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Aortic Stiffness and Compliance in Patients with Variant Form of Angina

Yuichi Ishikawa¹, Nobuhiro Miyazaki², Naoya Watanabe³, Yoshio Fujioka⁴, Takahiro Taniguchi⁴

We investigated the aortic stiffness in patients with variant form of angina by measuring pulse wave velocity and aortic compliance and compared them to those in patients with fixed coronary artery stenosis. The aorta was less stiff in patients with variant form of angina compared to patients with fixed coronary artery stenosis. Plasma cholesterol level in patients with variant form of angina was significantly lower than that in patients with fixed coronary artery stenosis. When patients with variant form of angina was divided to two groups, one with fixed stenosis and the other with no significant stenosis, aortic stiffness in variant form of angina patients without fixed stenosis is lower than that in variant form of angina patients with significant stenosis. And plasma level of HDL-cholesterol in variant form of angina patients without fixed stenosis was higher and atherogenic index in those patients was lower, compared with variant form of angina patients with significant stenosis.

In conclusion, even though the spectrum of angina is wide, the aortic stiffness is less and coronary risk factors are modest in patients with variant form of angina and especially those without fixed stenosis. These vascular characteristics may result more favorable prognosis of variant form of angina without significant fixed stenosis.

Key Words
Pulse wave velocity,
Aortic stiffness,
Aortic compliance,
Variant form of angina,
Coronary risk factors.

INTRODUCTION

The vascular stiffness increases with ageing and it is correlated to the degree of arteriosclerosis¹. We previously reported that the aortic pulse wave velocity (PWV), one of the parameters of aortic stiffness, was closely correlated with the aortic distensibility and the degree of coronary artery stenosis². The variant form of angina pectoris (VAP) is caused by the coronary arterial spasm and it has not been reported whether the coronary artery of VAP is as stiff as that of coronary artery disease (CAD) with fixed coronary stenosis. In this paper, we investigated the aortic stiffness in patients with VAP and compared it with that in patients with CAD with fixed coronary stenosis and chest pain syndrome. We also measured the plasma levels of cholesterol, triglyceride and HDL-cholesterol to
analyze the differences of the effects of plasma lipids levels on vascular stiffness. We found that the stiffness of aorta in patients with VAP was lower than that in patients with angina pectoris and chest pain syndrome.

The plasma level of cholesterol in patients with VAP was lower than those in patients with angina pectoris and chest pain syndrome.

SUBJECTS AND METHODS

SUBJECTS

Fifty eight patients (52 males and 6 females), range of age from 37 to 60 years old, who underwent diagnostic catheterization were investigated, 33 patients with old myocardial infarction and/or angina pectoris (OMI/AP) (mean age 54.0 ± 5.7 years old), 12 patients with variant form of angina pectoris (VAP) (mean age 50.7 ± 7.6 years old), 13 patients with chest pain syndrome (C) (50.6 ± 6.5 years old). VAP patients were further divided to two groups, 4 patients who had significant stenosis (VAP (+)) and 8 patients who had no significant stenosis (VAP (−)). All cases of VAP showed significant electrocardiographical ST elevation at spontaneous anginal attacks (Prinzmetal's variant form of angina). Patients with chest pain syndrome was diagnosed as the patients who had chest pain subjectively, but had no significant coronary stenosis angiographically. A significant stenosis was defined as more than 51% stenosis at least in one major coronary artery of coronary angiography.

MEASUREMENTS OF INVASIVE PULSE WAVE VELOCITY (PWV).

PWV was measured as previously reported2). Briefly, during diagnostic catheterization, we inserted a catheter-tip manometer (Miller 5F) from a femoral artery and advanced to the position 2 cm upper from the aortic valves and measured aortic pressure (reference position) and then drew back the manometer by 50 cm and measured aortic pressure. We recorded the aortic pressure curves and electrocardiogram (ECG) simultaneously using mingography (Siemens) at 100mm/sec of paper speed. We calculated PWV as following equation: the distance between each manometer/the time from the onset of Q wave in ECG to the onset of the upstroke of pressure curve.

MEASUREMENTS OF AORTIC COMPLIANCE

We calculated Tarazi's index3) which represented systolic aortic compliance as following equation: aortic pulse pressure at reference position / stroke volume. We calculated the stroke volume as following equation: cardiac output measured by thermodilution method / heart rate. We also calculated the diastolic blood pressure decay (DBPD)4) which represented diastolic aortic compliance as following equation: DBPD = time constant (T) / peripheral vascular resistance (R). T = diastolic time / 1nP1-1nP2. R = mean aortic pressure / cardiac output.
ASSESSMENTS OF CORONARY STENOTIC INDEX (CSI)

We underwent diagnostic coronary angiography by Judkins' method with multiprojections and three doctors who did not know the results of PWV analysed the degree of severity of coronary stenosis by the criteria of American Heart Association. We used two methods to calculate coronary stenotic index, namely Gensini's method and Balcon's method.5)

MEASUREMENTS OF PLASMA LIPID LEVELS

Blood samples were taken after 12 hours fasting. From the patients with myocardial infarction, blood samples were taken after more than one month interval from the onset. Total cholesterol (TC) and triglyceride (TG) were measured by the enzymatic methods using an autoanalyzer. High density lipoprotein-cholesterol (HDL-C) was measured by the heparin-calcium precipitation method. The atherogenic index (AI) was expressed as TC−HDL-C/HDL-C.

STATISTICAL ANALYSIS

We expressed the values as mean ± standard deviation. For statistical analysis, we used Student's t test. P value < 0.05 was considered significant.

RESULTS

CORONARY STENOTIC INDEX (CSI).

CSI in OMI/AP, VAP and C were 63.6 ± 20.8, 86.4 ± 12.4 and 98.1 ± 5.2, respectively (Tab. 1). There were significant differences between each group as shown in Fig 1. The degree of coronary stenosis in OMI/AP was the most severe in these three groups.

PWV.

PWV in OMI/AP, VAP and C were 7.81 ± 1.27 m/sec, 6.65 ± 0.99 m/sec and 7.10 ± 1.11 m/sec, respectively (Tab. 1). The slower PWV in VAP indicated that the aortic stiffness in VAP was significantly lower than that in OMI/AP (P<0.01) (Fig. 2).

AORTIC COMPLIANCE.

The Tarazi’s Index (PP/SV) in OMI/AP, VAP and C were 0.81 ± 0.30 mmHg/ml, 0.69 ± 0.25 mmHg/ml and 0.64 ± 0.17 mmHg/ml, respectively (Tab. 1). The DBPD in OMI/AP, VAP and CPS were 1.28 ± 0.38 ml/mmHg, 1.54 ± 0.53 ml/mmHg and 1.48 ± 0.33 ml/mmHg, respectively (Tab. 1). There were no significant differences in each index but there was a tendency that the vascular compliance in VAP was greater than that in OMI/AP (Fig. 2).
Table 1. Various Characteristics of Patients

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<th>OMI/AP (msec)</th>
<th>VAP (msec)</th>
<th>C (msec)</th>
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<tr>
<td>PWV</td>
<td>63.6 ± 20.8</td>
<td>86.4 ± 12.4</td>
<td>98.1 ± 5.2</td>
</tr>
<tr>
<td>Tarazi's Index</td>
<td>7.81 ± 1.27</td>
<td>6.65 ± 0.99</td>
<td>7.10 ± 1.11</td>
</tr>
<tr>
<td>DBPD (ml/mmHg)</td>
<td>0.81 ± 0.30</td>
<td>0.69 ± 0.25</td>
<td>0.64 ± 0.17</td>
</tr>
<tr>
<td>TC (mg/dl)</td>
<td>1.28 ± 0.38</td>
<td>1.54 ± 0.53</td>
<td>1.48 ± 0.33</td>
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<tr>
<td>HDL-C (mg/dl)</td>
<td>223.1 ± 47.7</td>
<td>192.4 ± 33.4</td>
<td>196.2 ± 19.8</td>
</tr>
<tr>
<td>AI</td>
<td>5.15 ± 2.04</td>
<td>3.95 ± 1.63</td>
<td>3.78 ± 1.18</td>
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OMI/AP: patients with old myocardial infarction or angina pectoris having significant fixed stenosis.
VAP: patients with variant form of angina.
C: patients with chest pain syndrome.
PWV: pulse wave velocity. DBPD: diastolic blood pressure decay.
TC: total cholesterol. HDL-C: high density lipoprotein cholesterol. AI: Atherogenic Index.

LIPIDS PROFILE.

The plasma levels of TC in OMI/AP, VAP and C were 223.1 ± 47.7 mg/dl, 192.4 ± 33.4 mg/dl and 196.2 ± 19.8 mg/dl, respectively (Tab. 1) and TC in VAP was significantly lower than that in OMI/AP (Fig. 3). The plasma level of HDL-C in OMI/AP, VAP and C were 37.8 ± 12.3 mg/dl, 42.8 ± 13.4 mg/dl and 43.6 ± 12.8 mg/dl, respectively.

The AI in OMI/AP, VAP and C were 5.15 ± 2.04, 3.95 ± 1.63 and 3.78 ± 1.18, respectively (Tab. 1). AI in OMI/AP was greater than that in C (Fig. 3).

COMPARISONS OF VARIOUS PARAMETERS IN VAP WITH AND WITHOUT SIGNIFICANT CORONARY ARTERY STENOSIS.

PWV in VAP (+) and VAP (−) were 7.24 ± 0.95 m/sec and 6.36 ± 0.92 m/sec, respectively (Tab. 2) and PWV in VAP (−) was significantly

Figure 1. Coronary Index in three groups. Values were expressed as mean ± standard deviation. OMI/AP: patients with old myocardial infarction or angina pectoris having significant fixed stenosis. VAP: patients with variant form of angina. C: patients with chest pain syndrome.
Figure 2. Comparisons of pulse wave velocity and aortic compliance. Values were expressed as mean ± standard deviation. PWV: pulse wave velocity. PP/SV: pulse pressure / stroke volume. DBPD: diastolic blood pressure decay. The other abbreviations are the same in Fig. 1.

slower than that in OMI/AP (P < 0.01). Tarazi' index in VAP (+) and VAP (−) were 0.84 ± 0.18 mmHg/ml and 0.62 ± 0.26 mmHg/ml, respectively (Tab. 2). DBPD in VAP (+) and VAP (−) were 1.23 ± 0.35 ml/mmHg and 1.69 ± 0.56 ml/mmHg, respectively (Tab. 2). DBPD in VAP (−) was significantly greater than that in OMI/AP (P<0.01). The aortic compliance in VAP (−) was significantly greater than that in OMI/AP (Fig. 4).

The plasma levels of TC in VAP (+) and VAP (−) were 178.3 ± 26.4 mg/dl and 199.1 ± 33.6 mg/dl, respectively (Tab. 2). The plasma concentrations of HDL-C in VAP (+) and VAP (−) were 29.8 ± 8.1 mg/dl and 48.4 ± 10.2 mg/dl, respectively (Tab. 2). AI in VAP (+) and VAP (−) were 5.28 ± 1.79 and 3.29 ± 1.14, respectively (Tab 2). The HDL-C level in VAP (−) was significantly higher and the AI was significantly lower than that in VAP (+) (Fig. 5).

DISCUSSION

The aortic stiffness increased with advancing age6). The age dependency could be accounted for by changes in arterial wall elastin and collagen contents1). It is controversial that the aortic stiffness is correlated to...
coronary arterial stiffness, coronary atherosclerosis or other arterial stiffness. We previously reported that aortic stiffness and compliance were correlated to the presence and the degree of coronary artery stenosis. Dart et al indicated that aortic distensibility assessed by echocardiography might be an indicator of coronary disease. Farrar et al also suggested that arterial stiffness can be used to predict the severity of diffuse asymptomatic atherosclerosis in monkeys.

However, to our best knowledge, there was no report on the relationship between aortic stiffness and variant form of angina pectoris. Here we reported that aortic stiffness measured by pulse wave velocity (PWV) in patients with VAP was lower than that in patients with OMI/AP who had fixed coronary narrowing. Among the patients with VAP, PWV in patients with VAP (−) was significantly lower and this indicated that coronary artery in VAP (−) may be softer and more compliant compared to coronary artery in OMI/AP who had fixed coronary stenosis. But PWV in patients with VAP with stenosis was equivalent to that in

Figure 3. Comparisons of pulse wave velocity and aortic compliance in patients with variant form of angina with or without significant fixed coronary stenosis. Values were expressed as mean ± standard deviation. VAP (+): patients with variant form of angina with significant fixed coronary stenoses. VAP (−): patients with variant form of angina without significant fixed coronary stenosis. The other abbreviations are the same in Fig. 1.
Table 2. Various Characteristics of Patients with VAP with and without fixed coronary stenosis

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<th>VAP (-)</th>
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<tbody>
<tr>
<td>PWV (m/sec)</td>
<td>7.24 ± 0.95</td>
<td>6.36 ± 0.92</td>
</tr>
<tr>
<td>Tarazi's Index (mmHg/ml)</td>
<td>0.84 ± 0.18</td>
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VAP(+) : patients with variant form of angina with significant fixed coronary stenosis.
VAP(-) : patients with variant form of angina without significant fixed coronary stenosis.
PWV: pulse wave velocity. DBPD: diastolic blood pressure decay.
TC: total cholesterol. HDL-C: high density lipoprotein cholesterol.
AI: Atherogenic Index.

OMI/AP with fixed stenosis. And also aortic compliance in VAP patients with stenosis was not different from that in OMI/AP with fixed stenosis. These results indicated that even if the aortic stiffness is correlated to coronary artery stiffness, vasospasm could occur irrespective of the degree of coronary artery stiffness.

Figure 4. Comparisons of plasma levels of lipid and atherogenic index. Values expressed as mean ± standard deviation. TC: total cholesterol. HDL-C: high density lipoprotein cholesterol. TG: triglyceride. AI: Atherogenic Index. The other abbreviations are the same in Fig. 1.
Figure 5. Comparisons of plasma levels of lipid and atherogenic index in patients with variant form of angina with or without significant fixed coronary stenosis. Values were expressed as mean ± standard deviation. The other abbreviations are the same in Fig 1, Fig 3 and Fig. 4.

The plasma TC level in patients with VAP without significant stenosis was significantly lower than that in patients OMII/AP. Scholl et al also showed that TC in VAP was lower than that in patients with CAD. There was no significant difference between TC levels in VAP with and without significant stenosis, but HDL level in VAP without stenosis was higher than that in VAP with stenosis. Kupari et al reported aortic stiffness positively correlated to the plasma level of HDL. Dart et al also reported that a group of patients with hypercholesterolemia had more distensible aorta than did normocholesterolemic controls. These data are contradictory to our data, but Hopkins et al showed the positive relation of aortic stiffness to LDL/HDL ratio.

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