Letter to the Editor

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Nephrocalcinosis Associated with Primary Aldosteronism

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Dear Sir,

It has been known that prolonged hypo-kalemia 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, PO2 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 G/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), metane-phrine 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

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<th>Basal Stimulated Aldosterone, ng/dl</th>
<th>Basal Stimulated</th>
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<tr>
<td>0.07 (0.15-2.33)</td>
<td>0.40(1.31-3.95)</td>
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<td>81.0(1-16)</td>
<td>61.5(4-31)</td>
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<td>0.22</td>
<td>1.4</td>
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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).

It has been known that chronic potassium deficiency results in progressive tubulo-interstitial injury. This is associated with augmented ammoniagenesis, and increased renal ammonia levels contribute to hypo-kalemic nephropathy through ammonia-mediated activation of an alternative complement 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 ammoniagenesis and medullary alkalization, common to conditions such as hypokalemia and distal tubular acidosis.

**Acknowledgement**

We would like to thank Dr. Torres at the Mayo Clinic for reviewing this case regarding primary aldosteronism as a cause of nephro-calcinosis.

**References**

