



RESEARCH ARTICLE

HYPERTENSION RELATED AORTIC REGURGITATION [HT -RELATED AR], ITS PREVALENCE AND PROGNOSTIC SIGNIFICANCE

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ABSTRACT

Aortic Regurgitation [AR] is diastolic flow of blood from aorta to left ventricle. AR could be acute or more commonly chronic. chronic AR could be congenital or acquired. it could be due to valve leaflet pathology, aortic wall defect or both. Severe, prolonged hypertension can cause aortic root dilatation and consequently lead to AR. The aim of this study is to estimate the prevalence of AR in prolonged hypertension i.e. hypertension related aortic regurgitation [HT-related AR] and prognostic significance of AR in hypertensive patients. 430 patients (206 female, 224 male) with history of hypertension for at least 5 years were enrolled in this study, same number of non-hypertensive patients of similar age and gender were used as control. Patients with AR due to obvious cause are excluded from the study. 2-D color Doppler ECHO study was done for both groups to detect AR in them. The result showed that patients with hypertension are more prone to develop AR than non-hypertensive patients, and HT-related AR was more common in males than females, and it is related to the duration and severity of hypertension. HT-related AR is also associated with other ECHO-bad prognostic criteria of hypertension like left ventricular hypertrophy (LVH) and left atrial enlargement, so we can consider HT-related AR as an additional bad prognostic ECHO criteria of hypertension.

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INTRODUCTION

Aortic Regurgitation AR is diastolic flow of blood from aorta to left ventricle. AR may be acute, presented with acute heart failure (Saura *et al.*, 2008), or more commonly chronic. Causes of acute AR include infective endocarditis, Dissecting Aneurysm or trauma to the chest. Chronic AR may be caused by valve pathology, Aortic wall diseases [Ascending Aorta] or both. Valvular abnormalities that may cause AR may be congenital or acquired. Bicuspid aortic valve is the most common congenital cause (Roberts *et al.*, 2012; Friedman *et al.*, 2008). Rheumatic heart disease was the most common cause of chronic valvular AR, but presently it is most commonly caused by infective endocarditis (Friedman *et al.*, 2008), which may lead to destruction or perforation of valve leaflet, vegetation can interfere with proper valve coaptation or causing prolapse of aortic valve which lead to AR (Braunwald, 1988; Giuliani, 1991; Kloner, 1990) it may be a cause of acute or chronic AR. Aortic valve replacement including transcatheter Aortic Valve Replacement [TAVR] is a common cause of both acute and chronic AR. (Sinning *et al.*, 2013), AR also may occur as a complication of Left

Ventricular Assist Device [VAD] implantation (Aggarwal *et al.*, 2013), other causes of acquired valvular AR are degenerative aortic valve disease and collagen vascular disease. Certain anorexogenic drugs like Dexfenfluramine may induce degenerative valvular changes that result in chronic AR. (Kanchera *et al.*, 1999). Systemic Lupus Erythematosis [SLE] can cause valvular fibrosis resulting in chronic AR (Jain and Halushka, 2009), Lupus can also cause Libman-Sachs sterile endocarditis usually involve mitral valve but sometimes it involve aortic valve lead to AR (Moysakis *et al.*, 2007; Lee *et al.*, 2009). Rheumatoid Arthritis rarely cause AR when rheumatoid nodule involve aortic valve leaflet (Chand *et al.*, 1999). Abnormalities of the ascending aorta in the absence of valve pathology include longstanding hypertension, idiopathic aortic dilatation, Marfan syndrome (Keane and Pyeritz, 2008), cystic medial necrosis, Ehlers-Danlos syndrome, Floppy aortic valve, Senile aortic ectasia, Syphilitic aortitis, Giant cell arteritis (Eberhardt and Dhady, 2007), Takayasu arteritis (Adachi *et al.*, 2007), Ankylosing Spondylitis (Palazzi *et al.*, 2008), Behcet disease (Schirmer *et al.*, 2003) and Whipple disease. (Jeserich *et al.*, 1997). Clinical signs of chronic AR depend on the severity of AR, it is caused by forward and backward flow of blood across the aortic valve leading to increase stroke volume (Babu *et al.*, 2003), these peripheral manifestations like water-Hammer pulse [collapsing

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pulse], pistol shot, neck nodding, wide pulse pressure usually occur in moderate and severe chronic AR. The murmur of AR is high – pitch early diastolic blowing murmur, loudest at left sternal border, the duration of the murmur correlated with severity of AR, functional systolic flow murmur may detected even in the absence of associated aortic stenosis (Babu *et al.*, 2003), Austine –Flint murmur [low- pitch mid – diastolic rumbling murmur heard at cardiac apex] may be heard in severe chronic AR due to regurgitant jet.

PATIENTS AND METHODS

430 patients with hypertension 206 female and 224 male above 50 years old, are enrolled in this native study, their hypertensive was for at least 5 years. Other 430 patients also 206 female and 224 male with matched age group, without hypertension or known cardiac problem who consult us for other causes were used as control. We exclude from the study patients with aortic regurgitation due to obvious cause like rheumatic valvular disease or bicuspid aortic valve. Basic investigations were done include CBC, blood sugar blood urea lipid profile, serum electrolytes. In addition to -12- Lead ECG. 2 –D – Color Doppler ECHO study was done with color Doppler to evaluate for evidence of aortic regurgitation (AR) The study was done at AL-Karaama teaching hospital and our private Cardiac Clinic at AL-Kutt--Wasit governorate, Iraq, the study was done during the period from January 2012 – April 2016. All group 1 patients have hypertension for at least 5years duration, with Blood Pressure more than 140-90 mmHg or on anti hypertension therapy. The control group (Group 2) of matching gender and age who are neither hypertensive nor have previous cardiac problem are prone to the same clinical examination and investigations including 2-D ECHO - Doppler study aiming to detect AR. The age and sex distribution of these patients shown in Table -1

Table 1. The age and sex distribution of hypertensive group patient

Age group	Male	Female	Total
50--60	56	39	95
61--70	79	63	142
71- 80	53	73	126
More than 80	36	31	67
total	224	206	430

The aim of the study is to estimate the prevalence of AR in patient with long standing hypertension [hypertension - related aortic regurgitation, HT –related AR], its significance and its relation to the prognosis.

RESULTS

From 430 patients in group 1 we found AR in 28 patients [6.51%] in whom their AR not related to other known cause of AR and labeled in our study as hypertension related Aortic Regurgitation [HT – related AR] where as we found AR in only 7 patients with group 2 i.e. control group [1.62 %], so AR is obviously occur more commonly in hypertensive patients than in non—hypertensive patients., as shown in Table –2

We found that HT-related AR was mild in 23 patients (82.14) and moderate in other 5 patients (17.85). We found that HT related AR occurs more commonly in male than in

female. [18 male 64.28%] Vs 10 female [35.71%]. This result is compatible with AR prevalence in general population, in the cohort from Framingham study AR was found in 13% of men and 8.5% of women. (Singh *et al.*, 1999)

Table 2. The prevalence, Gender and Age distribution of HT related AR in group 1 patients

Age group	Male	Female	Total
50--60	1	0	1
61--70	4	1	5
71- 80	5	3	8
More than 80	8	6	14
total	18	10	28

HT-related AR occur more commonly in older age group , i.e. its frequency increase with age. So one patient in (50-60) group, 5 patients in (61-70), 8 patients in (71-80) and 14 patients in above 80 years. AR prevalence in general population also increase with age. (Singh *et al.*, 1999)

HT- related AR correlated with duration of HT, So it occur in two patients in 5-10 years grup, 11 patients in 11-20 years group while occur in 15 patients with more than 20 years duration group. As shown in Table –3.

Table 3. Relation of HT-related AR to the duration of HT

Duration of HT in years	Male gender	Female	Total
5-- 10	2	0	2
11-20	7	4	11
Above 20	9	6	15
total	18	10	28

HT-related HT well correlated with severity of HT. as shown in table 4, so its frequency increase with severity of HT, only 2 patients in stage 1 (mild) HT), 10 in stage 2 (moderate HT) and 16 patients in severe HT

Table 4. The relationship of HT-related AR with severity of HT

Severity of HT[stage]	Male	Female	Total
Systole140-159 mmHg, diastole 99-99 mmHg	2	0	2
Systole 160-179 mmHg, diastole 100-109 mmhg	7	3	10
systole 180 mm Hg and above, diastole 110Hg mm and above	9	7	16
Total	18	10	28

Classical early diastolic blowing murmur of AR surprisingly very rarely occur in HT-related AR (detected clinically in only 2 patients), while no patient demonstrate any peripheral manifestation of AR like collapsing pulse, pistol shot or other manifestations. Nearly all patients with HT-related AR (27 patients) showed ECHO criteria of left ventricular hypertrophy (LVH) of different severity (with or without left atrial enlargement) as a complication of hypertension.

DISCUSSION

Chronic AR could be due to valve pathology, aortic wall defect or both. In prolonged hypertension, there is continuous stress on the aortic wall which lead to dilation of its annulus in addition to continuous stress on valve leaflet which can accelerate cusp calcification. This mechanical effect clearly explained the high frequency of AR in

patients with long history of hypertension, also explain its association with severity of hypertension. The present of LVH detected by ECHO study in nearly all patients with HT-related AR support the effect of long duration and severity on AR development. Also the association of HT-related AR with LVH, and left atrial [LA] enlargement [both of these two ECHO finding are indicators of bad prognostic criteria in hypertension patients], so we can consider HT-related AR as additional bad prognostic sign of hypertension. The lack of auscultatory findings and nearly absence of peripheral manifestation of classical AR in patients with HT-related AR can be explained by 1. mildness of AR in later group and as we know the duration of diastolic murmur correlated with AR severity, and as HT-related AR is usually mild, so we cannot expect detection of the murmur or other peripheral manifestations 2. Chronic AR specially severe one usually associated with wide pulse pressure due to elevated stroke volume during systole and the incompetence aortic valve allows the diastolic pressure within aorta to decline, so the severity of AR and consequently the duration of diastolic murmur and severity of peripheral signs of AR correlated with diastolic pressure gradient between aorta and LV, so more diastolic pressure gradient mean more severe AR and more severe peripheral signs. HT-related AR, usually associated with left ventricular hypertrophy [LVH] rather than left ventricular dilatation [due to associated hypertension], this may lead to stiffness of left ventricle and consequently decrease pressure gradient between aorta and left ventricle, so less severe AR and consequently less severe or absence of peripheral signs. These results will open new roads for researches firstly whether the mortality rate and the morbidity [frequency of complications] occur more commonly in HT related AR patients in comparison with hypertensive patients without AR, and Secondly whether the control of blood pressure in these patients will relieve or alleviate AR in the same manner of LVH improvement after control of HT, if so which medication is preferable.

Conclusion

HT-related AR is not uncommon condition, it is more commonly associated and correlated with duration and severity of hypertension, HT-related AR usually not associated with classical physical findings of usual AR, it may has adverse prognostic significance.

REFERENCES

- Adachi O, Saiki Y, Akasaka J, Oda K, Iguchi A, Tabayashi K. Surgical management of aortic regurgitation associated with takayasu arteritis and other forms of aortitis. *Ann Thorac Surg.*, 2007 Dec. 84(6):1950-3.
- Aggarwal A, Raghuvir R, Eryazici P, Macaluso G, Sharma P, Blair C, *et al.* The development of aortic insufficiency in continuous-flow left ventricular assist device-supported patients. *Ann Thorac Surg.*, 2013 Feb. 95(2):493-8.
- Babu AN, Kymes SM, Carpenter Fryer SM. Eponyms and the diagnosis of aortic regurgitation: what says the evidence?. *Ann Intern Med.*, 2003 May 6. 138(9):736-42.
- Braunwald E. Heart Disease: A Textbook of Cardiovascular Medicine. 3rd ed. Philadelphia, Pa: Saunders; 1988.
- Chand EM, Freant LJ, Rubin JW. Aortic valve rheumatoid nodules producing clinical aortic regurgitation and a review of the literature. *Cardiovasc Pathol.*, 1999, Nov-Dec. 8(6):333-8.
- Eberhardt RT, Dhady M. Giant cell arteritis: diagnosis, management, and cardiovascular implications. *Cardiol Rev.*, 2007 Mar-Apr. 15(2):55-61.
- Friedman T, Mani A, Elefteriades JA. Bicuspid aortic valve: clinical approach and scientific review of a common clinical entity. *Expert Rev Cardiovasc Ther.*, 2008 Feb. 6(2):235-48.
- Giuliani E. Cardiology: Fundamentals and Practice. 2nd ed. Philadelphia, Pa: Mosby Year Book; 1991
- Jain D, Halushka MK. Cardiac pathology of systemic lupus erythematosus. *J Clin Pathol.*, 2009 Jul 62(7):584-92.
- Jeserich M, Ihling C, Holubarsch C. Aortic valve endocarditis with Whipple disease. *Ann Intern Med.*, 1997 Jun 1. 126(11):920.
- Kanchera KM, Salti HI, Muliderink TA, *et al.* Echocardiographic prevalence of mitral and/ or aortic regurgitation in patients exposed to either fenfluramine – fentermine combination or to dexfenfluramine. *Am J Cardiol.*, 1999 Dec. 1; 84(11); 1335-8.
- Keane MG, Pyeritz RE. Medical management of Marfan syndrome. *Circulation*, 2008 May 27. 117(21):2802-13.
- Kloner R. The Guide to Cardiology. 2nd ed. New York: Le Jacq Communications; 1990.
- Lee JL, Naguwa SM, Cheema GS, Gershwin ME. Revisiting Libman-Sacks endocarditis: a historical review and update. *Clin Rev Allergy Immunol.*, 2009 Jun. 36(2-3):126-30.
- Moyssakis I, Tektonidou MG, Vasilliou VA, Samarkos M, Votteas V, Moutsopoulos HM. Libman-Sacks endocarditis in systemic lupus erythematosus: prevalence, associations, and evolution. *Am J Med.*, 2007 Jul. 120(7):636-42.
- Palazzi C, D' Angelo S, Lubrano E, Olivieri I. Aortic involvement in ankylosing spondylitis. *Clin Exp Rheumatol.*, 2008 May-Jun. 26(3 Suppl 49):S131-4.
- Roberts WC, Vowels TJ, Ko JM. Natural history of adults with congenitally malformed aortic valves (unicuspid or bicuspid). *Medicine (Baltimore)*, 2012 Nov. 91(6):287-308. [Medline].
- Saura D, Peñafiel P, Martínez J, de la Morena G, García-Alberola A, Soria F, *et al.* [The frequency of systolic aortic regurgitation and its relationship to heart failure in a consecutive series of patients]. *Rev Esp Cardiol.*, 2008 Jul. 61(7):771-4. [Medline].
- Schirmer M, Weidinger F, Sandhofer A, Gschwendtner A, Wiedermann C. Valvular disease and myocardial infarctions in a patient with Behçet disease. *J Clin Rheumatol.*, 2003 Oct. 9(5):316-20
- Singh JP, Evans JC, Levy D, *et al.* Prevalence and clinical determinants of mitral, tricuspid, and aortic regurgitation (the Framingham Heart Study). *Am J Cardiol.*, 1999 Mar 15. 83(6):897-
- Sinning JM, Vasa-Nicotera M, Chin D, Hammerstingl C, Ghanem A, Bence J, *et al.* Evaluation and management of paravalvular aortic regurgitation after transcatheter aortic valve replacement. *J Am Coll Cardiol.*, 2013 Jul 2. 62(1):11-20.