Clinical Research

Assessment of left atrial appendage function by transesophageal echocardiography in patients two weeks after acute coronary syndromes

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Background For many years in ischemic heart disease, ventricles rather than atria received attention so not much is known about left atrial function in left ventricular ischemia. Objective Our study aimed to evaluate left atrial appendage (LAA) function by means of transesophageal echocardiography in patients ten days after acute coronary syndromes (ACS). Method The study was performed on 16 adult patients (65.9±9.9 years old) in whom transesophageal echocardiography was done 10 days after ACS. The following left atrial appendage (LAA) planimetric parameters were analyzed: LAA transversal dimension, LAA longitudinal dimension, LAA maximal area, and LAA minimal area. LAA ejection fraction was calculated and analyzed. The following LAA Doppler parameters were analyzed: the peak LAA emptying and the peak LAA filling velocities. The control group consisted of 14 patients (43±14.6 years old) without cardiovascular diseases. Results Both LAA longitudinal dimension and LAA transversal dimension were significantly higher in patients with ACS than in control patients. The same was observed for LAA maximal area. Also LAA ejection fraction was higher in patients with ACS. LAA minimal area did not differ in the patients in either group. LAA peak emptying flow (LAAE) and LAA peak filling flow (LAAF) were significantly higher in patients of the study group than of the control group. Conclusion Our study shows that seven weeks after acute coronary syndrome LAA as a reservoir as well as a pump works at a higher level than it does in the control group. J Geriatr Cardiol 2005; 2(4):198-201.

Key Words: left atrial appendage; acute coronary syndromes; transesophageal echocardiography

Introduction

Except in supraventricular arrhythmia, atria have not been of great interest in heart pathology as well as in clinical cardiology. In ischemic heart disease, ventricles rather than atria received attention for many years. Function of the left atrial appendage (LAA) is even much less known, especially in acute coronary syndrome. However, the pivotal role LAA plays in both cardiovascular performance and pathological physiology is widely known. Although the LAA is an anatomical part of the left atrium, its physiology is quite distinct from LA in many aspects. First, LAA is a highly contractile pump with a pattern of contraction totally different from that of the main body of the left atrium (LA). Second, LAA is a more distensible chamber than LA. Third, the LAA is a place where thrombi are often formed and spontaneous echocardiographic contrast is often present.

How LAA works in acute left ventricular ischemia has not been previously explored. The purpose of this study was to examine LAA function by means of transesophageal echocardiography in patients 10 days after the acute coronary syndromes.

Patients and methods

Patients

The study group consisted of 16 adult patients (6 women and 10 men, aged 41 to 70 years [mean 56.9±9.9 years]) on whom transthoracic echocardiography (TEE) was performed 10 days after onset of the acute coronary syndrome, to evaluate their LAAs. This group consisted of 8 patients with unstable angina pectoris, 4 with recent anterior myocardial infarction, 3 with recent inferior myocardial infarction and 1 with recent non-Q wave myocardial infarction. Patients with valvular heart diseases, symptoms of congestive heart failure, arrhythmia (atrial fibrillation, supraventricular tachycardia), acute and chronic pulmonary diseases, hyperthyroidism, anemia, and neoplasm were excluded from this study. Laboratory results of the patients, such as blood morphology, bio-chemical blood and urine analyses were normal. All participants gave written informed consent.

The control group comprised 14 patients (6 women and
8 men, aged 24 to 69 years (mean 43.79±14.61) without cardiovascular diseases. They were examined by echocardiography to exclude atrial septal defect, atrial septal aneurysm or thrombus in the LAA.

Transesophageal echocardiography (TEE)

TEE was performed using a biplane probe (Acuson 127). Patients were studied in the morning, between 8:30 and 9:30 in a fasting state. Normally used drugs (nitrates, b-adrenergic blockers, calcium channel blockers) were stopped for at least 24 hours before TEE examination.

LAA images were acquired in horizontal views. The following LAA planimetric parameters were analyzed:

- LAA transversal dimension (LAA\_trans.) i.e. the width of the LAA neck was measured as a line drawn from the limbus of the confluence of the left upper pulmonary vein and the LAA outermost portion of the mitral annulus (Fig. 1).
- LAA longitudinal dimension (LAA\_long.) i.e. the length of the LAA was measured from the limbus of the confluence of the left upper pulmonary vein to the appendage apex (Fig. 1) (both dimensions were obtained at the onset of P wave of simultaneously recorded ECG).
- LAA maximal (LAA\_max.) i.e. diastolic area - measured at the onset of P wave of ECG and LAA minimal (LAA\_min.) i.e. systolic area – measured just after R wave of QRS complex of ECG. LAA areas were measured by manual planimetry by tracing a line starting from the top of the limbus of the left upper pulmonary vein along the whole LAA endocardial border as described previously. 5,8,9

The LAA ejection fraction (EF\_LAA) was calculated as:

\[
EF\_LAA = \frac{LAA\_max. - LAA\_min.}{LAA\_max. - LAA\_min.} 
\]

The LAA blood flow velocity was obtained by placing the pulsed Doppler sample volume into the outlet of the appendage blood cavity > 1 cm away from the left atrial cavity. The peak LAA emptying (LAAE) and the peak LAA filling (LAAF) velocities were recorded (Fig. 2) as described previously. 9,10

All images were recorded on VHS tape and were analysed using an off-line work station. Data from the average of three successive beats were analyzed.

Statistical analysis

Parameters of the LAA were compared between groups. The Student t-test and Cochrane-Cox tests were used for comparisons. P<0.05 was assumed as statistically significant.

Results

Mean values of analyzed LAA parameters are presented in Table 1.

Both LAA transversal dimension and LAA longitudinal dimensions were significantly higher in patients with acute coronary syndromes than in control patients (p<0.05). The same was observed for LAA maximal areas. Also LAA ejection fraction was higher in patients with acute coronary syndromes (p<0.05). LAA minimal area did not differ in the patient and control groups (p>0.05). LAAE and LAAF were significantly higher in patient group than of the control group (p<0.05).

### Table 1 Comparison of LAA parameters between 2 groups (X±SD)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Patient group (n=16)</th>
<th>Control group (n=14)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>LAA_trans.(cm)</td>
<td>1.93±0.59</td>
<td>1.32±0.24</td>
<td>0.00385</td>
</tr>
<tr>
<td>LAA_long.(cm)</td>
<td>4.03±0.88</td>
<td>3.11±0.66</td>
<td>0.00173</td>
</tr>
<tr>
<td>LAA_max. area (cm^2)</td>
<td>4.55±1.01</td>
<td>3.1±0.83</td>
<td>0.00035</td>
</tr>
<tr>
<td>LAA_min. area (cm^2)</td>
<td>1.93±0.97</td>
<td>2.08±0.65</td>
<td>NS</td>
</tr>
<tr>
<td>EF_LAA</td>
<td>0.58±0.17</td>
<td>0.33±0.08</td>
<td>0.000039</td>
</tr>
<tr>
<td>LAAE (m/s)</td>
<td>0.56±0.07</td>
<td>0.41±0.04</td>
<td>0.000039</td>
</tr>
<tr>
<td>LAAF (m/s)</td>
<td>0.51±0.09</td>
<td>0.42±0.04</td>
<td>0.00216</td>
</tr>
</tbody>
</table>

| LAA\_trans. - LAA\_trans. dimension, LAA\_long. - LAA\_long. dimension, LAA\_max. - LAA\_max. i.e. diastolic area, LAA\_min. - LAA\_min. i.e. systolic area |
| EF\_LAA - LAA ejection fraction, LAAE - peak LAA emptying flow velocity, LAAF - peak LAA filling flow velocity, NS - non-significant, p<0.05 statistical significance threshold |

Figure 1. Left atrium appendage, imaged by transesophageal echocardiography, longitudinal vision. LAA\_trans. and LAA\_long. dimensions measurement.

LAA\_trans. - transversal left atrial appendage dimension, LAA\_long. - longitudinal left atrial appendage dimension
The atria functions as a reservoir of blood during ventricular systole, as a passive conduit of blood during early ventricular diastole, and as a contractile chamber during later ventricular diastole.\textsuperscript{11,12,13,14,15} We divided LAA function (by analogy of LA function) into two components: LAA as a reservoir\textsuperscript{16} and LAA as a pump. We related planimetric parameters (LAA\textsubscript{mm}, LAA\textsubscript{im}, LAA\textsubscript{ss}, LAA\textsubscript{ns}) to the former LAA function component and Doppler parameters (LAAE, LAAF) and EF\textsubscript{LAA} to the latter one.

- This study showed that LAA function is more expressed in patients two weeks after the incidence of acute coronary syndromes than in the controls. The differences in configuration of analysed parameters show that LAA as a reservoir as well as a pump works at a higher level than in controls two weeks after acute coronary syndromes. LA work is functionally associated with the LV. LV function impairment is followed by changes in LA function. LA modulates LV work and this influence is highly expressed when LV work is impaired\textsuperscript{17}, which occurs in the case of LV ischemia. The same might be happening with LAA as a part of LA. An increase of pump and reservoir function of LA in LV ischemia, especially in myocardial infarction, was demonstrated.\textsuperscript{15,17,18,19} As LAA is an anatomical part of LA, despite its different physiology, it cooperates strictly with LA in changes of its work pattern. This cooperation was the subject of a few recent studies\textsuperscript{1,2,21,22}, which suggested such a dependent relationship. Recent in vivo studies confirmed that there were regional differences in LA function: specifically, the LAA shortens to a greater extent and is more distensible than is the body of the LA.\textsuperscript{1,3,20} LAA plays an important role in LA reservoir function in the presence of LA pressure and/or volume overload.\textsuperscript{8} It is suggested that large LAA compliance contributes greatly to the prevention of an elevation in intraventricular pressure and pulmonary congestion in many pathological and physiological conditions.\textsuperscript{3,8}

The fact of LAA enlargement and LAA mechanical function decrease (expressed by LAAE and LAAF decline) in atrial fibrillation is widely known.\textsuperscript{23} Atrial appendage flow velocities were noted to be significantly decreased after conversion of atrial fibrillation to sinus rhythm, which is the effect of so-called LAA stunning.\textsuperscript{10,24} LAA function in atrial fibrillation and LAA stunning phenomenon were investigated extensively.

There are few clinical studies evaluating LAA function in diseases other than atrial fibrillation. On the other hand, improvement of transesophageal echocardiography and its wide clinical use made it possible to visualize and evaluate LAA in a large number of patients.

Cornelius et al.\textsuperscript{3} estimated LAA to be 2.6 times as compliant as the LA. Ito et al.\textsuperscript{5} noticed that in patients after treatment of heart failure, LAA mechanical function improved significantly and the improvement generally was more significant in LAA than in LA or LV function. It is postulated\textsuperscript{6} that LAA works considerably through its intrinsic property, being independent of LA and LV to a great extent. Bilge et al.\textsuperscript{25} reported the LAA emptying velocities to be significantly reduced in hypertensive patients compared with controls as well as maximal LAA areas to be significantly larger. LV impairment due to its ischemia may involve LAA, resulting in an increase of LAA reservoir as well as systolic function.

To our knowledge no clinical studies have examined LAA function in LV ischemia. The great limitation of our study is the fact that TEE was performed two weeks after acute coronary syndromes. Applying TEE in acute ischemia in symptomatic patients is contraindicated because of its potential harm to the patients. This is why we used TEE to evaluate LAA only in stable, asymptomatic patients. The study primarily described a small number of patients with acute coronary syndromes. Therefore, the results cannot be generalized to all acute coronary syndromes patients. This aspect of LAA function in patients with acute coronary syndromes needs further thorough investigation.

**References**