

SUBGALEAL/SUBAPONEVROTIC HEMORRHAGE: A POTENTIALLY LETHAL CONDITION IN NEWBORNS

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Abstract: Subgaleal hemorrhage is determined by the rupture of the emissary veins, often associated with traumatic delivery. The blood loss can produce hypovolemic shock, disseminated intravascular coagulation (DIC), severe metabolic acidosis and death. **Material and method:** This is a case report about a newborn who died of hemorrhagic shock secondary to a subgaleal hemorrhage. **Results:** The newborn was admitted in the 3th day of life with a severe condition: hypovolemic shock, renal failure, DIC and a scalp swelling extended to the neck, eyelids and ears. **The treatment included:** repeated blood transfusions, inotropic agents, assisted ventilation. **The baby died on day 12, despite of intensive care management. Postmortem examination confirmed a massive subgaleal hemorrhage, and hypoxic-ischemic lesions of the brain. Conclusions:** Subgaleal hemorrhage must be diagnosed early, massive volume resuscitation has to be initiated as soon as possible, but often the affected newborns can not be saved because of multiorgan failure.

Cuvinte cheie: hemoragie subgaleală, traumatism obstetrical, șoc hipovolemic

Rezumat: Hemoragia subgaleală este determinată de ruptura venelor emisare, asociată frecvent cu nașterea traumatică. Sângerarea poate duce la șoc hipovolemic, coagulopatie intravasculară diseminată (CID), acidoză metabolică severă și deces. **Material și metodă:** Lucrarea este o prezentarea cazului unui nou-născut decedat în urma șocului hemoragic determinat de hemoragia subgaleală. **Rezultate:** Nou-născutul a fost internat în ziua a treia de viață cu stare generală gravă, semne de șoc hipovolemic, insuficiență renală, CID și edem hemoragic extins al scalpului. **Tratamentul a inclus transfuzii repetate de sânge, agenți inotropi, ventilație asistată. Nou-născutul a decedat în ziua a 12-a de viață în ciuda terapiei intensive susținute. Examenul anatomopatologic a evidențiat hemoragie subgaleală masivă și leziuni hipoxic-ischemice ale creierului. Concluzii:** hemoragia subgaleală necesită diagnosticare precoce, resuscitare volemică masivă inițiată cât mai curând posibil, susținerea funcțiilor vitale, însă frecvent decesul survine în ciuda acestor eforturi datorită insuficienței multiorganice.

INTRODUCTION

Literature review: Subgaleal hemorrhage is determined by the rupture of the emissary veins (the connections between the dural sinuses and the scalp veins). Blood is accumulated in the virtual space between the epicranial aponeurosis and the periosteum of the skull bones.⁽¹⁾

The prevalence of moderate-to-severe subgaleal hemorrhages is approximately 1,5 per 10 000 births.⁽²⁾ About 90% of cases are the result of a traumatic delivery: vacuum extraction, forceps application to the head in delivery, but it may also occur spontaneously. It may be associated with intracranial hemorrhage or skull fracture. In term babies, the subaponeurotic space may retain as much as 260 ml of blood (which is almost equivalent to the baby's blood volume).

The hemorrhage in the subgaleal space is gradual and it may not be clinically apparent at birth or during the first hours or days of life. The diagnosis is made by clinical examination and it appears as a fluctuant mass developing over the scalp. The mass extends forward to the orbital margins, backward to the nuchal ridge and laterally to the temporal fascia. The hematoma develops gradually, insidious and spreads across the whole calvaria.^(1,3,4,5,6) It may be initially confused with large caput succedaneum or cephalohematoma. Caput succedaneum (local

edema of the scalp with superficial echimosis) should regress by the first 48-72 hours of life, and cephalohematoma (subperiosteal hemorrhage) is limited to the individual skull bone, does not cross the midline and the resolution occurs over 2-3 weeks. Subgaleal hematoma may lead to anemia and severe hypovolemia - but blood loss may be massive before hypovolemia becomes evident. Clinical signs include: pallor, hyperbilirubinemia, tachycardia, increased respiratory rate, hypotension, oliguria, bleeding disturbances, changes of the neurological status. It may be complicated with hypovolemic shock, renal failure, disseminated intravascular coagulation, persistent metabolic acidosis, severe encephalopathy and death.^(1,5,6,7,8)

Management include: early recognition of subgaleal hemorrhage, vigilant observation to detect progression of the hematoma, measuring the circumference of the head and monitoring vital signs hourly, hemoglobin measurement every 4-8 hours, early institution of supportive care, and aggressive therapy for shock and anemia such as blood transfusion, volume support, and coagulation factors. Most of the newborns with subgaleal hemorrhage require intensive care: mechanical ventilation, boluses of crystalloid products, repetitive blood transfusions, inotropic agents, support of the vital functions,

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phototherapy.^(4,5,8,9) The volume of blood required may be estimated using the formula: 38 milliliters of blood for each centimeter by which the actual head circumference exceeds the head circumference at birth.⁽¹⁰⁾

In the absence of shock or intracranial injury, the long-term prognosis is generally good. The outcome of the newborns with subgaleal hemorrhage depends on early diagnosis, careful monitoring, prompt treatment, and associated pathology.

MATERIAL AND METHOD

We present the case of T.R., 34-35 weeker preterm female, born after spontaneous labour, in cranial presentation, through vaginal delivery, in a level I maternity, transferred to our neonatal intensive care unit in the 3rd day of life, diagnosed in our center with subgaleal haemorrhage, renal failure, DIC.

RESULTS

T.R., a female newborn was born at 34-35 weeks of gestation, after spontaneous labour in cranial presentation, vaginally, after a prolonged delivery, in a level I maternity. Anthropometric parameters at birth were: birth weight 1950 g, head circumference 31 cm, length 46 cm. She presented moderate caput succedaneum. Apgar scores were 5 at 1 minute, 6 at 5 minutes, and 8 at 10 minutes. The newborn required bag and mask ventilation for a short time. Initial cardio-respiratory adaptation was considered adequate, she was placed in incubator with oxygen supplementation as required. After 48 hours of life, the newborn was submitted in our institution (level III maternity) because of deterioration of the clinical status: tachypnea, apnea, pallor, jaundice, hyporeactivity, oliguria, swelling of the head.

On admission her vital signs were as follows: heart rate 90 beats/minute, mean blood pressure 28 mmHg, respiratory rate 50 breaths/minute on assisted ventilation, oxygen saturation 78% on FiO₂ of 100%, deep coma and bleeding at local punctions. On her head, a large, fluctuant swelling and extended echimosis were noted. The circumference of the head was 36,5 cm. (pictures 1,2)

Figures no. 1. and 2. Swelling and extended ecchymosis of the head



Blood gas analysis revealed an uncompensated metabolic acidosis (pH 6.90, pCO₂ 78 mmHg, pO₂ 32 mmHg, base deficit -22.1 mmol/l). Hemoglobin value was 2,9 g/dl, hematocrit 8,5%, platelets 28000/mm³, and liver and renal function were altered (ALT 115 U/l, total bilirubin 15,45 mg%, BUN 98 mg%, creatinine 1,92 mg%, albumine 2,47 g/dl, K 7,3 mmol/l). PT and PTT were prolonged.

Initial transfontanelar ultrasound examination showed difused hyperecogenity, reduced parenchimal differentiation, decreased Doppler values at the mean cerebral vessels, with RI= 0,47. (picture 3,4)

Serial head ultrasound showed intraventricular hemorrhage and multichistic lesions of the brain. (picture 5,6) A diagnosis of hemorrhagic shock secondary to a subgaleal hemorrhage was made.

Figure no. 3. Diffuse hyperecogenity



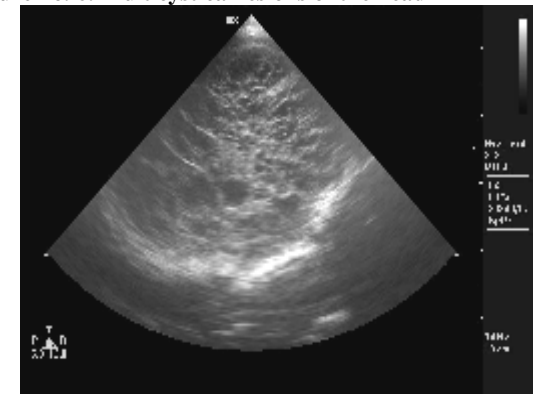
Figure no 4. Index of resistivity Doppler RI= 0,47



Figure no. 5. Intraventricular bilateral hemorrhage



Figure no. 6. Multicystical lesions of the head



After admission, our management was: temperature

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control (incubator with servocontrol), placement of umbilical vein catheter, assisted ventilation (IPPV).

Initially the newborn received 20 ml/kg bolus of crystalloid solution, later blood products (plasma, erythrocyte and platelet concentrates) and vitamin K. For maintenance of cardiac function and correction of severe low blood pressure, the infant received Dopamine (15 µ/kg/minute) and Dobutamine (10 µ/kg/minute). We tried to correct mixed acidosis with ventilation and sodium bicarbonate (8,4%). The hyperkalemia was corrected with i.v. 10% calcium gluconate, sodium bicarbonate and insulin administration. Despite full neonatal intensive care, the infant continued to deteriorate with severe encephalopathy and renal failure. She died at 12 days of age. Postmortem examination confirmed a massive subgaleal hemorrhage, subarahnoidian and intraventricular hemorrhage, hypoxic-ischemic changes of the brain, multichistic encephalomalacia, splenic and renal hemorrhage.

DISCUSSION AND CONCLUSIONS

The infants with subgaleal hemorrhage require early recognition and diagnosis, careful monitoring early and prompt treatment, to avoid morbidity and neonatal death. We believe that in our case subgaleal hemorrhage occurred after a moderate traumatic noninstrumental delivery. Unfortunately, the birth injury was underestimated. The diagnosis was delayed, so initial trauma was complicated with hypovolemic shock, renal failure, disseminated intravascular coagulation, and severe encephalopathy that lead to exitus, despite full neonatal intensive care and massive volume resuscitation.

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