Evaluation of mean platelet volume in patients with different degree of coronary collateral development

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Abstract
Aim: Coronary collateral vessels are an alternative source of blood supply to a myocardial area jeopardized by ischemia. As some patients have poor collaterals despite significant stenosis, it is thought that multiple factors affect collateral development beside coronary artery disease severity. Mean platelet volume is an indicator of platelet activation. Increased mean platelet volume is found to be related to worse prognosis in the coronary artery disease. In this study, we aimed to investigate the relationship between mean platelet volume and coronary collateral development. Material and Method: Patients with total occlusion in at least one coronary artery were enrolled in this study. Coronary angiography images of 367 patients without a history of revascularization were evaluated retrospectively, and coronary collateral development was graded according to Rentrop classification. Patients were divided into two groups based on Rentrop classification. Rentrop grade 0-1 was regarded as poor collateral development, and Rentrop 2-3 was regarded as good collateral development. Mean platelet volume was compared between these two groups. Results: Poor collateral development was found in 236 patients (64.3%), and good collateral development was found in 131 patients (35.7%). There was no statistically significant difference in mean platelet volume levels between two groups (9.9±1.2 fL and 10.3±1.3 fL, p=0.228). The 3-vessel disease was found to be a predictor of good collateral development (p=0.024). Discussion: In this study, it was found that there was no relationship between mean platelet volume and coronary collateral development.

Keywords
Coronary Collateral Development; Mean Platelet Volume; Coronary Artery Disease

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Mean platelet volume and coronary collaterals

Introduction
Platelets play an important role in the pathogenesis of coronary artery disease (CAD). Their reactivity is a key issue and platelet size, simply measured by mean platelet volume (MPV), is an indirect marker of platelet reactivity [1,2]. Large platelets have a greater content of granules, higher thrombotic potential, increased thromboxane synthesis and serotonin release [3-5]. It has been reported that elevated MPV is associated with increased mortality following myocardial infarction and increased cardiovascular events in patients undergoing either an elective or urgent percutaneous coronary intervention [2,6]. Coronary collateral vessels serve as an alternative source of blood supply to an ischemic myocardium. Poor collateral development was found to be associated with larger infract size and mortality in CAD patients [7,8]. One of the well-established determinants of collateral formation is coronary artery stenosis, but poor collateral development despite significant stenosis suggest that multiple mechanisms contribute to collateral formation [9]. Some mediators such as nitric oxide (NO), vascular endothelial growth factor, thromboxane, prostacyclin take part in the formation of collateral vessels [10-12].

In this study, we aimed to evaluate the relationship between MPV and coronary collateral formation in CAD patients.

Material and Method
The present study is a retrospective cross-sectional study. Between July 2011 and August 2012, 367 patients who underwent coronary angiography at our institution were enrolled in this study. All patients underwent coronary angiography because of suspicion of CAD based on their symptoms or diagnostic tests. Patients with at least one totally occluded major epicardial coronary artery were included in the study. Demographic and clinical data including age, gender, the prevalence of DM, hyperlipidemia, hypertension, and smoking history. The demographic and laboratory characteristics among poor collateral and good collateral groups are shown in Table 1.

Results
The mean age of the study population was 61.6±12.4, and 74% of the patients were male. 94 patients had a diagnosis of stable angina pectoris, 97 patients had unstable angina pectoris or non-ST elevation myocardial infarction, 176 had ST elevation myocardial infarction. Of the 367 patients, 131 patients (35.7 %) had good collateral development, and 236 patients (64.3 %) had poor collateral development. The two groups did not differ for age, gender, diabetes mellitus, hyperlipidemia, hypertension, and smoking history. The demographic and laboratory characteristics of the groups are shown in Table 1.

Table 1. Clinical and laboratory characteristics among poor collateral and good collateral groups

<table>
<thead>
<tr>
<th>Variables</th>
<th>All patients n=367</th>
<th>Good collateral n=131</th>
<th>Poor collateral n=236</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>61.6±12.4</td>
<td>62.8±11.4</td>
<td>61.7±12.0</td>
<td>0.939</td>
</tr>
<tr>
<td>Men (%)</td>
<td>74.6</td>
<td>71.7</td>
<td>76.3</td>
<td>0.453</td>
</tr>
<tr>
<td>Diabetes mellitus (%)</td>
<td>31.8</td>
<td>35.1</td>
<td>30.1</td>
<td>0.874</td>
</tr>
<tr>
<td>Hypertension (%)</td>
<td>45.7</td>
<td>42.7</td>
<td>47.5</td>
<td>0.602</td>
</tr>
<tr>
<td>Any smoking history (%)</td>
<td>33.8</td>
<td>32.1</td>
<td>34.7</td>
<td>0.401</td>
</tr>
<tr>
<td>Dyslipidemia (%)</td>
<td>49.3</td>
<td>49.6</td>
<td>49.1</td>
<td>0.821</td>
</tr>
<tr>
<td>Median ejection fraction (%)</td>
<td>40 (15-55)</td>
<td>40 (15-55)</td>
<td>40 (20-60)</td>
<td>0.506</td>
</tr>
<tr>
<td>Three-vessel disease (%)</td>
<td>25.1</td>
<td>35.1</td>
<td>19.5</td>
<td>0.016</td>
</tr>
<tr>
<td>Mean MPV (fL)</td>
<td>10.0±1.3</td>
<td>10.3±1.3</td>
<td>9.9±1.2</td>
<td>0.228</td>
</tr>
<tr>
<td>Mean hemoglobin (g/dL)</td>
<td>13.8±1.9</td>
<td>13.5±1.8</td>
<td>14.1±1.9</td>
<td>0.169</td>
</tr>
<tr>
<td>Mean white blood cell count (1000/μL)</td>
<td>7.157±3.06</td>
<td>6.470±2.89</td>
<td>9.890±3.14</td>
<td>0.021</td>
</tr>
<tr>
<td>Mean platelet count (1000/μL)</td>
<td>248.2±64</td>
<td>244.6±61</td>
<td>250.2±65</td>
<td>0.083</td>
</tr>
</tbody>
</table>
mean, and has a favorable impact on the prognosis of patients with coronary artery disease [8,14]. Collateral vessels are a valuable source for alternative blood supply to ischemic myocardium especially in case of unachievable revascularization. But, there is notable variation in the degree of coronary collateral development. Collateral development is a multifactorial process, and it is important to define the factors that facilitate collateral development.

Platelets play a crucial role in the pathogenesis of atherosclerotic complications, and they are important targets for the treatment of coronary artery disease. MPV is an indirect marker of platelet activity. Larger, metabolically, and enzymatically more active platelets have greater prothrombotic features [5]. Elevated MPV was associated with worse clinical outcomes in patients with CAD [2]. Whether platelets with elevated MPV affect outcomes for worse by collateral formation or not hasn’t been explained clearly.

Previous studies about this issue are controversial. In a study of patients with the acute coronary syndrome, high MPV on admission was found to be associated with the presence of coronary collateral formation [15]. In contrast, in another study, elevated MPV was found to be a predictor of inadequate collateral development [16]. Some studies revealed that MPV levels were not related to coronary collateral development [17–19]. We also found that there wasn’t any significant relation between the collateral formation and MPV. Our study differed from the studies mentioned above in some aspects. Patients with >50, >80 or >90 stenosis were enrolled in some of the previous studies. As the variations of the severity of the stenosis may affect collateral formation, we enrolled patients with at least one totally occluded major coronary artery. Also, there is a difference in classification of patients according to Rentrop classification. Rentrop 2–3 collaterals were accepted as good collateral formation in our study, whereas only Rentrop 3 was accepted as adequate collateral development in some studies. In a study by Tan et al. platelet activation was measured by soluble CD40 ligand, soluble P-selectin, and soluble glycoprotein V. They reported that the correlation between the degree of collateralization and these platelets activation markers was not significant [20]. Thromboxane A2 and serotonin have been shown to cause vasoconstriction of the collateral vessels [21]. Large platelets have a greater content of granules, increased thromboxane synthesis and serotonin release. It is possible that activated platelets within collateral vessels could cause vasoconstriction and decrease collateral flow. It has been reported that platelet activating factor caused a decrease in coronary collateral flow with the participation of thromboxane A2 [12]. It has been reported that serotonin blocker augments flow reserve of the collateral circulation in anginal patients [22]. Collateral development is a multifactorial process, and the further studies are required to understand the role of activated platelets in collateral formation.

## Discussion

In our study, we investigated whether MPV is related to collateral development in patients with CAD. We found that the MPV levels were not related to coronary collateral development. A well developed coronary collateral limits the ischemia, reduce the size of myocardial infarction, preserve left ventricle function, and has a favorable impact on the prognosis of patients with coronary artery disease [8,14]. Collateral vessels are a valuable source for alternative blood supply to ischemic myocardium especially in case of unachievable revascularization. But, there is notable variation in the degree of coronary collateral development.

Collateral development is a multifactorial process, and it is important to define the factors that facilitate collateral development. Platelets play a crucial role in the pathogenesis of atherosclerotic complications, and they are important targets for the treatment of coronary artery disease. MPV is an indirect marker of platelet activity. Larger, metabolically, and enzymatically more active platelets have greater prothrombotic features [5]. Elevated MPV was associated with worse clinical outcomes in patients with CAD [2]. Whether platelets with elevated MPV affect outcomes for worse by collateral formation or not hasn’t been explained clearly.

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## Conclusion

In conclusion, MPV levels were not related to coronary collateral development in a group of patients with either stable coronary artery disease or acute coronary syndrome. Our study has some limitations. First, it is a retrospective study. Because of its retrospective design, there were no available data about previous antiplatelet drug use. It is possible that previous usage of antiplatelet drugs may have modulated MPV levels. Secondly, angiographically visible collaterals represent only a fraction of the total collateral vessel amount.

Animal and human rights statement

No animal studies were carried out by the authors for this article. The study was approved by the institutional ethics committee. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Scientific Responsibility Statement

The authors declare that they are responsible for the article’s scientific content including study design, data collection, analysis and interpretation, writing, some of the main line, or all of the preparation and scientific review of the contents and approval of the final version of the article.

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