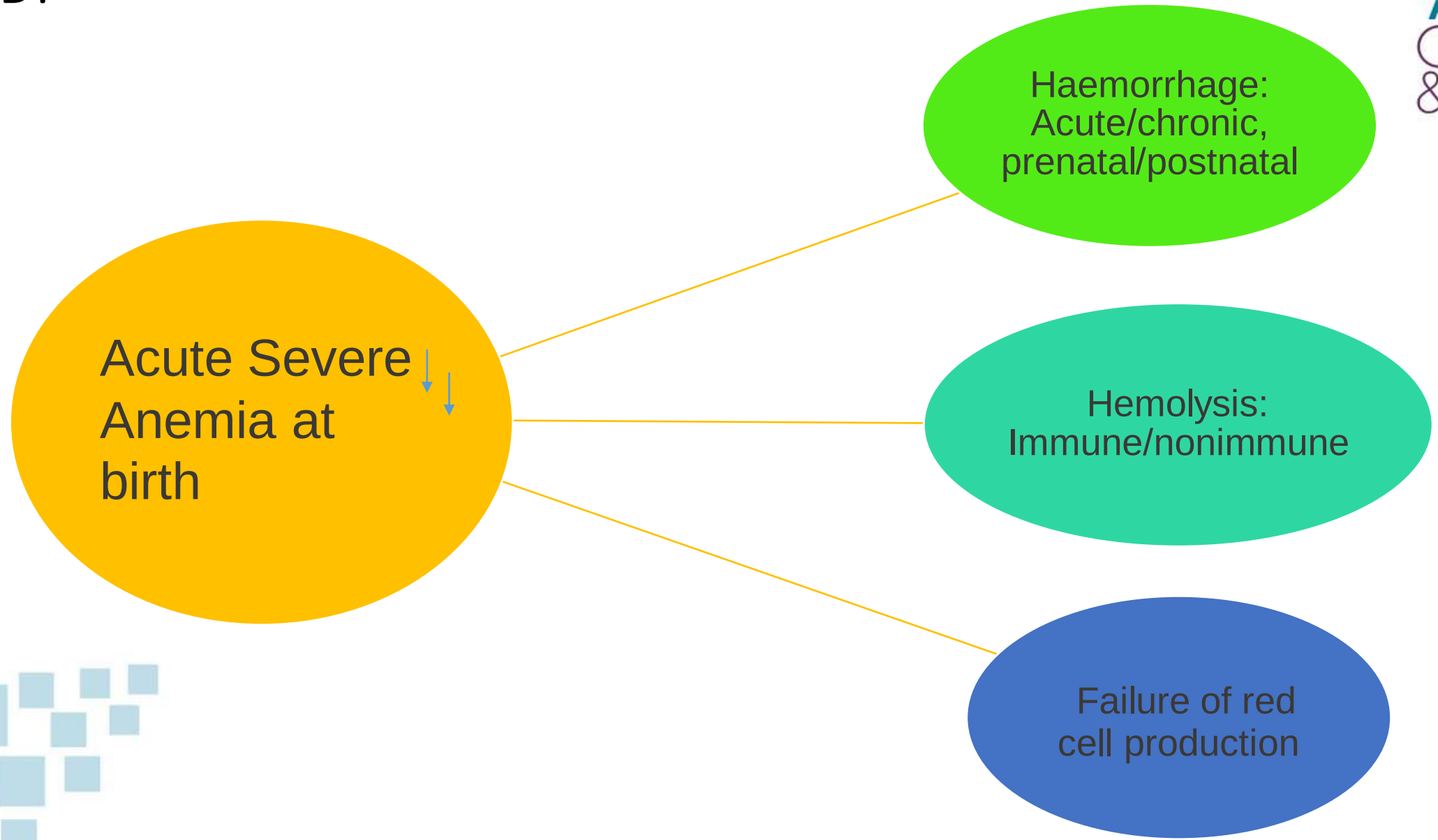
Abd- soft, no organomegaly

CNS- tone started to improve by 20 min with “towards normalcy” & at around 30-45 min response to normal tactile stimulus by limb withdrawl & wincing present

Pupils- both reacting to light

TERM/AGA/LBW/PERINATAL ASPHYXIA(DEPRESSION)/ SEVERE ANEMIA

D/D:


Acute Severe Anemia at birth
Haemorrhage: Acute/chronic, prenatal/postnatal
Hemolysis: Immune/nonimmune
Failure of red cell production

BLOOD LOSS

Prenatal hemorrhage may be caused by

- Fetal-to-maternal hemorrhage, Twin-to-twin transfusion
- Cord malformations, Placental abnormalities
- Diagnostic procedures

- **Perinatal hemorrhage** Precipitous delivery (ie, rapid and spontaneous delivery, which causes hemorrhage due to umbilical cord tearing)
- Obstetric accidents (eg, incision of the placenta during cesarean delivery, birth trauma)
- Coagulopathies

- **Hemorrhagic disease of the newborn**

DECREASED PRODUCTION

Congenital-

- Diamond Blackfan
- Fanconi's

Acquired-

- Infections: malaria, rubella, syphilis, HIV, cytomegalovirus, adenovirus, bacterial sepsis, Congenital parvovirus B19

BREAKDOWN

Hemolysis

- Immune-mediated disorders
- Red blood cell (RBC) membrane disorders- HS, HE
- Enzyme deficiencies- G6PD, Pyruvate Kinase
- Hemoglobinopathies
- Infections

INVESTIGATION

- Hb-3.2 g/dl,
- Platelet= 105 x $10^3/mm^3$
- Leucocyte count=50.8 x 10^3
- Total bilirubin of 0.5 mg/dl
- Blood group O positive
- DCT was negative..(double checked)
- Peripheral smear- not suggestive of hemolysis
- Serum LDH sent
- Serology for Parvovirus B 19- awaited
- Urine for CMV DNA PCR was sent

- Point of care- USG Cranium and abdomen -Normal
- Histopathological examination of placenta was normal
- Kleihauer Betke test was sent on the mother’s blood

- Infant received another 20 ml/Kg packed RBC transfusion in the next 24 hrs with a Hb of 10.4 gm/L post transfusion.

- Other complications of perinatal depression such as following were observed and managed appropriately

1. *Renal injury (hematuria 2+) with anuria,*
2. *deranged liver enzymes*
3. *hypocalcemia were routinely managed with good response over next 72 hrs.*

- Nasogastric partial feeds started on Day 2 and slowly progressed to full feeds by day 8.

- Infant was extubated on day 3 of life to nasal CPAP and to room air by day 8 of life.

KB Test Report- ratio of 3 fetal RBC's to every 100 of maternal corresponding to a fetal blood loss of 165 ml ("massive volume")

FINAL DIAGNOSIS:

TERM/SGA/LBW/ PERINATAL ASPHYXIA/ SEVERE ANEMIA DUE TO MASSIVE FETOMATERNAL HEMORRAGE

- Ultrasound cranium at 24 hrs and prior to discharge on day 12 was normal.
- A complete neurological examination at the time of discharge with Hammersmith recording was normal
- Baby was discharged on day 13 of life on breastfeeding and with appropriate weight gain.
- MRI in follow up at 4 weeks revealed no abnormality with normal neurological follow up

DISCUSSION

- Spontaneous fetomaternal hemorrhage is defined as fetomaternal bleeding with no antecedent history of trauma and no clinical/histopathological evidence of abruption. (suggested in 50% of all pregnancies)

- Vast majority of spontaneous FMHs are small volume bleeds(<2 ml) of no hemodynamic significance

- Spontaneous massive FMH is much less common.

-If acute, it can result in rapid fetal hemodynamic collapse and death.

-If chronic, it can result in fetal anemia and hydrops fetalis

- Volumes from 10 to 150 mL have been proposed to define massive FMH.

INCIDENCE

- Incidence of spontaneous massive FMH is unknown

- FMH greater than 80 mL and greater than 150 mL is estimated to occur in 1 in 1000 deliveries and 1 in 5000 deliveries, respectively

- A FMH of 20 mL/kg, which represents 20% of the fetoplacental blood volume is considered massive because it is associated with significant fetal/neonatal morbidity or mortality.

PATHOGENESIS

- Pathogenesis of spontaneous massive FMH is unclear.

- Breach in the integrity of the placental circulation.

- Histological study of Placenta revealed that retroplacental and parenchymal hemorrhage ,intervillous thrombi increased the likelihood of fetal cells presence in maternal circulation

Spontaneous versus Traumatic

- “Spontaneous” FMH is defined as one without antecedent history of trauma and no clinical or histopathological evidence of disruption.

- “Traumatic” injuries to the placental surface is caused by invasive diagnostic procedure(amniocentesis, chorionic villous biopsy, fetoscopy) blunt abdominal trauma (External cephalic version ,fall, Motor Vehicle crash)

CLINICAL PRESENTATION

- Spontaneous massive FMH can occur at any time during pregnancy or at delivery.

- Fetal death may be the presenting sign of a massive acute bleed.

- Hydrops and/or abnormal fetal heart rate patterns and/or decreased fetal movement may be the presenting signs of a massive FMH

- Some massive FMHs have no signs or symptoms.

- Severe Neonatal anemia

- Fetal heart rate monitoring in this setting may show sinusoidal wave pattern, recurrent late deceleration or fetal tachycardia

- Ultrasound may yield a low biophysical profile score.

Candidates for FMH testing

- Nonimmune hydrops fetalis with MCA-PSV ≥1.5 MoMs
- Sinusoidal fetal heart rate (FHR) pattern with MCA-PSV ≥1.5 MoMs
- Fetal demise/stillbirth
- Neonatal anemia

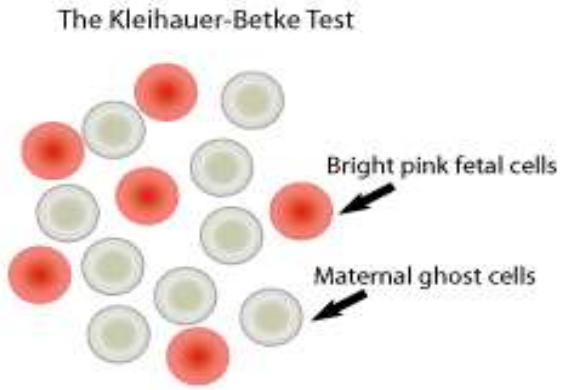
LAB EVALUATION

- Kleihauer-Betke acid elution assay
- Flow cytometry

KLEIHAUER BETKE ACID ELUTION ASSAY

- Detection and quatification of FMH
- Red blood cells from the maternal circulation are fixed

  to a slide that is exposed to an acidic pH solution
- Adult red blood cells become "ghost" cells (because of absence of staining)
- Fetal RBC's remain pink as hemoglobin F is stable at pHs in this range
- The volume of fetal whole blood (mL) in the maternal circulation is =



- *Percentage of fetal cells = Number of fetal cells X 100 / Total number of RBCs*

FLOW CYTOMETRY

- A monoclonal antibody to hemoglobin F is conjugated to a fluorochrome and used to detect fetal hemoglobin in permeabilized cells as they pass through the channel of a flow cytometer

- Increased cost and decreased availability of laboratory technicians

Management of antenatally detected cases

≥32 weeks of gestation –

Immediate cesarean delivery, with blood available for prompt neonatal transfusion

<32 weeks of gestation

- acutely correct fetal anemia, intravascular intrauterine transfusion (IVT) of donor red blood cells ,this transfusion is probably associated with lower morbidity and mortality
- daily Kleihauer-Betke or flow cytometry testing, MCA-PSV, and biophysical profiles until IVTs are no longer necessary.

Outcome

"Maternal"

- mother is at risk of alloimmunization to red cell antigens.

"Fetal/neonatal"

- Depends upon the rapidity of fetal blood loss and the volume of the hemorrhage relative to total fetoplacental blood volume
- Massive FMH is fatal if blood loss occurs over minutes rather than hours, days, or weeks..
- Hydrops if bleeding is over a long period with chronic anemia

Long-term prognosis for infants after massive fetomaternal haemorrhage

Chrystèle Rubod , Philippe Deruelle, Francoise Le Goueff, Virginie Tunez, Martine Fournier, Damien Subtil

- *48 patients*
- *Fetomaternal hemorrhages of 20 mL/kg or more significantly increased the risk of fetal death, induced preterm delivery, transfer to NICU, and neonatal anemia requiring transfusion. Long-term follow-up was not associated with neurological sequelae*

Apollo Cradle & Children's Hospital™

THANKS FOR LISTENING!!