CASE REPORT

A RARE CAUSE OF HYPERTENSION IN A NEWBORN: ADRENAL HEMORRHAGE

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INTRODUCTION

Hypertension is rarely seen in the neonatal period. Although it most commonly develops due to renal artery thrombosis, it may also be caused by an array of factors.[1] Adrenal hemorrhage is a rare cause of hypertension during neonatal period. To the best of our knowledge, it has been reported only in a few cases in the English-language literature.[2-4] Here, we present a newborn who had a history of birth trauma and adrenal hemorrhage, and consequently developed hypertension. Birth trauma is a complication that sometimes occurs despite good obstetric care due to difficult labor, abnormal presentation, or large birth weight. It may sometimes cause adrenal hemorrhage and rarely hypertension as a result of it. Therefore, increased awareness about complications associated with birth trauma will increase the chances of early diagnosis and treatment.

CASE REPORT

A male baby was born with normal spontaneous vaginal delivery at 40 weeks of gestation, weighing 4300 g in another hospital before referral to us on the first day of life. The labor was difficult and prolonged, baby was macrosomic and had brachial plexus injury associated with birth trauma. Parents did not have a history of consanguineous marriage, and family history was unremarkable for known diseases. The mother stated that it was her first pregnancy, that she was regularly followed during pregnancy, and that no abnormality was detected. APGAR scores of the baby were unknown. Physical examination revealed caput succedaneum, which was 5 × 6 cm in diameter in the right parietooccipital region of the head, flaccid paralysis of the right upper extremity, and a palpable mass in the right middle area of the abdomen. Examination for newborn reflexes showed that mororeflex in the right arm and grasping reflex in the right hand were absent. Blood pressure of the patient was measured in the right upper extremity as 147/99 mmHg, and his pulse was 140 beats per minute with good femoral pulses. Blood pressure results were obtained by an oscillometer and good correlation between upper- and lower-extremity readings were observed. There was no sign such as vomiting, convulsions, lethargy, and respiratory distress, to suggest hypertension. In laboratory findings, serum creatine kinase level was 5771 U/L (130–1200) and aspartate aminotransferase level was 146 U/L (35–140); other blood test values were within normal ranges. Ultrasonography investigation of the flank mass revealed a hemorrhage (28 × 33 × 38 mm in size) in the right adrenal gland, and no abnormality was observed in the Doppler flow study of the aorta and renal arteries. Echocardiography and skeletal radiographs showed no pathological findings. Propranolol was started at a dose of 1 mg/kg/day for treatment of hypertension. Vital signs and
hematocrit and blood glucose levels were normal. Owing to persistent hypertension, the dose of propranolol was gradually increased up to 4.5 mg/kg/day. At 12th day of the treatment, blood pressure became stable; then drug dose was gradually reduced and it was discontinued on day 25. The paralysis of the right upper extremity was defined as mix brachial plexus palsy (Erb–Duchenne palsy + Klumpke palsy) and physical therapy exercises were prescribed. In terms of possible complications that could develop secondary to hypertension, ocul fundus examination and echocardiography were performed and no abnormality was observed. Intermittent abdominal ultrasonography showed a progressive decrease in the size of adrenal hemorrhage, and on day 26 it was reduced to $16 \times 24 \times 26$ mm in size. When the patient was 27 days old, he was discharged. During the 1-year follow-up, there was no recurrence of hypertension; renal functions remained normal; adrenal US was normal; and an improvement in brachial plexus palsy symptoms was observed.

**DISCUSSION**

The frequency of hypertension in the newborn period is estimated to be 2%. The variability of limits for normal based on the baby’s birth weight, gestational age and postconceptional age, also differences in measurement techniques, makes it difficult to determine the actual incidences of hypertension. The most common cause of neonatal hypertension is renal artery thromboembolism, which develops secondary to umbilical artery catheterization. In our patient, renal function tests, urine output, and glomerular filtration rate were found to be normal. In addition, except right adrenal hemorrhage, no abnormality was observed in the ultrasonography of the kidneys with Doppler flow study of the aorta and renal arteries. Other common causes of neonatal hypertension such as hypervolemia due to oliguric renal damage; structural renal diseases such as polycystic kidney disease, aortic coarctation, and obstructive uropathy; medications such as corticosteroids; endocrine disorders; and bronchopulmonary dysplasia may be considered.

It was thought that the etiology of hypertension in our patient was due to bleeding of the adrenal gland. Transient hypertension developed as a result of acute renal obstruction, which was due to direct pressure on the proximal ureter by an adrenal hemorrhage or secondary to displacement of the kidney by the mass, resulting in curling of the proximal ureter. Hypertension is not a common complication of adrenal hemorrhage. In the four cases reported in the English-language literature, adrenal hemorrhage, partial or complete ipsilateral renal function loss and hypertension were observed, and hypertension was improved following the resolution of bleeding. Sherer et al. presented a case with transient neonatal hypertension due to in utero adrenal hemorrhage. Sirota et al. reported a case of newborn with adrenal hemorrhage associated with transient compression of the kidney and hypertension. They thought the cause of the hypertension was the acute renal compression by the hemorrhagic mass. Starinsky et al. had two similar cases with hypertension that caused by adrenal hemorrhage and a poorly functioning or non-functioning kidney on the same side. Hypertension is asymptomatic in every one of three patients in clinical presentation. Moreover, it may be represented with nonspecific symptoms such as feeding difficulties, irritability, and lethargy; or cardiovascular symptoms such as heart failure, vasomotor instability, and perfusion abnormalities; or neurological symptoms such as tremor, convulsion, apnea, and muscle tone changes. Any of these symptoms were not seen in our patient; hypertension was detected during the routine monitoring of vital signs.

Effective treatment of hypertension prevents from secondary damage to brain, kidneys, and heart. However, during treatment, to prevent the development of ischemia in the brain and the optic disk, blood pressure should be lowered gradually. Widely used drugs in neonatal hypertension are captopril, diuretics, β-blockers, and calcium channel blockers. We preferred propranolol, which is a non-selective β-blocker and it blocks the action of epinephrine and norepinephrine on both β1- and β2-adrenergic receptors. The patient’s blood pressure values became regulated synchronously with the resolution of hematoma, and his treatment was discontinued gradually.

**CONCLUSION**

As neonatal hypertension may progress asymptotically, it should be kept in mind in infants of diabetic mothers, macrosomic infants, and
difficult births, especially in neonates who develop adrenal hemorrhage. To protect the patient from complications of hypertension, it is important to pay attention to blood pressure monitoring even though its measurement procedure and evaluation are difficult.

REFERENCES