The Relationship Between the Gensini Score and Complete Blood Count Parameters in Coronary Artery Disease

Koroner Arter Hastalığında Gensini Skoru ve Tam Kan Sayımı Parametreleri Arasındaki İlişki

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ABSTRACT

Introduction: The aim of this study was to evaluate the relationship between the extend and severity of coronary artery disease (CAD) determined by the Gensini score and complete blood count parameters (white blood cell, hemoglobin, platelet, mean platelet volume, lymphocyte, neutrophil).

Patients and Methods: Ninety patients with CAD underwent coronary angiography (40 females, mean age 61 ± 1.2 years) were included in this study. Patients with acute coronary syndrome and prior cardiovascular disease excluded from the study. The association between the extent and severity of CAD, which were assessed by the Gensini score, and complete blood count parameters was analyzed by a correlation analysis.

Results: Coronary angiography revealed, 6 (6.7%) patients had three, 16 (17.8%) patients had two, and 24 (26.7%) patients had single-vessel disease; 44 (48.9%) patients had non-critical stenosis. The mean Gensini score was 19.1 ± 2.1. We found a relationship between white blood cell and neutrophil counts and the Gensini score. There was no relationship between Gensini score and the mean platelet volume and other parameters.

Conclusion: The present study supports the hypothesis that inflammation is one of the main component in the pathogenesis of CAD.

Key Words: Coronary artery disease; blood platelets; leukocyte count; neutrophils; blood cell count.

Received: 20.06.2012  Accepted: 12.07.2012
INTRODUCTION

Coronary artery disease (CAD) is a progressive inflammatory disease which atherosclerosis plays a major role in its etiology. CAD is a leading cause of morbidity and mortality worldwide, and the incidence of CAD has been gradually increasing. Platelets and white blood cells (WBCs) secrete and express a large number of substances that are crucial mediators of inflammation. Thus, they are involved in the development of cardiovascular disease and atherosclerotic process. Several studies have investigated the role of both platelets and WBCs in the development of CAD.

After clinical presentation of CAD, the diagnostic and treatment methods applied are of high cost. Therefore, the determination of the presence and extend of CAD by easy and applicable methods before the onset of the disease are of high importance. The aim of this study was to evaluate the relationship between the extend and severity of CAD determined by the Gensini score and complete blood count parameters.

PATIENTS and METHODS

Ninety patients (40 females, mean age 61 ± 1.2 years), who were admitted to our clinic with chest pain and scheduled for coronary angiography based on non-invasive stress tests, were included in the study. The study protocol was approved by the ethics committee of our institution and written consent was taken from all participants.

Basic demographic data were collected including age, gender, presence of diabetes mellitus, and the presence of traditional major cardiovascular risk factors (age, sex, hypertension, dyslipidemia, family history of premature cardiovascular disease (CVD), and current smoking). Patients with acute coronary syndrome and prior CVD (history of myocardial infarction, coronary artery bypass surgery, percutaneous coronary intervention, stroke and peripheral arterial occlusive disease) were excluded from the study.

Serum biochemistry and complete blood count parameters were measured in all patients. Venous blood samples were taken from patients after an 8 to 10-hour fast and were analyzed for complete blood count parameters (WBC, hemoglobin, platelet, mean platelet volume (MPV), lymphocyte, neutrophil), total cholesterol, high-density lipoprotein cholesterol, triglyceride, and glucose levels. Low-density lipoprotein cholesterol levels were calculated using the Friedewald formula. Patients were considered to have type II diabetes mellitus if they were previously diagnosed and treated for diabetes and/or if they had a fasting blood glucose level of ≥ 126 mg/dL. Patients were considered to have hypertension if they had previously known hypertension, or if they were on antihypertensive therapy, or if they had a systolic blood pressure of ≥ 140 mmHg and a diastolic blood pressure of ≥ 90 mmHg, which were calculated as the mean of two measurements taken on each arm.

Angiographic evaluations were done by two experienced cardiologists, who were blinded to the study. Discrepancies were solved by consensus. The extent and severity of CAD were assessed by the Gensini score. The Gensini score was calculated by multiplying the severity coefficient, which was assigned to each coronary stenosis according to the degree of luminal narrowing (reductions of 25%, 50%, 75%,...
90%, 99%, and complete occlusion were given Gensini scores of 1, 2, 4, 8, 16, and 32, respectively, by the coefficient identified based on the functional importance of the myocardial area supplied by that segment: the left main coronary artery, 5; the proximal segment of the left anterior descending coronary artery, 2.5; the mid segment of the left anterior descending coronary artery, 1.5; the apical segment of the left anterior descending coronary artery, 1; the first diagonal branch, 1; the second diagonal branch, 0.5; the proximal segment of the circumflex artery, 2.5 (if right coronary artery dominance exist 3.5); the distal segment of the circumflex artery, 1 (if dominant, 2); the obtuse marginal branch, 1; the posterolateral branch, 0.5; the proximal segment of the right coronary artery, 1; the mid segment of the right coronary artery, 1; the distal segment of the right coronary artery, 1; and the posterior descending artery, 1.

### Statistical Analysis

Statistical analysis was conducted using the Statistical Package for the Social Sciences (SPSS) for Windows (version 15; SPSS Inc., Chicago, IL, USA). Means were given together with standard errors, and frequencies were expressed as percentages. The association between the extent and severity of CAD, which were assessed by the Gensini score, and complete blood count parameters was analyzed by a correlation analysis.

### RESULTS

The significant CAD (> 50%) was established in 46 (51.1%) of 90 patients undergoing coronary angiography. Of all patients, 6 (6.7%) patients had three, 16 (17.8%) patients had two, and 24 (26.7%) patients had single-vessel disease; 44 (48.9%) patients had non-critical stenosis. The mean Gensini score was 19.1 ± 2.1.

Considering the risk factors, of the patients 65% had hypertension, 35% had diabetes mellitus, 22% had family history of premature CVD and 16% were smokers. Serum biochemistry and complete blood count parameter levels showed in Table 1. There was no correlation between the Gensini score and the MPV. We found a relationship between WBC and neutrophil counts and the Gensini score. There was no relationship between the other parameters (Table 2).

### DISCUSSION

Atherosclerosis and its most important complication, acute coronary syndrome, cause significant morbidity and mortality in men and women all over the world, despite the major advances in the treatment of cardiovascular diseases[1]. Inflammation plays a pivotal role in the progression of atherosclerosis, and WBC count is a marker of inflammation. WBCs play a major role in the development of CAD through different mechanisms, such as mediation of inflammation, induction of proteolysis and oxidative damage to the endothelial cells, plugging the microvasculature, induction of hypercoagulability, and infarct expansion[6]. Numerous studies have demonstrated that leukocytosis is an independent predictor of future cardiovascular events, both in healthy individuals free of CAD and in subjects with CAD[8,7]. In addition, the severity of CAD is shown to be related to the WBC count[8,9]. In this study, a relationship was found between the extent of CAD and WBC count, which is also consistent with the literature. These findings suggest that the WBC count may be a parameter that indicates the prevalence and the severity of CAD.

Table 1. Clinical characteristics of the group (mean ± SE)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose (mg/dL)</td>
<td>135.9 ± 7.7</td>
</tr>
<tr>
<td>LDL-C (mg/dL)</td>
<td>107.3 ± 3.6</td>
</tr>
<tr>
<td>HDL-C (mg/dL)</td>
<td>41.8 ± 1.2</td>
</tr>
<tr>
<td>TC (mg/dL)</td>
<td>176.5 ± 5.0</td>
</tr>
<tr>
<td>TG (mg/dL)</td>
<td>162.7 ± 12.1</td>
</tr>
<tr>
<td>WBC (10³/mm³)</td>
<td>7.6 ± 0.2</td>
</tr>
<tr>
<td>Gensini score</td>
<td>19.1 ± 2.1</td>
</tr>
<tr>
<td>Hemoglobin (g/dL)</td>
<td>12.8 ± 0.2</td>
</tr>
<tr>
<td>Platelet (10³/mm³)</td>
<td>228 ± 8.5</td>
</tr>
<tr>
<td>MPV (fl)</td>
<td>8.3 ± 0.09</td>
</tr>
<tr>
<td>Lymphocyte (10³/mm³)</td>
<td>2.1 ± 0.06</td>
</tr>
<tr>
<td>Neutrophil (10³/mm³)</td>
<td>4.5 ± 0.15</td>
</tr>
</tbody>
</table>

LDL-C: Low density lipoprotein cholesterol, HDL-C: High density lipoprotein cholesterol, TC: Total cholesterol, TG: Triglycerides, WBC: White blood cell, MPV: Mean platelet volume.

Table 2. Pearson correlations between the Gensini score and complete blood count parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>p</th>
<th>r</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC (10³/mm³)</td>
<td>0.030</td>
<td>0.230</td>
</tr>
<tr>
<td>Hemoglobin (g/dL)</td>
<td>0.395</td>
<td>-0.091</td>
</tr>
<tr>
<td>Platelet (10³/mm³)</td>
<td>0.493</td>
<td>-0.074</td>
</tr>
<tr>
<td>MPV (fl)</td>
<td>0.180</td>
<td>0.143</td>
</tr>
<tr>
<td>Lymphocyte (10³/mm³)</td>
<td>0.227</td>
<td>0.132</td>
</tr>
<tr>
<td>Neutrophil (10³/mm³)</td>
<td>0.018</td>
<td>0.254</td>
</tr>
</tbody>
</table>

WBC: White blood cell, MPV: Mean platelet volume.
den cardiac death are caused by thrombosis of the coronary arteries. Thrombus formation is triggered either by the rupture or the ulceration of the atherosclerotic plaque. In the subsequent process, the role of platelets and especially the thrombogenic state, due to the increased reactivity of circulating platelets, are very important. Platelets are heterogeneous in size and density. MPV, the most commonly used measure of platelet size, is a potential marker of platelet reactivity\(^{10}\). Although there is still uncertainty about the most precise methodology for the MPV measurement, it is routinely and cost effectively available in the clinical and outpatient setting. The increased platelet reactivity results in shortening of the bleeding time and increased platelet volume. The large platelets are metabolically and enzymatically more active and have an increased thrombotic potential. Recent studies have shown that the MPV is increased in acute coronary syndromes\(^ {10-12}\). However, the role of the platelets in the pathophysiology of stable CAD is still controversial. Platelets plays an important role at the beginning of the atherosclerotic lesions. The platelet-derived growth factor, released from the \(\alpha\)-granules of the platelets, stimulates the growth of the vascular smooth muscle, and has a chemotactic effect on the inflammatory cells and the fibroblasts\(^ {13}\). From this perspective, the platelets and their functional cursor MPV are important indicators of the presence, the prevalence and the severity of the stable CAD. There are several studies indicating the role of platelets in the development of stable CAD whereas other studies have suggested that platelets have no effect on the development of CAD\(^ {11,14}\). In this study, no relationship was found between the severity and extend of CAD and MPV.

CONCLUSION

To summarize, the association between higher total WBC and neutrophil counts and the severity and extent of coronary atherosclerosis was confirmed in this study which is also consistent with the literature. We found no relationship between the Gensini score and MPV. The present study supports the hypothesis that inflammation is one of the main component in the pathogenesis of CAD. However, these findings suggest that the WBC count may be a parameter that indicates the prevalence and the severity of CAD.

Limitations

Limitations of this study are its cross-sectional design and small number of patients. In addition, our patients had low Gensini score. These may be cause of the lack of correlation between the MPV and Gensini score.

REFERENCES