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New Peripheral Signs of Chronic Aortic Regurgitation seen in Five Patients in the North of Nigeria and Literature Review

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Abstract

These patients presented with cough productive of whitish frothy sputum, dyspnoea, orthopnoea and paroxysmal nocturnal dyspnoea with mild pitting pedal oedema. These five patients presented with established known signs of chronic aortic regurgitation such as: Quinke's, Hill's, Corrigan, De'musset, Duroziez, Positive wide pulse pressure, Rosenbach's, Locomotor branchialis, Ladolfi, Muellers, Ashrafian and Minervini. Auscultation revealed aortic regurgitation and ejection systolic murmurs. Hypertension was found to be their risk factor. Investigations for complication of hypertension and to rule out common chest infections such as tuberculosis eg FBC and Differentials, AAFB X 3, Mantoux, S/E/U/Cr were normal. Cxray done showed enlargement of the heart by measuring the CTR (Cardiothoracic ratio). Drugs such as Amlodipine, Furosemide, Aldactone, Moduretic, Lisinopril and Digoxin were used for treatment. A thorough clinical cardiovascular and general examination from head of the patients to helm was done on them and a diagnosis of chronic aortic regurgitation was established vis-a-viz already known signs of chronic aortic regurgitation. This aims at bringing to the fore of practicing clinicians these new signs seen in patients with chronic aortic regurgitation especially in areas without advanced medical equipments such as the hinterlands of Tropical Africa. Patients did well on follow up.

Keywords: *Aortic regurgitation, signs and symptoms, Eponymous and New Eponym signs and clinical examination.*

Introduction

Aortic regurgitation is due to incompetence of the aortic valve or disturbance of any of the valvular apparatus (eg leaflets,annulus of the aorta or the ascending aorta or aortic root[32]) resulting in leakage of blood into the left ventricular chamber during diastole[3,32]. Aortic regurgitation can be Congenital[1] or Acquired,Chronic or Acute presenting as heart failure[2]. Acquired causes usually affect the valves and include rheumatic fever, infective endocarditis[3,5,6], collagen vascular disease[3,10,12,13,14,15,16,17], trauma[32], post surgical[7,32,8],and degenerative aortic valve disease[32] or aortic wall such as hypertension[31,32,34], rheumatoid arthritis[13,34] while biscupid aortic valve is a congenital cause[1,9]. Most common cause of aortic regurgitation used to be rheumatic fever but currently infective endocarditis in developed countries[3,5,6] while biscupid valve is the most common congenital cause in developed countries[1,9]. Rheumatic heart disease remains highly prevalent in Asia,middle Eastern and North African countries[19]. Estimates of the prevalence of AR of any severity range from 2-30% but only 5-10% of patients with AR have severe disease, resulting in an overall prevalence of severe AR of less than 1% in the general population[20]. Age of detection of AR in the USA is 40-60yrs. AR is more common in men than in women. In the cohort from the Framingham study,AR was found in 13% of men and 8.5% women[18]. The greater prevalence of AR in men may reflect in part the preponderance of underlying conditions, such as marfan syndrome[20] or biscupid aortic valve in males[21].

Chronic regurgitation often begins when they are in their late 50s but most documented in patients older than 80yrs. The incidence of clinically significant aortic regurgitation increases with age,typically peaking in the fourth to sixth decade of life. Severe chronic AR is uncommon before age of 70yrs[18]. Patients with biscupid aortic valve and especially marfan syndrome tend to present much earlier[17,21]. The mechanism of aortic incompetence comprises the pressure in the left ventricle falling below the pressure in the aorta, the aortic valve is not able to completely close. The percentage of blood that regurgitates back through the aortic valve due to AR is known as the regurgitant fraction. The regurgitant flow causes a decrease in the diastolic blood pressure in the aorta and therefore an increase in pulse pressure. Since

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some of the blood that is ejected during systole regurgitates back into the left ventricle during diastole, there is a decreased effective forward flow in AR[5,6]. While Diastolic blood pressure is diminished and the pulse pressure widens, the systolic blood pressure generally remains normal or can even be slightly elevated, this is because the sympathetic nervous system and the renin-angiotensin-aldosterone axis of the kidneys compensate for the decreased cardiac output. Occasionally the diastolic blood pressure becomes high due to heart failure but widens on treating the heart failure[34]. Catheco- lamines increase the heart rate and does increase the strength of ventricular contraction which directly increases the cardiac output[7]. Cathecolamine cause peripheral vasoconstriction which causes increased systemic vascular resistance and ensures that organs are well perfused[8]. AR causes both volume overload(elevated preload) and pressure overload of the heart (elevated afterload)[11]. Acute AR leads to increased blood volume in the left ventricular during diastole. The Left ventricule of the heart doesn't have sufficient time to dilate in response to the sudden increase in volume. This rapid increase in end-diastolic volume increases pulmonary venous pressure and a decrease in the coronary blood flow dynamics leading to dyspnoea and pulmonary oedema and decreased myocardial perfussion and cardiogenic shock.

Chronic aortic regurgitation causes gradual left venticular overload that leads to a series of several compensatory changes, including left ventricular enlargement and eccentric hypertrophy. Left ventricular dilatation occurs through the addition of sarcomeres in series(resulting in longer myocardial fibres). As a result, the left ventricular becomes larger and more compliant, with greater capacity to deliver a large stroke volume that can compensate for the regurgitant volume. The volume overload, due to elevated pulse pressure and the systemic effects of neuroendocrine hormones causes left ventricular hypertrophy(LVH). There is both concentric and eccentric hypertrophy in AR[32]. The conc- entric hypertrophy is due to increased left ventricular pressure overload associated AR while the eccentric hypertrophy is due to volume overload caused regurgitant fraction[32].

The physical examination of an individual with aortic insufficiency involves auscultation of the heart and would hear a soft S1 in acute AR[32]. In chronic regurgitation listen for the murmur of aortic insufficiency and the S3 heart sound. There early diastolic decrescendo murmur which is best heard in the 3rd intercostal space in chronic regurgitation and may radiate along the sternal border. An ejection systolic murmur[31,32,34] may also be present following an increased stroke volume in the left ventricle. There are numerous peripheral signs(eponyms) associated with chronic aortic insufficiency which are related to the high pulse pressure and the rapid decrease in blood pressure during diastolic cycle due to blood returning to the heart from the aorta through the incompetent aortic valve but those mentioned in this article are those seen in patients encountered which are[4]:- An **Austin Flint murmur**[31,32] due to regurgitant jet from severe aortic insufficiency that renders partial closure of the anterior mitral leaflet. The **Wide pulse pressure**(pulse pressure greater than 60mmHg and usually greater than the diastolic pressure)[31,32,34], **Locomotor branchii** (pulsation of the branchial arteries)[34], **Corrigan's** pulse(dancing carotid)[20,31,32,34], **Landolfi** sign(a change in pupillary size of both eyes in accordance with the cardiac cycle and not related to light)[22,34], **DeMusset's** sign(head bobbing with each heart pulsation)[21,31,32,34], **Becker's sign** (pulsation of the retinal artery)[22,34], **Gerhardt's** sign(pulsation over enlarged spleen)[21,34], **Mueller's** sign (systolic pulsations of uvula)[21,34], **Lincoln** sign(popliteal artery pulsation)[22,34], **Sherman** sign(dorsalis pedis pulsation is quickly located and unexpectedly prominent in age >75yrs)[22],**Palmar click sign**(pulsation of the palm/palpable abrupt flushing of the palms)[37], **Rosenbarch's** sign(the pulsation of the liver)[22,34], **Ashfrasian** sign (pulsatile pseudoproptosis)[33],**Traube's** sign(pistol shot sound heard over the femoral artery)[22,31,32,34], **Duroziez's** sign(systolic murmur heard over the femoral artery when it is compressed proximally and a diastolic murmur when it is compressed distally using a bell)[21,31,32,34],**Hill's** sign(popliteal cuff systolic pressure exceeds branchial cuff pressure by 20mmHg)[22,34],**Mayne's** sign(diastolic pressure >15mmHg drop on raising the upper limb up)[22,34],**Quinke's** sign(capillary pulsations-its detected by pressing a slide on the patients nailbed)[21,31,32,34],**Lighthouse** sign(blanching and flushing of the forehead of patient)[34].**Minervini's** sign(pulsation of the tongue)[37]. Doing investigations to rule out the cause and confirm diagnosis include Cxray-PA which would show aortic unfolding and Cor Bovinum(gross cardiomegaly)[34], an ECG[31,32,34],Cardiac chamber catheterization[31,32,34] and transthoracic echocardiography[23,24] for assessing severity and any left ventricular function[31,32] and multidetector CT[25] and MRI[26]. Treatment can either be the use of medical or surgical[16,28] treatment. Medical[31,32,34,35] treament include angiotensin converting enzyme inhibitor or angiotensin receptor blockers, calcium channel blockers, hydralazine, diuretics, digoxin, reduction in salt intake and strenuous exercise[2,3,24,25] and B-blockers[30,34,35]. Patients with aortic regurgitation could be treated using surgical method that would entail valve replacement[3,16,27,28,29]. AR has various mortality percentages in individuals who do not undergo surgical treatment for this condition depending on the grade or class. The prognosis of aortic regurgitation depends on dilation of the left ventricle, ejection fraction and symptoms[36].

Case Report

Case 1

A 72yr old Female housewife, known hypertensive for about 8years who presented on account of two(2)weeks history of recurrent cough which was productive of whitish frothy sputum, dyspnoea and bilateral leg swelling. There was associated orthopnoea and

paroxysmal nocturnal dyspnoea.There was no facial and abdominal swelling. No fever, drenchy nightsweat.The patient had no contact with a chronically coughing adult or with a pulmonary tuberculous patient.

O/E: An elderly woman who is chronically ill-looking,afebrile,not pale,anicteric,not cyanosed,no significant peripheral lymphadenopathy,no finger clubbing with pitting pedal oedema.

CVS:-PR-84bpm regularly irregular,bounding and collapsing

Positive Locomotor branchialis
JVP-raised
BP-160/60mmHg
Apex beat- 6LICS lateral to midclavicular line, heaving which was downwards and out- wards.

HS:-S1S2S3 with a 3/4 AR murmur, ESM at the root of the neck.

Positive wide pulse pressure-100mmHg
Positive Quinke's sign
Positive Hill's sign-24mmHg
Positive Corrigan sign(Dancing carotid)
Positive De'Musset sign
Positive Duroziez's sign and murmur
Positive Rosenbach's sign

Nwosu P.U's sign-Oscillating (bobbing) hands which synchronized with the bobbing of the head and pulsation of the radial artery.

Nwosu P.U's sign: Pulsation of the superficial temporal artery at the Right/left parotid/zygomatic and temporal areas.

RS:RR-24cpm
Trachea central
Percussion note resonant
Fine bibasal crepitations

Abdomen:
Full and moved with respiration
No area of tenderness
Liver was enlarged-6cm below costal margin,tender,firm, smooth surface and regular edge which was pulsatile.
Liver span-18cm
No ascitis
CNS-normal

Diagnosis:-Congestive cardiac failure secondary to HHDx with grade 2/4 AR murmur and ESM.

Investigations:
Cxray-PA:cardiomegally-CTR:60%
Aortic unfolding
Distended pulmonary veins
Opacities bilaterally
FBC-normal
S/E/U/Cr-normal
AAFB X 3-negative
Mantoux-negative

Plan:-1)IV Frusemide 40mg dly x 3/7 then
2)Tabs frusemide 40mg dly
3)Tabs aldactone 25mg dly
4)Tabs Lisinopril 5mg dly
5)Tabs Atenolol 25mg dly

Patient improved and was discharged and followed up at the MOPD for about a year.

Case 2

A 68yr old man,known hypertensive for 10years and has been on moduretic, Lisinopril, Amlodipine but not regular presented with increased frequency in micturiting,nocturia, hesitancy,straining for about 4weeks duration. There was also dyspnoea and orthopnoe, paroxysmal nocturnal dyspnoea of 2weeks duration with occasional cough productive of whitish sputum with no streak of blood. There was occasional fever,weight loss but no drenchy nightsweat or contact with anyone with chronic cough.

O/E:- An elderly man chronically ill-looking,dyspnoiec (flaring of ala nasa),trachea tug, afebrile, not pale,anicteric,not cyanosed,no thrush,no significant peripheral lymphadenopathy, not dehydrated,no finger clubbing with mild pitting ankle oedema.

CVS:PR-74bpm regular,bounding,and collapsing.
Positive locomotor branchialis
JVP:-Not raised
BP-150/70mmHg
Apex beat:-5LICS Lateral to midclavicular line,heaving, downward and outward
HS-S4S1S2,ESM on the neck,2/4 AR murmur on the left 3rd intercostal space.
Positive wide pulse pressure-80mmHg
Positive Duroziez's sign and murmur
Positive Rosenbach's sign

Nwosu P.U's sign-sweating/diaphoresis and drying on the forehead following each stroke volume (increased systolic pressure) and run-off (decreased diastolic volume).This sign is very important especially in blacks since flushing and blanching on the forehead of patient (Lighthouse, Morton and Mahon sign) would be difficult to observe.

Nwosu P.U's sign-There is a marked rise and fall of radial pulse volume with upward (expiration) and downward (inspiration) movement of the tracheal prominence

respectively in a patient with coexisting respiratory disease.

RS:RR -30cpm
Trachea-central
Percussion note-resonant
Fine bibasal crepitations
Random wheeze
Vesicular breath sound.

Abdomen:Full and moves with respiration
No area tenderness
Liver:-3cm below costal margin,firm,smooth,regular edge and pulsatile.
DRE-prostate enlarged,hard,irregular with obliteration of the median sulcus.
Investigations:-
Cxray-PA showed cannon ball metastasis
Aortic unfolding
Cardiomegally:CTR-0.55
S/E/U/Cr,Ca2+,PO4 are normal
FBC,ESR and differentials-normal
AAFB X 3-negative
Mantoux-negative
Diagnosis-Metastatic cancer of the prostate with hypertensive heart disease with AR.
Treatment:-1.Tabs amlodipine 5mg dly
2.Tabs moduretic 1 dly
3.Tabs Lisinopril 5mg dly
4. Patient died after 5 days on admission

Case 3

He is a 58year old male Security/Farmer who presented at Jahun General Hospital on account of recurrent dry cough and dyspnoea of 6months haven received treatment from Hadeja general hospital for a week for Upper respiratory tract infection. A known hypertensive for about 6months and has been on moduretic but not regular.There is associated orthopnoea but no paroxysmal nocturnal dyspnoea. No history of fever, drenchy nightsweat,contact with a chronically coughing person or on tuberculosis drugs. He attested to being treated for pulmoary tuberculosis about a year ago with marked improvement-weight and appetite albeit a negative AAFB test.

O/E: An anxiously looking middle-aged man,dyspnoeic (flaring of ala nasi),afebrile,not pale, anicteric,no significant peripheral lymphadenopathy,no digital clubbing with mild pitting pedal oedema and palmar erythema.

CVS:PR-78bpm regular and normal volume,bounding and collapsing.
Positive Locomotor branchialis
JVP-not raised but had distended neck veins.
BP-140/70mmHg
Apex beat-5LICS Lateral to the midclavicular line,heaving which was downwards and outwards.
HS-S1S2S3 with a Loud A2,1/4 AR and ESM at the neck.
Positive wide pressure-70mmHg
Positive Rosenbach's sign
Positive Duroziez's sign and murmur
Positive De'Musset's sign

Nwosu P.U's sign:Oscillating (bobbing) hands with each heart beat which synchronized with the bobbing of the headand pulsation of the radial artery.
Nwosu P.U's sign: Pulsation of the eyelids(due to pulsation of the zygomaticofacial artey).

RS-RR-20cpm
Trachea deviated to the right
Dullness on the RT middle and Lower lung zones anterioly.
Bronchial breath sound on the RT mddle lung zone anteriorly.
Rhonci on the left middle lung zone posteriorly.
Other zones vesicular.

Abdomen:-Full and moves with respiration
No area of tenderness
Hepatomegaly:-10cm below costal margin,firm,smooth, regular and pulsatile.
No Ascitis.

Investigations

FBC-normal
S/E/U/Cr-normal
CXray PA:-Trachea deviated to the Right
Aortic unfolding
CTR:0.57%
RT middle lobe opacity
RT border of the heart silhouted
Opacities in both Right and Left lung zones.

Diagnosis:-Right Pulmonary fibrosis with Congestive cardiac failure secondary to hypertensive heart dx with AR.

Plan:1)Tabs moduretic 1 dly
2)Tabs prednisolone was titrated from10mg tds for a month then 10mg b.d and currently on 5mg dly.
3)Intranasal O2 5-6L PRN
4)Patient discharged against medical advice on financial grounds.

Case 4

Mr.O.J,a 76yr old man retired civil servant who presented on account of inability to use the Left side of the body and aphasia of 3days duration.He was at rest while it was noticed. No complaint of prior headache,vomiting, fever, neck stiffness,or trauma to the head.He had a previous stroke affectng the Right side about 5yrs ago with mild

residual function. He is a known hypertensve but not regular on his medication-moduretic and amlodipine.

O/E:An elderly man who is not febrile, not pale,anicteric,not cyanosed,mouth open with mild pitting pedal oedema.
CVS: PR-70 bpm regular,bounding and collapsing
JVP-slightly raised
Positive Locomotor branchialis
BP-140/60mmHg
AB-6LICS Lateral to midclavicular line,heaving which was downwards and outwards.
HS:-S4S1S2 with ESM at the neck,AR,and a Austin Flint murmurs
Positive wide pulse pressure-80mmHg
Positive Quinke's sign
Positive Palmar sign
Positive Rosenbach's sign
Positive Landolfi sign
Positive Mueller's sign
Positive Minervini's sign

Nwosu P.U's sign:-Increased palpable pulsation of the digital arteries on the palmar surface of the fingers and intermetacarpal arteries on the dorsum of the hands. **Nwosu P.U's sign:**Pulsation of the superficial temporal artery at the Right/left parotid/ zygomatic and temporal areas.

RS:RR-18cpm
Trachea central
Equal chest expansion
Resonant percussion note
Fine crepitations bibasally.

Abdomen:Full and moves with respiration
No area of tenderness
Liver was 4cm below costal margin,nontender,smooth surface, regular but pulsatile.
No palpable spleen or bilaterally ballotable kidneys.
No Ascitis.
CNS:-Conscous and alert
No meningeal signs
GCS-E-4
V-2
M-3(RT side of the body)

	RUL	RLL	LUL	LLL
Tone:	normal	normal	hypotonia	hypotonia
Power:	4	4	0	0
Reflexes:	normal	normal	hyperreflexia	hyperreflexia

Normal swallow reflex

Diagnosis:1)Right hemispheric stroke with left hemiplegia ?? Haemorrhagic
2) Congestive cardiac failure secondary to hypertensive heart disease with AR.

Investigations:
1)CXray-could not be done because of his state.
2)S/E/U/Cr/Ca/PO4-normal
3)FBC,ESR and differentials-normal
Plan:1)IV N/S 1L 6hrly with 10mls of Vit.Bco in each litre.
2)Dissolved tabs Vit C 200mg tds
3)Regular turning 2hrly
4)Stop all hypertensives.
5)Adviced to put the limbs through exercises to improve muscle function.
Patient was discharged after a week on admission.

Case 5

Mr. D.T is a 57yr old Businessman diagnosed of being hypertensive 2months ago in Lagos where he was treated for Upper respiratory tract infection and heart failure.He presented on account of recurrent dyspnoea,cough productive frothy whitish sputum but no streak of blood and swelling of both legs. There was associated history of orthopnoea and paroxysmal nocturnal dyspnoea. No history of fever,chest pain,drenchy night sweat,weight loss or contact with anyone with chronic cough or PTB. A positive history of alcohol intake on an average of 10units a week and cigarette smoking of 10 packyears but stopped immediately after he was diagnosed of the above ailment.

O/E:He is a middle-aged man who is dysnoeic (flaring of ala nasi) and afebrile,anicteric, not cyanosed,no significant lymphadenopathy,finger clubbing,no sacral oedema with pitting pedal oedema upto the upper third of the shank.
CVS:-PR-80bpm,irregularly irregular,bounding and collapsing
JVP-raised
BP-140/110mmHg
AB-6LICS anterior axillary line,heaving which was downwards and outwards.
HS:S1S2S3 with ESM and 2/4 AR murmurs
Positive corrigan sign
Positive Locomotor branchialis
Positive De'Musset's sign
Positive Duroziez's sign
Positive Ashrafian sign
Positive Minervini's sign

Nwosu P.U's sign:-bobbing of the abdomen and legs with each increased systolic pulsation.
Nwosu P.U's sign: Pistol shot sound of the carotid artery.
RS:RR-28cpm
Trachea is central
Equal chest expansion

Resonant percussion note
Fine bibasal crepitations
Abdomen:Full and moves with respiration
No area of tenderness
Liver was 6cm below costal,nontender,firm,smooth surface,regular edge and pulsatile.
Spleen not palpable below costal margin and nonballotable kidneys.
Mild Ascitis.

Diagnosis:Congestive cardiac failure secondary to Hypertensive heart disease with ESM and AR murmurs KIV Alcoholic cardiomyopathy.

Investigations:-
FBC-normal
S/E/U/Cr-normal
CXray PA:-Aortic unfolding
Distended pulmonary veins
Enlarged heart(cor bovinum)-CTR:60%
Right pleural effusion.
AAFB X 3-negative
Mantoux-negative
Plan:1)Tabs frusemide 60mg bd
2)Tabs aldactone 25mg dly
3)Tabs Digoxin 0.25mg dly
4)Tabs Lisinopril 5mg dly
5)Tabs atenolol 25mg dly
Patient was on follow up for about 6months and was doing well before I left the institution.

Discussion

Case 1: Shepresented with chronic aortic regurgitation following hypertension[31,32,34] of 8 years duration.On examination the patient had a bounding and collapsing pulse[34],locomotor branchialis[34],increased pulse pressure of 100mmHg which was greater than the diastolic pressure[31,32,34], positive Quinke's sign[21,31,32,34], Hill's sign of 20mmHg[22,24], a Corrigan sign of the carotid artery[20,31,32,34], a positive DeMusset's sign[21,31,32,34], Duroziez's sign and murmur[21,31,32,34] and a positive Rosenbach's sign[22,34] which are known documented signs of chronic aortic regurgitation. Apex beat that was heaving, down and out[31,32,34],Ejection systolic murmur[31,32,34] on the neck are seen in her and are features of chronic aortic regurgitation. **Nwosu P.U's sign**-Oscillating (bobbing) hands of the patient which synchronized with the bobbing of the head(DeMusset's sign) was seen in her-this hasn't been documented in literature as a sign of chronic aortic regurgitation.Essential tremors was thought of in this patient based on her age but it was unlikely because it wouldn't have synchronized with her head bobbing and each systolic pulsation and would worsen on action. There was no sign of parkinsons disease or cerebellar disease in her. **Nwosu P.U's sign**-Pulsation of the superficial temporal artery of the Right/left temporal region and at the parotid/zygomatic area synonymous to the Shermans or Lincoln sign seen in chronic aortic regurgitation. This pulsation of the superficial temporal artery has not been documented.

Case 2: Patient is a known hypertensive[31,32,34] for 10 years which is a recognised risk factor for chronic aortic regurgitation. The patient had a positive bounding and collapsing pulse[34], locomotor branchialis[34], also a wide pulse pressure of 80mmHg which was greater than the diastolic pressure[31,32,34], had Duroziez's sign and murmur[21,31,32,34] and Rosenbach's sign[22,34] which are signs of chronic aortic regurgitation in him. **Nwosu P.U's sign**-Sweating and drying on the forehead following increased systolic volume and diastolic run-off. This has not been reported in literature and most important in blacks since it is difficult to appreciate the blanching and flushing (Lighthouse phenomenom, Morton and Mahon sign) in blacks. Had a heaving apex beat which was downward and outward and a grade 2/4 AR mumur are in keeping with chronic aortic regurgitation. **Nwosu P.U's sign**-There is a marked rise and fall of radial pulse volume with upward(expiration) and downward(inspiration) movement of the tracheal prominence respectively in a patient with coexisting respiratory disease. During inspiration blood is been carried to the lungs leading to a decreased systolic blood volume and the amplitude of the pulse while in expiration the pulse volume is increased.This is exagerated in AR because of the run-off during diastole together with the decreased blood volume during inspiration leading to a reduction in pulse volume in inspiration and the increased systolic volume in AR leads to an increased pulse volume in expiration.

Case 3: She is a known hypertensive[31,32,34] for 6months a known risk factor for chronic aortic regurgitation. Positive bounding and collapsing pulse[34], locomotor branchialis[34], Wide pulse pressure of 70mmHg which is equal to the diastolic pressure of the patient but more than the normal pulse pressure[21,32,34], also had a positive Duroziez's sign and murmur[21,31,32,34], De Musset's, Rosenbach's[22,34] are signs of chronic aortic regurgitation in her. **Nwosu P.U's sign**:-Oscillating (bobbing) hands which synchronized with the head bobbing (De Musset's sign) was seen in her-this hasn't been documented as a sign of chronic aortic regurgitation. Essential tremors in her was thought of based on patients age but was unlikely because it wouldn't have synchronized with head bobbing and each systolic pulsation and would worsen on action. No sign of parkinsons disease or cerebellar disease made it unlikely. .**Nwosu P.U's sign**-pulsation of the eyelids is due to the increased pulsation of the zygomaticofacial artery due to increased systolic volume.This has not been documented in literature.It is similar to the Quinke's sign seen in the nail bed.

Case 4: He is a known hypertensive[31,32,34] for a duration not ascertainable, a known risk factor for chronic aortic regurgitation. He had positive bounding and collapsing

pulse[34], locomotor branchialis[34], Wide pulse pressure of 80mmHg which is equal to the diastolic pressure but it was more than the normal pulse pressure[31,32,34],Mueller's sign[21,34], Palmar click sign[37],Ladolfi sign[22,34], Rosenbach's sign[22,34],grade ¼ AR murmur, a Austin Flint murmur[31,32] and an Apex beat that is downward and outward[31,32,34] in him are all signs of chronic aortic regurgitation. **Nwosu P.U's sign**-increased palpable pulsation of the palmar interdigital arteries and the intermetacarpal arteries-this has not been documented. Arises due to increased systolic volume which is the cause of the Sherman and Lincoln signs **Nwosu P.U's sign**-Pulsation of the superficial temporal artery at the Right/left temporal region and at the parotid/zygomatic area synonymous to the Shermans or Lincoln sign seen in chronic aortic regurgitation.This pulsation of the superficial temporal artery has not been documented and follows an increased systolic volume.

Case 5: He is a known hypertensive[31,32,34] for two months presenting with signs of chronic aortic regurgitation.His examination revealed a bounding and collapsing pulse[34],narrow pulse pressure of 30mmHg and measured diastolic pressure of 110mmHg because of cardiac failure[34],had a positive Corrigan sign[20,31,32,34] of the carotid artery,DeMusset's sign[21,31,32], Duroziez's sign and murmur[21,31,32,34] and a positive Ashrafian sign[33] in the patient which are all documented signs of chronic aortic regurgitation. His Apex beat was heaving, down and out[31,32,34],a 2/4 AR murmur and Ejection systolic murmur[31,32,34] on the neck and a cor bovinum[34] were seen in him which are in keeping with chronic aortic regurgitation.**Nwosu P.U's sign**-bobbing of the abdomen and legs with each systolic pressure which synchronized with the bobbing of the head.This is as a result of the increased systolic volume from the run-off during diastole leading to increased pulsation of the abdominal aorta and femoral artery. This has not been documented in literature.**Nwosu P.U's sign**-pistol shot sign of the carotid artery.This has not been documented.This is similar to the Traube sign.

All the cases were treated with vasodilators-either calcium channel blockers or ACE inhibitor and antifailure regimen-diuretics and digoxin and mild exercise as docum- mented[2,3,24,25,30,34,35].

Conclusion

Aortic regurgitation is commonly caused by hypertension in our environment and could be prevented with regular check of blood pressure. Several signs have been associated with it and the above new signs named hereinafter me should be looked out for inorder to make a diagnosis of chronic aortic regurgitation clinically.

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