Transient Hyperkalemia and Hypoaldosteronism in a Patient with Acute Glomerulonephritis

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Abstract

The authors describe a 7-year-old boy with acute glomerulonephritis, who developed acute renal failure in the early course of his disease. While the renal function and other clinical manifestations gradually improved, hyperkalemia occurred unexpectedly, and returned to normal level spontaneously after a short period of symptomatic treatment. With the result of a low transtubular potassium gradient (TTKG) level, it was concluded that hypoaldosteronism was the major cause of hyperkalemia in this patient.

Key word : Hyperkalemia, Acute Glomerulonephritis, Hyporeninemic Hypoaldosteronism

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Postinfectious glomerulonephritis is the most common form of glomerulonephritis in children. The most common causative agent is group A β-hemolytic streptococci following acute pharyngitis or skin infection. The characteristic symptoms are hematuria, edema and hypertension. The prognosis is excellent, since all features, including edema, hypertension and gross hematuria, usually resolve within a few weeks. Less than 1 per cent of these patients present with a rapidly progressive disease and renal failure, which is characterized by azotemia, acidosis, hyperkalemia and circulatory overload (1). It is not unusual when hyperkalemia occurs along with other manifestations of severe renal insufficiency, however, few cases of transient hyperkalemia have been reported in patients with acute glomerulonephritis, who only had mild to moderate renal insufficiency. The pathogenetic mechanism for

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this phenomenon was reported to be due to transient hyporeninemic hypoaldosteronism. In some patients the hyperkalemia improved when nephritis resolved, but some needed furosemide or fludrocortisone to control their serum potassium level until spontaneous resolution occurred[2].

CASE REPORT

A seven-year-old boy was referred from a provincial hospital with the diagnosis of acute glomerulonephritis and acute renal failure. During four days admission, he had hypertension (BP130/90), edema and oliguria. The urinalysis revealed a specific gravity 1.035, pH 5, protein 1+, wbc 3-5/hpf, rbc 3-5/hpf. The blood chemistry at the time referred revealed BUN = 44 mg/dl, Cr = 5.4 mg/dl, Na = 131 mEq/L, K = 5.4 mEq/L. The level of plasma chloride and total CO_2 were not mentioned.

The positive findings of a physical examination at Chiang Mai University Hospital were hypertension (BP130/90), puffy eyelids and non-pitting edema of the lower extremities. The initial laboratory data revealed : Hb = 9.6 g/dl, Hct = 31 per cent, WBC = 12,000/mm^3, N = 69 per cent, L = 31 per cent, Platelet = 320,000/mm^3. Urinalysis: yellow clear, Sp gr = 1.020, pH = 5, protein 2+, glucose -ve, no wbc, no rbc. C_3 complement 165 μg/ml (550-1200 μg/ml). ASO titer 883 IU/ml (≤200 IU/ml). LE negative, ANA negative. BUN = 57 mg/dl, Cr = 5.5 mg/dl, Na = 130mEq/L, K = 5.3mEq/L, Cl = 107 mEq/L, total CO_2 = 15mEq/L.

The result of a kidney biopsy performed on the third day of admission was diffuse proliferative glomerulonephritis. No crescent formation was noted. The result was compatible with postinfectious glomerulonephritis.

Symptomatic treatment with a low salt diet, restriction of fluid intake, nifedipine and furosemide was given for twelve days and discontinued when the patient had diuresis and his blood pressure returned to normal.

The creatinine level gradually decreased in the second week despite a suspending high BUN level. The patient's overall condition was good. Hyperkalemia together with hyperchloremic metabolic acidosis was detected on the eleventh day of admission, while furosemide and nifedipine were still being given. Blood chemistry revealed BUN = 74 mg/dl, Cr = 1.0 mg/dl, Na = 139 mEq/L, K = 7.1 mEq/L, Cl = 121 mEq/L, total CO_2 = 17 mEq/L. Electrocardiography did not show any abnormality. Symptomatic treatment of hyperkalemia with 50 per cent glucose, insulin, kayexalate and sodium bicarbonate was given on that day. Kayexalate was continued for the next seven days and stopped one day before discharge. During ten days of treatment, potassium levels ranged from 4.6 mEq/L to 7.4 mEq/L with average level of 6.0 mEq/L. The patient was discharged on the twenty-first day with BUN = 30 mg/dl, Cr = 2.0 mg/dl, Na = 138 mEq/L, K = 5.6 mEq/L Cl = 116 mEq/L, total CO_2 = 23 mEq/L. His home medication was hydrochlorothiazide at 25 mg twice daily.

Since the renin and aldosterone assay were not available at Chiang Mai University Hospital, a TTKG (transtubular potassium gradient) was carried out instead to assess the aldosterone effect.

$$\text{The TTKG is } \frac{(U_{K^+} \div U_{OSM}/P_{OSM})}{P_{K^+}}$$

$$\text{In this case it was } \frac{(18.1 \div 386/302)}{6.3}$$

which was equal to 2.25. (serum K = 6.3 mEq/L, urine Na = 90 mEq/L, urine K = 18.1 mEq/L, urine osmolality = 386 mOsm/L, serum osmolality = 302 mOsm/L).

The patient was followed-up at the OPD one week later. The blood chemistry revealed BUN = 13mg/dl, Cr = 0.5 mg/dl, Na = 141 mEq/L, K = 4.2 mEq/L, Cl = 101 mEq/L, total CO_2 = 26 mEq/L and the urinalysis revealed protein 1+ with numerous rbc. Dihydrochlorothiazide was discontinued and no other medication followed. In the next three months, the patient's urinalysis and blood chemistry returned to normal, as shown in Table 1.

At the time of this report the patient had been followed-up regularly for 15 months without event.

DISCUSSION

Hyperkalemia is a common finding in the majority cases of acute renal failure with anuria or oliguria. It is also a well known finding in end-stage chronic renal failure with a GFR of less than 10 ml/min/1.73m^2. In acute glomerulonephritis, the prognosis is excellent, with less than 1 per cent of patients presenting with a rapidly progressive disease and renal failure[1]. For this reason, hyperkalemia is not a distinguished problem in this disease. However, in a few cases of acute glomerulonephritis,

Table 1. Results of urinalysis and blood chemistry during the follow-up period.

Time from onset	Urinalysis	Blood chemistry
1 month	numerous rbc/hpf, wbc–ve, protein1+	BUN = 13 mg/dl, Cr = 0.5 mg/dl, Na = 141 mEq/L, K = 4.2 mEq/L Cl = 101 mEq/L, Total CO_2 = 26 mEq/L
3 month	rbc 0-1/hpf, wbc–ve, protein –ve	BUN = 11 mg/dl, Cr = 0.4 mg/dl, Na = 139 mEq/L, K = 4.5 mEq/L Cl = 105 mEq/L, Total CO_2 = 26 mEq/L

hyperkalemia did occur in mild to moderate renal insufficiency, due to transient hyporeninemic hypoaldosteronism(2).

This syndrome has been reported increasingly, especially in adults with mild to moderate renal failure and clinical characteristics of interstitial nephritis. The most common underlying disease is diabetes mellitus, followed by interstitial nephritis, hypertension, gout, glomerulonephritis, nephrolithiasis, analgesic nephropathy, urinary tract obstruction, and mixed cryoglobulinemia(3-5). Most patients are asymptomatic and hyperkalemia is discovered during laboratory evaluation(3). Other cases such as chronic pyelonephritis, steroid resistant nephrotic syndrome(6,7), acquired immune deficiency syndrome (8), multiple myeloma(9), and sickle cell disease(3) have also been reported. In children, reported cases are from glomerulonephritis(2), chronic renal failure with or without hyperkalemia(10) and systemic lupus erythematosus(11). This syndrome can occur in the infancy period, presenting with lactic acidosis, deafness, mental retardation(12) or urinary salt wasting(13).

The explanations for decreased renin secretion include juxtaglomerular apparatus pathology (14), defective prostacyclin production, impaired conversion of prorenin to renin(15), and chronic expansion of extracellular fluid volume(16). The explanation for decreased aldosterone is not clear. Three major possibilities are postulated. Firstly, it is secondary to hyporeninemia. Secondly, there is a primary defect in the adrenal gland. Lastly, there are two coexistent primary defects: one in the adrenal gland and the other in the kidney(3).

According to this patient, although plasma renin and aldosterone assay are not available at our hospital, the calculation of TTKG (transtubular potassium gradient) is valid as a useful indicator of renal aldosterone bio-activity(17). The principle of TTKG is to determine the maximum tubular fluid K^+ concentration which presents at the end of the cortical collecting tubule. At this site the urine osmolality is similar to that of the plasma. No further K^+ secretion or excretion occurs. The TTKG in normal subjects is 8 to 9. It should be higher in the case of hyperkalemia due to increased K^+ secretion. A value below 7 and particularly below 5 in a hyperkalemic patient is highly suggestive of hypoaldosteronism(18). The low TTKG value of our patient (2.25), despite high serum potassium, indicated that the most likely cause of hyperkalemia was hypo- or pseudohypoaldosteronism.

The mainstay of treatment for this condition is mineralocorticoid that is usually in the form of fludrocortisone acetate. Other kinds of therapy include those that can lower serum potassium such as diuretics, sodium bicarbonate and sodium polystyrene sulfonate(3). In the present patient, short term administration of glucose, insulin, sodium polystyrene sulfonate, sodium bicarbonate and hydrochlorothiazide successfully lowered serum potassium without the use of fludrocortisone acetate. After discontinuation of these drugs, serum potassium remained stable in the normal range. This can be explained by the nature of the causative disease, acute glomerulonephritis, which is self-limiting and has a favorable recovery rate.

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REFERENCES

1. Cole BR, Salinas-Madrigal L. Acute proliferative glomerulonephritis and crescentic glomerulonephritis. In: Barratt TM, Avner ED, Harmon WE, eds. Pediatric nephrology. 4th ed. Baltimore: William and Wilkins, 1999: 669-89.
2. Don BR, Schambelan M. Hyperkalemia in acute glomerulonephritis due to transient hyporeninemic hypoaldosteronism. Kidney Int 1990; 38: 1159-63.
3. De Fronzo RA. Hyperkalemia and hyporeninemic hypoaldosteronism. Kidney Int 1980; 17: 118-34.
4. Villaria JG, Nunez JFM, Miralles JM, del Pozo DC, Romo T. Hyporenicnemic hypoaldosteronism in diabetic patients with chronic renal failure. Am J Nephrol 1988; 8: 127-37.
5. Schambelan M, Sebastian A, Biglieri EG, et al. Prevalence, pathogenesis, and functional significance of aldosterone deficiency in hyperkalemic patients with chronic renal insufficiency. Kidney Int 1980; 17: 89-101.
6. Weidmann P, Reinhart R, Maxwell MH, Rowe P, Coburn JW, Massry SG. Syndrome of hyporeninemic hypoaldosteronism and hyperkalemia in renal desease. J Clin Endocrinol Metab 1973; 36: 965-77.
7. Usberti M, Gazzotti RM. Hyporeninemic hypoaldosteronism in patients with nephrotic syndrome. Am J Nephrol 1998; 18: 251-5.
8. Kalin MF, Poretsky L, Seres DS, Zunoff B. Hyporeninemic hypoaldosteronism associated with acquired immune deficiency syndrome. Am J Med 1987; 82: 1035-8.
9. Shaked Y, Blau A, Shpilberg O, Samra Y. Hyporeninemic hypoaldosteronism associated with multiple myeloma : 11 years of follow-up. Clin Nephrol 1993; 40: 79-82.
10. Rodriguez-Soriano J, Vallo A, Sanjurjo P, Castillo G, Oliveros R. Hyporeninemic hypoaldosteronism in children with chronic renal failure. J Pediatr 1986; 109: 476-82.
11. Kozeny GA, Hurley M, Fresco R, Vertuno LL, Bansal VK, Hano JE. Systemic lupus erythematosus presenting with hyporeninemic hypoaldosteronism in a 10-year-old girl. Am J Nephrol 1986; 6: 321-4.
12. Shuper A, Eisenstein B, Stark H, Varsano I. Hyporeninemic hypoaldosteronism in a child with lactic acidosis, deafness and mental retardation. J Pedaitr 1982; 100: 769-72.
13. Landier F, Guyene TT, Boutignon H, Nahoul K, Corvol P, Job J-C. Hyporeninemic hypoaldosteronism in infancy : A familial disease. J Clin Endocrinol Metab 1984; 58: 143-8.
14. Phelps KR, Lieberman RL, Oh MS, Carroll HJ. Pathophysiology of the syndrome of hyporeninemic hypoaldosteronism. Metabolism 1980; 29: 186-99.
15. Nadler SL, Lee FO, Hsueh W, Horton R. Evidence of protacyclin deficiency in the syndrome of hyporeninemic hypoaldosteronism. N Engl J Med 1986; 314: 1015-20.
16. Oh MS, Carroll HJ, Clemmons JE, Vagnucci AH, Levison SP, Whang ESM. A mechanism for hyporeninemic hypoaldosteronism in chronic renal disease. Metabolism 1974; 23: 1157-66.
17. Rodriguez-Soriano J, Ubetagoyena M, Vallo A. Transtubular potassium concentration gradient : A useful test to estimate renal aldosterone bio-activity in infants and children. Pediatr Nephrol 1990; 4: 105-10.
18. Rose BD. Clinical physiology of acid-base and electrolyte disorder, 5th ed. New York : McGraw Hill Inc, 2001: 912-3.

ภาวะโปแตสเซี่ยมสูงในเลือดชั่วคราวร่วมกับฮัยโปอัลโดสเตอโรนิซึมในผู้ป่วยไตอักเสบเฉียบพลัน : รายงานผู้ป่วย 1 ราย

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ผู้ป่วยเด็กชายไทย อายุ 7 ปี ได้รับการวินิจฉัยว่าเป็นไตอักเสบเฉียบพลัน ร่วมภาวะกับไตวายเฉียบพลันในระยะเริ่มแรกของโรค แต่ในขณะที่การทำงานของไตและอาการต่าง ๆ ของผู้ป่วยเริ่มดีขึ้น กลับตรวจพบระดับโปแตสเซี่ยมในเลือดสูงขึ้นชั่วคราว และกลับเป็นปกติเองหลังจากได้รับการรักษาแบบประคับประคอง โดยอาศัยผล transtubular potassium gradient (TTKG) ที่ต่ำลง ผู้รายงานสรุปว่าสาเหตุของโปแตสเซี่ยมในเลือดที่สูงนี้ เกิดจากภาวะ hypoaldosteronism

คำสำคัญ : ระดับโปแตสเซี่ยมสูงในเลือด, ไตอักเสบเฉียบพลัน, ระดับเรนินและอัลโดสเตอโรนต่ำในเลือด

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