CASE REPORT

Bilateral Adrenal Hemorrhage in a Large for Gestational Age Neonate Due to Obstructed Labour and Birth Asphyxia

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ABSTRACT

Neonatal adrenal hemorrhage is a rare condition presenting as a unilateral or bilateral adrenal mass. Large size of the gland and increased vascularity makes it prone to hemorrhage. Abdominal ultrasound helps in confirmation of diagnosis and also differentiates it from other causes of abdominal masses. We present the case of a neonate who was large for gestational age delivered through SVD and developed adrenal hemorrhage secondary to obstructed labour and birth asphyxia. Diagnosis was confirmed by abdominal ultrasound and he was discharged home successfully on steroids on 13th day of life. However, his follow up could not be done as he expired after one month of discharge from hospital due to aspiration pneumonia. We conclude that conservative management is important and early surgical intervention should be avoided. Our aim is to emphasize radiological and clinical features associated with NAH for early diagnosis and management.

Key Words: *Adrenal hemorrhage, Obstructed labour, Birth asphyxia*

INTRODUCTION

Adrenal hemorrhage is a relatively uncommon phenomenon in neonates with varying clinical presentation ranging from asymptomatic minimal bleeding to fulminant hemorrhage due to adrenal insufficiency leading to death.[1] The incidence ranges between 1.7 and 3 cases per 1000 live births and this rarity makes it particularly important.[2] Associated risk factors include birth trauma due to obstructed labour which can occur in large for gestational age neonates, perinatal asphyxia, shock, septicemia or coagulation defects.[3] Large size of the adrenal gland to body weight and increased vascularity leads to mechanical compression and changes in venous pressure during delivery.[4] Clinical manifestations include mild anemia, poor feeding, vomiting, unexplained jaundice, lethargy, hypotonia, abdominal mass, discoloration, scrotal swelling and hypertension.[3] In this study we aim to describe the data of a neonate who had bilateral adrenal hemorrhage secondary to obstructed labour.

CASE REPORT

Male baby of 4.5 kg (large for gestational age) born at gestational age of 39^{+2} weeks to $G_6P_5A_0$ mother via SVD with obstructed labour due to shoulder dystocia. No antenatal risk factors were identified. Baby received with no cardiorespiratory effort and APGAR score of 0/10 at 1, 5 and 10 minutes. Immediate CPR was started in form of chest compressions and ambu bagging. Revival was achieved after 10 minutes of CPR. He initially had bradycardia but then cardiac activity was restored to normal with one shot of injection

adrenaline. He was hooked to CPAP on 4 cm H_2O and shifted to NICU for post-resuscitation care and management. First line IV antibiotics and IV fluids (10% dextrose) were commenced. His blood pressure was 89/39 mmHg, heart rate 132 bpm, respiratory rate 59/min. Neonatal reflexes were poor and he developed grade 3 hypoxic ischemic encephalopathy with focal clonic seizures for which phenytoin was added in maintenance dose after initial loading dose. Shoulder dystocia led to fracture of the left humerus for which orthopedic surgery review was done and cast applied. Investigations were done on day 1 of life which were as follows: pH 7.18, pCO_2 32.3, HCO_3 12, pO_2 136. Initial Hb 16.3 g/dl, TLC 23.5x10^9/L, platerets (Plt) 177x10^9/L, serum sodium 136 mmol/L, potassium 4.8 mmol/L, chloride 106 mmol/L, serum calcium 2.8 (2.1-2.65 mmol/L), urea 3.8 mmol/L (1.4-4.3 mmol/L), creatinine 75 µ mol/L (4-40 µ mol/L), STB 100 µ mol/L, ALT 520 U/L, AST 1480 U/, ALP 234 U/L, blood sugar (BSR) 74 mg/dl. Coagulation profile was normal. On 3rd day of life he became oliguric. RFTs were done which showed urea 8.7 mmol/L and creatinine 361 µ mol/L; therefore antibiotics were adjusted to renal doses, restricted fluids were given according to AKI protocol and urine output was monitored. Dopamine infusion was also started owing to poor perfusion, cold peripheries, feeble pulses and CRT > 2 seconds. His CPAP was weaned off and gradually shifted to 1 litre O_2. He also developed sclerema on lower limbs. Repeat labs showed Hb 10.7 g/dl, TLC 13.4 x 10^9/L, Plt 132 x 10^9/L and serum calcium 1.4 for which whole blood transfusion was done and IV calcium gluconate was replaced.

Abdominal and cranial ultrasound was done to acsertain the cause of anemia. Cranial ultrasound was unremarkable however abdominal ultrasound/color doppler done on day 4 revealed bilateral adrenal hemorrhage. Both supra-renal regions showed cystic areas with internal septations measuring 24 x 40 mm on right side and 28 x 30 mm on left side. The areas showed no flow on color doppler. Tab fludrocortisone 0.2mg once daily was started. On 8th day of life (DOL), he started deteriorating with oxygen desaturation and gasping breathing for which shifted to mechanical ventilator on PC-SIMV mode. CRP was markedly elevated (253 mg/L). Antibiotics were stepped up and stress dose of oral hydrocortisone was given. Repeat Hb was 9.4 for which RCC transfusion was done. After one day he was extubated and shifted to CPAP 5cm H_2O. Dopamine infusion was stopped as he had good volume pulses and was well perfused. He gradually started improving and shifted to oxygen and then became oxygen free and started taking expressed breast milk orally. RFTs were in normal range and urine output was adequate. The patient did not require further blood transfusions. He was finally discharged on 13th DOL on tab hydrocortisone, fludrocortisone, syp phenytoin and oral supplements. His follow up could not be done as he expired after one month of discharge from hospital due to aspiration pneumonia.

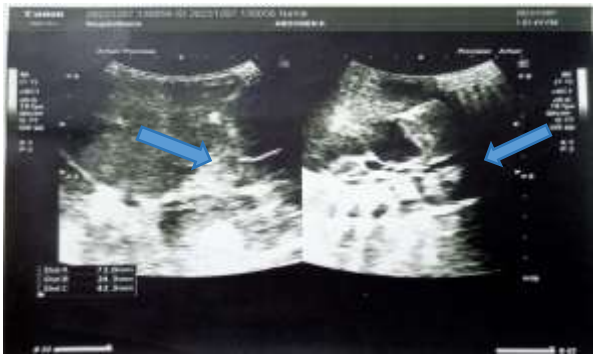

Fig 1: Abdominal ultrasound revelaed bilateral suprarenal masses with internal septations measuring 24 x 40 mm on right side and 28 x 30 mm on left side but without flow on color Doppler

DISCUSSION

Neonatal adrenal hemorrhage (NAH) is the most common cause of adrenal mass in neonates with most cases occurring unilaterally. However 10% cases are bilateral.[5] Incidence of NAH is higher in large for gestational age neonates who have obstructed labour. Two studies showed the association of increased birth weight and perinatal hypoxia with adrenal hemorrhage.[6,7] The right adrenal gland is the frequent site of NAH as it becomes trapped between the liver and spine causing mechanical compression and hemorrhage. The right adrenal vein drains generally directly into the inferior vena cava and is exposed to changes in venous pressure which can also lead to hemorrhage.[8] This condition may be an incidental finding or may present as an abdominal mass, scrotal hematoma or hypotension on exam, as well as unexplained

anemia, jaundice, and/or abdominal calcification on laboratory investigations and radiological imaging.[5] The ultrasound findings vary at different stages of bleeding. It appears solid, enlarged and echogenic in the early stage. After 1-2 weeks, liquefaction occurs and the mass shows mixed echogenicity with a central hypoechoic region and some internal echoes, and then cystic changes occur gradually. After sometime hemorrhage starts shrinking and is left with a rim of calcification. It becomes anechoic with complete resolution after 2 months. If renal vein thrombosis occurs, there is echogenic enlarged swollen kidney with increased echogenicity of the interlobular vessels.[9] Differential diagnosis include neuroblastoma, Wilms tumor, adrenal abscess, congenital adrenal hyperplasia, adrenal or splenic cyst, and lymphangioma which have a similar appearance on ultrasound.[5] The mainstay of treatment are glucocorticoids and mineralocorticoids as adrenal hemorrhage can compromise the production of cortisol and aldosterone. Long term use of corticosteroids has a suppressive effect on the hypothalamic-pituitary-adrenal axis. Therefore it is recommended that steroids should be given for a short period of time with suspension after clinical or radiological improvement.[9] The majority of patients have a good prognosis and are conservatively managed with the adrenal hemorrhage fully resolving over a period of three to six months.[10] Surgery is indicated rarely only if there is suspected neuroblastoma, severe adrenal insufficiency, retroperitoneal hematoma, associated trauma or failure to resolve with medical management.[11,12] Most cases of NAH resolve spontaneously. In a study, out of total 65 cases, 6 resolved prenatally, 23 resolved postnatallly, 22 regressed in size after birth and only 14 persisted postnatally. 16 cases underwent postnatal surgical intervention. Neuroblastoma was suspected in 16 cases out of which only 1 was a confirmed case.[13]

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Authors' contribution

FF: Writing manuscript
SN: Drafting revision
AQ: Statistical analysis
RA: Conceptualization of project
MS: Literature search
HA: Data collection

All the authors have approved the final manuscript draft and accept the responsibility of research integrity.