ADRENAL HAEMORRHAGE IN A NEWBORN – A CASE REPORT

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ABSTRACT

A term female neonate with monolateral adrenal haemorrhage associated with haemorrhagic disease of newborn is described. Diagnosis and follow-up of adrenal haemorrhage was done clinically and sonographically which revealed reduction in the size of adrenal haematoma over a month with no evidence of adrenal insufficiency. She was discharged well and followed up.

Keywords: *adrenal haemorrhage, haemorrhagic disease of newborn follow up, vitamin K deficiency.*

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INTRODUCTION

Townsend, in 1894, first described the bleeding in infants during the first two weeks of life as haemorrhagic disease of the newborn[1] (HDN). The association of HDN to deficiency of vitamin K (VK) was recognised by Dam et al[2]. Lane and Hathaway described three groups of children with HDN: early, classical and late types[3]. The classical type of HDN due to VK deficiency is mostly idiopathic but at times is caused by asphyxia or maternal drug ingestion. The most common presenting feature of the classical type of HDN is the development of bleeding from the gastrointestinal tract, skin and nasal regions at 2 to 5 days of age. The less common features are intracranial and umbilical haemorrhage at 2 to 3 weeks of life if VK remains undetected[3].

We describe a term neonate with the classical type of HDN presenting as monolateral adrenal haemorrhage (ADH). The association of ADH with HDN is less frequently seen in day-to-day clinical practice.

CASE REPORT

A term 3750gm female baby at thirty-four hours of life, was admitted for haematemesis and melaena with abdominal distension. She was born to a 29-year-old mother, gravida four para three, by spontaneous vertex delivery at home and was not asphyxiated at birth. Antenatally, the mother was anaemic and was treated with parenteral haematinics. Her second conception ended in stillbirth. Physical examination on admission revealed she was drowsy, severely anaemic, jaundiced and tachycardic. The length and head circumference were appropriate for age (50 to 75th centile). Her pulse was 158/min, respiratory rate 56/min and blood pressure 74/54mmHg. Abdominal examination revealed a smooth non-tender, firm mass on the right flank with no evidence of free fluid. The external genitalia was normal.

Investigations on admission revealed the following: arterial blood gases: PH 7.385, PCO_2 19.0mmHg, PO_2 146mmHg, HCO_3 8.2, B.E. -16.2, O_2 sat 99.3%; serum eletrolytes: serum sodium 149 mmol/l, serum potassium >6.0 mmol/l, blood urea 11.9mmol/l, serum calcium 1.56 mmol/l, random blood glucose 5.4 mmol/l, Hb 2.5 g/dl, PCV 9.5%, red blood cell count 0.85 x 10^{12}/l, total leucocyte count 16.3 x 10^9/l; differential count: P 72%, L 21%, M 6%, E 1%, MCV 111.6 fl, MCH 30.5 pg, MCHC 27.3g/dl, reticulocyte count 7.5%, platelet count 83 x 10^9/l, blood group O positive, direct Coombs' test negative, prothrombin time > 60 sec, international normalised ratio (INR) >6.0 and partial thromboplastin time (PTT) > 60 secs, control 44.0 sec and serum bilirubin 83umol/l. Screening for glucose 6 phosphatase dehydrogenase was normal and blood culture was negative. Peripheral smear showed no evidence of toxic granules. X-ray of the abdomen showed a homogeneous opacity on the right half of the abdomen. Serum bilirubin dropped to normal from its initial level of 330 umol/l with five days of continuous phototherapy.

Abdominal sonogram done on the 3rd day of life revealed a well defined, egg shaped, predominantly hyperechogenic and uncalcified mass measuring 4.0 x 2.2 cm in size, in the right suprarenal area (Fig 1). The right kidney was displaced downwards and the inferior vena cava to the medial side while the left kidney was normal. These features were suggestive of a right adrenal haematoma. CT abdomen (Fig 2) depicted a suprarenal mass. The margin was moderately enhanced while the central area was hypodense and the left kidney was normal.

On admission, she was managed with 70ml of fresh blood transfusion, followed by 60ml of fresh frozen plasma, oxygen and intravenous fluids. 60ml of packed cells were given to the baby a day later. She was also treated with intravenous VK of 1mgm per day for three days. The haematemesis and melaena subsided six hours after starting the treatment. She was treated with intravenous benzyl penicillin 100,000 units/kg and gentamicin 2.5mgm/kg/dose 12 hourly for five days. On the fourth day of life, she was exposed to continuous phototherapy in view of increasing bilirubin level. On the sixth day of life, the repeat haemoglobin was 12.2 g/dl, PCV 36.2%, and platelet count 108 x 10^9/l. On the ninth day of

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Fig 1 – Abdominal sonogram showing a suprarenal mass of inhomogenous echogenicity.

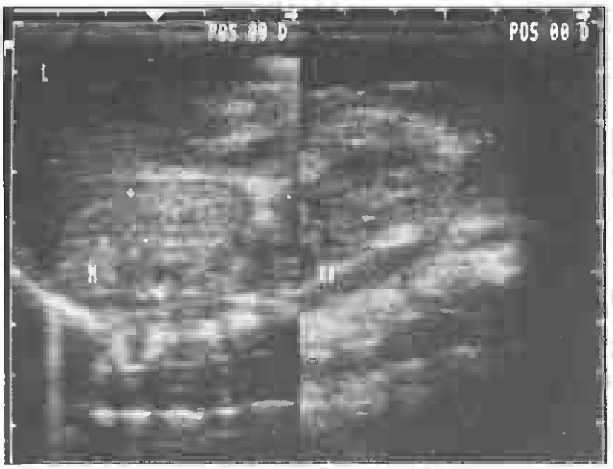


Fig 2 – Contrast CT abdomen showing right adrenal mass –a central area of hypodensity with enhanced margin and normal left kidney.

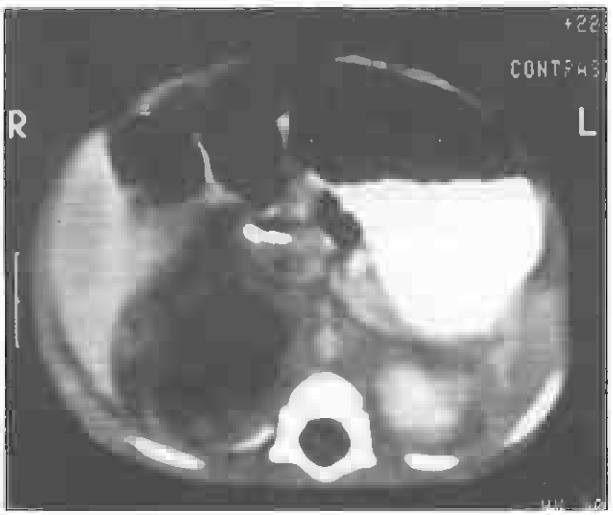


Fig 3 – Follow up sonogram of the same suprarenal mass with a well-defined hyperechoic capsule.

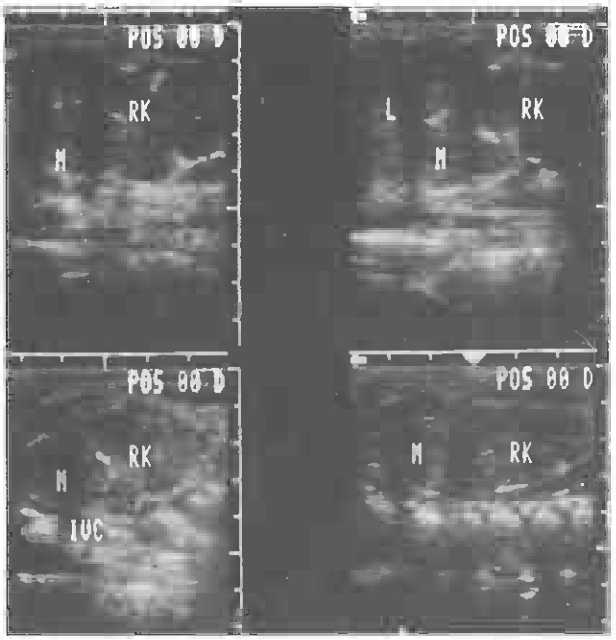
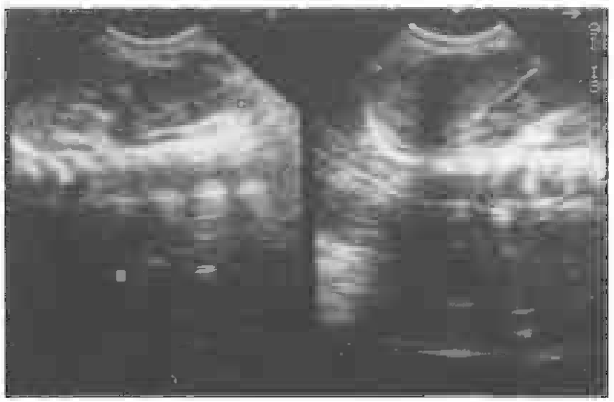


Fig 4 – Follow up sonogram of the same suprarenal mass with a rim of calcification



life, the sequential bilirubin level was 145umol/l. She was discharged well two days later and followed up.

Review of the child clinically as an outpatient on the sixteenth day of life showed reduction in size of right flank mass. Sequential sonogram revealed reduction in size of the heterogeneous mass measuring 2.8 x 2.0 cm with a well-defined hyperechogenic capsule (Fig 3).

Review of the child at one month of life revealed she was well with no evidence of adrenal insufficiency. Repeat sonogram showed a rim of calcification with further reduction in size of the heterogeneous echoic mass measuring 2.2 x 1.5cm (Fig 4).

DISCUSSION

The incidence of HDN has been reported to be 1 in 200 to 400 in term infants who were not given VK prophylaxis[3]. The genesis of HDN due to VK deficiency is believed to reflect a failure of conversion of glutamic acid to gammacarboxyglutamic acid with less of calcium binding sites on coagulative proteins, an increased level of noncarboxylated prothrombin in the circulation and failure of conversion of prothrombin to thrombin resulting in defective coagulation. This carboxylation process usually occurs in the endothelium of the hepatocytes. Risk factors for neonates to develop HDN include the low level of procoagulant factors (30%-60% of adult level) II, VII, IX and X, lack of established intestinal flora, inadequate intake of VK, solely breast fed babies, asphyxia and maternal drug ingestion. However, Blanchard et al[4] suggested the role of hepatic dysfunction in the genesis of HDN in addition to the presence of positive non-carboxylated prothrombin (PIVKA II).

The specific tests for VK deficiency include low levels of factors II, VII, IX and X, decreased ratio of factor II coagulant to factor II antigen, immunoassay for positive noncarboxylated prothrombin II[3]. The rapid correction of VK dependent coagulative factors after the therapeutic dose of VK still remains an excellent confirmation of VK deficiency[3].

ADH occurs more commonly following traumatic delivery, particularly difficult forceps, breech delivery, intrauterine asphyxia, vacuum extraction and large-for-date babies including infants of diabetic mothers[5]. Infants with unilateral ADH usually present with shock due to blood loss[6], while infants with bilateral haemorrhage present with the features of adrenal insufficiency as well as haemorrhagic

shock. Minor bleeds into the adrenals may result in anaemia, jaundice and abdominal mass while moderate to massive bleeds manifest with sudden collapse, cyanosis, limpness, jaundice, irregular respiration, flank mass and elevated or subnormal temperature.

ADH has to be differentiated from neonatal neuroblastoma, adrenal abscess, and cortical renal cyst[7]. ADH usually resolves in six weeks and the adrenal function is unimpaired with residual dystrophic calcification[7]. The sonographically important criteria for distinguishing ADH from adrenal malignancy are reduction in size, loss of echogenicity and formation of capsule within the first ten days of life.

Treatment of HDN consists of administration of intravenous, aqueous colloidal suspension of VK 1 to 2 mgm (maximum 5 mgm). This therapy, if effective, usually increases the level of clotting factors in 2 to 4 hours, approaching normal values at the end of 24 hours. For severe bleeding, fresh frozen plasma 10 ml/kg and packed red cells may be needed to correct the anaemia and shock.

The features suggestive of classical type of HDN in this neonate were haematemesis, dark coloured stools on day 2 of life, near normal platelet count, prolonged PT, PTT, rapid response to VK therapy and repeat haemoglobin 12.2 g/dl, PCV 36.2% and platelet count 108 x 10^9/L (on day 6 of life). The features suggestive of ADH in this neonate are an abdominal mass in the right flank, sono and contrast CT abdomen evidence of right adrenal bleed, and resolution of the adrenal mass over a one month period. The association of ADH with HDN is not commonly observed in routine clinical practice, particularly as a presenting feature. The role of sonography not only as a noninvasive tool for diagnosing monolateral ADH, but also for differentiation of adrenal tumours, and for follow-up ADH as described in this case report, is emphasised.

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