RUPTURE OF THE LIVER IN THE NEWBORN*

BY

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Rupture of the liver is a rare condition in newborn infants and most cases are diagnosed at autopsy. Only 11 instances of survival have so far been recorded, by Rogers (1934), Arden (1946, 1951), Potter (1952), Arden, Dique and Wrench (1955), Bret, Jamain and Coupé (1956), Greaves (1955), Mason Brown (1957) and Dennison, cited by Mason Brown. It therefore seems worth recording that we have encountered two cases of this condition within a period of four months. The first case was successfully treated by operation and survived, while the second died two days after operation from extensive subdural haemorrhage.

Case Histories

Case 1. (P.T.) This female infant was born spontaneously. The mother's pregnancy and labour had been uneventful. The child's birth weight was 7 lb. 6 oz. She was perfectly well during the first three days of life. At 80 hours of age, the infant suddenly turned pale and limp after being put to the breast. She was transferred to the surgical unit of the Children's Hospital at Bremen.

She was extremely pale and her breathing was shallow and irregular. One hour later the abdomen became distended. Blood picture: haemoglobin, 62%; erythrocytes, 2.8 million; leucocytes, 22,000; Coombs test negative. A diagnosis of intraperitoneal haemorrhage was made and the baby was given a transfusion of blood (200 ml) preoperatively.

At operation a large quantity of fluid blood escaped from the peritoneal cavity and was found to be coming from a tear about half an inch long near the lower border of the right lobe of the liver (Figs. 1a and b). The wound was sutured with chromic catgut.

During the 24 hours after operation the child vomited small amounts of bile-stained material. She was treated by intravenous infusion and tetracycline. She then made steady progress until 1 month of age when she was discharged home. She has since been followed up and remains well.

Case 2. (S.K.) This male infant was born spontaneously after an uneventful pregnancy and labour, weighing 8 lb. 11 oz. At birth the child was asphyxiated and pale, but spontaneous breathing started after insufflation of oxygen. There were carpopedal spasms and flexion of the wrists. On the first and second day of life the child vomited small amounts of haematin-stained fluid. On the third day he suddenly became very pale, collapsed, and was transferred to our care.

On admission the child was moribund with a markedly distended abdomen. The liver was palpable about one inch below the costal margin. Blood picture: haemoglobin, 78%; erythrocytes, 3.7 million; leucocytes, 22,000; Coombs test negative. The diagnosis of intraperitoneal haemorrhage was made, and, because of the experience and knowledge gained in connexion with Case 1, a rupture of the liver was suspected. The child's condition improved after a transfusion of blood (200 ml) and six hours after the onset of symptoms it was decided to operate.

At operation a large quantity of blood escaped from the liver region. A tear one inch long was found in the midst of a large subcapsular haematoma on the superior surface of the right lobe of the liver. The tear was repaired by inserting three stitches of chromic catgut.

The child stood the operation quite well, but postoperatively his condition deteriorated. He was treated by intravenous fluids, tetracycline, vitamin K and

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AC 76. He had minor convulsions and attacks of respiratory distress and two days after operation he died.

Autopsy. The body was that of a mature male neonate, 6 days of age, weight 8 lb. 5 oz., length 20 inches. Within the abdominal cavity was about 5 ml. of fluid blood. A large subcapsular haematoma was found on the superior surface of the right lobe of the liver. There was a tear (one inch long) near the distal margin of the right lobe. The site of the suture was intact. Another haematoma, the size of a half-crown, was found on the caudate lobe (Figs. 2 and 3). The dorsal wall of the stomach showed extensive haemorrhagic infarction of the mucosal and muscular layers. There were no abnormalities in the spleen. Both the kidneys showed a dark blue-reddish infarction of the medullary substance and papillae. There were perivascular haemorrhages and autolysis in the suprarenal glands, and bronchopneumonia and atelectases in the lungs. A cephalo-haematoma was found in the skull and there was a large subdural haematoma extending over the parietal and temporal lobe of the left cerebral hemisphere. Slight oedema of the brain was present. The anatomical diagnosis was: subcapsular haematoma of the liver with repaired rupture; haemorrhagic infarction of the stomach, both kidneys and suprarenal glands; cephalo-haematoma; subdural haematoma; and bronchopneumonia. The cause of death was subdural haematoma.

Discussion

Hodge (1870) was probably the first to report a case of fatal haemorrhage from the liver in an infant 2\frac{1}{2} days old. The question of trauma as an aetiological factor in the causation of hepatic haemorrhage has been discussed fully in the literature. Dittrich (1895) asserts that he had never known of a liver rupture without some external trauma. At that time the medico-legal aspect of the condition was widely discussed. Hedrén (1917) was the first to doubt if trauma was an aetiological factor in all cases of this condition, and he stated that hepatic rupture may be caused by physiological birth trauma.

It is impossible to discuss in full the series of events by which rupture of the liver may be brought about. Many theories have been advanced regarding the underlying cause, and the following includes some of the leading views. Gruenwald (1948) has described three possible mechanisms which may cause injury to the liver: (1) By pressure on the thorax the liver is pushed downward out of the hollow of the diaphragm which causes tension on the ligaments. He reported three cases in which the lesion was located at the coronary ligament and the hepato-duodenal ligament of the liver. (2) In cases where the lesion is found on the anterior surface of the liver, this may be caused by direct pressure of the costal margin. (3) Injuries which have no direct relationship to the ligaments or costal margin may be due to direct trauma to the liver itself.

Genell (1930) postulated that hepatic rupture may be caused by bending and compression of the foetal body. He reported a case of a woman with an oblique narrowing of the pelvis, who gave birth to two infants with hepatic rupture. Bret et al. (1956) collected 116 cases of hepatic haemorrhage from the literature and found a higher incidence of the condition in premature and in infants larger than average. Henderson (1941) postulated that difficult delivery and abnormal foetal positions are the principal causes of hepatic haemorrhage in newborn infants.

In our two cases none of the above-mentioned
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factors was present. Labour and delivery had been quite straightforward and the birth weights were normal.

Other factors which may play a part in the aetiology of this condition are asphyxia and increased bleeding tendency. Henderson (1941) in his series of 47 cases of hepatic haemorrhage found 35 cases which were severely asphyxiated at birth. Bělohradský (1937) and Arden (1946) implicated severe congestion of the liver in asphyxia as predisposing to rupture. The frequency of associated haemorrhages other than hepatic speaks in favour of an increased bleeding tendency, such as is present in haemorrhagic disease of the newborn. In Henderson's series 43% had associated haemorrhages. Our Case 2 showed multiple haemorrhages in other organs. Unfortunately the infant's poor condition gave no opportunity for laboratory investigations such as prothrombin estimations, determination of the bleeding time, clotting time and platelet count. During the first 48 to 72 hours of life the prothrombin and stable factor level fall sharply (Schulman and Currimbhoy, 1957). In most instances of liver rupture there is a symptom-free interval of one to seven days. This may be explained as the result of a two-stage rupture. During the first stage a haematoma arises from a contusion of the liver parenchyma, and, as all newborn infants are born with a moderate deficiency of both prothrombin and stable factor, a haematoma may arise quite easily from a trauma which otherwise has no consequences. In autopsies of newborn infants who died from other causes, a haematoma of the liver is quite often found as a secondary finding. After this symptom-free interval, when the prothrombin and stable factor deficiency is greatest, bleeding may start again with following rupture of the haematoma. In only rare instances the capsule may be torn by the original trauma and in these cases the infant dies in utero or immediately after birth.

It seems conceivable that hepatic haemorrhage, with and without following rupture, belongs in the group of manifestations of haemorrhagic disease of the newborn. The symptomatology of the condition is quite characteristic. There is a sudden onset of signs of severe illness after a symptom-free interval of one to seven days. According to Bret et al. (1956) most ruptures occur on the third or fourth day of life. Stingl (1948) and Lundquist (1930) reported cases in which the rupture occurred on the seventh day. After this lapse of time, when the baby seems to be in perfect health, there is a sudden appearance of severe anaemia with pallor and collapse. Abdominal distension, dullness to percussion in the right flank, and hepatomegaly are also found in most cases.

The following conditions may give the same clinical picture: (1) Haemorrhage of the suprarenal gland, which is usually associated with an extreme degree of pyrexia, a palpable mass in the loin, polypnorea and convulsions. (2) Congenital haemolytic anaemia, which can be excluded by a negative direct Coombs test. (3) Rupture of the spleen, which according to Gruenwald (1948) is much less common and only occurs in luetic and leukaemic spleens. (4) Intraventricular haemorrhage, which is associated with hypothermia and neurological symptoms.

Rogers (1934) recommends abdominal paracentesis to confirm the diagnosis.

The first case which recovered after surgical treatment was recorded by Rogers (1934). Arden (1951) and Arden et al. (1955) reported three cases which were treated by transfusion of blood only and recovered. Montalvo, Soto Pradera, Fusté, Rodriguez Molina, Diaz Rousselet, Alfonso, Silverio and Portela (1955) in their series of 24 cases had four infants in whom internal haemorrhage was diagnosed clinically; the children were treated by blood transfusion only and all of them died.

Because the extent of the tear and the amount of bleeding cannot be determined clinically, we feel that operation is the treatment of choice and should be carried out without delay as soon as the diagnosis of intraperitoneal haemorrhage is made.

Summary

Two cases of liver rupture during the neonatal period are described, one of which survived following operation. The aetiology, symptomatology and treatment are discussed and the literature briefly reviewed.

References

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