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Original Research

# Association of red Cell Distribution width with Characteristics of Coronary Atherosclerotic Plaques as Detected by Computed Tomography Angiography

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**ABSTRACT**
**Objective:** To evaluate the relationship between red blood cell distribution width (RDW) and the severity/morphology of coronary atherosclerotic plaques (CAPs).
**Methods:** We retrospectively analyzed 572 patients without a history of coronary artery disease (CAD) in whom dual-source 64-slice computed tomography angiography (CTA) was performed due to the suspicion of CAD.
**Results:** Critical CAPs were detected in 26.9% of subjects. The RDW value was higher in patients with critical CAPs than in those without (13.63±1.28 vs. 14.31±1.58, p<0.001). Patients with any type of CAP regardless of the morphology or severity revealed enhanced RDW levels compared with those with normal coronary arteries (p<0.001). In the multinomial logistic regression analysis, RDW was found as an independent predictor for the presence of severe CAP (odds ratio (OR): 1.40, 95% confidence interval (CI): 1.20–1.63, p<0.001). RDW was also found to be associated with the presence of non-calcified plaque (OR: 1.30, 95% CI: 1.08–1.57, p=0.006) and mixed plaque morphologies (OR: 1.47, 95% CI: 1.19–1.81, p<0.001) after adjusted for other variables.
**Conclusion:** Our findings suggested that RDW as a simple, available and inexpensive biomarker was significantly associated with both the severity and vulnerable morphology of CAPs in patients undergoing coronary CTA.
**Keywords:** Coronary atherosclerotic plaque, multidetector computed tomography angiography, red cell distribution width

## INTRODUCTION

Red blood cell distribution width (RDW) is a calculation of variableness in the dimensions of circulatory red blood cells. RDW as a costless and routine parameter of standard complete blood count (CBC) test is used for differential diagnosis of anemia (1). Chronic low-grade inflammation and enhanced oxidative stress are associated with defective erythropoiesis and consecutive erythrocyte degradation that results in increased RDW levels (2). It has been previously shown that enhanced RDW levels were related to adverse cardiovascular outcomes in patients with acute coronary syndrome and heart failure (3, 4). Furthermore, increased RDW values were shown to be associated with complex coronary artery lesions during conventional coronary angiography (5).

Multidetector computed tomography angiography (CTA) is an already known non-invasive diagnostic test that provides information about various characteristics of coronary atherosclerotic plaques (CAPs) (e.g., morphology and severity) (6-9). To our knowledge, the association of RDW levels with both the severity and morphological characteristics of CAPs as demonstrated by CTA has not been evaluated yet. The aim of the present study was to evaluate the relationship between RDW and the quantity and quality of CAPs as detected by dual-source 64-slice CTA in patients without a history of CAD.

## METHODS

### Study Population

A total of 572 patients in whom coronary dual-source 64-slice CTA was performed due to the suspicion of coronary artery disease (CAD) following a detailed clinical and laboratory evaluation were enrolled between January 2009 and June 2011. The mean age of the patients was 55±11 years, and 47.6% were men. Exclusion criteria were history of a documented CAD, history of blood transfusion, hematologic diseases including anemia, renal

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dysfunction (serum creatinine ≥1.5 mg/dL), hepatic disorders, malignancy, acute and/or chronic inflammatory and/or infectious diseases, and surgery or trauma within the previous month. The study was approved by our Institutional Ethics Committee (Hacettepe University, protocol no.: HEK 08/181) in accordance with the ethical issues as outlined in the Declaration of Helsinki. Written informed consent was obtained from each patient before study enrollment.

**Definitions and Risk Factor Assessment**

Baseline study parameters including age, gender, body mass index, family history of a premature CAD, smoking habits, history of diabetes mellitus, hypertension, hyperlipidemia, and medications were recorded. Hypertension was defined as a systolic blood pressure of ≥140 mmHg and/or a diastolic blood pressure of ≥90 mmHg in at least two measurements or already taking any antihypertensive agent (10). Dyslipidemia was defined as a total cholesterol of >200 mg/dL or triglycerides of >150 mg/dL. Diabetes mellitus was defined as a fasting plasma glucose level of >126 mg/dL or glucose level >200 mg/dL at any measurement or already taking an antidiabetic medication. Smoking was recorded as positive in only current smokers. Global CAD risk was estimated by the Framingham risk equation.

**Biochemical and Hematological Measurements**

Peripheral venous blood samples were collected after a 12-hour fasting interval. Biochemistry panel was processed using commercially available assay kits (Hitachi P800; Holliston, MA, USA). A CBC analysis including erythrocyte count, hemoglobin (Hb) level, RDW, mean corpuscular volume, and white blood cell count was calculated within 2 h of specimen collection using a Beckman Coulter (High Wycombe, UK) Gen-S automated hematology analyzer.

**Coronary CTA**

Coronary CTA studies were performed by using a dual-source 64-slice CTA scanner (Somatom Definition; Siemens, Erlangen, Germany). All participants were in sinus rhythm before scanning. Sublingual nitrate (5 mg isosorbide dinitrate (Isordil®; Fako, İstanbul, Turkey)) was administered 2-4 min before the test for coronary vasodilatation. The coronary angiographic scan was obtained following the injection of 80 mL nonionic contrast medium (350 mg I/mL Iomeprol, Iomeron®; Bracco, Milan, Italy) (adjusted for body weight of 1.25 cc/kg) at a flow rate of 6 mL/s followed by 50 mL saline solution with the same injection rate to wash out the contrast material from the right ventricle. Contrast administration was controlled with bolus tracking. The scan parameters were as follows: detector collimation, 32 mm × 0.6 mm; slice acquisition, 64 mm × 0.6 mm; gantry rotation time, 330 ms; temporal resolution, 83 ms; pitch, 0.2-0.47 adapted to the heart rate; tube-current, 390 m as per rotation; and tube potential, 120 kV. Scanning time was approximately 5.7-8.4 s, depending on cardiac dimensions and pitch, in a single breath held in the craniocaudal direction. Prospective electrocardiogram (ECG) tube-current modulation (ECG pulsing) for radiation dose reduction was used for all patients. Retrospective gating technique was used to synchronize data reconstruction with the ECG signal. The reconstructions were made in all cardiac phases at 50 ms intervals at a slice thickness of 0.75 mm and a reconstruction increment of 0.5 mm. The reconstruction interval with the fewest motion artifacts was selected and used for further analysis.

**CTA Evaluation**

All CTA data were evaluated by a radiology staff unaware of the clinical data of the patients. The intraobserver agreement was 95% for lesion severity and 96% for lesion morphology. CAP was defined as any clearly discernible structure attributable to the coronary artery wall in at least two independent image planes. The non-significant CAP was defined as stenosis causing ≤50% luminal narrowing, and the significant CAP was defined as stenosis causing >50% luminal narrowing. All of the CAPs were included in the analysis. For categorization of the CAPs, the coronary system was divided into 16 separate segments based on a modified American Heart Association classification using original axial images, thin slice, maximal intensity projections, and cross-sectional reconstructions orthogonal to the long axis of each coronary segment (0.75 mm thickness) (11). For each segment, CAPs were categorized as (1) none, (2) calcified, (3) non-calcified, and (4) mixed (Figure 1).

**Statistical Analysis**

Data analyses were performed using the SPSS software, version 20.0 (SPSS IBM Corp.; Armonk, NY, USA). Data are expressed as mean ± standard deviation or n (%). Kolmogorov-Smirnov test was used to assess the normal distribution of variables. Categorical and continuous variables between the two groups were compared using chi-square test and independent samples t-test, respectively. Kruskal-Wallis test was used to compare differences in

Figure 1. a-d. Contrast-enhanced multidetector computed tomographic image. (a) Normal right coronary artery; (b) significant, non-calcified plaque at the left main coronary artery extending to the ostium of the LAD (arrow); (c) non-significant, mixed plaque at the proximal segment of the LAD; (d) significant, dense calcified plaque at the middle LAD (arrow). LAD, left anterior descending artery

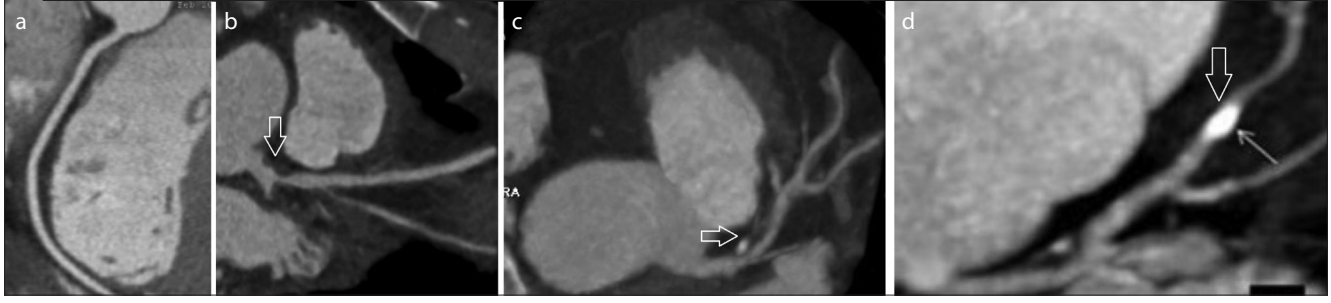


**Table 1.** Baseline demographic and clinical characteristics of patients due to the severity of coronary atherosclerotic plaques (n=572)

| Variables | Non-critical stenosis (n=418) | Critical stenosis (n=154) | p |
|---|---|---|---|
| Age (years) | 54±11 | 58±10 | <0.001 |
| Male gender (%) | 43.1 | 59.7 | <0.001 |
| BMI (kg/m$^2$) | 27.8±5.0 | 28.1±4.6 | NS |
| Hypertension (%) | 59.3 | 68.8 | 0. 038 |
| Diabetes mellitus (%) | 13.6 | 27.3 | <0.001 |
| History of smoking (%) | 25.4 | 41.2 | <0.001 |
| Family hx of CAD (%) | 8.4 | 11 | NS |
| Total cholesterol (mg/dL) | 208±43 | 202±44 | NS |
| Triglyceride (mg/dL) | 145±68 | 167±88 | 0.015 |
| HDL-cholesterol (mg/dL) | 52±14 | 50±16 | 0.023 |
| LDL-cholesterol (mg/dL) | 132±38 | 125±36 | NS |
| Serum creatinine (mg/dL) | 0.84±0.19 | 0.89±0.19 | 0. 002 |
| Hemoglobin (g/dL) | 14.56±1.45 | 14.31±1.89 | NS |
| RDW (%) | 13.63±1.28 | 14.31±1.58 | <0.001 |
| **Framingham risk score, n (%)** | | | |
| Low risk | 60.1 (251) | 44.4 (68) | |
| Intermediate risk | 29.5 (123) | 34.1 (53) | 0. 001 |
| High risk | 10.5 (44) | 21.5 (33) | |
| Medications | | | |
| Aspirin (%) | 50 | 66 | 0. 001 |
| Statin (%) | 34.6 | 40.5 | NS |
| Beta blocker (%) | 22.2 | 40.5 | <0.001 |
| CCB (%) | 12.1 | 17 | NS |
| ACE inhibitor (%) | 14.3 | 20.3 | NS |
| ARB (%) | 29.0 | 32.7 | NS |

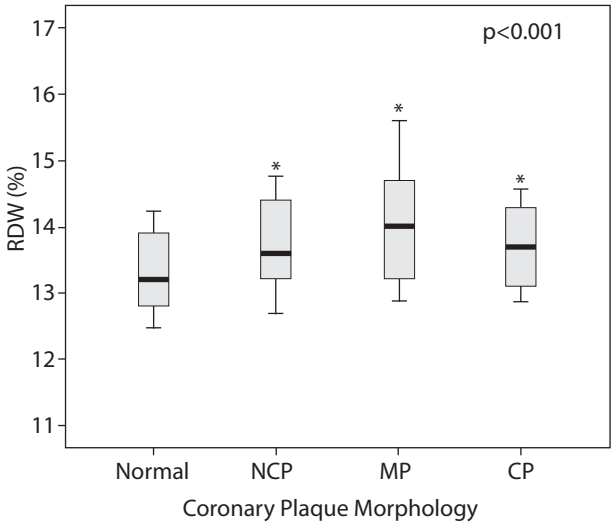
ACE: angiotensin-converting enzyme; ARB: angiotensin receptor blocker; BMI: body mass index; CAD: coronary artery disease; CCB: calcium channel blocker; HDL: high-density lipoprotein; hx: history; LDL: low-density lipoprotein; NS: non-significant; RDW: red blood cell distribution width

mean values between the groups where there were three or more independent groups. Logistic regression analysis was performed to assess the predictors of the presence and severity of CAPs. A two-tailed p-value <0.05 was accepted as statistically significant.

## RESULTS

Significant CAPs (luminal stenosis of >50%) were detected in 154/572 (26.9%) of subjects. The comparison of baseline parameters of the study groups according to the severity of CAPs as detected by CTA is presented in Table 1. Subjects with significant CAP were at an older age and predominantly male. Hypertension, diabetes mellitus, and history of smoking were also more prevalent in subjects with significant luminal stenosis (p<0.05). Moreover, the RDW level was found to be higher in patients with the significant CAP than in those without (13.63±1.28 vs. 14.31±1.58, p<0.001).

Figure 2. Comparison of RDW levels in between patients with normal coronary arteries and different atherosclerotic plaque morphologies




Furthermore, we assessed the relationship between RDW levels and the morphology of the CAPs (Table 2). A total of 345 (60.3%) patients with CAPs in their coronary arteries were stratified due to CAP morphologies as having primarily (defined as >70% of all segments) non-calcified plaque (NCP), mixed plaque (MP), or calcified plaque (CP). Percentages of patients with primarily NCP, MP, and CP were 32.8%, 11.9%, and 15.6%, respectively (Table 2). The RDW level was higher in patients with NCP, MP, and CP than in those with normal coronary arteries (13.96±1.47, 14.37±1.90, 13.80±0.95, and 13.51±1.25, respectively, p<0.001). Furthermore, the RDW level was significantly higher in patients with NCP and MP than in those with CP (13.96±1.47, 14.37±1.90 vs. 13.80±0.95, respectively, p<0.001) (Figure 2).

In the multinomial logistic regression analysis, RDW was found to be an independent predictor for the presence of significant CAPs at coronary CTA (odds ratio (OR): 1.40, 95% confidence interval (CI): 1.20-1.63, p<0.001) (Table 3). In addition to RDW level, age (OR: 1.03, 95% CI: 1.005-1.053, p=0.016), male gender (OR: 1.82, 95% CI: 1.08-3.03, p=0.023), smoking habit (OR: 2.52, 95% CI: 1.52-4.17, p<0.001), diabetes mellitus (OR: 1.83, 95% CI: 1.01-3.32, p=0.047), and serum triglyceride level (OR: 1.003, 95% CI: 1.000-1.007, p=0.049) were also found as significant predictors of the severity of CAP after adjustment for other risk factors (Table 3).

**Table 2.** Baseline characteristics of patients due to the morphology of atherosclerotic plaques (n=572)

| Variables | Normal (n=222) | NCP (n=188) | MP (n=68) | CP (n=89) | p |
|---|---|---|---|---|---|
| BMI (kg/m²) | 27.3±5.4 | 28.2±4.2 | 28.6±4.9 | 27.9±4.6 | 0.030 |
| Hypertension (%) | 50.9 | 66.8 | 62.3 | 78 | <0.001 |
| Diabetes mellitus (%) | 9.9 | 21.6 | 20.3 | 24.2 | 0.002 |
| Smoking (%) | 27.5 | 36 | 29 | 22 | NS |
| Family hx of CAD (%) | 9.5 | 10 | 7.2 | 7.7 | NS |
| Total cholesterol (mg/dL) | 206±40 | 206±45 | 204±38 | 210±52 | NS |
| Triglyceride (mg/dL) | 147±66 | 158±81 | 156±83 | 144±71 | NS |
| HDL–cholesterol (mg/dL) | 53±14 | 49±13 | 52±18 | 54±15 | 0.015 |
| LDL–cholesterol (mg/dL) | 130±34 | 130±40 | 126±32 | 132±43 | NS |
| Serum creatinine (mg/dL) | 0.84±0.19 | 0.88±0.18 | 0.82±0.15 | 0.85±0.24 | 0.049 |
| Hemoglobin (g/dL) | 14.55±1.48 | 14.49±1.75 | 14.29±1.64 | 14.49±1.40 | 0.04 |
| RDW (%) | 13.51±1.50 | 13.96±1.47 | 14.37±1.90 | 13.80±0.95 | <0.001 |
| **Framingham risk score** | | | | | |
| Low risk (%, n) | 71.2 | 37.3 | 62.9 | 46.2 | <0.001 |
| Intermediate risk (%, n) | 21.5 | 40.5 | 27.4 | 38.5 | |
| High risk (%, n) | 7.3 | 22.2 | 9.7 | 15.4 | |
| **Medications** | | | | | |
| Aspirin (%) | 43.7 | 58.5 | 63.2 | 65.2 | <0.001 |
| Statin (%) | 27.6 | 44.9 | 33.8 | 41.1 | 0.002 |
| Beta blocker (%) | 20.3 | 29.8 | 29.4 | 37.1 | 0.014 |
| CCB (%) | 7.7 | 17 | 13.2 | 20.2 | 0.007 |
| ACE inhibitor (%) | 15.3 | 17.6 | 14.7 | 14.6 | NS |
| ARB (%) | 24.8 | 32.4 | 29.4 | 38.2 | NS |

ACE: angiotensin–converting enzyme; ARB: angiotensin receptor blocker; BMI: body mass index; CAD: coronary artery disease; CCB: calcium channel blocker; CP: calcified plaque; HDL: high–density lipoprotein; hx: history; LDL: low–density lipoprotein; MP: mixed plaque (MP); NCP: non–calcified plaque; NS: non–significant; RDW: red blood cell distribution width

In the multinomial logistic regression analysis, RDW was also found to be a significant predictor of NCP (OR: 1.30, 95% CI: 1.08-1.57, p=0.006) and MP (OR: 1.47, 95% CI: 1.20-1.81, p<0.001) after adjustment for other risk factors (Table 4).

## DISCUSSION

The major findings of our study included: (1) the RDW level was significantly higher among patients with significant CAPs than among patients with non-significant CAPs, (2) the RDW levels were also higher in patients with predominantly NCP and MP than in those with CP and normal coronary arteries, (3) the RDW level was found as an independent predictor for the presence of significant CAPs, and (4) increased RDW levels were also significantly associated with either non-calcified or mixed morphology of CAPs. To our knowledge, this is the first study evaluating the relationship between RDW and both the severity and morphology of coronary atherosclerotic disease as detected by coronary CTA.

Previously, Lippi et al. (12) reported that proinflammatory biomarkers have been well correlated with the RDW levels independent from age, sex, Hb, and ferritin (12). Recent studies have also confirmed that RDW levels have closely had prognostic value in various cardiovascular diseases (13, 14). Additionally, another study has revealed that RDW might have a diagnostic importance in patients with acute coronary syndrome. This study has demonstrated that RDW has a sensitivity of 79% and a specificity of 50% for diagnosing acute coronary syndrome when the cut-off value is 14% (15). Recently, the association of RDW level with stable CAD has also received attention. Tonelli et al. (16) showed that RDW has been linked to major adverse cardiovascular outcomes in patients with

stable CAD with no concomitant heart failure symptoms. Isik et al. (17) also reported that RDW is significantly associated with the Syntax score as an indicator of the presence and complexity of stable CAD. Some studies have suggested that RDW is associated with cardiac syndrome X, atrial fibrillation, and heart failure (18-20).

The association of RDW with the severity of CAD has not been clarified yet. Among various hypotheses to explain the role of RDW in cardiovascular diseases, inflammation is accused at first (12) since recent findings have suggested that chronic low-grade ongoing inflammation plays a central role in atherogenesis (21). It is previously presented that increased proinflammatory markers, such as C-reactive protein and interleukin 6, have been correlated with the extent of lesions and the severity of CAD (21). Inflammation causes an increment of RDW levels by the suppression of erythropoietin gene expression and proliferation of erythroid progenitor cells, downregulation of erythropoietin receptor expression, or reduction of erythrocyte turnover (22). In addition, proinflammatory molecules block the maturation of erythrocytes, resulting in increased levels of RDW (23).

**Table 3.** Multinomial regression analysis demonstrating the association between cardiovascular risk factors and critical stenosis as detected in coronary computed tomography angiography

| Variables | OR (95% CI) | p |
|---|---|---|
| Age, years | 1.03 (1.01–1.05) | 0.016 |
| Male gender | 1.82 (1.08–3.04) | NS |
| Hypertension | 1.02 (0.59–1.76) | NS |
| Smoking | 2.52 (1.52–4.17) | <0.001 |
| Diabetes mellitus | 1.83 (1.01–3.32) | 0.047 |
| Serum creatinine | 1.76 (0.48–6.47) | NS |
| Triglyceride | 1.003 (1.000–1.007) | 0.049 |
| HDL–cholesterol | 1.01 (0.99–1.03) | NS |
| LDL–cholesterol | 0.99 (0.98–1.00) | NS |
| RDW | 1.40 (1.20–1.63) | <0.001 |

CI: confidence interval; HDL: high–density lipoprotein; LDL: low–density lipoprotein; NS: non–significant; OR: odds ratio; RDW: red blood cell distribution width

The composition of the CAP is an important predictor of the clinical progression and outcome in CAD. Plaques with abundant lipid and inflammatory cell content are more vulnerable to rupture and are often NCPs with low attenuation in coronary CTA (24). Previous studies showed that NCPs are also associated with the development of ACS more than the other plaque morphologies (25). Russo et al. (26) have demonstrated that among patients with suspected CAD, adverse cardiac outcomes are significantly higher in patients with NCPs and/or MPs than in patients with CPs. In our study, we have found that groups of patients with primarily CPs or normal coronary arteries had lower RDW levels than those of patients who primarily have NCPs or MPs. In addition, the RDW level is found to be an independent predictor for the presence of NCP or MP, but not CP after adjustment for other risk factors.

Our study has some limitations. First, adverse cardiovascular outcomes during follow-up have not been analyzed due to the cross-sectional and retrospective design of the study. Second, this was a single-center study; thus, our findings could not be generalized to whole populations. Third, levels of other parameters that may have effects on RDW including serum levels of iron, vitamin $B_{12}$, and folic acid have not been simultaneously measured in the present study.

## CONCLUSION

Our findings suggested that RDW as a simple, available and inexpensive biomarker was significantly associated with both the severity and vulnerable morphology of CAPs in patients undergoing coronary CTA. These data suggest that RDW might be an easily available biomarker for prediction of both the severity and mor-

**Table 4.** Multinomial regression analysis demonstrating the association between cardiovascular risk factors and the morphology of atherosclerotic plaque

| | Non–calcified plaque | | Mixed plaque | | Calcified plaque | |
|---|---|---|---|---|---|---|
| Variable | OR (95% CI) | p | OR (95% CI) | p | OR (95% CI) | p |
| Age, years | 1.07 (1.04–1.09) | <0.001 | 1.03 (1.01–1.06) | 0.016 | 1.06 (1.04–1.09) | <0.001 |
| Male gender | 2.18 (1.33–3.60) | 0.002 | 1.09 (0.57–2.10) | NS | 1.59 (0.87–2.89) | NS |
| LDL–cholesterol | 1.001 (0.996–1.007) | NS | 0.998 (0.991–1.006) | NS | 1.001 (0.994–1.008) | NS |
| HDL–cholesterol | 0.985 (0.969–1.000) | NS | 0.993 (0.974–1.013) | NS | 1.001 (0.984–1.019) | NS |
| Hypertension | 1.19 (0.74–1.91) | NS | 1.10 (0.59–2.05) | NS | 2.06 (1.11–3.83) | 0.023 |
| Diabetes mellitus | 1.45 (0.78–2.70) | NS | 1.59 (0.72–3.52) | NS | 1.75 (0.86–3.52) | NS |
| Smoking | 1.03 (0.64–1.67) | NS | 0.98 (0.51–1.88) | NS | 0.61 (0.32–1.16) | NS |
| RDW | 1.30 (1.08–1.57) | 0.006 | 1.47 (1.12–1.81) | <0.001 | 1.17 (0.93–1.48) | NS |

HDL: high–density lipoprotein; LDL: low–density lipoprotein; NS: non–significant; RDW: red blood cell distribution width

phology of coronary atherosclerotic disease in patients undergoing coronary CTA and may prove itself as a component of a scoring system for cardiovascular disease risk stratification.

**Ethics Committee Approval:** Ethics committee approval was received for this study from the ethics committee of Hacettepe University (protocol no.: HEK 08/181).

**Informed Consent:** Written informed consent was obtained from patients who participated in this study.

**Peer-review:** Externally peer-reviewed.

**Author Contributions:** Concept - M.U.Y., D.K., K.M.G.; Design - U.C., H.Y., A.H.A., T.H.; Supervision - H.Y., K.A., N.Ö., U.C.; Data Collection and/or Processing - U.C., M.U.Y., D.K., K.M.G.; Analysis and/or Interpretation - D.K., U.C., H.Y., T.H., K.A.; Literature Search - D.K., U.C., N.Ö., K.A.; Writing Manuscript - D.K., U.C., M.U.Y.; Critical Review - T.H., N.Ö., K.A., H.Y.

**Conflict of Interest:** The authors have no conflicts of interest to declare.

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