ELSEVIER

Clinical Research

Strain Pattern on Electrocardiogram Is Associated with Increased Carotid Intima-Media Thickness in Patients with Aortic Valve Stenosis

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Background: Coronary artery disease is present in at least 30% of patients with degenerative aortic stenosis (AS). Atherosclerosis also performs an important role in the progression of AS, because of the similarities of pathological mechanisms in both conditions. The electrocardiogram (EKG) strain pattern is associated with structural myocardial change and subendocardial ischemia and has been worldwide used as a marker of AS severity. We hypothesized that EKG strain pattern would be a marker of atherosclerosis as well in AS patients. The aim of this study was to associate the presence of EKG strain pattern in AS patients with the carotid intima-media thickness (CIMT).
Methods: Fifty-two consecutive patients referred from the cardiology clinic with moderate or severe AS were included in the study and underwent clinical evaluation, EKG, transthoracic echocardiography, and carotid ultrasonography, following statistical analysis of the results.
Results: There was a significant association between left ventricular EKG strain and increased CIMT ($P = 0.001$). The presence of strain increased the odds of abnormal CIMT ($P = 0.004$, odds ratio 9.7, 95% confidence interval 2.4−45.0), in a model adjusted for age and clinical diagnosis of systemic arterial hypertension. Additionally, EKG strain was associated with the presence of atherosclerotic plaque in at least one carotid artery ($P = 0.011$).
Conclusion: Our results suggest that AS patients with EKG strain pattern should be further investigated for the diagnosis of subclinical atherosclerotic disease.

INTRODUCTION

Degenerative aortic stenosis (AS) is a highly prevalent disease, and when presenting in a severe stage, is associated with increased morbidity and mortality.[1] Recent literature data indicate that at least 30−40% of AS cases are associated with coronary artery disease (CAD).[2] Atherosclerosis also performs

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an important role in the progression of AS. Current studies suggest that the development and progression of disease occurs due to a systemic cellular and molecular process very similar to atherosclerosis, with endothelial dysfunction and vessel calcification.[3–5]

The presence of electrocardiogram (EKG) left ventricle (LV) strain pattern in AS patients is a prognosis-worsening criterion and may be a marker of myocardial structural changes and subendocardial ischemia.[6] Recent study[7] performed in asymptomatic patients with AS showed that the presence of EKG strain is an independent predictor of poor prognosis. The authors argued that, first of all, it occurs because the presence of strain would indicate myocardial ischemia induced by the imbalance between oxygen demand and supply in the hypertrophied heart. Second, the presence of strain would also reflect AS-induced silent myocardial ischemia.

Also, recent data describing an association between severe AS and peripheral atherosclerosis[2,8] are consistent with the concept that atherosclerosis is a systemic disease. This information reinforces the idea that patients with clinically significant AS are at risk of peripheral atherosclerosis. The ultrasonography for evaluation of carotid intima-media thickness (CIMT) and detection of atherosclerotic plaque is considered a practical and relatively inexpensive noninvasive method to assess the presence of atherosclerosis.

In this context, the hypothesis of this study was that the presence of the left ventricular strain on the EKG of AS patients is a marker of subclinical atherosclerosis defined as an increased CIMT. The aim of this study was to investigate an association between EKG left ventricular strain of patients with moderate to severe AS and increased CIMT.

MATERIALS AND METHODS

This is a prospective study including 52 consecutive patients diagnosed with moderate or severe AS and referred from the outpatient cardiology service of Botucatu Medical School —UNESP from May 2012 to May 2013. All patients underwent a same day clinical evaluation, 12-lead EKG, transthoracic echocardiography, and carotid ultrasonography. This study was approved by the ethics committee of Botucatu Medical School (Protocol 4043-2011).

The inclusion criteria were: age over 18 years and presence of moderate or severe aortic valve stenosis. Exclusion criteria were: prosthetic heart valve; congestive heart failure due to other cause than AS; and other associated heart valves lesion greater than mild.

The criteria of unfavorable prognosis of AS[9] were: presence of angina, dyspnea, or syncope in clinical evaluation; echocardiogram revealing reduced LV ejection fraction (less than 0.5); or presence of LV hypertrophy (LV mass indexed to body surface area greater than 95 g/m^2 in women and greater than 115 g/m^2 in men).

The criteria of moderate to severity AS according to the latest guidelines of the American Society of Cardiology[9] were: echocardiogram revealing aortic valve area less than 1.5 cm^2, maximal aortic gradient greater than 36 mm Hg, or mean aortic gradient greater than 25 mm Hg.

Clinical Evaluation

Age, sex, race, and cardiovascular risk factors as defined by the American current guidelines,[10–14] such as hypertension, diabetes mellitus, obesity, dyslipidemia, smoking, CAD, and heart failure, were recorded besides physical examination.

Electrocardiogram

The standard 12-lead procedure was performed and the following variables were considered: cardiac rhythm, atrial or ventricular overload criteria, and left ventricular strain repolarization, according to the current guidelines of the American Society of Cardiology.[15] The left ventricular strain was defined as ST-segment changes on lateral leads (V5 or V6), showing the flattening ST segment depression $\geq$0.05 mV and asymmetric inverted T wave.

Transthoracic Echocardiogram

Complete echocardiograms were performed in all patients by the same examiner, in accordance with the standardization techniques recommended by the American Society of Echocardiography.[9,16]

Carotid Ultrasound

The CIMT was obtained by the automated method, in the posterior wall of the left and right common carotids, following the recommendations of the ''Consensus Statement from the American Society of Echocardiography''[17] and ''Mannheim Carotid Intima-Media Thickness Concensus.''[18] The values were adjusted for race and age, according to these consensuses. The detection and quantification of atherosclerotic plaques were also performed.[19]