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CASE REPORTS

Primary hyperaldosteronism- a resistant endocrine hypertension- A case report

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Abstract

A thirty years old lady presented with uncontrolled hypertension and dizziness during working for last 5 years. She had history of termination of pregnancy due to treatment-resistant hypertension. Even in the post-partum period her blood pressure remains uncontrolled with more than three antihypertensive drugs. Her serum K^+ level was low and USG of KUB & adrenal glands showed slightly increased right renal cortical echotexture; CT scan of abdomen revealed right Adrenal adenoma. She underwent unilateral adrenalectomy. Now, she has no complaints and her blood pressure is normal without any antihypertensive drugs. There is no target organ damage.

Key-words

Primary hyperaldosteronism, unilateral adrenal adenoma, resistant endocrine hypertension

Introduction

Recently, an increasing body of evidence suggests that aldosterone is an independent risk factor for cardio-vascular disease based on many clinical and experi-mental studies.[1–5] Growing lines of evidence have consistently supported the view that secondary hypertension due to primary aldosteronism is much more frequent (8–20%) than previously suspected .[6–10] It is important to diagnose primary aldosteronism during the early stage before the progression to end-organ damage because primary aldosteronism due to unilateral adrenal lesion, mostly aldosterone producing adenoma, can be cured by surgery.[11] We herein describe a case with primary aldosteronism due to right adrenal adenoma on CT scan- a resistant endocrine hypertension who was diagnosed and treated successfully in BSMMU.

Case summary

Mrs. Arifa begum, a non diabetic 30 years old house wife, mother of two children from Mirershari, Chittagong, experienced dizziness during working about 5 years back and found hypertensive (stage-02). Initially she was treated with two antihypertensive drugs (Amlodipin-5mg, and atenolol-50mg). But her blood pressure was poorly controlled with the above drugs. During first trimester of her second pregnancy, her blood pressure was difficult to control and she had to terminate at 6 months of her pregnancy due to treatment-resistant hypertension. Even in post partum period three antihypertensive drugs could not control her Blood pressure and she developed generalized weakness, polyuria, nausea and vomiting. Her physical examinations revealed hypertension and generalized motor weakness (grade-0/5) areflexia.

Her investigations revealed-Normal CXR; ANA-negative; and S. Electrolytes showed hypokalemia (serum electrolytes-Na^+ 146.6 mmol/L, K^+ 2.56 mmol/L, Cl^- 109.0 mmol/L and HCO_{3-} 25 mmol/L) and urinary electrolytes showed increased urinary excretion of potassium (Na^+ 63.6 mmol/l/24 hrs, k^+ 20.75mmol/l/24 hrs. serum aldosterone level of 2.5 nmol/l (normal: 0.1-0.97 nmol/l) in the presence of normal cortisol levels. Routine blood and urine analysis was normal; RA test: negative; S. creatinine 1.1 mg/dl, fasting blood sugar 5.5 mmol/L & random blood sugar 5.7 mmol/L; ESR-10 mm in 1st hr; USG of KUB & adrenal glands showed slightly increased right renal cortical echotexture. CT scan of abdomen revealed right Adrenal adenoma.

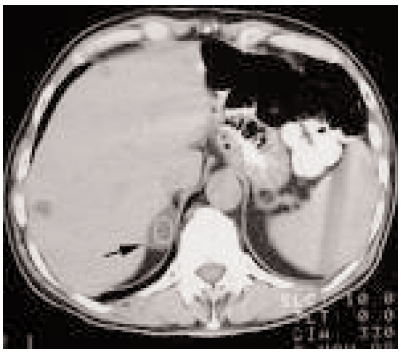

Figure : CT scan of the abdomen
(Arrow shows right adrenal adenoma)

So, she underwent unilateral right-sided adrenalectomy on 22.03.2008 in BSMMU.

Histopathology report

Specimen: Right adrenal gland: consists of a slice of gray white piece of tissue measuring 3.6×2 cm with a thickness of 0.3 cm, containing a nodular area measuring 1.6 cm in diameter. Cut surface was yellowish. Three blocks are embedded. Microscopic appearance of the specimen revealed a benign tumor composed of cells with vacuolated cytoplasm. Most of the tumor cells were uniform in size and shape. Some of the tumor cells showed mild pleomorphism. No capsular invasion was seen. Features were compatible with adrenocortical adenoma.

After operation, her Serum Electrolytes showed normal (Na^+ 145.4 mmol/L, K^+ 4.29 mmol/L, Cl^- 111.7 mmol/L); Echo: IVSd: 10mm, LVPWd 8mm, LVIDd 40mm EF 67.9%with no regional wall motion abnormality and fundoscopy showed normal findings .Now, she has no complaints and her blood pressure is normal without any antihypertensive drugs. There is no target organ damage.

Discussion

We present here a case of primary hyperaldosteronism- a cause of uncontrolled hypertension due to unilateral right-sided adrenal adenoma. The diagnosis of primary aldosteronism in the present case was most likely because of multidrug resistant hypertension associated with low serum k^+ level. Our patient has been hypertensive since last five years with past history of termination of pregnancy due to uncontrolled hypertension and her hypertension was poorly controlled by a number of antihypertensive drugs. The diagnosis of primary aldosteronism due to unilateral adrenal pathology, including unilateral adrenal hyperplasia (UAH), unilateral multiple adrenal micro-nodules (UMN), and unilateral adrenal adenoma is clinically important but difficult to localize with the conventional diagnostic procedures except CT scan. Now a days selective adrenal venous sampling (SAVS) is performed in those patients with primary aldosteronism with apparently normal adrenal gland whether hypersecretion of aldosterone is from unilateral or bilateral source .If it is bilateral, the diagnosis of IHA is made with first line of medical treatment using aldosterone receptor antagonists in combination with or without other antihypertensive drugs. If it is unilateral, the diagnosis of unilateral adrenal pathology including UMN, UAH and microadenoma and small adenoma is most likely, and surgical treatment (unilateral adrenalectomy) is recommended. Therefore, patients with unilateral adrenal adenoma may be misdiagnosed as either low-renin essential hypertension or IHA, and treated with antihypertensive drugs for long periods as in the present case. Distinct from IHA, hypertension caused by unilateral adrenal adenoma apparently responds to unilateral adrenalectomy.[12] Her pre operative treatment-resistant hypertension was well controlled without any antihypertensive drugs after removal of the affected right adrenal gland.

Conclusion

In conclusion, this young female patient was a case of primary aldosteronism due to unilateral adrenal adenoma, in which CT scan proved to be a useful diagnostic tool for its localization. It is important to diagnose primary aldosteronism during the early stage before the progression to end-organ damage because primary aldosteronism due to unilateral adrenal lesion, mostly aldosterone producing adenoma, can be cured by surgery. However, a long-term follow-up is needed to observe whether or not aldosterone hypersecretion may recur from the 'apparently normal' adrenal gland left intact.

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