

*Images in Nephrology*  
(Section Editor: G. H. Neild)

## A rare cause of secondary hypertension

Chin-Chi Kuo<sup>1</sup>, Vin-Cent Wu<sup>1</sup>, Ching-Wei Tsai<sup>2</sup>, Fan-Fen Wang<sup>3</sup>, Shih-Chieh Chueh<sup>4</sup> and Kwan-Dun Wu<sup>1</sup>

<sup>1</sup>Department of Internal Medicine, National Taiwan University Hospital, <sup>2</sup>Department of Internal Medicine, China Medical University Hospital Taipei Branch, <sup>3</sup>Division of Endocrinology and Metabolism, Taipei City Hospital Yang-Ming Branch and <sup>4</sup>Department of Urology, National Taiwan University Hospital, Taipei, Taiwan

**Keywords:** adrenocortical carcinoma; aldosteronism

A 20-year-old obese man was referred to our hospital with 1-year hypertension and mild hypokalaemia (potassium, 3.3 mmol/l). The ratio of postcaptopril plasma aldosterone concentration (PAC) to plasma renin activity (PRA) was high [25.8 (ng/dl)/0.33 (ng/ml/h) = 77.4]. The PAC post-saline loading test remained high (28.1 ng/dl). The 24-h urinary catecholamines and VMA (vanillyl mandelic acid) were within normal limits. Primary aldosteronism was diagnosed, and a computed tomography disclosed an iso-dense mass with a maximal diameter of 4 cm over the right adrenal gland (Figure 1). The patient then underwent right laparoscopic adrenalectomy. Grossly, a well-encapsulated 4.5 × 4.0 × 3.5 cm yellowish tumour with central necrosis was noted. Histologically, the tumour was composed of pleomorphic cells with high-grade nuclei, prominent nucleoli, and >1/3 of the tumour presenting patternless sheets of cells. Furthermore, the mitotic count was >5 in 50 high power fields with atypical mitotic figures (Figure 2a and b). Adrenocortical carcinoma (ACC) was diagnosed according to the modified Weiss classification system [1]. Subsequent immunohistochemical studies confirmed that the tumour was aldosterone producing (Figure 2c and d). Two months after the operation, PAC and

PRA were 32.7 ng/dl and 25.45 ng/ml/h, respectively, and the patient became normotensive.

ACCs are rare and account for an estimated 0.05–0.2% of all malignancies [2]. Hormonally functioning tumours occur in ~50% of ACC patients. Nevertheless, the aldosterone-producing ACC are even rarer. There is a bimodal occurrence by age, with a peak incidence at <5 years, and a second peak between 40 and 50 years [3]. The differentiation between benign and malignant neoplasms is often difficult by preoperative image. However, a tumour size of >4 cm should raise clinical suspicion of adrenocortical malignancy, even at a young age.

*Acknowledgement.* The authors would like to thank Dr Fan-Fen Wang for her medical technical support in immunohistologic staining. This report was financially supported by The Ta-Tung Kidney Foundation.

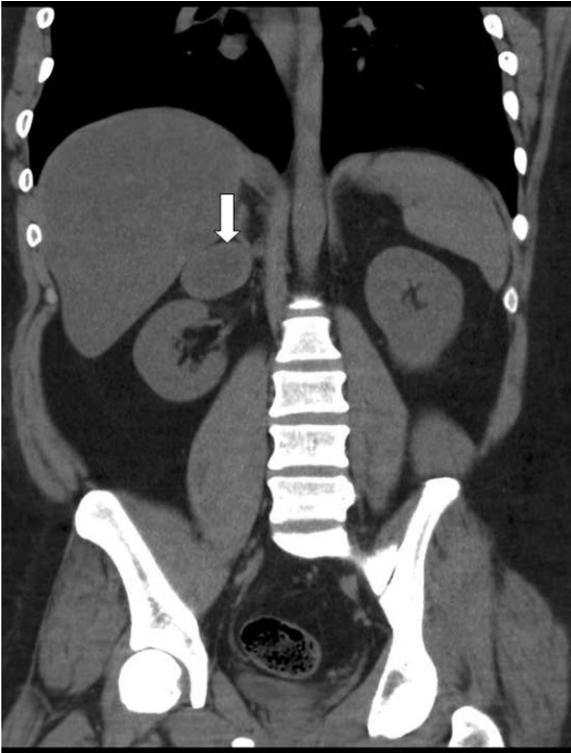
*Conflict of interest statement.* None declared.

### References

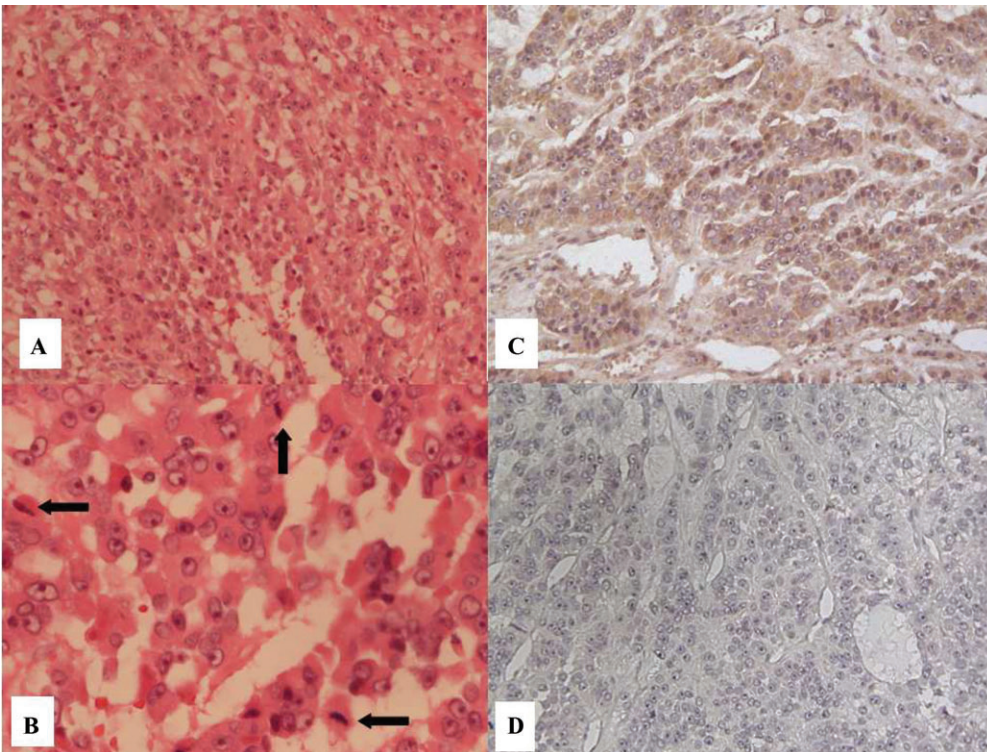
1. Medeiros LJ, Weiss LM. New developments in the pathologic diagnosis of adrenal cortical neoplasms. A review. *Am J Clin Pathol* 1992; 97: 73–83
2. Wajchenberg BL, Albergaria Pereira MA, Medonca BB, *et al.* Adrenocortical carcinoma: clinical and laboratory observations. *Cancer* 2000; 88: 711–736
3. Boushey RP, Dackiw AP. Adrenal cortical carcinoma. *Curr Treat Options Oncol* 2001; 2: 355–364

*Received for publication:* 8.12.08  
*Accepted in revised form:* 29.12.08

*Correspondence and offprint requests to:* Kwan-Dun Wu, Department of Internal Medicine, National Taiwan University Hospital, 7 Chung-Shan South Road, Taipei, Taiwan. Tel: +886-2-23123456-5014; Fax: +886-2-23934176; E-mail: kdwu@ntuh.gov.tw



**Fig. 1.** Adrenal tumour. Non-contrast CT scan shows an oval tumour, 4 cm in diameter, in the right adrenal region.



**Fig. 2.** Histopathology of adrenocortical carcinoma. (a) Pleomorphic tumour cells form patternless sheets of cells. (b) Tumour cells with eosinophilic cytoplasm, high nuclear grade and atypical mitotic figures (arrows) (haematoxylin and eosin stain, 100 $\times$ ; 400 $\times$ ). (c) Positive immunohistochemical staining for aldosterone (100 $\times$ ). (d) Substitution of non-immune serum for the primary antibody eliminated the signal as negative control (100 $\times$ ).