Role of Adrenal Venous Sampling and Radiological Imaging in Primary Hyperaldosteronism: A Case Report
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DOI: 10.36348/sjmps.2020.v06i05.006 | Received: 04.05.2020 | Accepted: 25.05.2020 | Published: 30.05.2020

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Abstract

We present a case of a 63-year-old man who was evaluated for consistent hypokalemia and longstanding uncontrolled hypertension. Iatrogenic hypokalemia was excluded. He underwent a series of laboratory investigations which showed high levels of aldosterone and increased aldosterone/renin ratio. Renal/adrenal ultrasound (US) showed a mass located in the right adrenal gland, which was presumed to be an adenoma. Adrenal Venous Sampling (AVS) was carried out to establish the diagnosis and secretory ability of the mass but the results showed a paradoxical low aldosterone secretion from the adrenal mass. The patient was further investigated with magnetic resonance imagining (MRI), which confirmed the adrenal mass and on the basis of which he underwent laparoscopic adrenalectomy that resulted in the normalization of aldosterone, serum potassium and blood pressure.

Keywords: Adrenal, Hyperaldosteronism, Hypertension, Incidentaloma, Aldosterone, Adrenal venous sampling, Adrenalectomy.

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INTRODUCTION

Primary hyperaldosteronism (PHA) is one of the most common causes of secondary hypertension [1]. It can cause considerable cardiovascular and cerebrovascular morbidity and mortality if it remains undiagnosed and untreated [2-4]. Patients with PHA usually have a vague presentation and therefore present a challenging scenario. The most common subtypes of primary aldosteronism are bilateral adrenal hyperplasia and aldosterone producing adenomas, other rarer forms such as unilateral adrenal hyperplasia and glucocorticoid responsive aldosteronism and the diagnosis of these subtypes can be clinical challenging [5]. Adrenal Venous Sampling (AVS), which is considered golden standard for the diagnosis of PHA, for confirmation of the secretory function of the adrenal lesion [6]. This case report is of a particular interest; firstly because of AVS showing paradoxical levels of aldosterone secretion, and secondly supplementing the role of AVS with radiological modalities which may be of great help in diagnosing and treating patients with such lesions.

PRESENTATION

A 63-year-old gentleman was referred to our clinic for the evaluation of unexplained hypokalemia. He had a history of uncontrolled hypertension for 20 years with two episodes of hypertensive emergency in the previous two years. He had no symptoms or other complains at presentation. He did not report any other medical disorder. He had no history of chronic ingestion of Licorice. His family history was not significant for hypertension, endocrine disease or renal disease and he did not report any allergies. He was taking Adalat (Nifedipine) 30 mg, Losartan 100 mg, Atenolol 100 mg and Potassium Chloride extended release 750 mg 2 tablets every 12 hours.

Physical examination did not yield any findings. However, his blood pressure (B.P) was slightly elevated (145/90mm Hg).

Laboratory investigations were conducted. Serum electrolytes showed a low level of potassium 3.2mmol/L (Normal: 3.5-5.3mmol/L), sodium 140 (Normal: 133-146mmol/L) and normal levels of chloride 103mEq/L (95-108mmol/L) and bicarbonate 28meq/L (22-29mEq/L).
Renal function tests showed a high normal level of serum urea 7.3 (2.5-7.8mmol/L) and a high level of serum creatinine 135microm/L (Normal: 64-104umol/L). There was a moderate decrease in estimated GFR (eGFR) 48ml/min/1.73m^2 (Normal: 90+). Urinalysis, Serum protein electrophoresis (SPEP) and 24hr urine creatinine level were normal.

Differential diagnosis under consideration were Cushing’s disease/syndrome, primary hyperaldosteronism and renal artery stenosis. Lack of clinical features and a normal cortisol level and Dexamethasone suppression test ruled out Cushing disease/syndrome. However, we found elevated serum aldosterone level 3818pm (Normal: 100-400pm) and a high aldosterone/renin ratio 1317 (Normal: <680).

We proceeded with renal/adrenal ultrasound (US) to look for an adrenal adenoma and/or hyperplasia. US demonstrated a right adrenal mass about 2.7x2.4x2.9cm over his right adrenal gland (Figure1). The patient was started on Spironolactone 100 mg besides Valsartan 100 mg and Atenolol 50 mg. His home BP readings improved as did his potassium level (about 3.7mmol/L).

We continued the investigations with AVS to confirm the right adrenal adenoma as a cause of his aldosterone excess and rule out a remote possibility of bilateral adrenal hyperplasia. To our surprise, his right AVS had the lowest concentration of aldosterone of all other locations sampled. His readings were as follows (in pmol/L):

Right adrenal sample: 455, Left adrenal sample: 1189
Right renal vein sample: 1217, Left renal vein sample: 1260

At this stage, we considered a diagnosis of bilateral adrenal hyperplasia with an incidental adenoma. We included Endocrinology team on board, in regard to his AVS results as the findings were paradoxical given the low aldosterone level on the right side. They considered a possibility of anomaly in sample taking and processing. They advised for further imaging for a detailed adrenal anatomy. The Radiology team deemed the MRI a reasonable imaging modality. MRI report showed a 2.9 x 2.5 cm right adrenal nodule with imaging features in keeping with an adenoma. The left adrenal gland was normal.

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DISCUSSION
Primary aldosteronism is one of the most common causes of secondary hypertension, accounting for 5% of cases of all patients with hypertension and for 10% of patients referred to hypertension units [1]. Diagnosis and treatment are of particular importance because primary aldosteronism is associated with a higher risk of cardiovascular and cerebrovascular events than is essential hypertension [2-4].

The diagnosis of primary aldosteronism comprises three steps: screening, confirmatory or exclusion testing, and subtype diagnosis [7-10]. This last step is of fundamental importance to allocate
patients to the correct management: unilateral adrenalectomy for aldosterone-producing adenoma and unilateral adrenal hyperplasia, or pharmacotherapy with mineralocorticoid receptor antagonists for individuals with bilateral adrenal hyperplasia or if the operation cannot be performed [7]. Subtype diagnosis comprises CT scanning and adrenal vein sampling [6].

Adrenal vein sampling is a demanding technique, requiring a skilled radiologist; however, for a procedure that is considered the golden standard in primary aldosteronism subtype diagnosis, reproducibility of the interpretation of the results between centers is poor, due to scarce standardization of protocols i.e. different criteria and protocols are used to define both cannulation of the adrenal veins and lateralization of aldosterone production [11-12].

The absence of a standardized and widely accepted adrenal vein sampling protocol for all centers has meant some clinicians are reluctant to do adrenal vein sampling or to refer patients to centers where this procedure is done [6].

A recent systematic review of the literature concluded that diagnosis of PHA, when it was only based on computed tomography (CT) or magnetic resonance imaging (MRI) data, led to inappropriate treatment in 37.8 % of patients [12]. However, AVS, when combined with CT or MRI may greatly enhance the ability to diagnose and treat primary hyperaldosteronism, as is seen in the present case.

CONCLUSIONS

AVS is considered gold standard for the diagnosis of primary hyperaldosteronism but due lack of standardization and uniformity in various diagnosing centers, the results may be difficult to interpret. However, AVS, when combined with advanced radiological imaging techniques such as CT or MRI, along with multi-disciplinary discussion of such patients with relevant subspecialists whenever the need arises, may greatly enhance the ability to diagnose and treat primary hyperaldosteronism.

ACKNOWLEDGEMENTS

The authors would acknowledge Dr. Majed Darraj, Dr. Nael Shaat and Dr. Mohammed Abdulrasak for their support.

Disclosure

The authors report no conflicts of interest.

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