The impact of diabetes mellitus and hypertension on pulse wave velocity

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Objective: brachial-ankle pulse wave velocity (baPWV) is widely used as a marker of arterial stiffness and increased arterial stiffness is an important risk factor for cardiovascular disease. Although diabetes mellitus (DM) and hypertension (HTN) are major risk factor for atherosclerosis, the impact of DM and HTN on pulse wave velocity (PWV) has not been well defined. Material and methods: DM with hypertensive patients (Group I, n=467, 232 men, 63±11 years) and hypertensive patients (Group II, n=348, 196men, 59±13 years) who undergone testing a baPWV were included. The measurement of the baPWV were performed in a quiet controlled room (22±1 °C) in the supine position with using an automated device (VP-2000, Colin, Co. Ltd, Komaki, Japan). Results: baPWV is significantly higher in Group I than Group II (1714±360 cm/sec vs. 1620±306 cm/sec, p<0.001). Multivariate linear regression analysis showed significant association of Ln(baPWV) with age (beta=0.008, p=0.001), mean arterial pressure (beta=0.004, p=0.001), heart rate (beta=0.004, p=0.001) and DM &HTN (beta=0.025, p<0.05), R2=0.036 in all study group. Conclusion: baPWV is higher in DM with hypertensive patients than hypertensive patients and this study suggest that DM and HTN may play a significant role in the arterial stiffness.

Extrinsic Compression of the Left Anterior Descending Coronary Artery by Rib in a patient with Progressive Left Ventricular Remodeling

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A 78 year old male with a history of dilated cardiomyopathy diagnosed 5 years ago was admitted to our clinic with chest pain. The initial ECG showed sinus tachycardia and left bundle branch block. Lab tests revealed elevated levels of troponin T (0.127 ng/mL), creatinine kinase-MB isoenzyme (9.8 ug/L). Transthoracic echocardiography revealed progressed left ventricle (LV) remodeling compared to previous echocardiography 2 years ago. The LV was severely dilated(LV end-diastolic dimension=76 mm) with decreased systolic function (ejection fraction=23%) and the territories of left anterior descending coronary artery (LAD) were akinetic and the other segments were hypokinetic. Non ST-elevation myocardial infarction was diagnosed. Coronary angiography showed distal LAD was cut off in diastole. Proximal and distal reference segments appeared normal.Intravasacular ultrasonography (IVUS) and optical coherence tomography (OCT) revealed distal LAD was deformed to a slitlike appearance, suggesting extrinsic compression. To evaluate the cause of extrinsic compression, cardiac multi-detector computed tomography (MDCT) was performed. MDCT showed enlarged LV which contacted with chest wall. Distal LAD was trapped between dilated LV and costochondral cartilage, and narrowed due to extrinsic compression by costochondral cartilage in diastole. Because of refractory angina, we performed percutaneous coronary intervention (PCI). Distal LAD was directly stented with an Endeavor 3.0x38mm stent. He was discharged free of angina. Extrinsic compression of coronary artery due to LV dilatation is extremely rare. In the present case, we demonstrated that LV dilatation resulted in extrinsic compression of coronary artery, which was confirmed by cardiac MDCT. There has been no data about optimal treatment of extrinsic compression of coronary artery due to dilated LV. In this case, compression leaded to significant myocardial ischemia. We decided PCI due to refractory angina. Successful stenting of LAD leaded to relief of symptoms. To our knowledge, progressive LV remodeling resulting in LAD compression by costochondral cartilage presenting with acute myocardial infarction, which was treated with successful PCI is the first in the literature.