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PRELIMINARY STUDY ON VENTRICULAR ARTERIAL COUPLING OF PATIENTS WITH HYPERTENSION BY ULTRASONOGRAPHY

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Background Ageing and chronic diseases, such as hypertension and diabetes, modify the structure and function of both the arteries and ventricles. The ventricular performance is influenced by arterial load and affects the effective arterial elastance. The interaction of ventricular and arterial system,

termed ventricular-arterial coupling, is a determinant of cardiovascular performance and cardiac energetics.

Objective To assess whether cardiovascular stiffness and its coupling of patients with hypertension can be evaluated by ultrasound; and to compare cardiovascular stiffness and ventricular-arterial coupling in controls and hypertensive patients.

Methods The 40 patients with essential hypertension and 40 age- and gender-matched subjects without hypertension, diabetes and other cardiovascular diseases as controls underwent comprehensive two-dimensional and Doppler evaluation. Left ventricular dimensions, wall thickness were measured from standard two-dimensional, M-mode echocardiography. Left ventricular (LV) ejection fraction (EF) and stroke volume (SV) were detected biplane Simpson's method. The flow velocities of mitral and left ventricle outflow tract (LVOT) were traced; mitral peak velocity of early (E), late (A) filling, peak velocity (v_{OT}) and its acceleration time of LVOT were obtained by Pulsed-wave Doppler. Mitral annular velocity was measured by Doppler tissue imaging. Early diastolic (e) was measured from the apical four-chamber view. End-systolic pressure (ESP) estimated as $ESP = (2 \times SBP + DBP) / 3$. Echo-Doppler-derived LV diastolic elastance (E_d , $(E/e)/SV$), LV end-systolic elastance (E_{es} , v_{OT}/AT), effective arterial elastance (E_a , ESP/SV), and ventricular-arterial coupling index (E_a/E_{es}) were calculated.

Results (1) Hypertensive patients had significant higher SBP and DBP, thicker interventricular septal and left ventricular posterior wall thickness, wider diameter of carotid artery (D) and faster carotid-femoral pulse wave velocity (C-FPWV) ($p < 0.001$); but had lower E/A and e/a ($p < 0.001$) compared with controls; the left ventricular diastolic end diameter, EF, FS and SV were similar between hypertensive and control group ($p > 0.05$). (2) In hypertensives, the E_a (2.60 ± 0.82 vs 2.14 ± 0.73 mm Hg/ml, $p = 0.019$), E_d (0.21 ± 0.08 vs 0.16 ± 0.05 /ml, $p = 0.008$) and E_a/E_{es} (1.77 ± 0.86 vs 1.38 ± 0.57 mm Hg. s^2 /ml/m, $p = 0.039$) were elevated compared with the controls'. However, the E_{es} did not differ between two groups ($p > 0.05$). (3) In all subjects, the E_{es} significantly correlated with EF ($r = 0.378$, $p = 0.005$). The E_a significantly correlated with C-FPWV ($r = 0.299$, $p < 0.001$). E_d inversely correlated with e/a ($r = -0.343$, $p = 0.018$). (4) Selected randomly 10 subjects and measured by two independent investigators, the correlation coefficient of E_a/E_{es} was 0.851 ($p < 0.001$), and average difference of E_a/E_{es} was (0.27 ± 0.54 mm Hg. s^2 /ml/m).

Conclusions (1) Compared with systolic ventricular elastance, the vascular elastance was vulnerable to damage. (2) The diastolic ventricular elastance was easier to be subjected to damage than systolic ventricular elastance. (3) Compared with the control group, the balance site of E_a/E_{es} in hypertension group moved upward. Arterial stiffness was associated with ventricular stiffness, and their matching relation can be applied to evaluate ventricular-arterial coupling.