A Rare Presentation of Adrenal Hemorrhage in Newborn: Case Report

Yenidoğanda Adrenal Hemorajinin Nadir Görülen Bir Prezentasyonu

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ABSTRACT Neonatal hyperkalemia is a frequent condition seen in very low-birth-weight infants and it may also be associated with hemolysis, renal failure, hemorrhage and aldosterone deficiency. Herein, we report two cases of hyperkalemia secondary to adrenal hemorrhage. First patient was admitted with dyspnea and hypoglycemia. During follow-up, hyperkalemia and hyperbilirubinemia appeared, and adrenal hemorrhage was detected on the abdominal ultrasonography. Although there was no adrenal insufficiency, hyperkalemia was thought to be related to hemorrhage. Second patient was a newborn of a diabetic mother and she had hypoglycemia. Hyponatremia and hyperkalemia appeared on the second day of her life and she was diagnosed as adrenal insufficiency. Abdominal ultrasonography showed adrenal hemorrhage. In conclusion, abdominal ultrasonography must be obtained to evaluate the etiology of hyperkalemia, and patients with adrenal hemorrhage should be followed closely for probable electrolyte imbalance.

Key Words: Adrenal insufficiency; hyperkalemia; hemorrhage; infant, newborn

ÖZET Neonatal hiperkalemi çok düşük doğum ağırlıklı yenidoğanlarda sık görülen bir durum olmakla beraber hemoliz, böbrek yetmezliği, kanama ve aldosteron yetersizliğinde de ortaya çıkabilir. Bu yazıda adrenal hemoraji nedeniyle hiperkalemi gelişen 2 olgu sunuldu. Solunum sıkıntısı ve hipoglisemi nedeniyle kabul edilen birinci olgunun takiplerinde hiperkalemi ve hiperbilirubinemi ortaya çıkması üzerine yapılan batın ultrasonografisinde, adrenal hemoraji saptandı. Adrenal yetersizlik gelişmeyen olguda hiperkaleminin hemorajiye sekonder olduğu düşünüldü. Diabetik anne çocuğu olan ve hipoglisemi nedeniyle yatırılan ikinci olguda ise yaşamının ikinci gününde hiponatremi ve hiperkalemi saptanması ve adrenal yetersizlik tanısı konması üzerine çekilen batın ultrasonografide, adrenal hemoraji görüldü. Sonuç olarak, hiperkalemi etiyolojisini belirlemede abdominal ultrasonografi mutlaka yapılmalı ve adrenal hemoraji saptanan tüm olgular elektrolit bozuklukları açısından yakından takip edilmelidir.

Anahtar Kelimeler: Adrenal yetmezlik; hiperkalemi; kanama; bebek, yenidoğan

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Neonatal hyperkalemia is a frequent detected condition especially in very low-birth-weight (VLBW) infants. Besides prematurity, renal failure, aldosterone deficiency, hemorrhage, tissue necrosis and administration of certain drugs may also cause hyperkalemia.[1] The fetal adrenal gland is a well-vascularized organ and hence, is more prone to hemorrhage. This condition may be observed in the newborns of diabetic mothers, neonates with perinatal asphyxia, trauma, coagulation disorders

and shock and septicemia, while no etiological cause may be identified in some patients.[2] Although patients classically present with jaundice, anemia and abdominal mass, a wide spectrum of clinical findings ranging from an asymptomatic state to adrenal insufficiency may be observed.[3] Here, we report two cases with hyperkalemia due to adrenal hemorrhage.

CASE REPORTS

CASE 1

The term female infant was admitted to newborn unit due to respiratory distress and hypoglycemia at the 3rd hour of life. The infant was born by Cesarean section from a gravida two para two mother at 39 weeks of gestation who had an uneventful pregnancy and delivery. Apgar scores were 7 and 9 at 1 and 5 minutes, respectively. Physical examination findings were as follows: Body weight: 2900 gr. (10-25p), height: 49 cm (25p), head circumference: 33.5 cm (10p) blood pressure: 75/40 (51) mmHg, heart rate: 138/R and she was tacyhpneic and dyspneic with normal breath sounds. Other systemic findings and chest roentgenogram were normal. With oxygen support, dyspnea of the patient with the diagnosis of transient tachypnea of newborn regressed rapidly. Blood glucose was regulated with 6.5 mg/kg/minute glucose perfusion. Laboratory findings were as follows: Na: 132 mmol/l, K: 7,6 mmol/l, Cl: 103 mmol/l, urea: 23 mg/dl, Cr: 1.05 mg/dl, LDH 2300 U/l, CRP: 5 mg/dl, Hct: 47.5%, Hgb: 15.2 g/dl, WBC: 38 500/mm^3, 26% neutrophils, 70% lymphocyte, 4% monocyte, platelet count: 139000/ mm^3. Calcium gluconate and sodium bicarbonate were given IV and sodium polystyrene sulfonate was administered orally. Control blood Na level was 136 mmol/l and potassium level started to decrease at the second day of this therapy. Blood sugar of the patient was regulated and enteral feeding was increased while IV fluid intake was decreased. Since urinary tests, blood chemistry results and urine output were normal, renal failure was eliminated. The cranial ultrasonography which was performed to invastigate any intracranial hemorrhage was reported as normal, on the other hand abdominal ultrasonography revealed adrenal hemorrhagic lesions in the right and left adrenal glands with the sizes of 30.2 x 18 mm and 30.7 x 21.8 mm, respectively (Figure 1). Values of PT and activated PTT levels were 14.2 sec and 43.6 sec, respectively. Follow-up blood pressure and blood sugar levels were found within normal limits and laboratory investigations for adrenal insufficiency revealed normal values as follows: Aldosterone: 198 pg/mL (normal values, 34- 273), renin: 27 pg/mL (normal values, 3-33), ACTH: 56 pg/mL (0-100), basal cortisol: 160 nmol/l (N: 138-690 nmol), and cortisol (after ACTH stimulation test): 439 nmol/l. The patient had hyperbilirubinemia at the second day of life had phototherapy on the 4th day of her hospitalization (total bilirubin: 21 mg/dL). In two days serum bilirubin level decreased to 14.3 mg/dL. Serum potassium level decreased gradually with anti-potassium treatment and antipotassium therapy was stopped on the 6th day. The patient was discharged on the 15th day of life with a normal potassium level. Abdominal ultrasonography perfor- after three months showed regression of the hematomas, however they still could be seen in the adrenal glands.

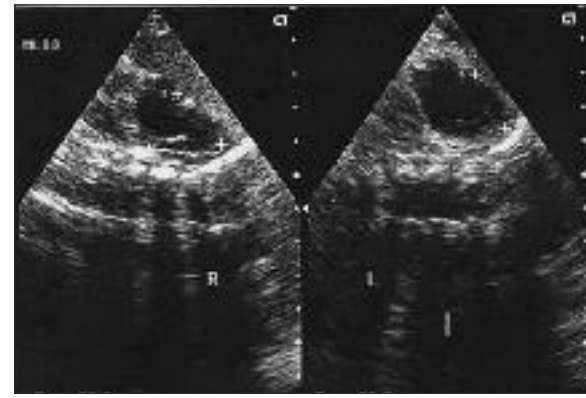

FIGURE 1: Adrenal hemorrhagic lesions in the right and left adrenal glands with the sizes of 30.2 x 18 mm and 30.7 x 21.8 mm in case 1.

CASE 2

A female infant was admitted to newborn unit with a blood sugar level of 20 mg/dl on the 2nd day of life. The infant was born from a gravida 2 para one mother by vaginal delivery at 40 weeks of the gestation and the mother had gestational diabetes mellitus. Apgar scores were 8 and 10 at 1 and 5 minutes, respectively. On admission, physical examination

findings were as follows: weight 3000 g (25p), height: 49 cm (25p), head circumference: 33.5 (10-25p), BP: 73/40(54) mm/Hg and there were hair on ears. The remainder of physical examination was normal. Laboratory results were as follows: Na: 126 mmol/l, K: 7.6 mmol/l, urea: 32 mg/dl, Cr: 0.64 mg/dl, LDH:1732 U/l, CRP: 3.19 mg/dl, Hct: 45.1%, Hgb: 14.9 mg/dl, WBC: 15100/mm^3, differential leukocyte count: 36% neutrophil, 49% lymphocytes, 4% band, 8% monocyte, platelet: 252000/mm^3. Intravenous 6.5 mg/kg/minute glucose perfusion was started for treatment of hypoglycemia. Then glucose infusion rate was increased to 8.1 mg/kg/min to maintain normoglycemia. Since the urine output and renal function tests were normal, probability of renal failure was eliminated. Meanwhile, phototherapy was started on the third day of life due to high serum bilirubin level (total bilirubin: 16.2 mg/dl). To investigate the etiology of hyperkalemia and hyponatremia laboratory tests performed and their results were as follows: Urine Na level: 32 mmol/l(normal values < 20), renin:158 pg/ml (normal values, 3-33), aldosterone: 285 pg/ml (normal values, 34-273) and ACTH: 236 pg/mL (normal values, 0-100), basal cortisol: 86 nmol/l (normal values, 138-690) and cortisol (after ACTH stimulation test):162 nmol/l. With these laboratory values, abdominal ultrasonography was performed to investigate adrenal insufficiency. Ultrasonographic examination showed a cystic hematoma of 33 x 20 mm size in the right adrenal gland and a solid hematoma of 35 x 25 mm size in the left adrenal gland (Figure 2). Hence, methyl prednisolone (1 mg/kg/day) treatment was initiated. Laboratory results obtained on the second day of treatment were as follows: Na: 135 mmol/L, K: 5.5 mmol/L. The blood glucose levels and blood pressure were within normal limits. On control abdominal ultrasonography which was performed in the second week, the size of the hematomas were 20.8 x 13.4 mm in the right and 22 x 15.9 mm in the left adrenal gland. Since cortisol level significantly increased during ACTH stimulation test at the 10th day of treatment, prednisolone therapy was gradually tapered and stopped in two weeks time.

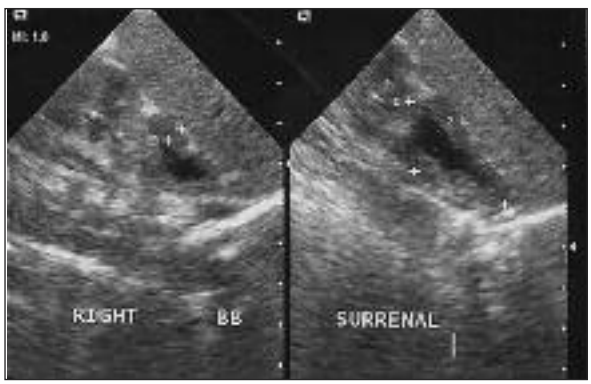

FIGURE 2: A cystic hematoma of 33 x 20 mm size in the right adrenal gland and a solid hematoma of 35 x 25 mm size in the left adrenal gland in case 2.

DISCUSSION

Hiperkalemia is common (approximately 30%) in infants under 1000 g of birth weight. Besides prematurity, hemolysis, excess potassium administration, metabolic or respiratory acidosis, renal failure, aldosterone deficiency, hemorrhage, tissue necrosis and administration of certain drugs may also cause hyperkalemia.[1]

Adrenal hemorrhage may occur particularly in newborns of diabetic mothers, neonates with asphyxia, trauma, coagulation abnormalities, shock and septicemia, while no etiologic cause may be found in some instances.[2] In the first case there was no risk factor, and the second one was a newborn of diabetic mother. Adrenal hemorrhage prevalence has been estimated to be approximately 3/100000.[3] The right side is affected more commonly than the left side, while 10-15% of the patients may present with bilateral hemorrhage.[4] Hemorrhage was bilateral in both of our patients.

The clinical spectrum of adrenal hemorrhage varies widely from being asymptomatic to life-threatening intraabdominal hemorrhage.[5] Classical clinical manifestations are abdominal mass, anemia due to bleeding, jaundice with an unknown etiology and less frequently, death due to bleeding and adrenal insufficiency in severe cases. Scrotal hematoma may also develop due to intraperitoneal leak resulting from rupture of the capsule of the adrenal gland.[6] Adrenal insufficency is a potential but extremely rare complication. Residual function in the subcapsular area and high regeneration capacity of adrenal gland may be possible explanations for the

rarity of adrenal insufficiency.[7,8] Both of our patients presented with hypoglycemia as well as hyperkalemia and hyperbilirubinemia. One of them had adrenal insufficiency. Hyperkalemia in the other patient without adrenal insufficiency was thought to be secondary to hemorrhage.

Other rare clinical manifestations are neonatal hepatitis and cholestasis due to insufficiency of growth hormone and cortisol.[8]

Adrenal hemorrhage should always be considered in the differential diagnosis of unilateral or bilateral abdominal mass. Ultrasonography is a useful diagnostic tool and may help clinicians in differentiating hemorrhage from other causes.[9] Complete resolution of hemorrhage can be detected usually in two months.[10] Although the hematoma in our patient decreased in size, it did not resolve completely until the third month of life.

In summary, hyperkalemia may be one of the clinical findings of adrenal hemorrhage. It should be kept in mind that hyperkalemia may develop due to hemorrhage without adrenal insufficiency in these patients. Therefore, abdominal ultrasonography should be performed for exclusion of adrenal hemorrhage in the work-up in order to define the etiology of hyperkalemia, and patients with adrenal hemorrhage should be closely follow up for a possible electrolyte imbalance. The first case is also important, since to our knowledge, a case who presented with hyperkalemia secondary to adrenal hemorrhage without an adrenal deficiency has not been reported in literature so far.

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