Intravascular volume dependency of left ventricular mass calculation by two-dimensional guided M-mode echocardiography

Mustafa Kilickap MD¹, Sibel Turhan MD¹, Tamer Sayin MD¹, Gokhan Nergizoglu MD², Sim Kutlay MD², Neval Duman MD², Uzeyir Rahimov MD¹, Deniz Kumbasar MD¹, Gunes Akgun MD¹, Cetin Erol MD¹

M Kilickap, S Turhan, T Sayin, et al. Intravascular volume dependency of left ventricular mass calculation by two-dimensional guided M-mode echocardiography. *Can J Cardiol* 2007;23(3):219-222.

BACKGROUND: Increased left ventricular mass (LVM) is an independent risk factor for cardiovascular morbidity and mortality, and may be used for risk stratification. Two-dimensional echocardiography, the most commonly used technique for estimation of LVM, uses the third power of the left ventricular internal diameter (LVID) for the calculation.

OBJECTIVES: To determine whether a decrease in intravascular volume after dialysis may cause inaccurate estimation of LVM by echocardiography.

METHODS: Thirty-eight patients undergoing hemodialysis due to chronic renal failure constituted the study group (14 women [37%] and 24 men [63%], mean age $\pm$ SD 38.7 $\pm$ 10.9 years). LVID, and inter-ventricular and posterior wall thicknesses were measured by two-dimensionally guided M-mode echocardiography. Stroke volume and cardiac output were calculated using left ventricular outflow tract diameter and the pulsed-wave Doppler time-velocity integral obtained from left ventricular outflow tract. LVM was calculated by using Devereux's formula, and was indexed for body surface area and height. All echocardiographic parameters were measured or calculated before and after dialysis (on the same day), and then compared.

RESULTS: There were no significant changes in wall thickness; however, LVID, LVM, the LVM/body surface index and the LVM/height index significantly decreased after dialysis ($P < 0.001$ for each parameter). There was a significant correlation between the change in LVID and the change in LVM ($P < 0.001$, $r = 0.59$). Stroke volume and cardiac output also decreased significantly after hemodialysis ($P < 0.001$ for each parameter).

CONCLUSIONS: Intravascular volume-dependent change in LVID causes inaccurate estimation of LVM, so volume status should be kept in mind, especially in serial assessment of LVM.

Key Words: Echocardiography; Hemodialysis; Intravascular volume; Left ventricular mass

It is well known that increased left ventricular mass (LVM) is an independent risk factor for cardiovascular morbidity and mortality (1-3). The attributable risk of left ventricular hypertrophy for all-cause mortality is greater than that of single- or multivessel coronary artery disease, or low ejection fraction (4). Moreover, regression of the LVM index has been found to be associated with lower rates of clinical cardiovascular events (5). Several authors suggest that it may be a useful parameter

La dépendance du volume intravasculaire du calcul de la masse ventriculaire gauche par une échocardiographie bidimensionnelle en mode M

HISTORIQUE : L'augmentation de la masse ventriculaire gauche (MVG) est un facteur de risque indépendant de morbidité et de mortalité cardiovasculaires et peut être utilisée pour stratifier le risque. L'échocardiographie bidimensionnelle, la technique la plus utilisée pour évaluer la MVG, fait appel au troisième pouvoir du diamètre interne ventriculaire gauche (DIVG) pour le calcul.

OBJECTIFS : Déterminer si une diminution du volume intravasculaire après la dialyse peut causer une évaluation inexacte de la MVG par échocardiographie.

MÉTHODOLOGIE : Trente-huit patients sous hémodialyse en raison d'une insuffisance rénale chronique formaient le groupe d'étude (14 femmes [37 %] et 24 hommes [63 %], âge moyen de 38,7 $\pm$ 10,9 années). Le DIVG et l'épaisseur des parois interventriculaire et postérieure ont été mesurés par échocardiographie bidimensionnelle en mode M. Le volume de l'accident vasculaire cérébral (AVC) et le débit cardiaque ont été calculés au moyen du diamètre de la chambre de chasse du ventricule gauche et du temps-vélocité Doppler pulsé intégral obtenu dans la chambre de chasse du ventricule gauche. La MVG a été calculée à l'aide de la formule de Devereux et indexée pour la zone de surface corporelle et la hauteur. Tous les paramètres échocardiographiques ont été mesurés ou calculés avant et après la dialyse (le même jour), puis comparés.

RÉSULTATS : L'épaisseur de la paroi n'avait pas changé de manière significative. Toutefois, le DIVG, la MVG, l'indice de surface de la MVG par rapport au corps et l'indice de la MVG par rapport à la hauteur ont considérablement diminué après la dialyse ($p < 0,001$ par paramètre). Il y avait une corrélation importante entre le changement du DIVG et le changement de la MVG ($p < 0,001$, $r = 0,59$). Le volume de l'AVC et le débit cardiaque diminuaient également considérablement après l'hémodialyse ($p < 0,001$ par paramètre).

CONCLUSION : Un changement du DIVG dépendant du volume intravasculaire s'associe à une évaluation inexacte de la MVG. Il faut donc tenir compte du statut du volume, surtout pendant l'évaluation sérielle de la MVG.

in risk stratification and guiding treatment, especially for hypertensive patients at low or medium risk (6,7). For these reasons, accurate estimation of LVM has crucial importance.

Echocardiography is the most commonly used technique for the estimation of LVM. Devereux et al (8,9) proposed a formula for the estimation of LVM and found that the LVM calculated by this formula was consistent with the necropsy data. Calculation of LVM by this formula requires some geometric assumptions.

¹Cardiology Department; ²Nephrology Department, Ankara University School of Medicine, Ankara, Turkey

Correspondence: Dr Mustafa Kilickap, Ankara Üniversitesi Tıp Fakültesi Kalp Merkezi, Kardiyoloji ABD, 06590 Dikimevi, Ankara, Turkey.

Telephone 90-312-595-60-00 ext 6286, fax 90-312-363-22-89, e-mail mkilickap@yahoo.com

Received for publication November 24, 2005. Accepted May 3, 2006

The formula uses the third power of the left ventricular internal dimension (LVID); therefore, change in LVID alone may result in inaccurate estimation of LVM. However, data are inconsistent on whether estimation of LVM by this formula is sensitive to changes in LVID caused by the changes in intravascular volume. In the present study, to detect whether echocardiographic estimation of LVM may be influenced by the change in intravascular volume or LVID, we calculated LVM before and after hemodialysis. Because LVM is not expected to change in one day, any difference in calculated LVM after hemodialysis suggests that this calculation is sensitive to a decrease in intravascular volume.

PATIENTS AND METHODS

Patients

Thirty-eight patients undergoing hemodialysis due to chronic renal failure constituted the study group. Exclusion criteria included technically poor quality of echocardiographic images precluding satisfactory imaging and measurement, presence of atrial fibrillation, asymmetrical hypertrophy and previous myocardial infarction.

Echocardiographic examination

All echocardiographic examinations were performed using Sonos 5500 (Philips Medical Imaging, USA). Interventricular septal thickness, posterior wall thickness and LVID at end-diastole were measured from the LV parasternal long-axis view using two-dimensional guided M-mode echocardiography according to the American Society of Echocardiography guidelines (10). Meticulous care was taken to correct the alignment of the cursor to precisely intersect the interventricular septum and posterior wall perpendicularly. All parameters were measured in at least three consecutive beats, and the arithmetic means were used for statistical analysis. LVM was calculated using the following equation, proposed by Devereux et al (9), in which IVST and PWT denote interventricular septal thickness and posterior wall thickness, respectively:

$$\text{LVM (g)} = ([\text{IVST} + \text{LVID} + \text{PWT}]^3 - [\text{LVID}]^3) \times 1.04 \times 0.8 + 0.6$$

The LVM index was calculated by dividing the LVM by body surface area (LVM/BSA [g/m²]) and height (LVM/h [g/m]). To demonstrate the possible effect of the change in intravascular volume on the calculation of LVM, all parameters were measured before and after dialysis (on the same day), and then compared. Stroke volume and cardiac output were also calculated before and after dialysis. Doppler-derived stroke volume was calculated as the product of the LV outflow tract cross-sectional area and the LV outflow tract time-velocity integral obtained by pulsed Doppler. Cardiac output was calculated as the product of stroke volume and heart rate.

To assess intraobserver and interobserver variability for LVM calculation, 17 randomly selected recordings that had been stored on videotape were remeasured by the same operator (MK) one month after the first set of measurements. Also, the same 17 recordings were analyzed by another operator (ST) in a blinded fashion to determine interobserver variability.

Statistics

Statistical analysis was performed by SPSS (version 11.5, SPSS Inc, USA). Data are expressed as arithmetic mean $\pm$ SD. Echocardiographic measurements before and after dialysis were compared using paired *t* tests. A correlation between the change in LVM and the change in LVID was assessed by Pearson's correlation analysis. *P* < 0.05 was considered statistically significant. Intraobserver and interobserver variabilities were assessed in

three ways: as the difference between two readings in per cent of the mean; by the intraclass correlation coefficient, which varies between 0 and 1, and values between 0.8 and 1.0 suggest almost perfect agreement (11); and by the Bland-Altman analysis to determine the mean difference between the measurements (bias) and 95% limits of agreement (mean difference $\pm$ 1.96 $\times$ SD) (12). The differences between the two measurements were plotted against their means.

RESULTS

The study group consisted of 14 women (37%) and 24 men (63%), and the mean age was 38.7 $\pm$ 10.9 years. Intraobserver variability was 2.1% and interobserver variability was 3.1%. The intraclass correlation coefficient was 0.94 for each of the intraobserver and interobserver agreements. The Bland-Altman analysis revealed that the mean difference in LVM was -3.32 g for intraobserver measurements and +4.84 g for interobserver measurements (Figures 1A and 1B).

Mean patient weight decreased after dialysis from 62.23 $\pm$ 12.64 kg to 59.85 $\pm$ 12.31 kg (*P* < 0.001). Stroke volume and cardiac output also decreased after dialysis, from 97.67 $\pm$ 28.40 mL/beat to 73.28 $\pm$ 20.53 mL/beat (*P* < 0.001), and from 6.87 $\pm$ 2.07 L/min to 5.86 $\pm$ 2.01 L/min (*P* < 0.001), respectively.

Although there were no significant changes in IVST and PWT, the LVID, LVM, LVM/BSA and LVM/h significantly decreased after dialysis (Table 1). There was a significant correlation between the change in LVID and the change in LVM (*P* < 0.001, *r* = 0.59).

DISCUSSION

Increased LVM is associated with increases in cardiovascular morbidity and mortality. Therefore, its measurement may be used as a surrogate parameter for the assessment of future patient cardiovascular risk. Conventional echocardiography has been the most widely applied method for the estimation of LVM; however, in contrast to three-dimensional echocardiography and magnetic resonance imaging, it uses geometric assumptions for the calculation. Because the third power of LVID is used in this formula, change in LVID alone may cause an erroneous estimation of LVM. In the present study, we measured LVID, interventricular septal and posterior wall thicknesses, and calculated LVM before and after hemodialysis. Although the wall thicknesses did not significantly change, LVID and LVM significantly decreased after dialysis. Moreover, there was a significant positive correlation between the change in LVID and the change in LVM. Because LVM is not expected to regress acutely after dialysis, we concluded that the change in LVM is a function of the change in LVID.

Our findings are consistent with several previous observations that demonstrated that the LVM calculation by echocardiography is sensitive to changes in effective intravascular volume (13,14), and suggest that volume status should be kept in mind when assessing LVM. Because LVM may be considered as a surrogate marker for cardiovascular risk, patients who are taking diuretics or undergoing hemodialysis may erroneously be considered to be at high or low risk, depending on their intravascular volume, if LVM is calculated by two-dimensional guided M-mode echocardiography. This issue is especially important if a temporal change in LVM (in other words, future cardiovascular risk) is being assessed.

Figure 1 Bland-Altman plots of intraobserver (A) and interobserver (B) differences in the calculated left ventricular mass (LVM) and 95% limits of agreement ($\pm 1.96 \times \text{SD}$). The mean differences in LVM were -3.32 ± 12.98 g for intraobserver measurements and $+4.84 \pm 11.46$ g for interobserver measurements

Hemodialysis causes a substantial decrease in plasma volume in a brief time, which is not expected to occur in a majority of patients who are evaluated in daily practice. For this reason, this may be considered a limitation of our study, and our suggestions may be considered to be valid only for patients undergoing hemodialysis. Prisant et al (13) evaluated the change in calculated LVM after administration of intravenous furosemide. Although the decrease in mean weight (and possibly intravascular volume) in their group seemed to be less remarkable than that in our patients, they also found a significant decrease in calculated LVM after intravenous furosemide administration. A decrease in LVM is possibly caused by a decrease in LVID (corresponding to intravascular volume), so it can be expected that the difference in LVM is correlated with the difference in weight, as in our study. Although our study did not evaluate the effect of diuretics on the estimation of LVM, based on our study

TABLE 1
Comparisons of weight, and echocardiographic measurements and calculations

	Before dialysis	After dialysis	P
Weight, kg	62.23 $\pm$ 12.64	59.85 $\pm$ 12.31	<0.001
Stroke volume, mL/beat	97.67 $\pm$ 28.40	73.28 $\pm$ 20.53	<0.001
Cardiac output, L/min	6.87 $\pm$ 2.07	5.86 $\pm$ 2.01	<0.001
Interventricular septal thickness, cm	1.00 $\pm$ 0.15	1.00 $\pm$ 0.15	0.61
Posterior wall thickness, cm	0.90 $\pm$ 0.14	0.91 $\pm$ 0.13	0.47
Left ventricular internal dimension, cm	5.29 $\pm$ 0.55	4.74 $\pm$ 0.69	<0.001
LVM, g	191.28 $\pm$ 60.42	160.36 $\pm$ 55.76	<0.001
LVM/body surface area ratio, g/m ²	115.64 $\pm$ 33.93	98.67 $\pm$ 31.78	<0.001
LVM/height ratio, g/m	115.94 $\pm$ 32.92	97.05 $\pm$ 30.37	<0.001

LVM Left ventricular mass

and the above-mentioned study, it can be speculated that patients who are taking diuretics for heart failure may also be prone to inaccurate estimation of LVM. However, according to the Frank-Starling law, failed hearts have unique hemodynamics, so one should be cautious when extending these findings to the patients taking diuretics for decompensated heart failure.

Similar to our findings, Harnett et al (14) found that the LVM index significantly decreased after dialysis; however, they did not find any correlation between the difference in LVM and the difference in weight. They concluded that this may be a function of their relatively crude assessment of intravascular volume.

In a well-designed study, Lantelme et al (15) demonstrated that a nitroglycerine-induced decrease in LVID had no effect on LVM. The decrease in effective intravascular volume in that study may have been smaller than in our group. Although they did not state change in body weight, stroke volume and cardiac output in their article, the change in mean LVID was less than that in ours (-0.21 versus -0.55). We think that there may be a critical volume that does not affect the calculated LVM significantly. However, our study did not address how much volume change results in miscalculation of LVM, but simply assessed the volume dependency of this formula.

CONCLUSIONS

The change in LVID caused by the change in plasma volume may result in inaccurate estimation of echocardiographic LVM, and this issue should be kept in mind in serial assessments of LVM. Three-dimensional echocardiography or cardiac magnetic resonance imaging may be more appropriate options for the calculation of LVM in patients who have a substantial change in their intravascular volume.

REFERENCES

- Levy D, Garrison RJ, Savage DD, Kannel WB, Castelli WP. Prognostic implications of echocardiographically determined left ventricular mass in the Framingham Heart Study. *N Engl J Med* 1990;322:1561-6.
- Schillaci G, Verdecchia P, Porcellati C, Cuccurullo O, Cosco C, Perticone F. Continuous relation between left ventricular mass and cardiovascular risk in essential hypertension. *Hypertension* 2000;35:580-6.
- Casale PN, Devereux RB, Milner M, et al. Value of echocardiographic measurement of left ventricular mass in

- predicting cardiovascular morbid events in hypertensive men. *Ann Intern Med* 1986;105:173-8.
 4. Chobanian AV, Bakris GL, Black HR, et al; Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. National Heart, Lung, and Blood Institute; National High Blood Pressure Education Program Coordinating Committee. Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. *Hypertension* 2003;42:1206-52.
 5. Devereux RB, Wachtell K, Gerds E, et al. Prognostic significance of left ventricular mass change during treatment of hypertension. *JAMA* 2004;292:2350-6.
 6. European Society of Hypertension-European Society of Cardiology Guidelines Committee. 2003 European Society of Hypertension-European Society of Cardiology guidelines for the management of arterial hypertension. *J Hypertens* 2003;21:1011-53. (Errata in 2003;21:2203-4, 2004;22:435).
 7. Schillaci G, De Simone G, Reboldi G, Porcellati C, Devereux RB, Verdecchia P. Change in cardiovascular risk profile by echocardiography in low- or medium-risk hypertension. *J Hypertens* 2002;20:1519-25.
 8. Devereux RB, Reichek N. Echocardiographic determination of left ventricular mass in man. Anatomic validation of the method. *Circulation* 1977;55:613-8.
 9. Devereux RB, Alonso DR, Lutas EM, et al. Echocardiographic assessment of left ventricular hypertrophy: Comparison to necropsy findings. *Am J Cardiol* 1986;57:450-8.
 10. Schiller NB, Shah PM, Crawford M, et al. Recommendations for quantitation of the left ventricle by two-dimensional echocardiography. American Society of Echocardiography Committee on Standards, Subcommittee on Quantitation of Two-Dimensional Echocardiograms. *J Am Soc Echocardiogr* 1989;2:358-67.
 11. Landis JR, Koch GG. The measurement of observer agreement for categorical data. *Biometrics* 1977;33:159-74.
 12. Bland JM, Altman DG. Statistical methods for assessing agreement between two methods of clinical measurement. *Lancet* 1986;1:307-10.
 13. Prisant LM, Kleinman DJ, Carr AA, Bottini PB, Gross CM. Assessment of echocardiographic left ventricular mass before and after acute volume depletion. *Am J Hypertens* 1994;7:425-8. (Abst)
 14. Harnett JD, Murphy B, Collingwood P, Purchase L, Kent G, Parfrey PS. The reliability and validity of echocardiographic measurement of left ventricular mass index in hemodialysis patients. *Nephron* 1993;65:212-4.
 15. Lantelme P, Bouchayer D, Gayet C, Lievre M, Gessek J, Milon H. Influence of a rapid change of left ventricular dimensions on the echocardiographic measurement of left ventricular mass by the Penn convention. *J Hypertens* 1999;17:1323-8.
-
-