The Correlation between Aortic Stiffness and Left Ventricular Mass Index in Hypertensive Patients: A Cardiac MRI Study

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Background and Objective: *In hypertensive patients, increased left ventricular (LV) mass and impaired aortic stiffness are independent predictors for cardiovascular events. There were some prior studies whichestablished the correlation between left ventricular hypertrophy and aortic stiffness; nevertheless, there are limited data in hypertensive patients. Furthermore, few studies applied cardiac MRI which is a promising technique for LV mass assessment. The authors sought to assess the correlation of LV mass and impaired aortic stiffness, as measured by cardiac MRI.*

Material and Method: *A total of 113 hypertensive patients (mean age 68.9 $\pm$ 10.3 years, female 51.3%) who underwent cardiac MRI study were enrolled. Left ventricular mass was obtained by summation of multiple slice technique and then calculated into the left ventricular mass index. Aortic stiffness was measured as aortic pulse wave velocity (PWV) by distance divided by time delay between mid-ascending and mid-descending aorta. Pearson correlation analysis was applied to determine the correlation of aortic stiffness and left ventricular mass index.*

Results: *Mean left ventricular mass index was 53.39 $\pm$ 18.32 g/m^2, mean PWV was 11.72 $\pm$ 5.11 m/s. No correlation was found between PWV and LV mass index (r = 0.085, p-value = 0.37). However, aortic stiffness had significant correlation with age(r = 0.469, p-value < 0.001).*

Conclusion: *There was no significant correlation between LV mass index and aortic stiffness in hypertensive patients. This may be explained by the relatively less severe left ventricular hypertrophy in our patients.*

Keywords: *Aortic stiffness, Pulse wave velocity, Left ventricular mass, Hypertension, Magnetic resonance imaging*

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Hypertension is one of major risk factors of cardiovascular disease, leading to target organ damage such as left ventricular hypertrophy, albuminuria, etc. There is extensive evidence that indicates those target organ damages are the marker of high risk for cardiac events.

Arterial stiffness is the condition of decreased vascular elasticity in many different sizes of blood vessels causing increase in systolic blood pressure, pulse pressure and left ventricular mass and decrease in diastolic blood pressure, as well as coronary perfusion abnormality[1]. Arterial stiffness can be detected before manifested cardiovascular disease and may playarole as a marker for the development of atherosclerotic disease[2].

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There are many vascular changes implicate stiffness such as decreased elastin fibers, collagen deposition, vascular inflammation, medial smooth muscle necrosis, and substitution of macromolecule in vessel wall[3-5]. Factors affected arterial stiffness, such as age, smoking[6], obesity[7], hypertension[8,9], hyperlipidemia[10,11], metabolic syndrome[12], type 1 and type 2 diabetes[12], hyperhomocyteinemia[13] and high C-reactive protein level[14,15], have been established from several studies and also showed that arterial stiffness is an independent predictor for cardiovascular events in the general population[16,17], in elderly patients[18], in hypertension[19,20] and in end-stage renal disease (ESRD)[21,22].

Various methods can be used to assess the arterial stiffness. Pulse wave velocity (PWV) is a technique that can provide information about the distensibility of the vessel being studied. It represents the speed of blood flow along the aorta through the distal branches. PWV will be increased when the vessel becomes stiffened or haspoor compliance. PWV

commonly measured by ultrasonography,which has alimitation for the evaluation of large arteries, such as aorta, due to poor acoustic penetration; Cardiac Magnetic Resonance Imaging (MRI) should be the preferred alternative method that can accurately measure the PWV because it provides cross-sectional images of aorta with high resolution and can directly measure the length of aorta. This technique overcomes the limitation of other modalities that merely use rough estimation of vessel length.

There were some prior studies which established the correlation between left ventricular hypertrophy and aortic stiffness; nevertheless, there are limited data in hypertensive patients[23-25]. Furthermore, few studies applied cardiac MRI which is the promising technique for LV mass assessment. We sought to assess the correlation of LV mass and impaired aortic stiffness, as measured by cardiac MRI.

Material and Method

The present study was cross-sectional, single center study. A total of 113 hypertensive patients who underwent cardiac MRI study at Siriraj hospital were enrolled. Inclusion criteria were: male or female age 18 or above with hypertension defined as 1) systolic blood pressure more than 140 mmHg, or diastolic blood pressure more than 90 mmHg at rest or 2) treated with antihypertensive medication. Exclusion criteria were these: unable to perform due toferromagnetic prosthesis or claustrophobia, pregnancy, aortic aneurysm, hypertrophic cardiomyopathy, severe aortic stenosis, severe aortic regurgitation, severe mitral regurgitation, severe LV systolic dysfunction. The present study protocol was approved by ethic committee and each subject gave informed consent. The primary objective was to assess the correlation of LV mass and impaired aortic stiffness.

MRI analysis of aortic stiffness

PWV was measured as an indicator of aortic stiffness. Cardiac MRI was performed using a 1.5-T Phillips Achieva XR scanner (Phillips Medical System, Best, the Netherlands). Cardiac axis was located by ECG-triggered non breath hold black blood single shot sequentive 100 slides. The scanning parameters were: echo time (TE) 20 ms; repetition time (TR) 1,800 ms, refocusing flip angle 90°, slice thickness 8 mm; field of view in x axis (FOVx) 240 to 360 mm; field of view in y axis (FOVy) 250 to 280 mm; typical matrix size 118 x 115; and typical acquired spatial resolution 1.59-1.86 x 2.17-2.43 mm.

PWV was assessed by using velocity encoded MRI (VE-MRI) technique as the through plane flow in the mid ascending and descending aorta at the level of pulmonary trunk with the following scanning parameters: Echo time (TE) 3.6 ms, Repetitive time TR 5.3 ms, Refocusing flip angle 12°, slice thickness 8 mm; FOVx 320 mm; FOVy 270 mm, typical matrix size 160 x 132, typical acquired spatial resolution 2.0 x 2.04 mm,temporal resolution 10 to 12 ms and velocity encoding 170 cm/s.

Image analysis

Cardiovascular imaging software (Extended workspace) was used to draw the contours of the mid-ascending and -descending aorta. The flow at these 2 levels was obtained in all phases of the cardiac cycle. next, the corresponding flow time curve was created. The arrival time of pulse wave was measured as the point of interception of linear extrapolation of baseline and steep early systolic stage. From the reconstructed sagittal view corresponding to the same level as VE-MRI image obtained, the centerline was drawn from the level of the mid-ascending aorta to the mid-descending aorta to obtained the aortic path length[10] (Fig. 1). The PWV between the mid-ascending and mid-descending aorta was calculated according to the following formula:

$$PWV = Dx/Dy \quad (m/s)$$

where Dx = aortic path length between the mid-ascending and mid-descending aorta(m) Dy = timedelay(s)

A higher pulse wave velocity indicated a stiffer aorta.

Assessment of Left ventricular mass index and other parameters

Leftventricular mass index was measured as the indicator of left ventricular hypertrophy. Study was performed using 1.5-T MRI scanner (Phillips, Achieva, Netherlands) withbalanced-turbo-fast-field-echo (b-TFE) technique invertical long-axis, four-chamber and multiple slice short-axis view. Default parameters were: repetition time/echo time/number of excitations = 3.7/1.8/2, 390 x 312 mm field of view, 256 x 240 matrix,1.25 x 1.25 x 8 cm^2 reconstruction pixel, 8 mm slice thickness, and 60° flip angle, 25 cardiac phases.

Left ventricular volume was obtained by summation of left ventricular cross-sectional area multiplied by slice thickness from each short-axis slice; then were calculated the left ventricular end-diastolic volume (LVEDV), left ventricular end-systolic volume

(LVESV) and left ventricular ejection fraction (LVEF).

Left ventricular mass was quantified by summation of end-diastolic myocardial volume and then converted volume to mass by constant density factor of 1.05 g/mL. Finally, the LV mass was divided by body surface area to obtain LV mass index.

Intraobserver and interobserver reproducibility of PWV was performed in 15 randomly selected patients.

Statistical analysis

The statistical analyses were performed by SPSS version 13.0. Continuous data were expressed as mean ± SD. Categorical data were presented as numbers and percentages. Pearson's correlation coefficient was used to compare 2 continuous variables. For assessment of agreement within single observer and between observers, a Bland-Altman plot was performed and intraclass correlation coefficients (ICC) were calculated. P-value < 0.05 was considered statistically significant.

Results

Baseline characteristics

Patient characteristics and MRI parameters are shown in Table 1 and 2. There were 113 patients included in the present study with mean age 68.9 ± 10.3 years, female 51.3%. Mean PWV was 11.72 ± 5.11m/s. (Table 1).

Table 1. Clinical Characteristics of the study patients (n = 113)

Variables	Number (%) or Mean ± SD
Age (yrs)	68.88 ± 10.29
Men/Woman	55 (48.7)/58 (51.3)
Height (cm)	156.90 ± 14.82
Weight (kg)	67.31 ± 14.82
Body surface area (m^2)	1.71 ± 0.20
Systolic blood pressure (mmHg)	154.41 ± 21.72
Diastolic blood pressure (mmHg)	81.12 ± 13.17
Heart rate (bpm)	78.45 ± 18.26
Underlying disease	
Diabetes mellitus	52 (46.02)
Dyslipidemia	94 (83.18)
Smoking	20 (17.70)
Medications	
Aspirin	45 (39.82)
Clopidogrel	15 (13.27)
Beta blocker	45 (39.82)
Angiotensin-converting enzyme inhibitor	13 (11.50)
Angiotensin receptor blocker	22 (19.46)
Calcium channel blocker	23 (20.35)
Statins	52 (46.02)

Continuous data represented as mean ± SD
Categorical data represented as number (percent)

Table 2. Cardiac MRI parameters

Parameters	Mean ± SD
Pulse wave velocity(m/s)	11.52 ± 5.11
LVEDV (ml)	134.89 ± 42.78
LVESV (ml)	42.28 ± 34.28
LVEF(%)	67.40 ± 11.28
LV mass (g)	92.14 ± 37.56
LV mass index (ml)	53.59 ± 18.32

LVEDV: left ventricular end-diastolic volume
LVESV: left ventricular end-systolic volume
LVEF: left ventricular ejection fraction

Correlation analysis

No correlation was found between PWV and LV mass index (r = 0.085 , p-value = 0.37). However, aortic stiffness and age have reasonable correlation (r = 0.469, p-value < 0.001) (Fig. 2).

Reproducibility of PWV measurements

There was good intra observer reproducibility of PWV as well as inter observer reproducibility in general (Table 3, Fig. 3 and 4).

Discussion

Reaching theprimary objective of the present study, no correlation was found between left ventricular mass index and aortic stiffness measured as aortic pulse wave velocity (PWV) determined by cardiac MRI. However there was significant correlation between aortic PWV and age which supports previous data from several studies.

To our knowledge, this is the first study that used cardiac MRI to assess the correlation between aortic PWV and left ventricular mass index in hypertensive patients. Previous studies that show such a positive correlation used a different method from our study for assessment. Bouthier et al[26] evaluate the left ventricular mass/volume ratio measured byechocardiography and aortic stiffness as Carotid-femeral pulse wave velocity (PWVcf) using doppler ultrasonography in patient with hypertension.

Another study from Nitta et al[27], shows significant correlation between arterial PWV obtained by brachial-tibial pressure waveform analysis and left ventricular mass index evaluated by echocardiography in end stage renal disease patients.

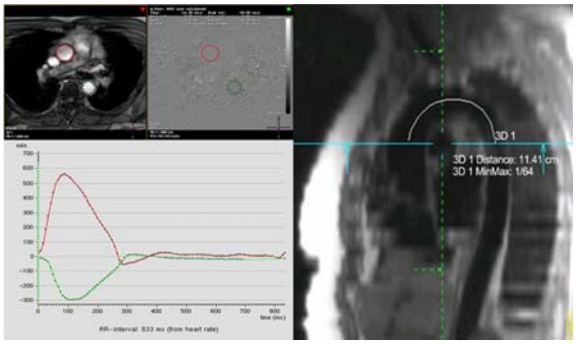

Fig. 1 Measurement of time delay and aortic path length between mid ascending and descending aorta. (Left Upper) Velocity-encoded MRI acquired at mid-ascending (dark circles) and mid-descending thoracic aorta (pale circles). (Left Lower) Corresponding measurement of flow at 2 sites (mid-ascending as black line and mid-descending thoracic aorta as dotted line). (Right) Measurement of aortic path length from multiplanar reconstruction

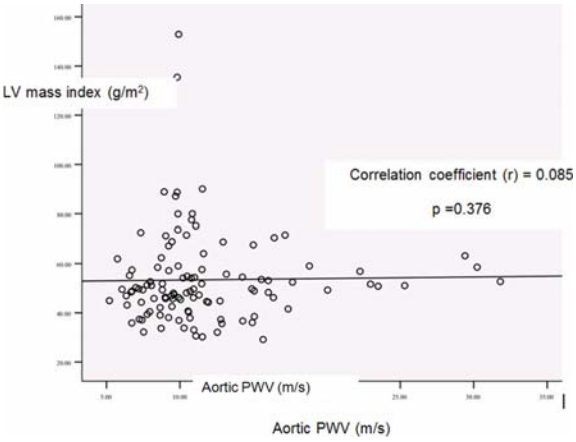

Fig. 2 Pearson correlation of aortic PWV and LV mass index

There is a possibleexplanationfor our result that no correlation was observed with left ventricular mass index. Patients in the previous study by Nitta et al[27], had significant left ventricular hypertrophy 185.7 $\pm$ 35.7 g/m^2 compare with 53.39 $\pm$ 18.32 g/m^2 from our study.

The present study had some limitations. First, the authors could not apply our result to the all hypertensive patients,because the patients being studied have some predisposing factors towards coronary artery disease. Second, many patients in the

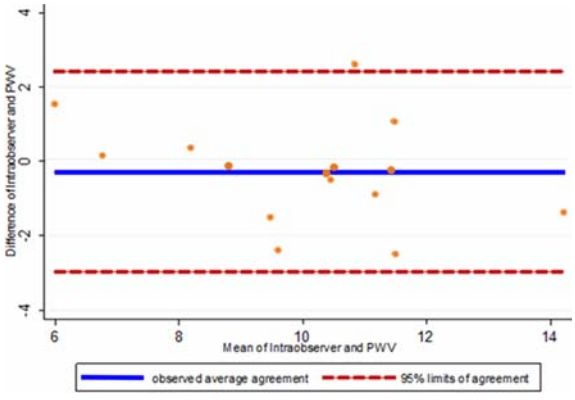

Fig. 3 Bland-Altman plot of Intraobserver PWV measurement

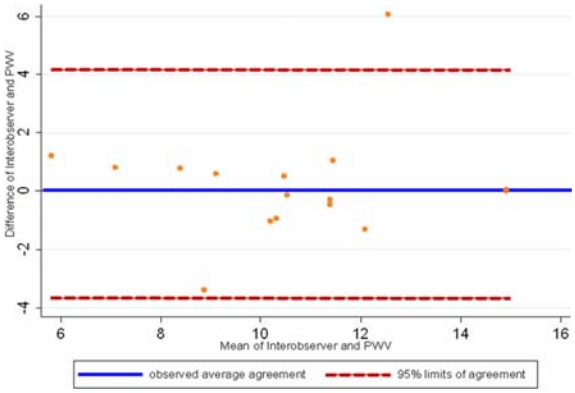

Fig 4. Bland-Altman plot of Interobserver PWV measurement

Table 3. Bland-Altman analysis and intraclass correlation coefficients (ICC) of the intraobserver and interobserver agreement of aortic PWV

Variables	Difference of average (95% Limit of agreement)	ICC (95% CI of ICC)
Intraobserver-PWV	-0.28 (-2.98 to 2.42)	0.80 (0.50 to 0.93)
Interobserver-PWV	0.24 (-3.68 to 4.16)	0.67 (0.26 to 0.88)

present study received some medication, that was not stoppedbefore testing,and thismight affect both aorticstiffness such as ACE inhibitor[28], ARBs[29], betablocker[30], statins[31]. Therefore, there could an effect on the differentsetof result between patients who hadreceived and who not received these medications.

However, our study had some strengths. First, we use aortic PWV measure by MRI which is a well accepted indicator of arterial stiffness without assumptions about central pressure and vessel length. Second, left ventricular mass index assessed by MRI was highly accurate and reproducible. Both of these study protocols could be performed in the same period of timewithout additional instrument use.

Perspectives

Data from previous studies shown significant correlation between aortic stiffness and left ventricular mass. The present study does not confirm this correlation. However larger population needs to be evaluated by this method including those with more severe left ventricular hypertrophy, in order to demonstrate any positive correlation.

Conclusion

The present study cannot demonstrate the significant correlation between LV mass index and aortic stiffness measured by cardiac MRI in hypertensive patients.

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Potential conflicts of interest

None.

References

1. Laurent S, Cockcroft J, Van Bortel L, Boutouyrie P, Giannattasio C, Hayoz D, et al. Expert consensus document on arterial stiffness: methodological issues and clinical applications. Eur Heart J 2006; 27: 2588-605.
2. Mitchell GF, Hwang SJ, Vasan RS, Larson MG, Pencina MJ, Hamburg NM, et al. Arterial stiffness and cardiovascular events: the Framingham Heart Study. Circulation 2010; 121: 505-11.
3. Laurent S, Boutouyrie P, Lacolley P. Structural and genetic bases of arterial stiffness. Hypertension 2005; 45: 1050-5.
4. Dobrin PB. Vascular mechanics. In: Shepherd JT, Abboud FM, editors. Handbook of physiology. Section 2, Vol 3: The cardiovascular system. Washington, DC: American Physiological Society; 1983: 65-102.
5. Lakatta EG, Levy D. Arterial and cardiac aging: major shareholders in cardiovascular disease enterprises: Part I: aging arteries: a "set up" for vascular disease. Circulation 2003; 107: 139-46.
6. Kool MJ, Hoeks AP, Struijker Boudier HA, Reneman RS, Van Bortel LM. Short- and long-term effects of smoking on arterial wall properties in habitual smokers. J Am Coll Cardiol 1993; 22: 1881-6.
7. Ferreira I, Henry RM, Twisk JW, van Mechelen W, Kemper HC, Stehouwer CD. The metabolic syndrome, cardiopulmonary fitness, and subcutaneous trunk fat as independent determinants of arterial stiffness: the Amsterdam Growth and Health Longitudinal Study. Arch Intern Med 2005; 165: 875-82.
8. O'Rourke MF. Arterial function in health and disease. Edinburgh: Churchill Livingstone; 1982.
9. Simon AC, Levenson J, Bouthier J, Safar ME, Avolio AP. Evidence of early degenerative changes in large arteries in human essential hypertension. Hypertension 1985; 7: 675-80.
10. Aggoun Y, Bonnet D, Sidi D, Girardet JP, Brucker E, Polak M, et al. Arterial mechanical changes in children with familial hypercholesterolemia. Arterioscler Thromb Vasc Biol 2000; 20: 2070-5.
11. Wilkinson IB, Prasad K, Hall IR, Thomas A, MacCallum H, Webb DJ, et al. Increased central pulse pressure and augmentation index in subjects with hypercholesterolemia. J Am Coll Cardiol 2002; 39: 1005-11.
12. Schram MT, Henry RM, van Dijk RA, Kostense PJ, Dekker JM, Nijpels G, et al. Increased central artery stiffness in impaired glucose metabolism and type 2 diabetes: the Hoorn Study. Hypertension 2004; 43: 176-81.
13. Bortolotto LA, Safar ME, Billaud E, Lacroix C, Asmar R, London GM, et al. Plasma homocysteine, aortic stiffness, and renal function in hypertensive patients. Hypertension 1999; 34: 837-42.
14. Booth AD, Wallace S, McEniery CM, Yasmin,

Brown J, Jayne DR, et al. Inflammation and arterial stiffness in systemic vasculitis: a model of vascular inflammation. Arthritis Rheum 2004; 50: 581-8.
15. Yasmin, McEniery CM, Wallace S, Mackenzie IS, Cockcroft JR, Wilkinson IB. C-reactive protein is associated with arterial stiffness in apparently healthy individuals. Arterioscler Thromb Vasc Biol 2004; 24: 969-74.
16. Shokawa T, Imazu M, Yamamoto H, Toyofuku M, Tasaki N, Okimoto T, et al. Pulse wave velocity predicts cardiovascular mortality: findings from the Hawaii-Los Angeles-Hiroshima study. Circ J 2005; 69: 259-64.
17. Willum-Hansen T, Staessen JA, Torp-Pedersen C, Rasmussen S, Thijs L, Ibsen H, et al. Prognostic value of aortic pulse wave velocity as index of arterial stiffness in the general population. Circulation 2006; 113: 664-70.
18. Boutouyrie P, Tropeano AI, Asmar R, Gautier I, Benetos A, Lacolley P, et al. Aortic stiffness is an independent predictor of primary coronary events in hypertensive patients: a longitudinal study. Hypertension 2002; 39: 10-5.
19. Laurent S, Boutouyrie P, Asmar R, Gautier I, Laloux B, Guize L, et al. Aortic stiffness is an independent predictor of all-cause and cardiovascular mortality in hypertensive patients. Hypertension 2001; 37: 1236-41.
20. Laurent S, Katsahian S, Fassot C, Tropeano AI, Gautier I, Laloux B, et al. Aortic stiffness is an independent predictor of fatal stroke in essential hypertension. Stroke 2003; 34: 1203-6.
21. London GM, Blacher J, Pannier B, Guerin AP, Marchais SJ, Safar ME. Arterial wave reflections and survival in end-stage renal failure. Hypertension 2001; 38: 434-8.
22. Shoji T, Emoto M, Shinohara K, Kakiya R, Tsujimoto Y, Kishimoto H, et al. Diabetes mellitus, aortic stiffness, and cardiovascular mortality in end-stage renal disease. J Am Soc Nephrol 2001; 12: 2117-24.
23. Asmar RG, Pannier B, Santoni JP, Laurent S, London GM, Levy BI, et al. Reversion of cardiac hypertrophy and reduced arterial compliance after converting enzyme inhibition in essential hypertension. Circulation 1988; 78: 941-50.
24. Boutouyrie P, Laurent S, Girerd X, Benetos A, Lacolley P, Abergel E, et al. Common carotid artery stiffness and patterns of left ventricular hypertrophy in hypertensive patients. Hypertension 1995; 25: 651-9.
25. London GM, Pannier B, Guerin AP, Marchais SJ, Safar ME, Cuche JL. Cardiac hypertrophy, aortic compliance, peripheral resistance, and wave reflection in end-stage renal disease. Comparative effects of ACE inhibition and calcium channel blockade. Circulation 1994; 90: 2786-96.
26. Bouthier JD, De Luca N, Safar ME, Simon AC. Cardiac hypertrophy and arterial distensibility in essential hypertension. Am Heart J 1985; 109: 1345-52.
27. Nitta K, Akiba T, Uchida K, Otsubo S, Otsubo Y, Takei T, et al. Left ventricular hypertrophy is associated with arterial stiffness and vascular calcification in hemodialysis patients. Hypertens Res 2004; 27: 47-52.
28. Benetos A, Vasmant D, Thiery P, Safar M. Effects of ramipril on arterial hemodynamics. J Cardiovasc Pharmacol 1991; 18 (Suppl 2): S153-6.
29. Karalliedde J, Smith A, DeAngelis L, Mirenda V, Kandra A, Botha J, et al. Valsartan improves arterial stiffness in type 2 diabetes independently of blood pressure lowering. Hypertension 2008; 51: 1617-23.
30. Agabiti-Rosei E, Porteri E, Rizzoni D. Arterial stiffness, hypertension, and rational use of nebivolol. Vasc Health Risk Manag 2009; 5: 353-60.
31. Pirro M, Schillaci G, Mannarino MR, Savarese G, Vaudo G, Siepi D, et al. Effects of rosuvastatin on 3-nitrotyrosine and aortic stiffness in hypercholesterolemia. Nutr Metab Cardiovasc Dis 2007; 17: 436-41.

การศึกษาความสัมพันธ์ของ Aortic stiffness และ Left ventricular mass index ด้วยวิธีคลื่นแม่เหล็กไฟฟ้าในผู้ป่วยความดันโลหิตสูง

อนุชิต วงศ์เพ็ญ, ธนัญญา บุณยศิรินันท์

วัตถุประสงค์: *ในผู้ป่วยความดันโลหิตสูงการที่มีหัวใจห้องซ้ายล่างหนาตัวขึ้นบ่งบอกถึงความเสี่ยงต่อการเกิดโรคหัวใจในอนาคต มีการศึกษาที่พบความสัมพันธ์ของหัวใจห้องล่างซ้ายหนาตัวกับการแข็งตัวของหลอดเลือดแดงใหญ่ (Aortic stiffness)โดยใช้วิธีการวัดแบบต่างๆกัน อย่างไรก็ตามข้อมูลการวัดที่ได้จากการตรวจด้วยวิธีคลื่นแม่เหล็กไฟฟ้าซึ่งมีความแม่นยำสูงและการศึกษาเจาะจงเฉพาะผู้ป่วยความดันโลหิตสูงยังมีน้อยมาก การศึกษานี้มีวัตถุประสงค์เพื่อหาความสัมพันธ์ของภาวะหลอดเลือดแดงใหญ่แข็งตัวและการหนาตัวของหัวใจห้องล่างซ้าย*

วัสดุและวิธีการ: *เป็นการศึกษาแบบ cross-sectional study คัดเลือกผู้เข้าร่วมการวิจัยจากผู้ที่มารับการตรวจหัวใจด้วยด้วยคลื่นแม่เหล็กไฟฟ้าที่โรงพยาบาลศิริราช ในช่วงตั้งแต่เดือนเมษายน พ.ศ. 2554 ถึง กันยายน พ.ศ. 2554 ผู้เข้าร่วมการวิจัยจะได้รับการตรวจหัวใจด้วยคลื่นแม่เหล็กไฟฟ้าและประเมินภาวะหลอดเลือดแดงใหญ่แข็งด้วยวิธีวัด pulse wave velocity (PWV), ตรวจวัดการหนาตัวของหัวใจห้องล่างซ้ายด้วยการวัด left ventricular mass index แล้วจึงหาความสัมพันธ์ระหว่างการหนาตัวของหัวใจห้องล่างซ้ายกับภาวะหลอดเลือดแดงใหญ่แข็ง*

ผลการศึกษา: *ผู้เข้าร่วมการวิจัยจำนวน 113 คน มีอายุเฉลี่ย 68.9±10.3 ปี, เพศหญิงร้อยละ 51.3 การหนาตัวของหัวใจห้องล่างซ้าย (LV mass index) เฉลี่ย 53.39±18.32 กรัมต่อตารางเมตรไม่พบความสัมพันธ์อย่างมีนัยสำคัญทางสถิติระหว่างการหนาตัวของหัวใจห้องล่างซ้ายกับภาวะหลอดเลือดแดงใหญ่แข็ง(r =0.085 , p-value =0.37) อย่างไรก็ตามพบว่าภาวะหลอดเลือดแดงใหญ่แข็งมีความสัมพันธ์กับอายุ (r = 0.469 , p-value <0.001)*

สรุป: *ในการศึกษานี้ไม่พบความสัมพันธ์ระหว่างการหนาตัวของหัวใจห้องล่างซ้ายกับภาวะหลอดเลือดแดงใหญ่แข็งโดยการตรวจด้วยคลื่นแม่เหล็กไฟฟ้า ซึ่งอาจอธิบายจากกลุ่มผู้เข้าร่วมการศึกษามีการหนาตัวของหัวใจห้องล่างซ้ายไม่รุนแรง*