Incidentaloma

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Abstract

An incidentaloma is a benign tumor found coincidentally without relevant clinical symptoms or suspicion, diagnosis of which has increased with increased use of modern imaging technology becoming a common management scenario. Incidentaloma (Conn’s syndrome) is a disease of the adrenal glands involving the excess production of aldosterone and patient usually presents with clinical features of hypokalemia and hypertension. Atypical clinical presentations with a combination of normotension and normokalemia are described in few cases. In this case report, the patient presented with a headache, abdominal pain, and essential hypertension only. To rule out other pathology ultrasonography was performed and underlying adrenal adenoma was found. Vigorous control of blood pressure using adequate antihypertensive medications subsequently lead to symptomatic relief.

Key words: Adrenal adenoma, Essential hypertension, Normokalemia, Primary hyperaldosteronism, Ultrasonography

INTRODUCTION

The finding of adrenal incidentalomas (AI) is reported to be as high as 8% and 4% in autopsy series and radiologic series, respectively.¹,² Primary hyperaldosteronism (HA) is the most common form of endocrine hypertension and is now considered as the most common cause of secondary hypertension.³ It usually presents with features of hypokalemia such as muscle cramps, weakness, tetany, and severe or resistant arterial hypertension. However, there is a varied spectrum of presentation with clinical and biochemical variations in primary HA. In particular, hypokalemia is not evident in up to one-third of cases, and normotensive primary HA is rare.⁴,⁵ Other than these variations, primary HA rarely presents in an emergency room with a catastrophic complaint constituting a diagnostic dilemma.⁶ The consideration of size and radiographic appearance of the mass are of particular importance. Suspicious adrenal masses of ≥4 cm are advised surgical removal. Masses <4 cm if found to be hormonally active, should also be removed but usually recommended for observation for an increase in size.⁷

CASE REPORT

A 70-year-old female arrived in the Medicine Department with a recurrent headache since 2 years associated with myalgia, backache, history of on and off abdominal pain, and bilateral swelling on legs. History of hypertension was recorded on a couple of occasions during past 2 years, but no treatment of hypertension was prescribed and advised follow-up. There was no history of thyroid illness, connective tissue disease, and similar complaints. The patient had undergone hysterectomy and cholecystectomy 21 and 10 years back, respectively. Clinical examination revealed that it is a case of hypertension with pitting edema and no palpable abdominal mass. Neurological examination revealed full strength in all muscles with normal deep tendon jerks. The patient was under observation for blood pressure monitoring, and it was recorded a maximum of 164/90 mm of Hg and a minimum of 146/80 mm of Hg without any use of antihypertensive drugs. Serum electrolytes reports of sodium, potassium, and chloride were 134, 4, and 92 meq/L, respectively. Thyroid function tests, urine routine microscopy, and renal function tests were normal. Electrocardiography showed left bundle branch block without changes of hypertension or hypokalemia.
Ultrasonography (USG) of the abdomen showed a 2.3 cm × 1.3 cm hypoechoic, round lesion in the left adrenal gland. Computed tomography scan abdomen confirmed the left adrenal adenoma with nonhomogenous enhancement (Figure 2). Plasma renin activity (PRA) was 2.0 ng/mL/h, and plasma aldosterone was 405.4 pg/mL. Overnight dexamethasone test revealed suppressible cortisol levels. 24 h urinary vanilmandelic acid measured 1.12 mg/24 h. Various biochemical markers measured with optimum precautions, their levels are adrenocorticotropic hormone 25 pg/ml, adrenaline 1.52 pg/ml, noradrenaline 1.42 pg/ml, and cortisol 109 ng/ml. Diagnosis of Conn’s syndrome was established by hypertension, raised plasma aldosterone, decreased PRA, and adrenal adenoma on magnetic resonance imaging.

Management
The patient was prescribed a combination of the antihypertensive medications angiotensin receptor blocker and diuretic (telmisartan and hydrochlorothiazide, 40/12.5 mg). The reduction in blood pressure lead to a subsequent headache relief within 7 days. The patient has been advised a routine USG every 6 months to check progression of the tumor for further management of disease.

DISCUSSION
Patients suspected to have primary HA used measures the plasma aldosterone concentration (PAC) to PRA ratio as the first investigation to support a suspicion of the disease. A high ratio of PAC to PRA (202.73), i.e., >25 signifies HA if the aldosterone concentration is >15 ng/dL. HA should be screened for in every hypertensive patient with AI. Traditionally, HA has been clinically associated with hypokalemia and hypertension. However, normokalemia occurs in up to 50% of patients with HA. Thus, hypokalemia is not a useful marker for the purpose of screening. The best screening test is upright PAC to plasma aldosterone renin ratio. Primary HA is now increasingly being recognized as the underlying cause in patients suffering with essential hypertension.
The likelihood of primary HA seems directly related to the severity of hypertension with increased incidence in patients with severe hypertension and lesser (<2%) with mild hypertension. An initial increase in sodium reabsorption related to the effect of aldosterone on distal renal tubules explains the arterial hypertension of primary HA. Aldosterone effect on the central nervous system, increased peripheral vascular resistance and increased vascular sensitivity to vasopressors such as angiotensin and adrenaline are factors responsible for higher blood pressure. The occurrence of normal blood pressure in primary HA is rare; in case of middle-aged females, the diagnosis is usually raised by clinical features of hypokalemia (fatigue, paresthesia, and tetany).

CONCLUSION

The varying clinical presentations of primary HA present a diagnostic dilemma to the clinicians. The usual approach of hypertension with hypokalemia to suspect this condition may be missing in many borderline hypertensive and normotensive cases. Atypical presentations are increasingly being described. All cases of hypertension must be screened for this condition.

REFERENCES


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