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Research Paper

A Case of Unilateral Adrenal Adenoma Presenting As Uncontrolled Hypertension and Hypokalemia.

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ABSTRACT
Primary hyperaldosteronism or Conn's syndrome should be considered as antihypertensive drug resistant hypertension associated with hypokalemia.70 % of primary hyperaldosteronism is due to a single adrenal cortical adenoma. Identifying primary hyperaldosteronism is important because of its prevalence and association with a higher rate of cardiovascular morbidity and mortality.
KEYWORDS *Primary hyperaldosteronism*

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I. INTRODUCTION

Clinically primary hyperaldosteronism or Conn's syndrome should be considered as antihypertensive drug resistant hypertension associated with hypokalemia.The diagnosis can be challenging but important to diagnose because as in the patient in this case, the hypertension can often be cured by surgery. Approximately 70 % of primary hyperaldosteronism is due to a single adrenal cortical adenoma and most of the rest is due to bilateral idiopathic adrenal hyperplasia.We hereby present a case of primary hyperaldosteronism due to adrenal cortical adenoma in 39 year old male who was being treated for hypertension in whom the diagnosis and surgical management get him relieved of his symptoms.

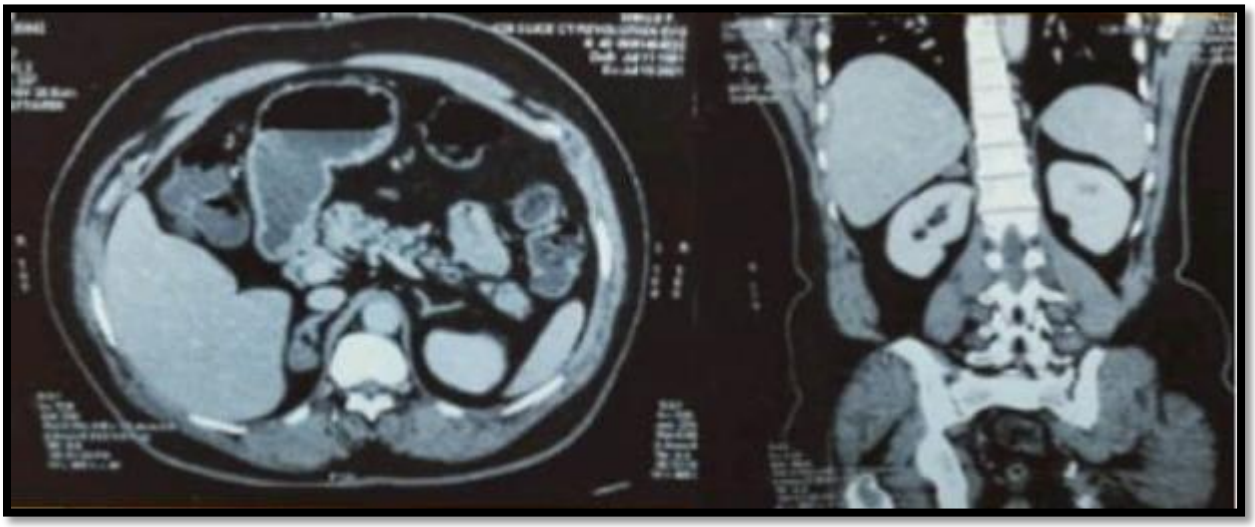
II. CASE PRESENTATION

A 39 year old male with hypertension for 8 years on medication was presented to the hospital initially with bilateral lower limb pain and weakness. On examination blood pressure was 220/110 mm Hg.Abdomen was soft and non tender, no organomegaly.

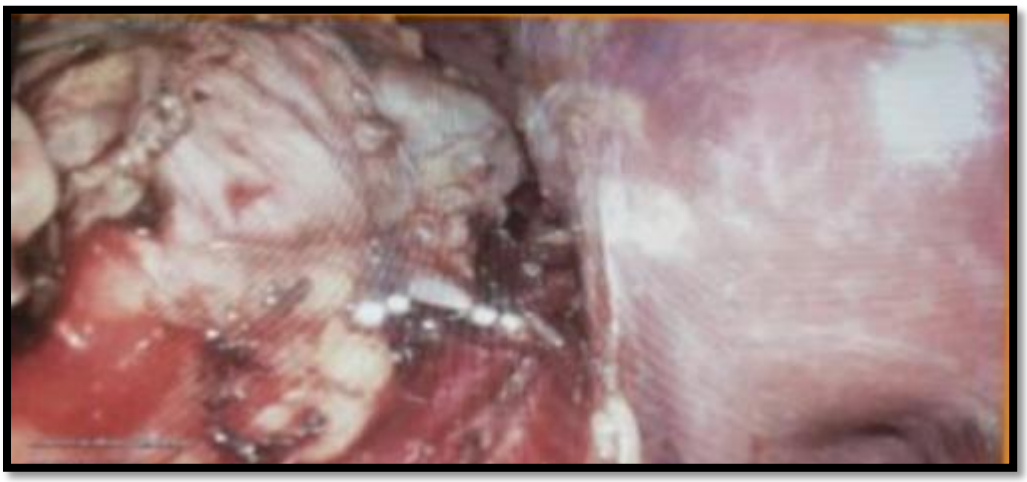
Blood investigations showed hypokalemia (1.9 mEq/L), metabolic alkalosis, serum ACTH-16 picograms (<46 picograms), free metanephrines -24 nanomol/ml (7.9-88), Nor metanephrines-2.5 nanomol/ml (20-135), 24 hour Urine metanephrine-99 microgram /24hour 24 hour urine nor metanephrine-391.47 microgram/24 hour, Aldosterone/renin ratio -1.004, Serum aldosterone-10.10 ng/ml (21-35.30), Renin direct-10.06 miU/U (4.40 46.10), Serum cortisol-morning 8am -10.25 microgram/dl and Midnight -9.44 microgram/dl.

Ultrasound abdomen showed right hypo echoic suprarenal lesion.

CECT abdomen shown well defined lobulated lesion of approximate size 18 x 15 mm involving lateral limb of right adrenal gland. Left adrenal gland – normal prostate-normal, no ascites, no significant lymph nodes. Lesion shows the following morphological and contrast enhancement. Washout characteristics include-Precontrast average attenuation of the lesion-12 Hounsfield unit(HU). Post contrast enhancement noted. Portal venous phase attenuation value -59 HU.Absolute washout -63%.Relative washout -49%.



Patient underwent laparoscopic right adrenalectomy on 22/4/2022 and was stable post operatively , Discharged on fourth day.Histology report was consistent with adrenal adenoma.



III. DISCUSSION

The usual presenting features of primary hyperaldosteronism are hypertension and hypokalemia, but potassium levels are frequently normal in day to day cases of primary hyperaldosteronism. It has been estimated that only 9% to 37% of patients with primary hyperaldosteronism have low potassium levels. This is probably because of early diagnosis as more patients with hypertension are being screened for primary hyperaldosteronism. The most common subtypes of primary hyperaldosteronism are aldosterone-producing adenomas and bilateral idiopathic hyperaldosteronism. Less common forms of hyperaldosteronism include familial hyperaldosteronism, unilateral adrenal hyperplasia, pure aldosterone producing adrenocortical carcinomas and ectopic aldosterone-producing tumors. Very recently, the Endocrine Society updated their clinical practice guidelines for the diagnosis and treatment of primary hyperaldosteronism. The guidelines recommend case-detection testing in patient groups who have a relatively high prevalence of primary hyperaldosteronism. However, there is evidence that the screening should include patients who have systolic blood pressure higher than I5O mmHg and diastolic blood pressure higher than 100 mmHg. Additionally, the guidelines stress the importance of a more timely diagnosis and treatment of such patients, given its high prevalence in hypertensives and its role in cardiovascular and kidney damage. While making the diagnosis it is also Important to rule out other causes which might cause hypokalemia and hypertension, like renovascular disease (hypersecretion of renin leading to increased aldosterone secretion) and diuretic therapy. Less frequent causes are Cushing's syndrome, Licorice ingestion, congenital adrenal hyperplasia, Liddle's syndrome, and renin- secreting tumors.

IV. CONCLUSION

Identifying primary hyperaldosteronism is important because of its prevalence and association with a higher rate of cardiovascular morbidity and mortality when compared to age and sex-matched patients with primary hypertension and the same degree of blood pressure elevation. We were able to diagnose this patient in a stepwise manner ruling out many diagnoses before arriving at the correct diagnosis. In patients diagnosed with primary hyperaldosteronism, treatment of the mineralocorticoid excess results in reversal or improvement of the hypertension and resolution of the increased cardiovascular risk.

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