The Chest Radiograph

A Useful Investigation in the Evaluation of Hypertensive Patients

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Background: The assessment of target organ damage is important in the evaluation of a hypertensive patient as it provides information on the severity of the hypertension and the cardiovascular risk assessment. The aim of our study was to determine the usefulness of the chest radiograph in the assessment of target organ damage in hypertensive patients.

Methods: Unselected patients attending an academic hypertension clinic were studied. The cardiothoracic ratio and the aortic knob width were measured and compared to other markers of target organ damage. The aortic width was measured in age- and sex-matched controls.

Results: Seventy-two hypertensive and 77 age- and sex-matched normotensives were evaluated. There was a highly significant difference the aortic knob width between the normotensive and hypertensive patients (3.28 cm *v* 3.69 cm, $P < .0001$). The aortic knob width was significantly correlated with age in normotensive and hypertensive patients, systolic and diastolic blood pressure (BP), and all markers of target organ damage except the electrocardiogram (ECG) voltage. The cardiothoracic ratio was also significantly correlated with age and other markers of target organ damage, but not clinic BP. Multiple regression analysis revealed that only the cardiothoracic ratio ($r = 0.34$, $P < .02$) and the ECG voltage ($r = 0.58$, $P < .00005$) were independently correlated with left ventricular mass.

Conclusions: The chest radiograph provides important predictive information of associated target organ damage in hypertensive patients. Am J Hypertens 2004;17: 507–510 © 2004 American Journal of Hypertension, Ltd.

Key Words: Target organ damage, hypertension, aortic knob width, left ventricular hypertrophy.

The assessment of target organ damage is important in the evaluation of a hypertensive patient as it provides important information on the severity of the hypertension and the cardiovascular risk assessment.[1] Traditional markers of target organ damage are fundal changes, renal function, albuminuria, and left ventricular hypertrophy (LVH), as assessed either by the electrocardiogram (ECG) or echocardiography. In our clinic patients routinely undergo chest radiography, and it is our anecdotal experience that patients with severe hypertension have an increased cardiothoracic ratio and more dilatation or ectasia of the thoracic aorta than normotensives. Hypertension guidelines do not recommend the chest radiograph in the routine evaluation of hypertensive patients.[1] This is probably because the cardiothoracic ratio has been considered an unreliable investigation in the assessment of LVH in hypertensives. The ECG and echocardiography are the preferred investigations for assessment of LVH. The Losartan Intervention For Endpoint (LIFE) study recorded that >70% of patients with hypertension and ECG criteria for LVH had LVH on echocardiography. In addition, concentric and eccentric hypertrophy was seen in 25% to 29% and 45% to 51% of patients, respectively.[2] Eccentric hypertrophy is the dominant finding in hypertensives. Cardiothoracic ratio measured by chest radiograph may be a useful pointer to eccentric hypertrophy, and in conjunction with the ECG assist in the assessment of LVH.

A MEDLINE search revealed no studies evaluating thoracic aortic dilatation using the chest radiograph in hypertensive populations, although several studies have reported an association with aortic root dilatation and LVH assessed by echocardiography in patients with hypertension.[3–7] For the primary care practitioner echocardiography is not readily available and there is a need for simple and inexpensive tests to assist in the evaluation of patients with hypertension. With this in mind we decided to evaluate the usefulness of the cardiothoracic ratio and the dilatation of the ascending aorta in predicting LVH and other markers of target organ damage, and assess the relationship with both systolic and diastolic blood pressure (BP). In addition, the thoracic aortic width would be compared between hypertensives and normotensive controls.

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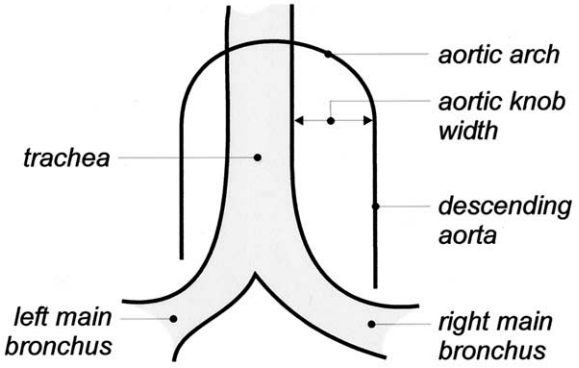

FIG. 1. Method of measurement of aortic knob width.

Methods

Unselected new patients attending the hypertension clinic at Groote Schuur Hospital were studied. All patients underwent routine clinical evaluation. Blood pressure was measured in the seated position after 5 min of rest using a mercury sphygmomanometer according to the standard recommendations. All patients had a standard 12-lead ECG, chest radiograph, standard two-dimensional echocardiography with measurement of the intraventricular septum, posterior ventricular wall, internal ventricular diameter, and aortic root, and a blood test to determine serum creatinine levels. The ECG voltage was calculated by adding the R in V1 to the S in V5, and the cardiothoracic ratio (CTR) by dividing the maximal horizontal width of the heart by the horizontal inner width of the rib cage. Fundal changes were classified as grade 1 (arteriolar narrowing), grade 2 (increased light reflex or arteriovenous nipping), grade 3 (hemorrhages and exudates), and grade 4 (grade 3 plus papilledema). The widest width of the ascending aortic knob was measured in a horizontal line from the point of the left edge of the trachea to the left lateral wall of the aortic knob (Fig. 1). Left ventricular mass on echocardiography was calculated by the following formula:

$$1.04 \times ([\mathrm{LVID_d} + \mathrm{IVS_d} + \mathrm{LVPW_d}]^3 - \mathrm{LVID_d}^3) - 13.6\ \mathrm{g},$$[8]

where LVID = left ventricular internal diameter, d = diastole, IVS = intraventricular septum, and LVPW = left ventricular posterior wall; 1/creatinine was used as a measure of renal function because of the inverse relationship between creatinine and creatinine clearance.[9]

Patients were excluded if the chest radiograph was not properly centered, if there was any deviation of the trachea or shift of the mediastinum, and if there was underlying cardiac disease not due to hypertension or any known disease of the aorta (eg, Takayasu's arteritis).

Normotensive age- and sex-matched controls undergoing routine chest radiographs were evaluated. The horizontal width of the aortic knob was measured in the same manner as hypertensives.

Table 1. Baseline parameters of the normotensive and hypertensive patients

Parameter	Hypertensive (SD)	Normotensive (SD)
Number	72	77
Age (y)	40.5 (11)	37.2 (14)
Aortic knob width (cm)	3.69 (0.72)*	3.28 (0.46)*
Systolic BP (mm Hg)	164.8 (28.2)	—
Diastolic BP (mm Hg)	103.8 (13.4)	—
Creatinine (μmol/L)	103.2 (48.5)	—
Cardiothoracic ratio	0.48 (0.049)	—
ECG voltage	38.3 (14.2)	—
Left ventricular (LV) mass (g)	281.9 (129.3)	—

* $P < .0001$.
BP = blood pressure; ECG = electrocardiogram.

Statistical Analysis

The horizontal width of the aorta in normotensives and hypertensives was compared using the Student *t* test. The cardiothoracic ratio and horizontal width of the aorta was also compared with BP and other markers of target organ damage by univariate regression analysis, and multiple regression analysis was used to determine the independent correlations with left ventricular (LV) mass calculated by echocardiography.

Results

Seventy-two hypertensive and 77 age- and sex-matched normotensives were evaluated. The baseline characteristics of the two groups are listed in Table 1. No patient had evidence of coarctation of the aorta. There was a highly significant difference in the aortic knob width between the normotensive and hypertensive patients (3.28 cm *v* 3.69 cm, $P < .0001$). In addition, using univariate linear regression analysis aortic width was significantly correlated with age in both normotensive and hypertensive patients ($r = 0.53$, $P < .000001$ and $r = 0.53$. $P < .000002$). The aortic root measured by echocardiography was also correlated with age ($r = 0.18$, $P < .01$).

The aortic knob width was compared with other markers of target organ damage and BP levels by univariate linear regression analysis (Table 2). The aortic knob width was significantly correlated with both systolic and diastolic BP, and all markers of target organ damage except the ECG voltage calculation. In contrast, there was no correlation between clinic BP and markers of target organ damage including LV mass.

The cardiothoracic ratio was also significantly correlated with age and other markers of target organ damage, but not clinic BP (Table 3).

Table 2. Univariate linear regression analysis comparing aortic knob width with blood pressure and markers of target organ damage

Parameter	*R* Value	R^2	*P*
Age	0.52	0.28	< .000002
Aortic root	0.4	0.16	< .002
Systolic BP	0.39	0.16	< .0006
Diastolic BP	0.38	0.14	< .001
1/creatinine	−0.39	0.15	< .0006
Fundi	0.36	0.13	< .007
Cardiothoracic ratio	0.36	0.13	< .002
ECG voltage	0.04	0.001	.72
LV mass	0.32	0.1	< .02

Abbreviations as in Table 1.

However, multiple regression analysis revealed that only the cardiothoracic ratio ($r = 0.34$, $P < .02$) and the ECG voltage ($r = 0.58$, $P < .00005$) were the independently correlated with LV mass (Table 4).

Discussion

This small study has several important observations. Aortic knob width on the chest radiograph correlated significantly with age in both normotensives and hypertensives, and the aortic root on echocardiography, with age in hypertensive patients. In addition, the aortic knob width was significantly more dilated in hypertensives than in normotensives, and was directly related to both systolic and diastolic BP in hypertensives. These observations support the concept that progressive dilatation of the thoracic aorta is part of the aging process, which is exacerbated by hypertension.

The highly significant correlation of aortic width, but not LV mass, with both systolic and diastolic BP is an interesting observation. This suggests that aortic dilatation is much more dependent on pressure, whereas the mechanisms for LVH are far more complex. Blood pressure is one component in the genesis of LVH, and local factors

Table 3. Univariate linear regression analysis comparing cardiothoracic ratio with other markers of target organ damage

Parameter	*R* Value	R^2	*P*
Age	0.24	0.06	< .05
Aortic root	0.0	0.0	.98
Systolic BP	0.05	0.0	.63
Diastolic BP	0.16	0.03	< .17
1/creatinine	0.0	0.0	< .98
Fundi	0.42	0.17	< .001
Aortic width	0.36	0.13	< .002
ECG voltage	0.29	0.08	< .02
LV mass	0.29	0.08	< .03

Abbreviations as in Table 1.

Table 4. Multiple regression analysis to determine independent correlation with left ventricular mass

Parameter	*R* Value	*P*
Age	0.22	.19
Aortic root	0.08	.57
Aortic width	−0.12	.45
Systolic BP	−0.21	.21
Diastolic BP	0.21	.21
1/creatinine	−0.28	.053
Cardiothoracic ratio	0.34	< .02
ECG voltage	0.58	< .00005

Abbreviations as in Table 1.

such as the renin-angiotensin-aldosterone system and transforming growth factor-β1 (TGF-β1)are important cofactors.[10]

Aortic knob width is also a good marker of target organ damage as it correlated with 1/creatinine, fundal changes, cardiothoracic ratio, and LV mass on echocardiography by univariate regression analysis. In addition, the aortic width correlated with the aortic root measurement on echocardiography ($r = 0.4$, $P < .001$) supporting it as a reliable measurement of caliber of the thoracic aorta. Our tentative suggestion is that an aortic knob width >3.6 cm should be considered a marker of target organ damage.

Left ventricular hypertrophy is considered the most powerful determinant of cardiovascular outcome in hypertensive patients.[11] Echocardiography provides reliable measurement of LV mass, but this investigation is usually only available in specialist practice. It is, therefore, important to have predictors of LVH using investigations available to the primary care practitioner. Multivariate analysis was used to determine predictors of LVH in our study. The ECG voltage and cardiothoracic ratio were the only independent predictors (Table 3). The LIFE study has clearly shown that the ECG voltage can predict LVH hypertrophy on the echocardiogram,[2] but this study, to our knowledge, is the first to demonstrate the relationship between cardiothoracic ratio and LVH on echocardiography. Although the aortic knob width was not an independent predictor, it does not diminish its importance as a marker of target organ damage.

The chest radiograph may also have other important benefits in the evaluation of a hypertensive patient. It may identify coarctation of the aorta (an important secondary cause of hypertension), features of cardiac failure (an important complication of hypertension), aortic calcification of aortic stenosis, and smoking-related complications like emphysema, which may influence the choice of antihypertensive therapy.

With regard to frequency of repeating the chest radiogram, there is no information on the regression of aortic changes. This is an important area for further research but aortic dilatation is unlikely to regress rapidly and it is suggested that the radiograph be repeated after 1 year. The regression of LVH is an important issue, but the echocar-

diogram is probably a more reliable test to assess this. However, the finding of cardiomegaly on chest radiography should prompt a request for echocardiography.

In conclusion, the chest radiograph provides important predictive information of associated target organ damage in hypertensive patients. Also in conjunction with the ECG it may provide important information on the presence of LVH, a powerful marker of risk in hypertensive patients.

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References

1. Guidelines Subcommittee: 1999 World Health Organization–International Society of Hypertension guidelines for the management of hypertension. J Hypertens 1999;17:151–183.
2. Devereux RB, Bella J, Boman K, Gerdts E, Nieminen MS, Rokkedal J, Papademetriou V, Wachtell K, Parinicas M, Okin PM, Roman MJ, Smith G, Dahlof B: Echocardiographic left ventricular geometry in hypertensive patients with electrocardiographic left ventricular hypertrophy: the LIFE Study. Blood Press 2001;10:74–82.
3. Kim M, Roman MJ, Cavallini MC, Schwartz JE, Pickering TG, Devereux RB: Effect of hypertension on aortic root size and prevalence of aortic regurgitation. Hypertension 1996;28:47–52.
4. Bella JN, Wachtell K, Boman K, Palmieri V, Papademetriou V, Gerdts E, Aalto T, Olsen MH, Olofsson M, Dahlof B, Roman MJ, Devereux RB: Relation of left ventricular geometry to aortic root dilatation in patients with systemic hypertension and left ventricular hypertrophy (the LIFE Study). Am J Cardiol 2002;89:337–341.
5. Elsheikh M, Casadei B, Conway GS, Wass JA: Hypertension is a major risk factor for aortic root dilatation in women with Turner's syndrome. Clin Endocrinol 2001;54:69–73.
6. Iarussi D, Caruso A, Galderisi M, Covino FE, Dialetto G, Bossone E, de Divitiis O, Cetrufo M: Association of left ventricular hypertrophy and aortic dilation in patients with thoracic aortic dissection. Angiology 2001;52:447–455.
7. Palmieri V, Bella JN, Arnett DK, Roman MJ, Oberman A, Kitzman DW, Hopkins PN, Parinicas M, Rao DC, Devereux RB: Aortic root dilatation at sinuses of valsalva and aortic regurgitation in hypertensive and normotensive subjects: the Genetic Epidemiology Network Study. Hypertension 2001;37:1229–1235.
8. Liu JE, Devereux RB: Clinical assessment of cardiac hypertrophy, in Sheridan DJ (ed): Left Ventricular Hypertrophy, 1st ed. London, Churchill Communications, 1998.
9. Bleyer AJ: A reciprocal graph to plot reciprocal serum creatinine over time. Am J Kid Dis 1999;34:576–578.
10. Weber KT, Sun Y, Dhalla AK, Guntaka RV: Extracellular matrix and fibrosis in cardiac hypertrophy, in Sheridan DJ (ed): Left Ventricular Hypertrophy. 1st ed. London, Churchill Communications, 1998.
11. Kannel WB, Cobb J: Left ventricular hypertrophy and mortality: results from the Framingham study. Cardiology 1992;81:291–298.