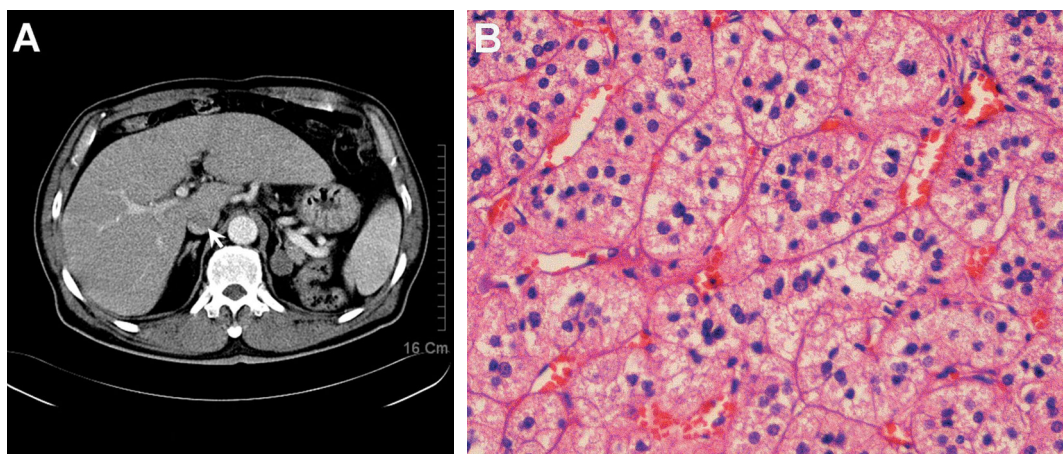


## Primary Aldosteronism Presenting with Hypertension and Quadriparesis

Cheng-Yang Hsieh and Tzu-Tung Tsai



**Figure.** Abdominal CT with contrast showed a nodule, 1.5 cm in size, in left adrenal gland (arrow in A). Microscopic finding (200X) of excised left adrenal gland tumor showed packed polygonal cells with foamy cytoplasm, compatible with adrenal adenoma (B).

A 64 years old hypertensive man presented with progressive quadriparesis for 1 week. Review of his medication revealed Nifedipine 30 mg/day only. On examination, he had high blood pressure (179/115 mm Hg) with weakness of neck extensor and proximal four limbs symmetrically. Significant laboratory abnormalities included serum potassium 2.1 mmol/L and creatine kinase 1089 U/L (normal reference [NR]: 50-350 U/L). Serum creatinine, glucose, calcium, chloride, sodium, cortisol and thyroid hormone were normal. Twenty-four-hour urine collection revealed increased urinary potassium excretion (138.0 mmol/day). Analysis of arte-

rial blood gas (ABG) under room air showed metabolic alkalosis with respiratory compensation ( $\text{pH} = 7.58$ ,  $\text{pCO}_2 = 51.2$  mmHg,  $\text{HCO}_3^- = 48.3$  mEq/L, and  $\text{SaO}_2 = 97.5\%$ ).

The basal aldosterone was high (972 pg/mL, NR: 37-240 pg/mL), with suppressed plasma renin concentration (0.28 ng/mL, NR: 3.10-37.00 ng/mL). Abdominal computed tomography showed a nodule, 1.5 cm in size, in the left adrenal gland (Fig. A). Adrenalectomy was done and the pathological diagnosis was adrenocortical adenoma (Fig. B). His weakness, hypertension and hypokalemia all got cured after opera-

From the Department of Neurology, Cheng-Kung University Hospital, Tainan, Taiwan.

Received June 6, 2007. Revised and Accepted June 7, 2007.

Reprint requests and correspondence to: Tzu-Tung Tsai, MD, Department of Neurology, National Cheng Kung University Hospital, No. 138, Sheng-Li Road, Tainan, Taiwan. E-mail: ttttsai@mail.ncku.edu.tw

tion.

Primary aldosteronism is hypersecretion of mineralocorticoid aldosterone due to adrenal gland pathology and its mostly frequent cause is unilateral adrenal adenoma<sup>(1)</sup>. It increases excretion of potassium and hydrogen ion via urine, mild retention of sodium and water in the body, and results in hypertension, metabolic alkalosis and hypokalemia in our patient. Hypokalemic weakness, either presents in the form of persistent or periodic paralysis, is a common symptom of primary aldosteronism<sup>(2)</sup>. But on the contrary, primary aldosteronism is a less frequent etiology of hypokalemic weakness encountered in daily practice, though no definite frequency available in the literature. Thorough survey of patient's hypertension and hypokalemia leads to the diagnosis of such a surgically curable disease. Further studies for aldosteronism

may be needed for patients presenting with hypokalemic weakness and high blood pressure, particularly in the oriental population<sup>(3)</sup>.

#### References:

1. Williams GH, Dluhy RG. Disorders of the adrenal cortex: Primary aldosteronism. *Harrison's Principle of Internal Medicine*, 15ed. New York: McGraw-Hill, 2001:2095-7.
2. Ropper AH, Brown RH. The metabolic and toxic myopathies: primary aldosteronism. *Adams and Victor's Principles of Neurology*, 8ed. New York: McGraw-Hill, 2005;1238.
3. Huang YY, Hsu BR, Tsai JS. Paralytic myopathy-a leading clinical presentation for primary aldosteronism in Taiwan. *J Clin Endocrinol Metab* 1996;81:4038-41.