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Rupture of the spleen in erythroblastosis fetalis

Rupture of the spleen in the newborn is a rare occurrence. Giedion (1963), reviewing this problem, could find only 25 published cases, and subsequently Leape and Bordy (1971) collected reports on only 18 neonates successfully treated for rupture of the spleen. Breech presentation or difficult vertex delivery has been held responsible for this injury in some cases, but more often no specific type or complication of delivery can be incriminated. Though there are more reports of rupture of normal spleen, it is well known that a pathologically enlarged spleen, usually due to erythroblastosis or in the past to syphilis (Lundquist, 1930), can rupture. Sirola (1967) has reported the only successfully treated newborn infant who had erythroblastosis, splenic rupture, and successful management including splenectomy. The following case is reported as representing a further example of an infant with similar pathology who has survived.

Case report

A male infant was born by spontaneous vertex delivery at 36 weeks' gestation after induction of labour because of severe rhesus isoimmunization. The mother, a gravida 2 para 1 and abortus 1, was group 0 rhesus negative $\bar{c}de/\bar{c}de$. There had been a marked rise in antibody titre to anti C+D from 29 to 36 weeks' gestation, the titre in saline and indirect antiglobulin test reaching 1/512. The optical density difference of the amniotic fluid had risen from lower zone II of Liley's curve at 31 weeks to upper zone II at 34 weeks.

The infant weighed 3080 g and his general condition was satisfactory with an Apgar score of 4 at 1 minute and 9 at 5 minutes. The liver was not enlarged and the spleen was just palpable. A left hydrocele was noted. The cord blood was group 0 rhesus positive, direct antiglobulin test positive, antibody titre by indirect antiglobulin test 1/128 for anti C+D, and serum bilirubin 6·8 mg/100 ml. Capillary haemoglobin was 12·0 g/dl, packed cell volume 0·37, and platelet count 192 000/mm^3 with normal RBC morphology and thrombotest 100%.

At 2½ hours the infant's condition deteriorated with the development of circulatory failure. Immediate exchange transfusion was followed by improvement in the infant's condition. The haematological data are given (see Table). Phenobarbitone and phototherapy were started. At 14 hours petechiae on the trunk were noted, the abdomen was slightly distended, and the left hydrocele felt more firm. Hypoglycaemia was treated and a second exchange transfusion was carried out at 24 hours to correct the anaemia and remove antibodies. By 48 hours the haemoglobin had fallen again to

TABLE

Haematological and biochemical data

	Age (h)	Fragmentation of RBC	Hb (g/dl)	Platelets (per mm^3)	Coagulation factors	FDP (mg/ml)	Serum bilirubin (mg/100 ml)	True blood glucose (mg/100 ml)
Cord Blood	Birth	0	12·0 (capillary sample)	192 000	—	—	6·8	—
1st exchange transfusion								
Pre	3	+	9·7	100 000	I, V, VIII ↓	>40 <80	6·9	292
Post		0	14·3	75 000	VIII ↓	$>$*40* <80	5·0	156
	14	—	—	—	—	—	—	2
	18	+	9·5	57 000	—	—	6·9	—
2nd exchange transfusion								
Pre	24	+	6·5	45 000	I±VII ↓	$>$*160*<320	5·8	270
Post		0	13·6	49 000	Normal	$>$*40* <80	4·7	62
	40	0	8·1	58 000	—	—	6·7	—
	48	±	7·2	90 000	I, VIII±VII ↓	—	—	—

RBC, red blood cells; FDP, fibrin degradation products.

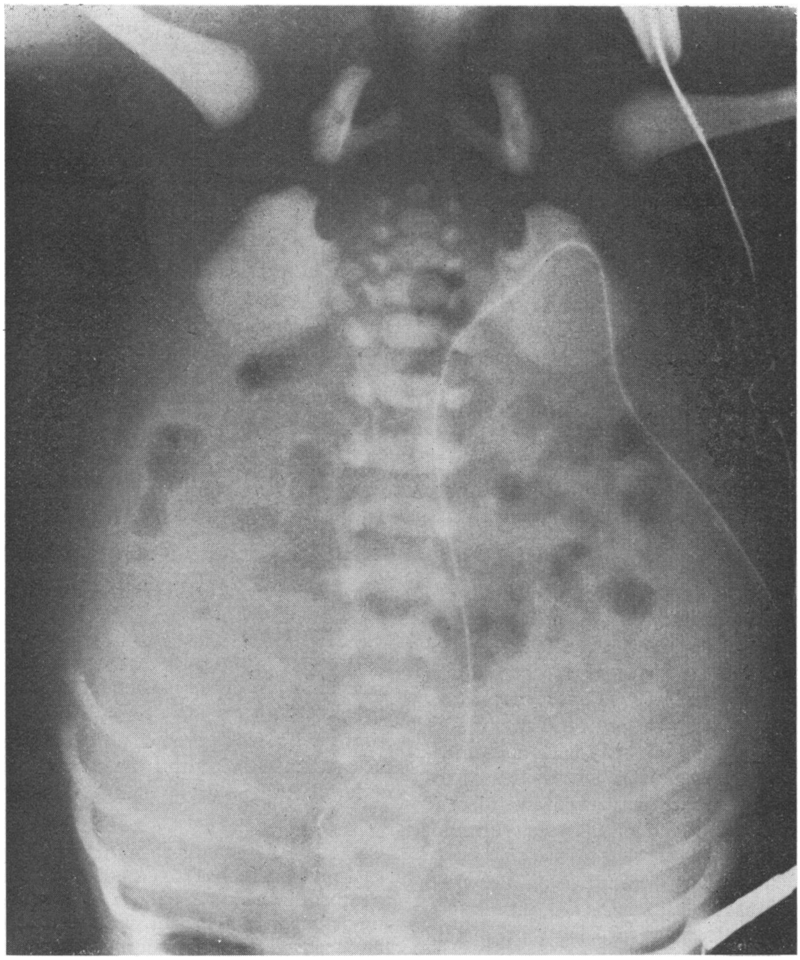

FIG.—*Opacity in left hypochondrium, displacement of stomach to the right with angulation of the nasogastric tube.*

7·2 g/dl, but the bilirubin remained low at 6·7 mg/100 ml. The presence of haemoperitoneum was confirmed by an abdominal paracentesis in the left lower quadrant. *X*-ray of the abdomen (see Fig.) showed displacement of the stomach and the bleeding was presumed to come from a ruptured spleen. A transfusion of 50 ml partially packed cells was given and the baby transferred to the Royal Hospital for Sick Children. Laparotomy revealed a large volume of blood in the peritoneal cavity originating from the spleen, the capsule of which was torn in the region of the spleno-lieno-renal ligament. Also there was a large left suprarenal haemorrhage displacing the kidney and a smaller right suprarenal haemorrhage. Splenectomy was performed and postoperative progress was satisfactory. Plasma cortisol levels were normal, as was the response to Synacthen. The spleen weighed 14 g. Histologically it showed distended sinusoids which contained erythroblasts and focal subcapsular haemorrhage with at one point rupture through the capsule. At 3 weeks of age a pyelogram showed that the left kidney was still displaced, presumably secondary to the adrenal haemorrhage. The infant has progressed normally since discharge from hospital. In view of the increased susceptibility of the splenectomized infant to severe infections (Diamond, 1969), prophylactic penicillin has been continued.

Discussion

Early recognition of this uncommon complication of erythroblastosis with institution of appropriate treatment is essential to a successful outcome. In the Royal Maternity Hospital, Glasgow, there have been only two other cases of ruptured spleen in the last 23 years, both had erythroblastosis and both died, and at the Royal Hospital for Sick Children no case of perinatal splenic rupture has been admitted in the last 14 years.

Diagnosis is not easy in the infant who has had exchange transfusion as some abdominal distension is common after such a procedure. The diagnosis of haemoperitoneum in this patient was confirmed by paracentesis. Abdominal tap is of value when positive, but if negative is not always reliable. In this case the unusual feature was the low serum bilirubin even considering the effect of phototherapy. Presumably the large amount of the infant's blood which was in the peritoneal cavity was unavailable for breakdown and conversion to bilirubin.

The thrombocytopenia, fragmentation of RBC, consumption of factors, particularly I, V, VIII, and later probably VII, together with the release of fibrin degradation products (FDPs) was most likely due to the local stimulation of coagulation from thromboplastins released from the ruptured spleen. Though causing a further fall in platelets, the exchange transfusion with ACD blood seemed to halt the consumption of factors and to lower the level of FDPs. There may also have been an element of disseminated intravascular coagulation (Chessells and Wigglesworth, 1971) but hypotension and local coagulation were probably the major factors in this case. The coagulopathy with consumption of platelets and coagulation factors was probably responsible for the continuation of the haemorrhage, initiating a vicious circle (Digilio, Bacchetta, and Ferreri, 1960).

In the present case it was difficult to define when the suprarenal haemorrhages occurred. The hypotension and coagulopathy were probably important contributory factors.

Summary

A male infant of 36 weeks' gestation, weighing 3080 g, with erythroblastosis, ruptured spleen, and bilateral suprarenal haemorrhages is described. The infant survived after exchange transfusions and splenectomy.

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Hypertension, oedema, and suppressed renin aldosterone system due to unsupervised salt administration

Hypernatraemia is recognized as a complication of incorrectly prepared hyperosmolar infant feeds, especially in the presence of acute diarrhoeal disease (Taitz and Byers, 1972). We report an infant who was given a glucose-salt-water mixture on medical advice and developed hypertension and oedema after being given an excess of salt.

Case report

A previously healthy, bottle-fed Asian girl, aged 3 months, developed diarrhoea. After 3 days the parents sought medical help and were advised to give her clear fluid feeds prepared by adding 5 teaspoons of glucose and 1 saltspoon of salt to 600 ml (20 fl oz) water. Having no saltspoon in the house, they prepared each feed by adding 2 teaspoons of glucose and 5 pinches of salt to 240 ml (8 fl oz) water. The baby drank eagerly one litre of the glucose saline solution every 24 hours. Urine output appeared normal and she had 5 loose stools a day. During the next 3 days she developed swelling of the face, abdomen, and legs, and was admitted to The Hospital for Sick Children.

She had gained 450 g in weight, had facial and leg oedema, and her blood pressure was 140/80 mmHg. Investigations showed plasma sodium 144 mEq/l, potassium 3·6 mEg/l, urea 5 mg/100 ml, urine osmolality 66 mOsm/l, and 24-hour urine sodium 20 mEq. Plasma creatinine and albumin were normal. Plasma renin activity (PRA) was subnormal on admission (55 pgAI/ml per h) and was associated with a low plasma aldosterone concentration (PAldo) of 11·9 ng/100 ml. Mean values for children of this age are 1392 (range 472-3130) pgAI/ml per h and 20·2 (range 8·3-75) ng/100 ml, respectively (Dillon and Ryness, 1974).

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