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TRANSFORMING GROWTH FACTOR -B1: A MARKER OF LEFT VENTRICULAR HYPERTROPHY IN HYPERTENSION
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Left ventricular hypertrophy (LVH) develops in approximately 15-20% of patients with hypertension and is an independent risk factor for subsequent cardiovascular morbidity and all-cause mortality. Factors such as age, gender, weight and blood pressure explain only 41% of LV mass variability. Non-hemodynamic factors have been postulated to play a role in LVH development. Transforming growth factor-β1 (TGF-β1), a cytokine produced by virtually all cell types, has been implicated as a key mediator in cardiovascular hypertrophy in animal studies. Its overproduction stimulates collagen synthesis and inhibits collagen degradation, leading to extracellular matrix proliferation and excess fibrosis. We therefore investigated the determinants of ventricular hypertrophy and compliance in a cohort of patients with hypertension and LVH diagnosed by the electrocardiographic criteria of S in V1 or R in V5/V6 > 35 mm or R in AVL > 11 mm. Sixty-seven patients enrolled in the Echocardiographic study of the effect on Cardiovascular Hypertrophy of Olmesartan (ECHO) were investigated. Demographic, clinical, and laboratory data obtained at study entry after at least one week off anti-hypertensive therapy were compared to the patient’s baseline LV mass and ventricular compliance measured by 2-D echocardiography. Using linear regression analysis, serum TGF-β1 correlated significantly with LV mass indexed to body surface area (r = 0.67, p = 0.246, p = 0.019). This correlation was enhanced in patients with concentric hypertrophy (r = 0.58, p = 0.302, p = 0.0092). The strongest correlation was seen in patients with concentric hypertrophy who were not on hydroxymethylglutaryl-coenzyme A (HMG-CoA) reductase inhibitors at study entry (r = 0.353, p = 0.0075). We therefore conclude that serum TGF-β1 predicts severity of LVH in hypertension, particularly in patients with concentric hypertrophy not taking HMG-CoA reductase inhibitors.

Key Words: Hypertension, Left Ventricular Hypertrophy, Transforming Growth Factor Beta-1

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AUGMENTED ACE2 EXPRESSION IN ISCHEMIC CARDIOMYOPATHY
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Previously, we showed that myocardial ischemia increased angiotensin (Ang)-(1-7) in cardiac myocytes. Since angiotensin converting enzyme 2 (ACE2) has been implicated in the production of Ang-(1-7) we investigated whether cardiac remodeling post myocardial infarction was also associated with elevated ACE2 expression. Experiments were performed in Lewis rats that were subjected to ligation of the left main coronary artery (LIG) or sham (SHAM) ligation. Rats were given a 4 week recovery period during which time cardiac hypertrophy associated with cardiac dysfunction was characterized by significant elevations in left ventricular end-diastolic pressure (p< 0.01) and heart weight to body weight ratio (p<0.05) in the LIG group. At the end of this 4 week period the rats were sacrificed and heart tissue processed for ACE2 immunoreactivity. ACE2 immunoreactivity was evident in cardiac myocytes and coronary vessels. However, there was markedly less ACE2 immunoreactivity in the infarcted region of the heart. The intensity of ACE2 immunoreactivity in septal tissue was quantified using algorithms in Photoshop™. This analysis revealed significantly (p<0.05) greater ACE2 immunoreactive staining in the LIG versus the SHAM group.

These results support the hypothesis that augmented ACE2 expression contributes to increased Ang-(1-7) production in the remodeled myocardium. The augmented ACE2 staining may be a post-transcriptional event since we have previously reported that ACE2 mRNA was not increased in this model of cardiomyopathy.

Key Words: Cardiac Remodeling, Cardiomyopathy, Myocardial Ischemia

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UTILITY OF CORNELL VOLTAGE-DURATION PRODUCT FOR DETECTION OF LEFT VENTRICULAR HYPERTROPHY. THE ELECTROTENS STUDY
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Since echocardiography is usually not feasible in primary care for detection of left ventricular hypertrophy (LVH), the interest on new ECG criteria for diagnosis of LVH is markedly increasing. Of them, the most relevant criterion, mainly due to its application in LIFE trial, is Cornell voltage-duration product (VDP). In fact, this criterion has been included in the European guidelines on Hypertension 2003 as an elective method to detect LVH. With the aim to determine the utility of this method in daily clinical practice we performed the ELECTROTENS study.

For this multicenter trial five consecutive hypertensive patients ≥18 years who attended a Cardiologic outpatient clinic were recruited. An ECG was performed to all the patients and all the records were sent to a core lab for a central VDP calculation. 1624 patients were initially recruited, but after a strict clean up procedure only 782 patients were finally evaluated. Population: 51% males; age 66.1 ± 10.4 yrs; BMI 28.2 ± 6.1 kg/m², BP 155.7 ± 7.7/ 90.8 ± 10.6 mmHg (48% grade Hypertension, 39% grade II, 13% grade III). History of hypertension: 6.3 ± 5.9 years.

Results: LVH was detected by VDP in 23.4% of the patients. Some factors were predictive of LVH by ECG-VDP: age, male gender, systolic BP and presence of hypertensive retinopathy.

Conclusions: Almost one of four hypertensive patients who daily attend a Cardiologic outpatient clinic exhibit LVH detected by VDP. This rate is significantly higher than that observed when other classical ECG voltage criteria, such as Sokolow-Lyon or Cornell, are used. Therefore, the VDP criterion appears to be a very useful method to detect LVH in daily clinical practice. Since echocardiography is not always easily feasible, the new ECG criteria, not only based on QRS voltage, should be considered a very useful tool to improve the detection of LVH, mainly in primary care medicine.

Key Words: Cornell Voltage-Duration Product, Electrocardiography, Left Ventricular Hypertrophy

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EFFECTS OF CANDESARTAN ON LEFT VENTRICULAR GEOMETRY AND DIASTOLIC FUNCTION IN HYPERTENSIVE PATIENTS WITH LEFT VENTRICULAR HYPERTROPHY. THE VIPE STUDY
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The VIPE study was designed to assess the efficacy of candesartan on hypertensive echocardiographic left ventricular hypertrophy (LVH) at

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