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Conn's Syndrome in a Male Adult with Uncontrolled Hypertension; A Case Report

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Abstract:- Adrenal hyperplasia is a rare cause of secondary hypertension[1]. Our index case Is 54 year old community health extension worker, known hypertensive of 20 years who presented on account of uncontrolled hypertension with background family history of hypertension in both parents and siblings.
Methods: we retrieved the case file of the patient from UATH Data and Record unit.
Results: Patient blood pressure during the period of evaluation was persistently elevated with mean blood pressure of 185/115 + 10mmHg while on triple antihypertensive. Biochemistry result showed severe hypokalaemia of 2.2mmol/l, aldosterone level was markedly elevated with value of 33.90ng/dl, serum renin level of 0.68ng/dl and aldosterone renin-ratio is 49.9, abdominal CT showed features of bulky adrenal gland worse on the left.
Conclusion: Clinical impression of Conn's syndrome was made based on the above findings. Patient was placed on spironolactone- an aldosterone antagonist with other antihypertensive. Patient symptoms resolved, his blood pressure became normal and the electrolyte abnormalities corrected at the 6-8 weeks of treatment.

Keywords:- *Adrenal Gland, Conn's Syndrome, Hypertension, Hypokalaemia.*

I. INTRODUCTION

Primary hyperaldosteronism (Conn's syndrome) is one of the endocrine causes of secondary hypertension and accounts for 0.5-2% of patients with secondary hypertension. Screening with the use of Plasma Aldosterone/Plasma Renin activity ratio followed by aldosterone suppressing confirmatory testing has given much higher prevalence estimates (5.13% of all hypertensive) for primary aldosteronism[1]

Primary hyperaldosteronism has a number of causes, 2/3 cases(66%) are due to adrenal adenoma, Bilateral idiopathic adrenal hyperplasia (33%), Primary adrenal hyperplasia(2%), Aldosterone producing adrenocortical carcinoma(<1%), Familiar hyperaldosteronism(<2%), Primary aldosteronism is present in about 10% of people with high blood pressure (BP). It occurs most often in women, often begins between 30 and 50years[2]

Conn's syndrome is named after Jerome W.Conn (1907-1994) an American endocrinologist who first described adenoma as a cause of the condition in 1955 in a patient with unilateral aldosterone producing adenoma.[3]

It is characterised by excess production of aldosterone independent of renin angiotensin system causing increase sodium and water retention, hypertension, hypokalaemia and metabolic alkalosis.[3] Hypertension associated with hypokalaemia in a patient not on diuretics should be investigated for primary hyperaldosteronism, so also refractory hypertension, hypertension before 40yrs and strong family history of hypertension in first degree relative.

II. CASE REPORT

54 year old community health worker, known hypertensive of 20yrs on 10mg of Lisinopril, 5mg nevibiolol referred from peripheral hospital in account of uncontrolled hypertension, chest pain and dry cough. Chest pain was not related to activity or meals. There was no history of exertional dyspnoea, orthopnoea or paroxysmal nocturnal dyspnoea. There was no associated palpitations, headache, syncopal attack or loss of consciousness. No vomiting abdominal pain or swelling, no facial or leg swelling. No reported history of heat intolerance or increased irritability, no anterior neck swelling, no increased bruisability. However, there was a family history of hypertension in both parents and siblings.

Physical examination was remarkable for pulse rate of 68bpm regular, BP 185/115mmHg, apex was displaced and heaving, no jugular venous distention. No palpably enlarged abdominal organ, no demonstrable abdominal or renal bruit.

A clinical assessment of severe hypertension with possibility of secondary cause was entertained. Some Investigations were requested; transthoracic echocardiography revealed left ventricular hypertrophy, grade 1 diastolic function and normal left ventricular systolic function in keeping with hypertensive heart disease.

Electrolyte, urea and serum creatinine at presentation showed Na- 136mmol/L, K- 2.2mmol/L (hypokalaemia), Chloride 95mmol/L, Bicarbonate 31mmol/L (alkalosis) , Urea 6.6mmol/L, Creatinine 92mmol/L.

Abdominal CT Showed bulky adrenal gland worse on left, right adrenal gland with post IV contrast (HU=21)

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measuring15.8mm, left measuring 22mm in axial dimension with enhanced post IV Contrast (HU=38), Aldosterone 33.90ng/dl, Renin 0.68nm/ml, Aldosterone :Renin Ratio 49.9(0-20), Renin/Aldosterone 0.0200.

Patient was initially on Tab Telmisartan 80mg daily, Tab Nifedipine 30mg daily, and Tab Bisoprolol 2.5 mg daily prior to outcome of some of the investigations.

Sequel to above investigations a diagnosis of hyperaldosteronism (Conn's Syndrome was made and Tab spironolactone 25mg daily was added to his medication. Patient is presently stable with good BP control.

III. DISCUSSION

Conn's syndrome is associated with excessive production of aldosterone independent of renin angiotensin system causing increase sodium and water retention, hypertension, hypokalaemia and metabolic alkalosis.

The above case review is a case of secondary hypertension that was thought to be essential hypertension, investigation done revealed hypokalaemia, bulky adrenal glands (L>R), Low renin, high aldosterone, high aldosterone renin ratio.

A case of Conn's syndrome was reported in Port Harcourt in a 39 year old business woman and a known hypertensive of 4yrs who presented in the emergency department of university of Port Harcourt Teaching Hospital with history of extreme weakness and inability to work, had cardiac arrest while being evaluated and was successfully resuscitated, Abdominal CT Scan revealed a right adrenal mass, serial E/U/CR revealed persistent hypokalaemia with metabolic alkalosis and was commenced on a spironolactone and other antihypertensive. A similar case was reported in a 58 year old male Turkish who had sudden collapse due to ventricular fibrillation while on vacation. His abdominal CT Showed right adrenal mass[1].

Conn's syndrome is said to be rare in Nigeria reason for paucity of literature review on our case study. One of the endocrine cause of hypertension is primary hypersldosteronism accounting for about 0.05-2.5 % of secondary hypertension[1], studies have shown increase prevalence of up to 5.13% of all hypertensive secondary to hypersldosteronism with the use of Plasma Aldosterone/Plasma renin ratio followed by aldosterone suppression confirmatory test1.Hypertension with hypokalaemia in a patient who is not on diuretics should be investigated as well as patient with resistant hypertension, hypertension occurring before 40 year in a patient with family history of hypertension in 1st degree relatives[4]

Hypokalaemia may be severe in patient with Conn's syndrome though it could be normal and this does not exclude primary hyperaldosteronism[5].

The diagnosis is made through both laboratory and imaging findings, the screening test for conns syndrome include plasma Aldosterone/Plasma renin ratio, serum electrolyte with focus on the potassium (hypokalaemia) and metabolic alkalosis, this is followed by aldosterone suppression confirmatory test1. Aldosterone/Renin ratio of >20 and Aldosterone >15ng/dl make diagnosis of Conn's syndrome probable. This is followed by confirmatory test using 24 hour urine aldosterone level obtained after 3 day of salt loading, this is the most frequently used test for confirmation. Instructions can be given to patient to maintain a sodium intake of at least 200mEq/day (one teaspoon of salt 3 times daily) for 3 days, value of >14mcg of a 24 hour aldosterone excretion with concomitant 24-hour urine sodium excretion >200mEq) is diagnostic of primary aldosteronism. Other test for confirmation includes postural studies though cumbersome is done with saline infusion test with 2 litres of normal saline infused over 2-4hrs with sample taken before and after for plasma aldosterone, plasma aldosterone reduces in patient with essential hypertension while in hyperaldosteronism it fails to suppress[2, 3]

Imaging of choice is Abdominal CT with sensitivity of 67-85%. MRI has a higher sensitivity, however, in patient with equivocal findings, adrenal venous sampling following ACTH stimulation test is indicated[4]

In the index case we were able to assay the aldosterone and renin levels which showed elevated serum aldosterone of 39ng/dl (3-16 ng supine) aldosterone/renin ratio of 49.9 (0-20) with low serum renin 0.68 (0.98-4.18), electrolyte checked showed hypokalaemia of 2.2mmol/l (3-5), while abdominal CT revealed bulky adrenals left greater than the right with differential post IV Contrast enhancement which strongly suggests Conn's syndrome.

Patient was treated with Spironolatone- an aldosterone antagonist- a drug of choice for medical treatment of Conn's syndrome 5, other antihypertensive with good cardiovascular profile were added to optimise blood pressure control. Patient symptoms resolved, his blood pressure became normal and the electrolyte abnormalities corrected at the 6-8 weeks of treatment.

IV. CONCLUSION

There is need for a high index of suspicion for secondary hypertension in a young hypertensive patients, patient with resistant hypertension and patient with strong family history of hypertension in first degree relatives.

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