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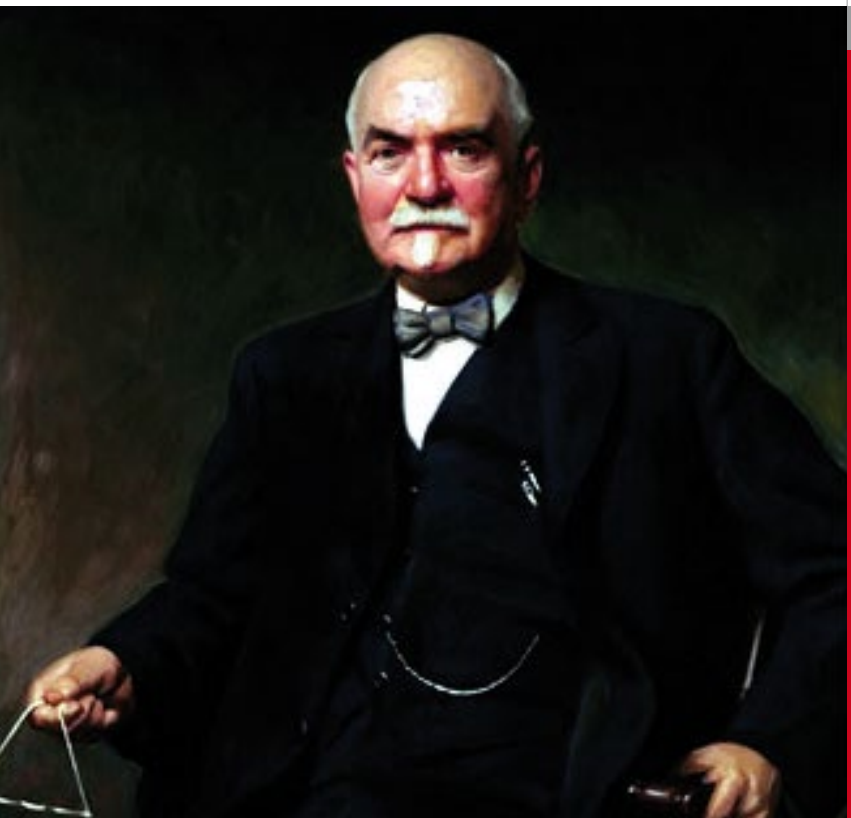
Severe anemia due to sub-capsular hepatic hematoma – an unexpected revelation



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After an uneventful pregnancy, a 26-year-old woman of African origin delivered a boy at 40 2/7 weeks of gestation weighing 3680 g. Induction of labor was prolonged over 48 hours. Apgar score was 9, 10, and 10 at 1, 5, and 10 minutes, respectively. Delivery was traumatic with subconjunctival hemorrhages and a cephalohematoma. In addition, the baby transiently showed decreased movements of the right arm. Oral vitamin K prophylaxis was administered by the age of 4 hours.

The boy was then transferred to the maternity ward. Two days later, the mother noticed that her baby seemed to be in pain and had an enlarged abdomen. Conservative measures failed to control what was interpreted as abdominal cramps. The baby was groaning and refused feedings. On the 4th day of life, when the newborn screening was performed, the boy appeared pale. Because the blood was reported to look very watery, a hematocrit was obtained and found to be 14 %. Vital signs were reported to be normal (heart rate and pulse quality), however, no blood pressure was measured. The baby was transferred by ambulance to the Neonatal Intensive Care Unit at our institution.

On admission, clinical examination revealed a pale boy with weak inguinal pulses. His heart rate was 160 bpm and his blood pressure was 61/34 mmHg (mean 47 mmHg). There was a 2/6 non-specific systolic heart murmur. He had a left-sided parieto-occipital cephal-

hematoma measuring 3 cm in diameter. His abdomen was distended and painful. The liver appeared to be slightly enlarged extending to about 4 cm below the costal margin. There was a small bruise just above the umbilicus, interpreted as being a positive Cullen's sign (Fig. 1).

A CBC showed hemoglobin of 31 g/l and a hematocrit of 9.9 %. The erythrocyte count was $0.98x10^6$/µl with normal indices, and the reticulocyte count was normal, but with an elevated HFR (high fluorescence ratio) percentage of 27%. The platelets, the white cells count as well as coagulation screen were unremarkable. There was a significant metabolic acidosis (pH 7.12) with a BE of -15.8 mmol/l and an elevated lactate of 18 mmol/l. Blood types of mother and baby were A positive, and a direct Coombs' test was negative. The total serum bilirubin was 139 µmol/l. Liver enzymes were normal and LDH was elevated to 1142 U/l.

A total volume of 40 ml/kg of packed red blood cells was transfused in two aliquots over 4 hours each. The transfusions were well tolerated with a rise in hematocrit to 38% and rapid normalization of the lactic acidosis (BE -1.4 mmol/l, lactate 1.8 mmol/l).

Abdominal, cerebral and cardiac ultrasonographies were performed. The cerebral ultrasound was normal and the echocardiogram showed a normal anatomy and ventricular function with no signs of cardiac failure.

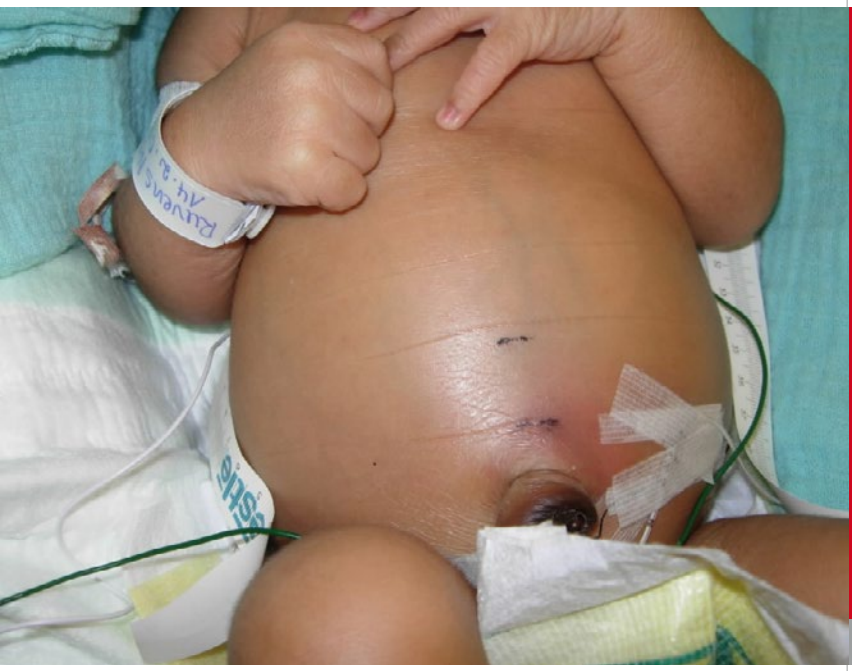

Fig. 1

Distend abdomen with positive Cullen's sign.

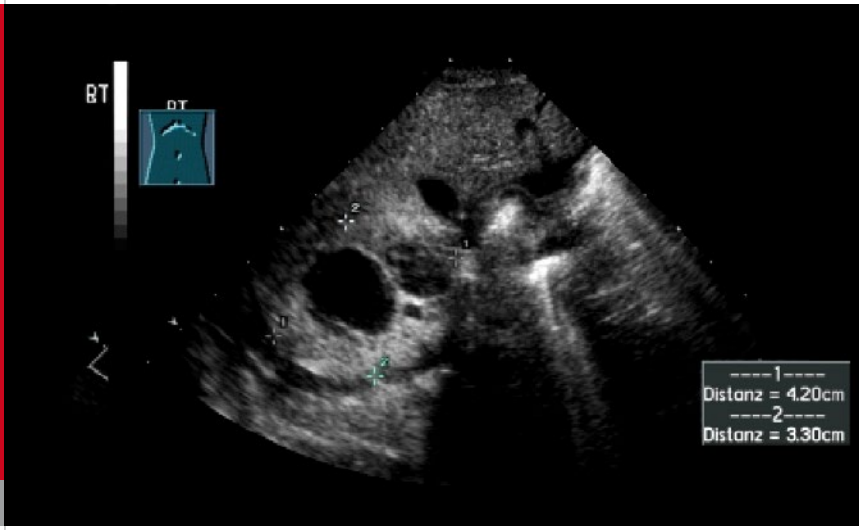

Fig. 2

Ultrasonsgraphy of the abdomen: solid and cystic hepatic lesion measuring 5 by 3 cm in size.

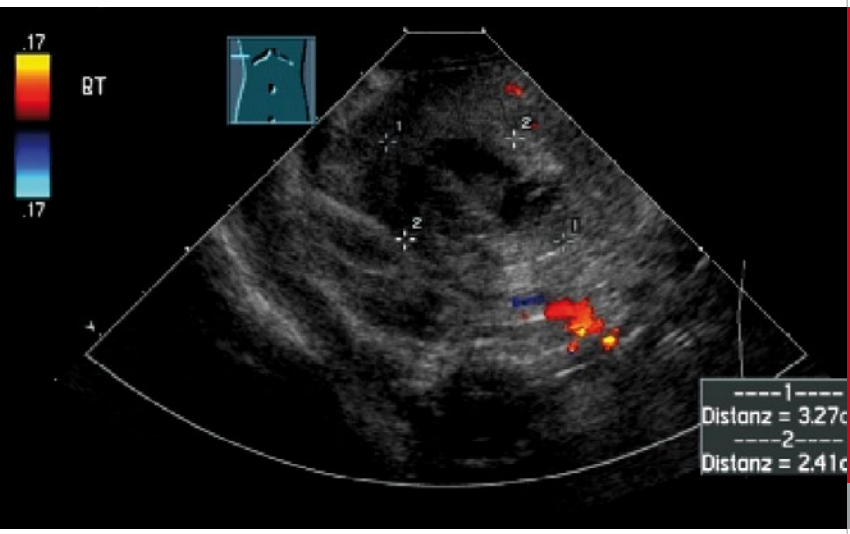

Fig. 3

Ultrasonsgraphy of the abdomen: Color Doppler study showing no signs of perfusion of the lesion.

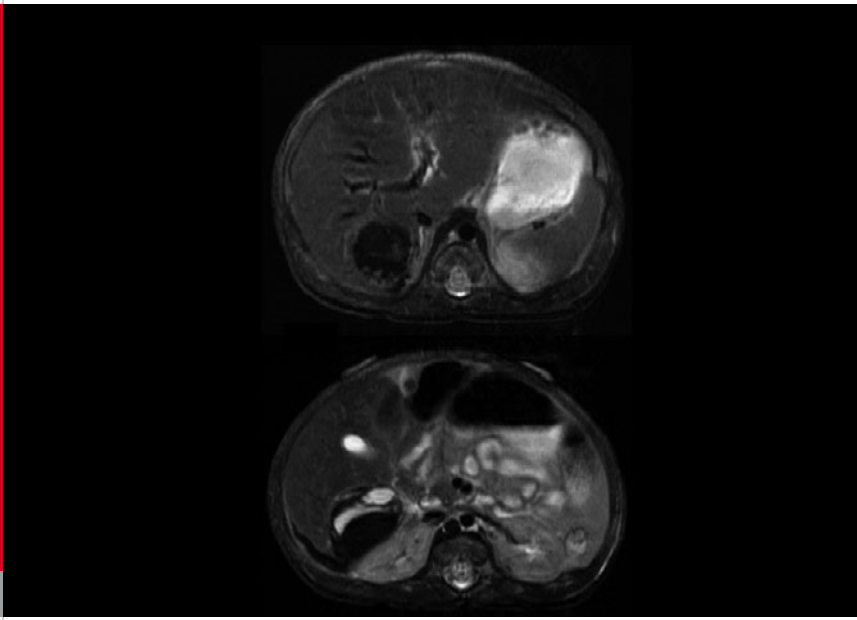

Fig. 4

T2-weighted axial MRI of the abdomen showing the posteriorly located hepatic lesion and hematoascites.

Abdominal ultrasonography revealed a partially cystic, partially solid structure measuring 5 by 3 cm in size within the liver, located posteriorly, very close to the surface (Fig. 2). On color Doppler, there were no visible vascular structures within the lesion (Fig. 3). Spleen, kidneys and adrenal glands were normal. There was a significant amount of free fluid interpreted as hematoascites due to its non-echo-free appearance. The cystic structure was interpreted as a hepatic subcapsular hematoma.

The further course was uneventful with stable hematocrit and normalization of the LDH. On the 7th day of life, an MRI of the abdomen was performed that confirmed a partially cystic, partially solid non-enhancing lesion within the liver (Fig. 4). The capsule of the liver was displaced by the lesion but not infiltrated. There was fluid of intermediate intensity in the subcapsular region as well as in Morrison's pouch and in the paracolic grooves. These findings were interpreted as blood degradation products, consistent with status post subcapsular and intraabdominal hemorrhage. Hepatic tumors (e.g., hamartoma, lymphangioma), however, could not be excluded. Follow-up ultrasound examination showed regression of the lesion and disappearance of free intraabdominal fluid.

The infant was discharged on the 9th day of life. Ultrasound follow-up examinations were performed regularly until complete disappearance of the pathological

findings. The boy is now one year of age and doing remarkably well with normal development (Fig. 5).

DISCUSSION

We were confronted with a baby on the 4th day of life with pre-shock and severe anemia. The main differential diagnoses of anemia were: diminished production, hemolysis, or bleeding, and could have been of acute, subacute or chronic onset. Significant hemolysis was unlikely since bilirubin levels were not elevated. Diminished blood production also was unlikely. A very low hematocrit with a moderately elevated reticulocyte count (mostly very young reticulocytes) made a subacute hemorrhagic event most likely. Blood loss may have started a few days earlier, probably during delivery.

The ultrasongraphic finding of an intrahepatic tumor has a broad differential: subcapsular hematoma, hemangioma, mesenchymal hamartoma, hepatoblastoma, or metastasis of a neuroblastoma. In our case, lack of vascularity of the lesion and the presence of hematoascites were felt to be consistent with spontaneous rupture of a subcapsular hepatic hematoma. Since patients with subcapsular hematomas frequently have cerebral germinal matrix hemorrhages (1), a cerebral ultrasound was performed showing normal anatomy and no bleeding.

Fig. 5

The patient is doing well at one year of age.

Subcapsular hematomas are rarely suspected and diagnosed clinically. Most diagnoses of subcapsular hematomas are made at autopsy. The frequency of subcapsular hepatic hematomas in the newborn ranges from 2.3 % (2) to 6.9 % and even up to 15% (3). First reports were by Hodge and date back to 1870.

Subcapsular hematomas are usually found in term infants and are most likely due to trauma at birth. Compression of the fetal liver during passage through the maternal pelvis is possible, as noted by Mangurten (4). LGA and breech delivery put the baby at a particularly high risk. In recent years, this entity has also been reported in preterm and SGA infants. Possible causes are complications of umbilical vein catheterization (5) or sepsis (1) with group B streptococcus being the most frequent organism isolated. Clotting disorders as well as thrombocytopenia may be other contributing factors. Cerebral germinal matrix hemorrhage is frequently associated and needs to be excluded.

Management consists of hemodynamic stabilization, correction of any coagulation abnormalities and close monitoring. Once the liver capsule ruptures, the baby is in great danger of live threatening hemorrhage. If bleeding is stopped by these measures, conservative therapy can be continued. Surgery should be reserved for cases in which medical treatment fails (6).

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