I. NEONATOLOGY

SEVERE POSTASPHYXIC SYNDROME FOLLOWING A MASSIVE FETOMATERNAL BLEEDING

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Abstract

The fetomaternal bleeding might be an important cause of anaemia in the neonate. It occurs in 8% of all pregnancies and in 1% of them the volume may be as large as 50 ml. These losses may be acute or chronic. If the blood loss is recent, it may lead to a neonatal hypovolemic shock along with it's drastic consequences. If the bleeding is chronic, the baby develops an iron deficiency anaemia in which situation he is pale, the hematocrit level is low but his peripheral pulse and perfusion are good.

The normal level of hematocrit does not put aside a massive acute haemorrhage or a chronic fetomaternal bleeding on it's way to burst!

ABO incompatibility may have a masking effect over a neonatal anaemia due to the foetal bleeding. This is why it is mandatory to take into consideration this possibility.

The fetomaternal bleeding is confirmed by the Kleihauer – Betke preparation of the mother's blood. A 50 ml loss of foetal blood into the maternal circulation will show up as 1% foetal cells in the maternal circulation. Be aware that the test is considered non valid when there is evidence of a maternal hemoglobinopathy. There are some records that the foetal anaemia is frequently associated with a particular situation – the umbilical cord wrapped around the baby – and this concludes with a neonatal anaemia dued to a possible compression of the umbilical vein and secondary the decrease of foetal venous blood flow.

Key words: fetomaternal bleeding, Kleihauer – Betke test.

There has to be emphasized the major role of the severe ischemia and hypoxia that come along together. They add a burden on the medical judgement in the aspects of diagnosis and therapy to be chosen. At this moment, there is necessary to pinpoint some issues:

- The diagnosis is not always revealed from the very beginning
- The complete evaluation is difficult
- The treatment is controversial
- The prognosis (ethical and medical) is complex

CASE REPORT

The baby P.I. is a male, born on the 10th of July, 2003 at 11.30 am. His birth weight was 2900gr, cranial circumference – 32 cm, thoracic circumference – 31cm and 47 cm in length. His mother, P.O. was 24 years old, with no history of other pregnancies or abortions, 0I, Rh +, married to the healthy father aged 29 years. She was a house wife and they live in the city. Her first visit to the doctor took place when she was two months pregnant and she kept on going monthly afterwards. The date of her last period was the 10th of October, 2002 so the gestational age of her baby was approximately 39 weeks. At the moment of her admittance in our hospital, she was considered to do well, having no fever, blood pressure 120/80 mmHg, the uterus's cervix was 3 cm dilated, the amniotic membranes were ruptured 3 hours prior to this moment. The obstetrician decided to continuously monitor her and her baby and started Ocitocine iv. She was closely watched during this procedure so that the foetal distress that rapidly underwent (persistent bradychardia) could be traced from the very beginning. There was no delay in deciding to put to an end to this pregnancy by performing an emergency caesarean section.

The baby boy delivered by caesarean section, head first scored Apgar as follows: 1 at 1 minute, 1 at 5 minutes, 3 at 15 minutes and 4 at 20 minutes. There were no evidences of macroscopic placental pathology (malformation or sectioning), no evidence of umbilical cord pathology and no evidence of maternal bleeding.

First evaluation:

- the baby was pale,
- non reactive, limp

- no respiratory effort, bradycardic (heart rate < 60 bpm),
- fixed mydriasis,
- no peripheral pulse, impossible to detect the blood pressure.

Gathering the information we could get so far in the delivery room, we had established the *first diagnosis*:

1. term newborn baby boy with intrauterine growth restriction
2. neonatal shock probably dued to a hypovolemic process

Resuscitation in the delivery room: the baby was placed under a radiant heater (to reduce heat loss by radiation and conduction), he was then dried and wrapped in pre-warmed towel (to prevent evaporative heat loss) meanwhile assessing his colour, muscle tone, heart rate and breathing efforts. The airway was clear but yet there was no effective breathing so the baby was intubated using an endotracheal tube. He was given inflation breaths, receiving 100% O_2 with continued support, breathing efforts were almost satisfactory but the heart rate did not respond. Giving the very slow heart rate and not increasing despite good lung inflation as judged by good chest movement, it was necessary to start chest compressions (to provide means of moving oxygenated blood from the lungs to the heart and coronary arteries). Nevertheless, these resuscitative measures were not enough to produce an increase in heart rate so we chose the next step – drugs (Adrenaline via endotracheal tube, repeatedly). In order to have a rapid venous access, there was placed an umbilical venous catheter and several boluses of normal saline 0,9% and sodium bicarbonate were pushed in.

Second evaluation (after 20 minutes of resuscitation):

- extremely pale,
- non reactive, no spontaneous breathing,
- heart rate >100 bpm, blood pressure 60/30 mmHg,
- O_2 saturation 80-85% while receiving 100% O_2 via endotracheal tube,
- Astrup parameters (arterial blood sampled): pH=7,15, P_aO_2=35, P_aCO_2=65, BE=-15, HCO_3^- =18,
- Referred to the Neonatal Intensive Care Unit for assisted ventilation (IPPV) with the start parameters as follows: PIP=24-26-29 cm H_2O, PEEP=4cmH_2O, ET/IT=0,3/0,9, volume=7l/minut, respiratory rate=55-57rpm, FiO_2=100% (they were adjusted according to the Astrup values and the chest X-ray performed, sequentionally)
- The main goal was to sustain a viable circulation for the baby giving him Dopamine + Dobutamine along with prophylactic antibiotics (Ampicilline + Gentamycine), drugs to prevent further bleeding, vitamin E and several transfusions of packed RBCs of the same group and Rh as the baby's.

Second diagnosis (after admitting in the Neonatal Intensive Care Unit):

1. term newborn baby boy with intrauterine growth restriction
2. neonatal shock- severe acute hypoxia
3. multiorganic failure
4. acute post bleeding anaemia.

Differential diagnosis between neonatal asphyxia and acute blood loss.

FEATURES	NEONATAL ASPHYXIA	ACUTE BLOOD LOSS
Heart rate	Low	High
Respiratory rate	Low	High
Intercostal retractions	Present	Absent
Colour of the skin	Pale with cyanosis	Pale, no cyanosis
Response to O_2 or assisted ventilation	Improving	No significant changes

Third evaluation

- the neurological examination stated the 2nd / 3rd stage of SARNAT and SARNAT , the baby being lethargic/comatose, mildly hypotonic, absent complex reflexes, miosis, spontaneous respirations but occasionally apnoeic, heart rate normal/tachycardia, generalized tonic - clonic seizures (day 2).
- Because there was no identifiable blood loss coming from a placental cause, an umbilical cord pathology and no post delivery bleeding, we targeted our attention towards a foetal bleeding.

So, there were performed additional investigations:

► Hb=9,6g%, Ht=30%, H=2630000/mm^3, L=11000/mm^3, BIII, Rh (-)

► Astrup –severe metabolic acidosis

► low Ca level in the serum, urea=53U/L, Creatinine=0,9mg%, Na^+=126mEq/l, K^+=3,88mEq/l, blood sugar level 181mg%….40mg%

► chest X-ray – cardiomegalia (cardiothoracic index>0,67), increased vascular shadow on the pulmons (Fig. 1)

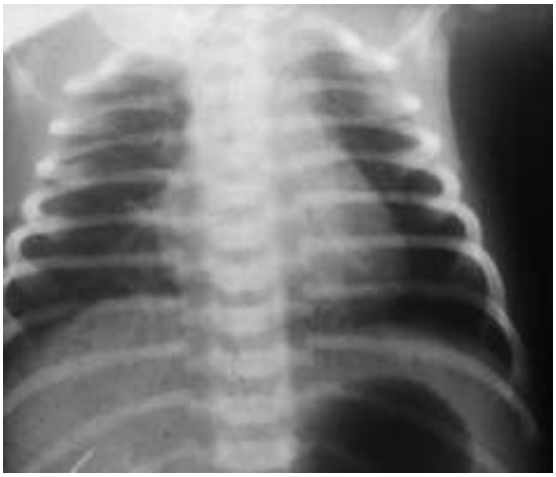

Fig. 1. Chest X-ray: cardiomegalia, increased vascular shadow on the pulmons.

► transfontanellar ultrasound – ischemic encephalopathy, multifocal cortical necrosis with cerebral oedema
► the maternal blood sample is coloured using acid fluids; the foetus's erythrocytes turn dark while the mother's do not and this is why these last ones are named "phantom cells"
► the volume of fetomaternal transfusion = (number of foetal erythocytes/number of maternal erythrocytes) x 2400

The outcome:

-day 2-extremely reduced activity, generalized seizures which did not ceased after triple anticonvulsant therapy (Phenobarbitone + Valproic Acid + Midazolam).

-day 4-stuporos/comatose, absent spontaneous respirations, acute renal failure-rapidly progressing, repeatedly decreases of O_2 saturations in need of bag-and-mask ventilation and *finally cardio-respiratory arrest.*

Anatomo-pathological aspects:

- severe cerebral oedema with plane cerebral circumvolutions, interhemispheric patterns roughly recognizable; pointish haemorrhages on the cerebral mass, cerebellum, cerebral trunchus; pulmonary tissues evidenced shock, liver and spleen were affected by the stasis phenomenon; the kidneys had extended areas of necrosis on their cortex and medulla (hemorrhagic necrosis).

Conclusions:

- The fetomaternal haemorrhage with acute onset may mimic a severe obstetrical asphyxia
- Continuously monitoring the mother and her foetus was beneficial for choosing the wise moment to perform the caesarean section
- Tough target it was to identify in the delivery room the initial cause of the severe foetal distress!
- Initially, the Hb level of the baby might be slightly low because of the vasoconstrictive process
- The clinical aspects of a respiratory distress (pallor, tachycardia, tachypnea, weak pulse, low blood pressure) may as well be signs of an obstetrical asphyxia
- BE AWARE when you have a pale baby with no evidence of cyanosis, at the very beginning of a respiratory distress and KEEP IN MIND the possibility of a fetomaternal bleeding especially if the baby in his poorest condition does not respond to O2 therapy or to conventional mechanical ventilation (high parameters)
- The poor outcome of this case may be explained by an antenatal asphyxia because of a massive acute blood loss certified by Kleihauer-Betke test
- The major 3 factors involved in the asphyxic process (hypoxia due to anaemia, hypercapnia, mixed acidosis) led to consequences affecting multiple areas: pulmonary level, cerebral level (cerebral oedema, signs of hypoxic-ischemic encephalopathy), renal tubular necrosis with acute renal failure, mesenteric hypo perfusion, lactic acidosis, glycaemia, electrolyte and thermal disorders

NOTA BENE

**this was a case of massive acute fetomaternal bleeding, demonstrated by the Kleihauer-Betke test (HbF>4% states for a foetal loss of 200ml);
**the pregnant woman was closely monitored during her labour;
**even if the foetal distress was captured from the very first possible moment, it's consequences were devastating;
**giving Ocitocin in order to sustain the labour of the mother associated with a wrapped umbilical cord might increase the blood loss from the foetus to his mother.

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