Nephrocalcinosis Associated with Primary Aldosteronism

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Dear Sir,

It has been known that prolonged hypokalemia is associated with renal interstitial fibrosis, renal cyst formation and impairment of renal function [1]; however, nephrocalcinosis associated with primary aldosteronism is rarely reported [2]. A 45-year-old woman was admitted to our hospital because of intermittent muscle cramps, polyuria and polydipsia which developed 7 years earlier. At admission, blood pressure was 200/110 mm Hg. Biochemical findings indicated sodium 146 mEq/l, potassium 2.2 mEq/l, chloride 109 mEq/l, BUN 8.6 mg/dl, creatinine 0.8 mg/dl, total protein 7.8 g/dl, albumin 4.4 g/dl, AST 19 IU/l, ALT 13 IU/l, alkaline phosphatase 113 IU/l, calcium 8.9 mg/dl, phosphorus 2.5 mg/dl, magnesium 2.2 mg/dl. Twenty-four hour urine collections indicated sodium 135 mEq, potassium 50 mEq, calcium 102 mg, phosphorus 168 mg, magnesium 4.2 mg. Arterial blood gas analysis showed pH 7.42, pO₂: 89.5 mm Hg, HCO₃⁻ 29.5 mm Hg, PCO₂ 44.7 mm Hg, and urine gas analysis showed pH 6.87, P0₂ 119.4 mm Hg, PCO₂ 36.6 mm Hg, HCO₃⁻ 9.2 mm Hg. The hormone study revealed PTH 0.24 ng/ml (normal: <0.88 ng/ml), 25-(OH) vitamin D 11.6 ng/ml (normal: 9-42 ng/ml), ACTH 3.0 pg/ml (normal: <37 pg/ml), 24-hour urine cortisol 57.8 µg/day (normal: 10-80 µg/day), metanephrine 1.0 mg/day (normal: up to 1.2 mg/day), VMA 4.1 mg/day (normal: below 5 mg/day). Plasma renin activity and aldosterone concentration before and after adrenalectomy are shown on table 1.

Intravenous pyelography findings were unremarkable. The computed tomography of the abdomen showed bilateral medullary calcification and adrenal mass (2×1 cm) consistent with adrenal tumor (fig. 1, 2). Adrenalectomy was performed on the 9th hospital day, and clinical symptoms, blood pressure and hypokalemia improved shortly after operation.

This case shows that prolonged hypokalemia in primary aldosteronism can be one of the causes of nephrocalcinosis. The causes of nephrocalcinosis are primary hyperparathy-

Table 1. Laboratory finding
Preoperative

Postoperative

PRA, ng/ml/h

Basal

Stimulated Aldosterone, ng/dl

Basal

Stimulated

0.07 (0.15-2.33) 0.40(1.31-3.95)

81.0(1-16) 61.5(4-31)

0.22

1.4

Values in parentheses represent normal values. PRA = Plasma renin activity.

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Fig. 1. Abdomen CT shows multiple, patchy calcifications which are confined to the medulla.

Fig. 2. Abdomen CT shows a hypodense mass at left adrenal gland (arrows).

roidism, milk-alkali syndrome, vitamin D intoxication, sarcoidosis, renal tubular acidosis, primary oxalosis, renal infarct, renal tuberculosis, medullary sponge kidney, magnesium deficiency and prolonged use of diuretics. However, in our case, the listed causes were excluded by clinical history, biochemical findings, hormone and radiologic findings.

It has been known that chronic potassium deficiency results in progressive tubulointerstitial injury. This is associated with augmented ammoniogenesis, and increased renal ammonia levels contribute to hypo-kalemic nephropathy through ammonia-mediated activation of an alternative comple-

ment pathway [3]. The interstitial inflammation in the renal medulla, cyst formation, and calcification may all be part of the same process, possibly as a result of increased renal ammoniogenesis and medullary alkalization, common to conditions such as hypokalemia and distal tubular acidosis.

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Primary Aldosteronism and Nephrocalcinosis