ANOMALOUS RENAL ARTERY IS POTENTIAL CAUSE OF RESISTANT HYPERTENSION IN A 53 YEAR OLD PATIENT: CASE REPORT.

[1]Busari A.A and [2]Bello B.T

[1]Nephrology Unit, Department of Medicine, Gbagada General Hospital, Gbagada.
[2] Department of Medicine, Lagos University Teaching Hospital, Lagos

ABSTRACT

BACKGROUND:
Drug-resistant hypertension can be attributable to secondary hypertension and other causes. Anomalous renal artery is uncommon but can be a potential cause of resistant hypertension.

CASE REPORT:
We highlight the challenges in management of resistant hypertension and describe its unusual association with renal artery anomaly in 53 years old man who was referred to our nephrology clinic from a peripheral general hospital on account of poorly controlled hypertension. At presentation, BP was severely elevated at 208/100mmHg but no remarkable findings in the rest of the examination. Several investigations done including abdominal ultrasound scan and Computerised Tomography (CT) Renal angiogram revealed a Left anomalous renal artery. Patient declined all suggested urologic interventions and he was then managed conservatively.

CONCLUSION:
We found that anomalous renal arteries can be a potential cause of resistant hypertension. We therefore recommend ultrasound scan of the abdomen as a screening modality due to its being non-invasive.

Key-words: Renal artery, anomalous, resistant hypertension, stenosis

NigerJMed2016: 192-196

INTRODUCTION

Resistant hypertension is increasingly common in clinical practice and it is said to be present in about one in eight patients with hypertension. This condition occurs when there is a failure to achieve target blood pressure goal despite receiving three or more anti-hypertensive drugs at optimal tolerable doses that includes a diuretic. However, renal artery anomaly is uncommon; its association with resistant hypertension is even rarer. In this case report, we therefore highlight the challenges in management of resistant hypertension and describe its unusual association with renal artery anomaly.

Case History:

Mr E.K a 53year old book seller who was referred to our nephrology clinic from a peripheral General Hospital on account of poorly controlled hypertension despite being on 3 antihypertensive including a diuretic for 3 months. He was diagnosed hypertensive and diabetic 8yrs and 5yrs respectively prior presentation and claimed to be adherent to his prescribed regimen. He was taking lisinopril 20mg, amlodipine 10mg, and Co-amilozide (Amiloride 5mg/Hydrochlothiazide 50 mg) 1 tablet all daily. He was also taking Glucovance (Metformin500mg/ Glyburide 5mg) twice daily. At presentation in our clinic, he had no history of chest pain, blurring of vision or passage of frothy urine. He complained of occasional dry cough. There was a history of hypertension in his mother. He takes alcohol occasionally but claimed he never smoked.Examination revealed a middle aged man who was obese (BMI of 33.9kg/m2), not pale and had no peripheral oedema. His pulse rate was 84b/min regular and his blood pressure (BP) was 208/100mmHg. His apex beat was difficult to palpate due to the presence of a thick anterior chest wall. On auscultation, the 1st, 2nd and 4th heart sounds were heard and there were no murmurs. The rest of the physical examination was unremarkable. A chest X-Ray revealed Cardiomegaly and unfolding aorta while an electrocardiogram showed left ventricular hypertrophy with strain pattern. His serum urea & creatinine was 42mg/dl and 1.4 mg/dl respectively. Urinalysis revealed no abnormality; HbA1c was 5.1%

Correspondence: busarial@yahoo.com

but his lipid profile were deranged (total cholesterol– 220mg/dl, HDL cholesterol– 40mg/dl, LDL cholesterol– 103mg/dl and triglycerides– 76mg/dl). An assessment of resistant hypertension with Metabolic Syndrome to rule out Renal Artery Stenosis (RAS) was made. He was commenced on life-style modification and some of his medications were changed such that he was now to be on; Tabs Losartan 100mg daily (to replace lisinopril which was suspected to be the cause of ACEI –induced dry cough) other medication included Tabs Amlodipine 10mg daily, Co-amilozide (Amiloride 5mg /Hydrochlothiazide 50 mg), Tab Glucovance (Metformin500mg/ Glyburide 5mg) twice daily, Tabs clopidogel 75mg daily and Tab Atovastatin 20mg nocte. Ultrasound examination of his abdomen revealed asymmetric kidney sizes (Right kidney 10.1cm by 5.5cm; Left kidney 5.2cm by 3cm). The echo patterns were normal on the right but cortical echogenicity was increased on the left. There was no dilatation of the calyces or ureters. Resistivity Index [RI] of left renal vessel during Doppler was 0.73. Conclusion was suggestive of LT Renal Artery Stenosis.

Despite the modification of his antihypertensive regimen there was no significant change in his BP after 2 weeks with his clinic BP as high as 190/105mmHg, his medications were altered again as follows to include addition of Nifedipine XL 60mg morning and 30mg at night, Atenolol 50mg daily and Minoxidil 5mg twice daily while Amlodipine was discontinued. A Computerised Tomographic (CT) angiogram of the renal arteries was also done which revealed that the origin of the left renal artery from the aorta was 5.3cm distal to that of the right (Figure1),that the artery itself appeared foreshortened, 2cm prior to bifurcation, with tapering of dorsal and ventral branches (Figure 2&3). The left kidney was seen to be lying inferior to the right kidney and it was shrunken with irregular margins (Figure 1&2). There was also a persistent nephrogram phase with delay excretion in the left kidney. The right renal artery and kidney were within normal limit. The conclusion was that of Left Renal artery anomalous origin, distribution and bifurcation with Hypoplastic ectopic Left kidney. He was subsequently referred to urologist for possibility of Nephrectomy and renal denervation therapy was also discussed with him but declined both interventions citing fear of surgery and financial constraint. He was then advised to intensify on life style modification and adherence to medication. Subsequent clinic visits have been uneventful. BP became fairly controlled, ranging between 148/80mmHg – 160/ 90mmHg.Discussion:

The disease burden attributable to systemic arterial hypertension is substantial, accounting for 62% of all strokes and 49% of all cases of heart disease, culminating in an estimated 7.1 million deaths a year; equivalent to 13% of total death worldwide.[1,2] Although most cases of hypertension can be effectively treated with lifestyle changes, drugs, or both, however those with hypertension that is resistant to treatment are at the extreme end of the cardiovascular risk [1,2]

Resistant hypertension (RH) is said to occur when there is a failure to achieve target blood pressure goal despite receiving three or more anti-hypertensive drugs at optimal tolerable doses that includes a diuretic[3]. This phenomenon is increasingly common in clinical practice and it is said to be present in about one in eight patients with hypertension. Although the exact prevalence of resistant hypertension is unknown, recent published reports have suggested that the prevalence ranges from 10 - 20% of general hypertensive population.[4] Patients with RH have a 3- to 6-fold increased risk of fatal and non-fatal cardiovascular events compared with hypertensive patients whose blood pressure is maintained within normal levels 4-6. Patient or clinician-related factors may contribute to pseudo-resistant hypertension which include poor adherence to prescribed medication, suboptimal dosing of antihypertensive agents, inappropriate combinations of antihypertensive drugs, the white coat effect, and clinical inertia 4-6. Therefore management of resistant hypertension must begin with a careful evaluation of the patient to confirm the diagnosis and exclude factors associated with "pseudo-resistance". True resistant hypertension may be caused by obesity, alcohol, smoking, dietary sodium, concomitant use of drugs such as non-steroidal anti-inflammatory drugs oral contraceptives, sympathomimetics (decongestants, anorectics), adrenal steroids and antineoplastic drugs targeting the vascular endothelial growth factor (VEGF) pathway may contribute to the development of resistant hypertension[3-6]. Other conditions such as obstructive sleep apnea, renal artery stenosis, renal parenchymal disease,

primary aldosteronism, pheochromocytoma, Cushing's disease, thyroid and parathyroid dysfunction; and aortic coarctation also contribute to the development of resistant hypertension4-6. Renal artery stenosis (RAS), which could be in form of atherosclerotic or fibromuscular dysplasia is one of the common causes of secondary hypertension and resistant hypertension[6,7]. Anomalous renal artery is uncommon but can be a potential cause of resistant hypertension 8. Due to the presence of anomalous renal artery, there may be an alteration in the fluid dynamics that effect the respective kidney, which then develops kidney ischemia, thereby causes an increase in renin secretions and this leads to a reno-vascular, renin-

dependent hypertension, and a chronic kidney disease condition[8]. The most common variations of renal artery are excess (accessory) renal arteries and early bifurcation[9,10]. However, anomalous renal artery can occur when the renal artery can arises at unusual aortic origin such as above the superior mesenteric artery or celiac trunk, in which the renal artery may be entrapped in the medial arcuate ligament [8-10]. In this situation, the renal artery may be compressed and presented with symptoms of hypertension[8-10]. The compression to renal artery may occur in the presence of low insertion of median arcuate ligament or high origin of the renal artery[8-10]. In the case presented, although there was no accessory or multiple renal arteries but there was an anomalous left renal artery in terms origin. There was also ventral and dorsal tapering of the fore-shortened left renal artery with the resultant Hypoplastic left kidney (Figure2 & 3). The anomaly of the renal artery alone may not be the only contributory factor responsible for the resistant hypertension in this patient. The co-existing long-standing stenosis may explain in part the ischaemia and subsequent hypoplasia of the left kidney and resistant hypertension. Obesity and excess salt intake might have also contributed.

In the evaluation of the patient with suspected Renovascular hypertension, Duplex Doppler ultrasonography is an attractive option because it is a non-invasive test that is relatively inexpensive, does not require contrast medium, and can be used in patients with any level of renal function[11]. As with many of the non-invasive imaging tests, there are numerous parameters and abnormal criteria indicating possible renovascular disease[12]. The diagnosis of RAS is a frequent consideration when dealing with severe hypertension and renal failure; however, the choice of imaging modalities is controversial. Ultrasonography, Computed tomography angiography (CTA), and magnetic resonance angiography (MRA) are all used, and each method has its own advantages and drawbacks. Moreover, interpretation requires significant expertise. No consensus has been reached on the choice the imaging modality and the decision should be based on the level of expertise available[12]. In the case presented the patient had ultrasound scan done which served as a screening method. The revelation of shrunken left kidney and Doppler finding prompted further investigation of Computed tomography angiogram (CTA) which latter revealed renal artery anomaly. After the establishment of a hemodynamically significant stenosis that results in hypertension there exist multiple treatment options. The treatment options which are to be considered for a hemodynamically significant stenosis of an anomalous renal artery are embolization, balloon angioplasty, stenting and even partial nephrectomy. Renal sympathetic denervation is another treatment option. Evidence has shown that resistant hypertention may, at least in part, be mediated by chronic activation of the sympathetic nervous system (SNS)[13] and the subsequent emergence of percutaneous sympathetic denervation of the renal arteries—a novel intervention that could stimulate a paradigm shift in the way we manage not only treatment-resistant systemic hypertension, but also a myriad of pathophysiological entities associated with chronically augmented SNS activation.[14] We considered that our patient might have probably benefited from Nephrectomy or percutaneous sympathetic denervation since he already had a left hypoplastic kidney which makes stenting or revascularisation unnecessary. But patient declined both interventions citing fear of surgery and financial constraint. Renal sympathetic denervation (RSDN) has been shown to be both safe and effective, with a durability of action apparently sustained out to 3 years. A recent cost-effectiveness analysis of RSDN based on a Markov model and the Symplicity HTN-2 has suggested it to be a cost-effective intervention for resistant hypertension which might also result in lower cardiovascular morbidity and mortality[15].

Conclusion

Anomalous renal arteries can be potential cause of resistant hypertension. We recommend Ultrasound scan as a screening modality for renal artery stenosis and anomalous renal arteries due to its being non-invasive, free of radiation, low cost, and lack of contraindications because of renal failure and contrast allergy.

Figure 1; The left kidney was seen to be lying inferior to the right kidney and it was shrunken with irregular margins. (See arrow)

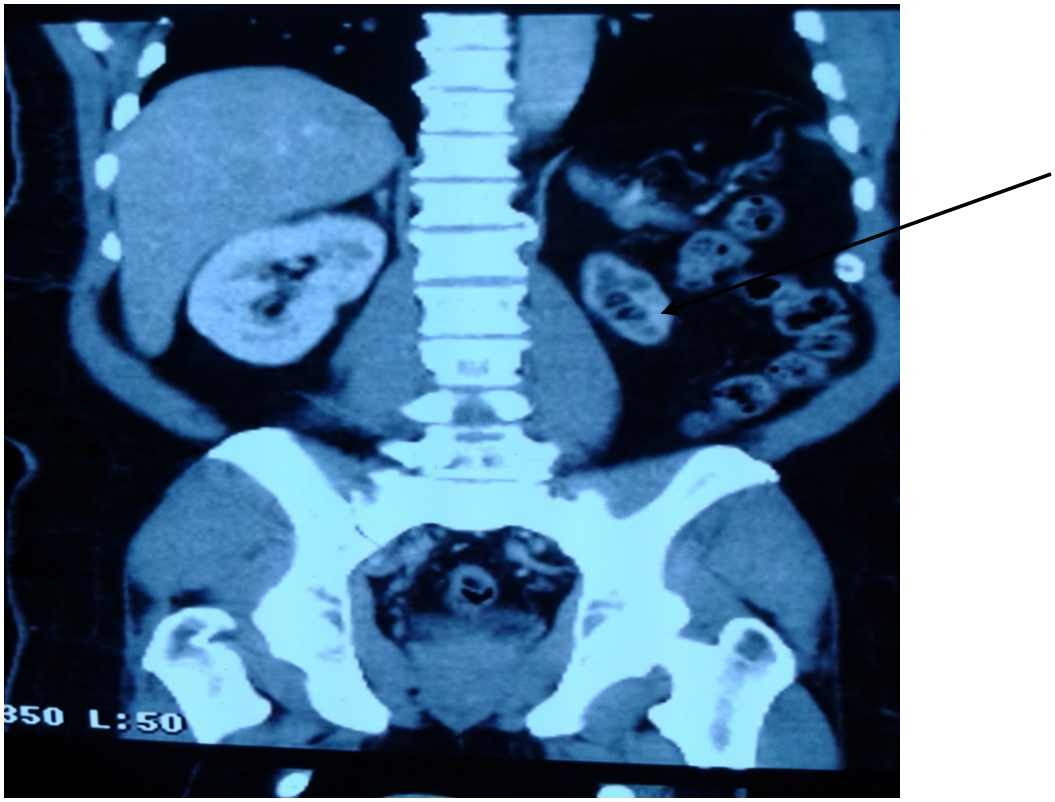


Figure 2; The left kidney was seen to be shrunken with irregular margins. (See arrow)

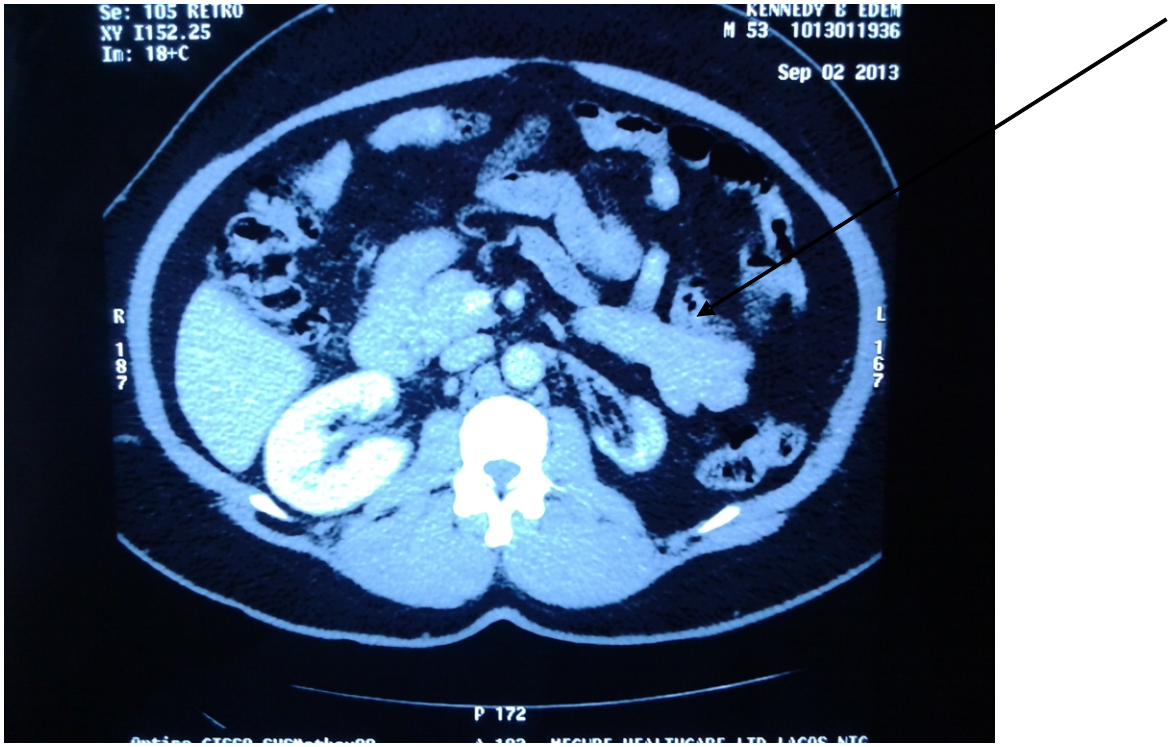
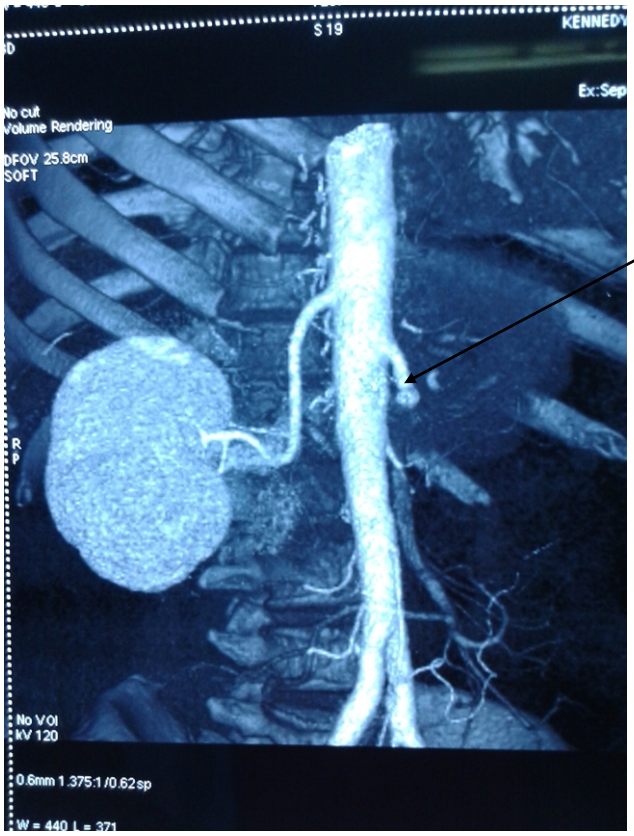


Figure 3. **The origin of the left renal artery from the aorta was 5.3cm distal to that of the right that the artery itself appeared foreshortened, 2cm prior to bifurcation, with tapering of dorsal and ventral branches noted (see arrow).**



REFERENCES:

1. World Health Organization. Global status report on non-communicable diseases. WHO. 2014;www.who.int/nmh/publications/ncd-status-report-2014/en
2. Makris A, Seferou M and Papadopoulos D.P. Resistant Hypertension Workup and Approach to Treatment. Int J Hypertens. 2010; doi: 10.4061/2011/598694.
3. Calhoun DA, Jones D, Textor S, et al. Resistant Hypertension: Diagnosis, Evaluation and Treatment: A Scientific Statement from The American Heart Association Professional Education Committee Of The Council For High Blood Pressure Research. Circulation. 2008;117(25):510–526.
4. Myat A, Redwood S.R, Qureshi A.C, Spertus J.A, Williams B. Resistant hypertension; Clinical Review. BMJ. 2012; 345: 7473 doi: 10.1136/bmj.e7473.
5. De la Sierra A, Segura J, Banegas JR, Gorostidi M, de la Cruz JJ, Armario P, et al. Clinical Features of 8295 Patients with Resistant Hypertension Classified on the basis of Ambulatory Blood Pressure Monitoring. Hypertension. 2011;57(5):898–902.
6. Pierdomenico SD, Lapenna D, Bucci A, Di Tommaso R, Di Mascio R, Manente BM, et al. Cardiovascular outcome in treated hypertensive patients with responder, masked, false resistant, and true resistant hypertension. Am J Hypertens. 2005;18(11):1422–8.
7. Garg JP, Elliott WJ, Folker A, Izhar M, Black HR. Resistant hypertension revisited: A comparison of two university-based cohorts. AJH. 2005;18(5 Pt 1):619-26.
8. Decker DO and Glenn Chapman, A Case of Back Pain with Resistant Hypertension resulting from an Anomalous Accessory Renal Artery; Health & Medicine. 2013; http://www.slideshare.net/Larkinhospital.
9. Patil UD, Ragavan A, Nadaraj Murthy K, Shankar R, Bastani B. Helical CT Angiography In Evaluation of Live Kidney Donors. Nephrol Dial Transplant. 2001;161: 900-4.
10. Sari S, Verim S, Sivrioglu AK, Bozlar U. A Rare Cause of Secondary Hypertension: Median Arcuate Ligament Compression of The Renal Artery Diagnosed By CT Angiography. BMJ Case Rep. 2013;12:114.
11. Postma CT, Van Aalen J, de Boo T, Rosenbusch G, Thien T. Doppler Ultrasound Scanning in the Detection of Renal Artery Stenosis In Hypertensive Patients. Br J Radiol. 1992;65(778):857–860.
12. O'Neill WC, Bardelli M, Yevzlin AS. Imaging for Renovascular Disease. Semin Nephrol. 2011;31(3):272-82.
13. Esler M. The Sympathetic Nervous System through the Ages: From Thomas Willis to Resistant Hypertension. Exp Physiol. 2011; 96:611–622.
14. DiBona GF, Esler M. Translational Medicine: The Antihypertensive Effect of Renal Denervation. Am J Physiol Regul Integr Comp Physiol. 2010; 298:245–253.
15. Geisler BP, Egan BM, Cohen JT, Garner AM, Akehurst RL, Esler MD, Pietzsch JB. Cost-Effectiveness and Clinical Effectiveness of Catheter-Based Renal Denervation for Resistant Hypertension. J Am Coll Cardiol. 2012; 60:1271–1277.