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# A Case Report on Adrenal Cortical Adenoma with Conn's Syndrome

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## Abstract

Adrenal cortical adenoma related primary aldosteronism being a rare case ata young age is amedical challenge for clinical diagnosis. A 31-year-old male patient is reported with uncontrolled hypertension even after adhering to medication therapy for past 10 years. His laboratory findings and CT scan revealed the underlying cause as hypertension with adrenal carcinoma confirmed by adrenal vein sampling showing elevated aldosterone/renin ratio and hypokalaemia. A laparoscopic surgical intervention subsided his uncontrolled hypertension for the past 10 years. This rare case report highlights the necessity of differential diagnosis and points out the importance of diagnostic techniques to rule out specific underlying causes of especially common diseases like hypertension.

**Keywords:** Aldosteronism • Hypertension • Cohns syndrome • Hypokalaemia • Laproscopy

## Introduction

Conn syndrome was named after J. W. Conn, described it in 1955. He was a patient who had hypertension with an aldosterone-producing adenoma. The adenoma usually characterizes increased aldosterone secretion from the adrenal glands, suppressed plasma renin, hypertension, and hypokalemia. Conn's Syndrome is also characterized by excess secretion of aldosterone from the adrenal gland, manifested by hypertension, hypokalaemia, and hyporeninemia. The prevalence of Primary Aldosteronism(PA) in the hypertensive population is 0.1% to 2%. Previous studies have shown that adenomas are the most frequent cause of primary aldosteronism (50% to 70%), followed by bilateral hyperplasia (30%). Usually, adenomas are managed surgically and bilateral hyperplasia medically.

The diagnosis of each subtype of PA remains a clinical challenge because although the two most common subtypes are bilateral adrenal hyperplasia and aldosterone-producing adenomas (APA), other rarer forms such as unilateral adrenal hyperplasia (UAH) and glucocorticoid responsive aldosteronism can also manifest.

Primary hyperaldosteronism is caused by aldosterone-producing adenomas, bilateral idiopathic adrenal hyperplasia, aldosterone-producing adrenal carcinoma or familial aldosteronism. The increased amount of aldosterone causes renal sodium reabsorption, water retention, and potassium excretion. The increased sodium reabsorption by the kidneys leading to plasma volume expansion which is the initiating mechanism for hypertension. This may further induce tissue inflammation and heightened sympathetic drive, with subsequent development of fibrosis in vital organs, like heart, kidneys, and vasculature. As a result, this may lead to the development of chronic kidney disease, atrial fibrillation, stroke, ischemic heart disease, and congestive heart failure [1-5].

## Case Description

A 31-year-old male patient with a 10-year past history of hypertension was on Spironolactone 125 mgOD and Prazosin 5 mg OD while he visited the clinic. Even as this medication was taken by the patient his BPlevel (160/90 mmHg) was high, along withit he had hypokalaemia (2.9 mEq/l), elevated plasma aldosterone (22 ng/dl) level, and suppressed plasma renin activity (0.69 ng/ml).

The 24-hour test of urine catecholamines (metanephrine) was 213.3 mcg/24 hr, ruling out Pheochromocytoma. The salivary cortisol was 4.27 mg/dl, ruling out Cushing's syndrome.

A CT scan of the abdomen and pelvis showed well-defined oval, a small, right adrenal lesion 14×10×13 mm from medial limb, which could be an adenoma. Adrenal vein sampling confirmed unilateral-right adrenal adenoma where left arterial showed to be normal. The aldosterone/renin ratio was calculated to be 31.88 ng/dl per ng/(ml/hour).

Laparoscopic right adrenalectomy was done in the patient where right adrenal was dissected from Inferior Vena Cava (IVC) and the specimen was sent for histopathological studies (Figure 1).

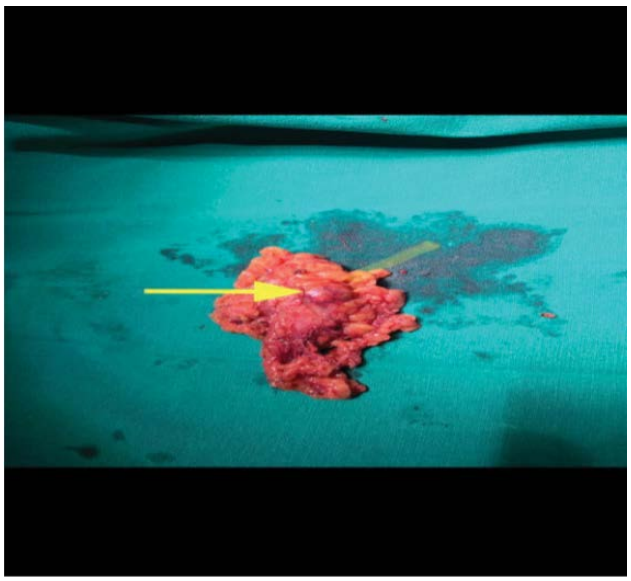

**Figure 1.** Adrenalectomy specimen.

Histopathological report of right adrenalectomy specimen showed features consistent with adrenal cortical adenoma – benign.

The patient was stable at the time of discharge and was asked to continue with Prazosin 5mg until normotensive.

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## Discussion

This case illustrates the difficulty of defining the aetiology of primary hyperaldosteronism and we review the biochemical and scanning techniques available to aid in diagnosis.

Severe hypokalemia remains unnoticed in otherwise healthy persons but can often present with severe manifestations in some patients. Paralytic myopathy in association with hypokalemia is a recognized feature of Conns' syndrome and is seen more commonly in Asian patients. The classical recommendation in patients with significant hypertension requiring 2 or more drugs with poor control, young hypertensives, and hypertension associated with hypokalemia is to investigate for the presence of primary hyperaldosteronism. Since, the risk factors leading to this condition is remaining a mystery the best choice is to appropriately diagnose the patients [6].

## Conclusion

Hypertension is unusual in adolescents and that too due to endocrine causes are very rare, but Conn's syndrome should always be considered in the differential diagnosis. The accurate diagnosis can minimize the consumption of unindicated drugs, improve the quality of life of the patient, and reduce the economic burden.

## Limitations and Future recommendations

The major limitation was that the past medication details of respective patient was not available in records as well as the patient was unaware of it. The recommendation that is to be suggested is more rigorous study in this field to achieve rapid accurate diagnosis of this syndrome in various conditions of patients with and without symptoms.

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