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Adrenocorticotrophic hormone independent macronodular adrenal hyperplasia

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DESCRIPTION

Figure 1 is of a CT scan showing bilateral macronodular adrenal glands in a patient with adrenocorticotrophic hormone-independent macronodular adrenal hyperplasia (AIMAH).

AIMAH was first described in 1964¹.

AIMAH is often discovered as a fortuitous radiological finding, as in our patient, or during investigation for endogenous hypercortisolism. AIMAH represents less than 1% of patients with endogenous Cushing's syndrome.

Most AIMAH patients are diagnosed in the fifth and sixth decades.

CT imaging normally shows bilateral adrenal nodules, which sometimes grow to as large as 5 cm, often with a combined adrenal weight of more than 60 g². Although recent AIMAH data suggest that larger adrenal nodules accumulate an increased number of genomic and transcript

abnormalities,³ AIMAH is a benign process that has so far not been shown to become malignant.

The secretion of cortisol in most patients with AIMAH is under the influence of hormones other than adrenocorticotrophic hormone, which stimulate aberrantly expressed receptors in the adrenal glands.² Examples include G-protein coupled receptors for glucose-dependent insulinotropic peptide, β -adrenergic receptors, vasopressin (V2, V3) receptors, serotonin (5-hydroxytryptamine) receptors and leptin receptors.

La Croix has described useful protocols for identifying various aberrant receptors as well potential medical treatment options.² Treatment for AIMAH includes specific blockade of aberrant receptors where applicable. When specific blockade of aberrant receptors is not possible, unilateral adrenalectomy is recommended in those patients with moderate steroid hormone excess. In patients with

Figure 1 CT scan of the adrenal glands.

very high levels of steroid hormone production, bilateral adrenalectomy is the treatment of choice.

Competing interests None.

Patient consent Not obtained.

REFERENCES

1. **Kirshner MA**, Powel RD Jr, Lipsett MB. Cushing's Syndrome: nodular cortical hyperplasia of adrenal glands with clinical and pathological

features suggesting adrenocortical tumour. *J Clin Endocrinol Metab* 1964;**24**:947–55.

2. **Lacroix A**. ACTH-independent macronodular adrenal hyperplasia. *Best Pract Res Clin Endocrinol Metab* 2009;**23**:245–59.
3. **Almeida MQ**, Harran M, Bimpaki EI, *et al*. Integrated genomic analysis of nodular tissue in macronodular adrenocortical hyperplasia: progression of tumorigenesis in a disorder associated with multiple benign lesions. *J Clin Endocrinol Metab* 2011;**96**:E728–38.

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