Vascular liver calcification in infants

A case report with some pathogenetic considerations

G. de Filippi¹ and P.G. Betta²

¹Department of Paediatric Radiology, Ospedale Infantile and ²Department of Pathology, Ospedale Civile, Alessandria, Italy

Abstract. A case of calcified portal vein thromboemboli in a neonate is described with special regard to the radiological and histological features. A possible pathogenetic mechanism involves a disseminated intravascular coagulation subsequent to placental release of thromboplastin.

Case report

A male infant was born at 28 weeks of gestation to a 38-year-old gravida IV, para 3, abortus 1, Rh-positive woman following premature onset of labour. Only one 8-year-old daughter was still alive, but was spastic and affected by retarded psychomotor development.

The pregnancy was unremarkable, except for a slight uterine haemorrhage at the third month. The parental histories were negative for smoking, exposure to drugs, or family history of cardiac, chromosomal, or congenital anomalies. Delivery was spontaneous 3 h following amnion rupture. The placenta and membranes were expelled simultaneously, the former in pieces and featuring patchy areas of infarct and necrosis. The amniotic fluid was dark.

The infant appeared in shock from birth, and suffering from diffuse cyanosis and severe dyspnea. Apgar scores were 0 and 4 at 1

Fig. 1. Plain film of the abdomen. Multiple linear and hieroglyphic-like peripheral calcifications in the right lobe of the liver

Fig. 2. Longitudinal sonogram. Multiple dense echoes due to calcifications along the hepatic surface
and 5 min, respectively. Oropharynx suction were performed repeatedly and assisted ventilation was started. Palpation revealed the liver to be enlarged. A chest X-ray showed the features of hyaline membrane disease. On abdominal radiography linear and hieroglyphic-like calcifications in the subcapsular segment of the right lobe of the liver were observed. They were scattered but with an overall semicircular distribution (Fig. 1). Ultrasonography confirmed their peripheral location (Fig. 2). Two hours after birth the infant was transferred to the intensive care unit and subjected to assisted ventilation with endotracheal intubation. Barbiturates, corticosteroids, and dopamine were also administered. The platelet count was $12 \times 10^9/l$ of blood. Positive cultures for *Pyocyaneus* were obtained from a sample of suction material. Thirteen hours after birth a first heart arrest occurred followed by a temporary recovery. A subsequent aggravation of the cardiopulmonary malfunction led the infant to death at 36 h of age.

The autopsy revealed both the lungs to be acutely congested, totally airless and of liver-like consistency on section, and on microscopic examination the dilated respiratory bronchioles were lined with well-developed hyaline membranes and the terminal sacs were congested and collapsed. Radiographs of the liver before dissection provided further evidence of the peripheral location of the calcifications (Fig. 3). On gross inspection of the cut surface of the liver multiple nodules and branching lines of a chalky-yellow colour were seen just beneath some shallow depressions of the capsule of the right lobe. They averaged 1 to 2 mm in width and 2 to 6 mm in length. Histologically these lesions corresponded to totally calcified thrombi of the subcapsular branches of the portal vein associated with ischaemic necrosis predominantly midzonal and centrilobular in distribution (Fig. 4). In a few portal tracts more or less confluent tiny calcifications were scattered. Recent fibrin thrombi were found in the main branches of the splenic artery. No evidence of congenital abnormalities or foetal infections was seen.

### Discussion

The most frequent cause of hepatic calcification in the perinatal period is meconium peritonitis, although peritoneal liver involvement is not constant within the typical picture of diffuse intra-abdominal calcification. Plastic peritonitis caused by a ruptured hydrometrocolpos shares the same radiological features.

Parenchymal hepatic calcifications have been reported as a nonspecific change in congenital infections, such as cytomegalovirus infection, rubella, toxoplasmosis and herpes simplex. In these cases, however, calcifications usually involve multiple organs and are frequently associated with congenital abnormalities. Parenchymal calcification may also be found in primary and secondary liver malignancies.

Vascular liver calcifications in the infant are seen in portal-vein thromboembolism [1], in which they are peripherally located, and vascular insufficiency with ischaemic infarcts [2], which exhibits a diffuse involvement of the liver.

The present case showed both radiological and microscopical evidence of peripheral distribution of hepatic calcifications involving the subcapsular and interlobular branches of the portal-vein. Thus these findings were consistent with the typical morphological appearances of portal-vein thromboembolism.

Coexisting hyaline membrane disease does not seem incidental since its occurrence has been previ-