SPONTANEOUS NEONATAL HAEMOTHORAX — A REPORT OF TWO CASES

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SYNOPSIS

Two cases of spontaneous haemothorax in the neonate are reported. In one of the cases, differentiation from a diaphragmatic hernia could not be easily made on the basis of the clinical examination and a plain x-ray film. The cause of haemothorax could not be determined with certainty, however vitamin K-dependent hypoprothrombinemia (haemorrhagic disease of the new born) could be an aetiologic factor. The treatment included thoracocentesis, blood transfusion for anaemia and vitamin K administration for hypoprothrombinemia.

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INTRODUCTION

Pleural effusion is a rare cause of acute respiratory distress in the neonate. Chylothorax is the commonest form of pleural effusion seen; it can be congenital[1-4] or iatrogenic following thoracic surgery.[5, 6] A prenatal pneumonitis[7] or a granilomatous infection such as tuberculosis[8] could also give rise to a pleural effusion. Haemothorax is the rarest form of pleural effusion in the neonate.

Two cases who were admitted to the Paediatric Unit of the alor Setar General Hospital are reported.

Case 1

A five-day-old Malay female neonate was admitted to the Paediatric Unit on the 19.11.1986 with a one-day history of breathlessness. She was the product of a normal vaginal hospital delivery. The Apgar Score at one and five minutes were nine and ten respectively. She was a full-term baby with a birth weight of 3150g. She was put to the breast four hours after birth and since then, was exclusively breast-fed. The baby passed urine and meconium on the first day of life. She was found to be normal on routine examination and was discharged from the hospital on the second day of life. Before discharge, she was immunized with BCG; however she was not given vitamin K injection.

She was well until one day before admission, she developed sudden onset of breathlessness and excessive sweating while being breast-fed. Following that, the breathlessness persisted and she was unable to suck. However, the baby did not turn blue. The following morning, a domiciliary nurse was called to see the baby in the house and she advised admission to the hospital.

There was no associated umbilical bleeding, rectal bleeding, haematemesis or bruising of the skin. The parents denied any history of trauma. The pregnancy was uneventful. There was no history of ingestion of anti coagulants or anti convulsants by the mother during pregnancy or confinement. The patient is the only child and there is no family history of a bleeding disroder.

On admission, she was afebrile. She had pallor. She was tachypnoeic with a respiratory rate of 80 minute, there were intercostal and subcostal recessions. The left hemithorax was noted to be 'hyperexpanded' and there were diminished chest movements on this side. There was dullness on percussion and reduced breath sounds on auscultation of the left hemithorax. The apex beat was prominently heard on the right chest and the rate was 140 per minute. There were no cardiac murmurs. The abdomen was noted to be 'scaphoid', the liver was palpable 1.5cm below the costal margin on the right. The spleen was not felt. Other systems were normal.

A provisional disgnosis of a left diaphragmatic hernia was made.

The results of the investigations done on admission are as follows:

Haemoglobin 9.4g%; total white blood cell count 14000 per mm3 (with a differential count of 42% polymorphs and 53% lymphocytes); platelet count 280,000 per mm3; blood urea nitrogen 11mg%; serum sodium 132 mmol per 1; serum potassium 4.6 mmol per 1; serum chloride 98 mmol per 1 and prothrombin time 23 s (control 15 s). Arterial blood gas analysis revealed a pH of 7.255, carbon dioxide tension of 37.7 mmHg, oxygen tension of 62.0 mmHg and an oxygen saturation of 87.5%. A portable chest x-ray film showed opacification of the left hemithorax with a few radiolucent areas in the left mid-zone. The mediastinum was displaced to the right (Fig. 1).

An umbilical venous drip was set up and oxygen was given via a head-box at the rate of 2 per minute. Three mgs of vitamin K was given intravenously after blood had been taken for the investigation of the prothrombin time (PT). Intravenous penicillin and intramuscular gentamycin were commenced. She was transfused with 60 mls of fresh blood. Soon after admission, the baby became increasingly tachypnoeic and had to intubated and ventilated in the Intensive Care Unit. Four hours after the administration of vitamin K, the PT was 17s (control 15s).

It could not be ascertained from the clinical and radiological findings as to whether the patient indeed had a left diaphragmatic hernia. A radiological contrast study of the upper gastrointestinal tract using diatrizoate melglumine (Gastrografin) was done as an emergency procedure. It showed that the radiolucent areas visualised in the initial plain x-ray film were not loops of bowel (Fig. 2). A diagnostic pleural tap was done and fresh blood was aspirated. A chest tube was inserted and 70mls of blood were drained from the left pleural cavity.

Meanwhile repeat investigations showed a haemoglobin concentration of 8.0g%, platelet count of 380,000 per mm3,

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blood urea nitrogen of 8mg%, serum sodium of 138 mmol per 1, serum potassium of 2.3 mmol per 1, serum chloride of 103 mmol per 1 and PT of 18s (control 15s). The bleeding time was 3 minutes and the clotting time was 6 minutes. Another 3 mgs of vitamin K was given intravenously and 60mls of fresh blood were transfused. Potassium chloride was added into the drip chamber in view of the low serum potassium level.

Following the drainage of blood, the baby's general condition improved. After 48 hours, she was weaned off the ventilator and the chest tube was removed. Oral feeding was started. A repeat chest x-ray film was normal. The PT and the activated partial thromboplastine time, which were done on the 7th hospital day (11th day of life) were normal. She was discharged woll on the 13th day of life.

CASE TWO

A four-day-old Malay male infant was referred to the Paediatric Unit on the 3.3.1987 with a history of respiratory difficulty and jaundice for one day. He was delivered at home by a government midwife. It was a normal vaginal delivery. The baby was notised to have cried immediately after birth; the Apgar score was not noted. He was a full-term baby with a birth weight of 7 pounds. He did not receive vitamin K. Since birth, he had been exclusively breast-fed.

He was apparently well till the day of his admission to hospital. On that morning, he developed sudden respiratory difficulty, following which he sucked poorly and ceased to be active. Jaundice was noted by the parents too. There was no associated umbilical or rectal bleeding, haematemesis or skin bruising. He was brought to see an Obstetrician at a Private Maternity Centre. The Obstetrician referred the baby to the hospital for further management. There was no history of trauma.

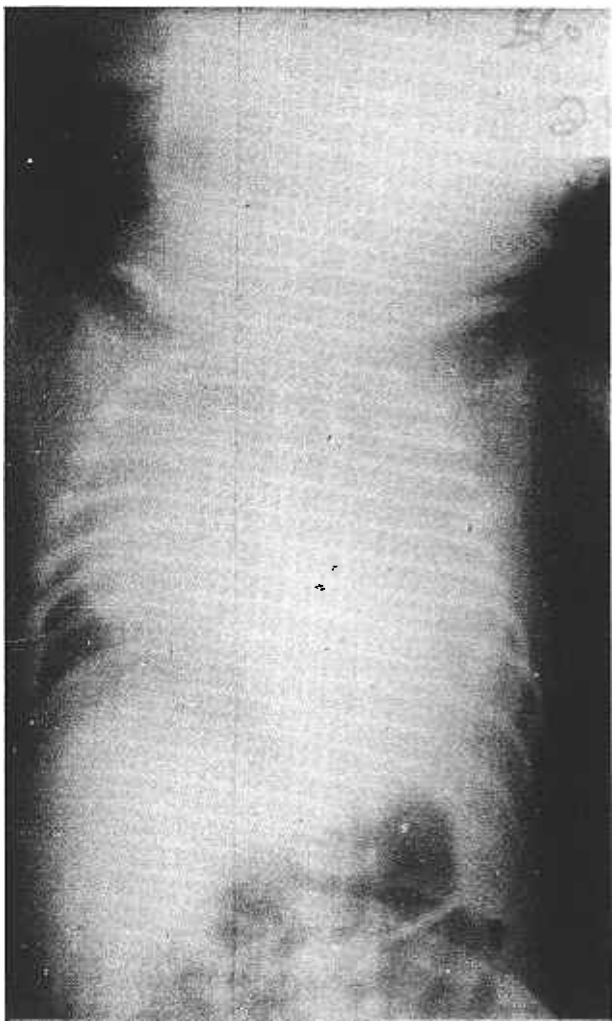

FIG. 1: X-ray film showing opacification of the left nemithorax with a few radiolucent areas in the left mid-zone and diaplacement of the mediastinum to the right.

The mother's pregnancy was unremarkable. She denied taking any anticonvulsants, anticoagulants or traditional medication during this pregnancy. The patient is the fifth child. The rest of the siblings are healthy. There is no family history of a bleeding disorder.

On admission, he looked ill and pale. His axillary temperature was 37.0°C. Jaundice was present. He was tachypnoeic with a respiratory rate of 60 minute, there were subcostal and intercostal recessions. There was dullness on percussion of the right hemithorax. On auscultation of the right side, the breath sounds were noted to be markedly reduced. The apex beat was 160 per min. There were no cardiac murmurs. The liver was palpable 1 cm below the costal margin and the spleen was tipped. Bowel sounds were normal. The Moro reflex was diminished and he had generalized hypotonia.

Investigations which were done on admission showed a haemoglobin concentration of 6.4g%, total white blood cell count of 7600 per mm3 (54% polymorphs and 46% lymphocytes), platelet count of 270,000 per mm3, blood urea nitrogen of 17mg%, serum sodium of 141 mmol per 1, serum potassium of 4.5 mmol per 1, serum chloride of 103 mmol per 1, blood sugar of 147mg%, serum bilirubin of 12.0mg% (conjugated fraction 0.6mg) and prothrombin time of 19 s (control 15 s). The results of a capillary blood gas analysis revealed a pH of 7.358, carbon dioxide tension of 38.4 mmHg, oxygen tension of 40.mmHg and oxygen saturation of 73%. A portable chest x-ray film showed a right pleural effusion with displacement of the mediastinum to the left (Fig. 3). The blood culture failed to show any growth.

An intravenous drip was set up and oxygen was given via a head-box at the rate of 2 l per minute. Intravenous penicillin and gentamycin were started. After blood had been taken for the investigation of the prothrombin time, 1mg of vitamin K was given intravenously. Sixty mls of fresh blood was transfused soon after. Fresh blood was aspirated from a disgnostic tap of the right pleural cavity. A chest-tube was inserted and 150mls of blood were drained. The patient became less tachypnoeic following the procedure. An additional dose of 3mgs of vitamin K was given intravenously.

The following day, he was active. However he developed a temperature of 38.0°C. He was still pale, the repeat haemoglobin concentration was 9.6g%. His serum bilirubin rose to 13.5mg%. The repeat prothrombin time was 16 s (control 15 s). Phototherapy was commenced. On the 3rd hospital day, another 50mls of blood was drained. He was transfused with another 60 mls of fresh blood. He was no longer tachpnoeic. His temperature had settled. The serum bilirubin was 14.6mg%. Feeding was started on the 4th hospital day and he tolerated the feeding well.

The results of the investigations which were done on the 5th hospital day showed a bleeding time of 3 minutes and an activated partial thromboplastin time of 39 s (control 35 s; normal range 35-45 s). The chest tube was clamped on this day; however he developed a right pneumothorax. The chest tube was resited. The pneuthorax subsequently disappeared and the chest tube was removed 3 days later. The antibotics were discontinued after 10 days. Phototherapy was stopped on the 7th hospital day. He was discharged well on the 11th hospital day; on discharge his haemoglobin concentration was 12.4g%.

DISCUSSION

Only 23 cases of neonatal haemothorax have been reported in the medical literature.[9-21] Seven of these cases were

attributed to Vitamin K — dependent Hypoprothrombinemia (haemorrhagic disease of the newborn).[11-16]

According to Weill et al,[12] the aetiologic factors that could give rise to neonatal haemothorax are generalized haemorrhagic diseases in which the pleural lesion is a localised manifestation, pleural base vascular malformations in which traumas initiates the bleeding and exaggerated hypoprothrombinemia usually occurring between the 2nd and the 6th day of life. An increased incidence of bleeding, particularly into the pleural or parietal cavities had been noted by Pomerance and Yaffe[22] in neonates when mothers took phenobarbitone during pregnancy.

In the 2 cases reported here, there was no history of trauma. Their mothers did not take anticonvulsants during pregnancy. There was no obvious evidence of a bleeding tendency such as umbilical or rectal bleeding, haematemesis or skin bruising. Both the babies were not given vitamin K at birth and they were exclusively breast-fed. The prothrombin times returned to normal after the administration of vitamin K. All these evidences suggest that vitamin K-dependent hypoprothrombinemia could be responsible for the development of haemothorax in the 2 cases. Unfortunately, coagulant factor assays could not be done in the hospital.

It has been shown by Sutherland and others[23-24] that vitamin K-dependent hypoprothrombinemia is more common in breast-fed babies who have not been given vitamin K at birth.

The common modes of presentation of haemorrhagic disease of the newborn are bleeding from the umbilical cord, gastro-intestinal tract, skin or the nostril. Haemothorax as the only manifestation of haemorrhagic disease of the newborn is extremely rare.

In case one, a left diaphragmatic hernia could not be ruled out on the basis of the findings on clinical examination and a portable x-ray film. Differentitation is important since the treatment of the two conditions is different; in congenital diaphragmatic hernia, emergency surgery is a life-saving measure while in haemothorax an urgent thoracocentesis is indicated to relieve the respiratory distress. A contrast radiologic examination of the gastrointestinal tract was used to differentiate the two conditions.

Neonatal haemothorax is treated by thoracocentesis. When the patient is anaemic, blood transfusion is indicated. Treatment should also be directed at correcting the underlying cause. Vitamin K is given to those with vitamin K-dependent hypoprothrombinemia.

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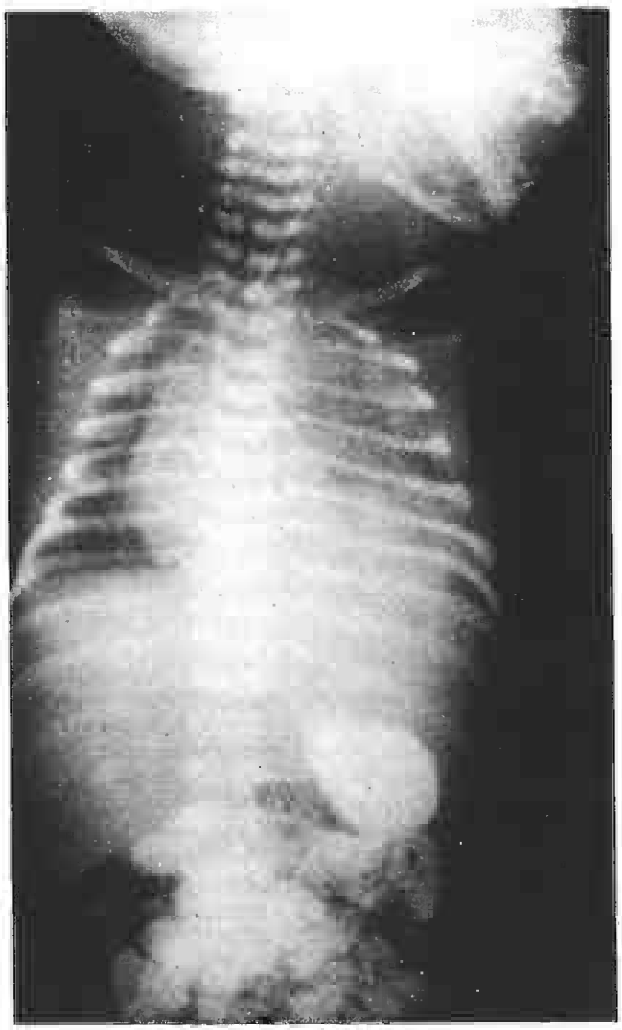

FIG. 2: X-ray film with contrast showing radiolucent areas visualised in the initial portable x-ray film were not loops of intestine.

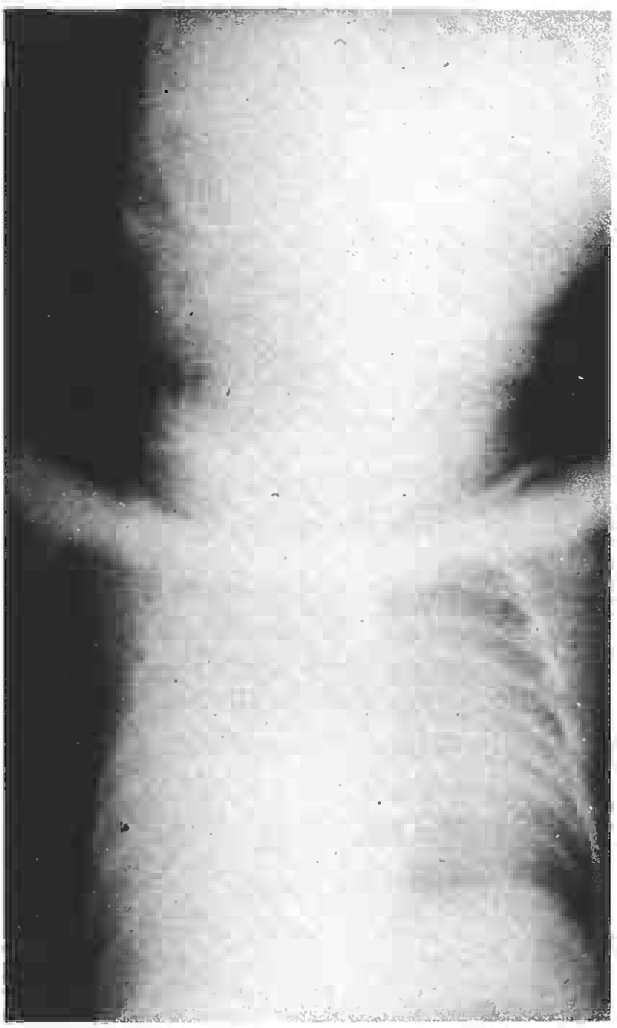

FIG. 3: X-ray film showing opacification of the right hemithorax and displacement of the mediastinum to the left.

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