Relationship between reduced elasticity of extracardiac vessels and left main stem coronary artery disease

We read with great interest the extended analysis by Weber et al. of their previous publication and the elegant work by Hadjinikolaou et al., concerning the relationship between arterial stiffness and elasticity and the presence of left main stem coronary artery disease.

Their findings agree with our observation that ascending aortic blood pressure-derived indices are independently related to coronary atherosclerosis. The values of these indices depend on the interaction between heart and vascular bed. Extended analysis of our study population provides more information about the relationship between left main stem atherosclerosis and blood pressure-derived indices. Among 423 study subjects with angiographically confirmed coronary artery disease and preserved left ventricular function, 16 were found to have left main stem stenosis of at least 50%, 76 patients had stenosis <50%, and 331 had no plaque detected on angiography. Patients with significant stenosis, as well as those with insignificant plaque in their left main stem, had significantly higher intra-aortic pulse pressure (77.3 ± 18.7 vs. 71.1 ± 18.5 vs. 65.6 ± 8.1 mmHg), aortic pulsatility (0.79 ± 0.20 vs. 0.76 ± 0.17 vs. 0.69 ± 0.15), and pulsatility index (1.10 ± 0.40 vs. 1.04 ± 0.32 vs. 0.92 ± 0.27) when compared with the group without any main stem atherosclerosis detectable on angiography. There was no significant difference in mean ejection fraction or heart rate among the groups, which suggests that the difference in vascular properties influences the outcome measures. Moreover, pulse pressure, pulsatility, and pulsatility index correlated with the per cent of left main stenosis (r = 0.15, P < 0.01; r = 0.20, P < 0.001; r = 0.21, P < 0.001, respectively). None of the other aortic blood pressure-derived indices or brachial pressure-derived indices was significantly correlated with left main stem atherosclerosis.

In conclusion, patients with left main stem atherosclerotic plaque are characterized by impaired mechanical properties of their vessels leading to increased aortic pulse pressure and pulsatility. Indeed, this can lead to poor prognosis of such patients.

References


Piotr Jankowski
Kalina Kawecka-Jaszcz
I Department of Cardiology
Collegium Medicum
Jagiellonian University
Krakow, Poland
E-mail address: piotr_jankowski@interia.pl

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Evident, but poorly defined: left ventricular hypertrabeculation/noncompaction and its diagnostic criteria

We read with interest the article by Murphy et al. about the natural history and familial characteristics of left ventricular hypertrabeculation/noncompaction (LVHT). In 45 patients with LVHT, they observed a survival from death or transplantation of 97% and conclude that LVHT is associated with a better prognosis than previously reported.

That LVHT is a disorder of endomyocardial morphogenesis, as stated by the authors, is only one of several hypotheses. Actually, the etiology of LVHT is not known. Arguments against the 'embryonal' hypothesis are patients in whom LVHT developed during life: three members of a family who subsequently developed LVHT did not show LVHT characteristic changes by fetal echocardiography. In a further case, LVHT was echocardiographically not present in the first days of life, but only at the age of 5 weeks. In two further cases with muscular dystrophy, LVHT was documented to develop during life. Disappearance of LVHT has been observed 2 years after a Coxiella burnetti infection.

Which criteria were applied for making the primary diagnosis of LVHT, and remained the criteria unchanged during the study period? Were echocardiograms of all 518 patients with idiopathic dilative cardiomyopathy reviewed and screened for LVHT? Were interobserver variability studies performed? When using Jenni's criteria in the short axis view, how did the authors distinguish between papillary muscles and trabeculations? We want to stress that further criteria of LVHT, applied in the largest series of 77 patients, overcome this problem by preferentially using the apical four-chamber view. These echocardiographic diagnostic criteria for LVHT are (i) more than three trabeculations protruding from the left ventricular wall, apically to the papillary muscles, visible in single image plane and (ii) intertrabecular spaces perfused from the ventricular cavity, visualized on colour Doppler imaging. Trabeculations are defined as structures with the same echogenicity like the myocardium and moving synchronously with the ventricular contractions.

It is reported that no patient had clinical evidence of skeletal myopathy. It is not known whether all patients were investigated by a neurologist. LVHT has not only been described in association with mutations in the G4.5, α-dystrobrevin and Cypher/ZASP gene, but also in Becker and Duchenne muscular dystrophy, myotonic dystrophy, myoadenylate-deaminase deficiency, and metabolic myopathies.

The authors present the largest ever-reported follow-up study of LVHT patients. Did they identify any baseline parameter as indicator for deterioration or death? Did the prognosis differ between patients in whom LVHT was associated with left ventricular dilatation and systolic dysfunction and patients with normal systolic function?