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ORIGINAL PAPER

Impact of white blood cell count on myocardial salvage, infarct size, and clinical outcomes in patients undergoing primary percutaneous coronary intervention for ST-segment elevation myocardial infarction: a magnetic resonance imaging study

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Abstract We sought to determine the relationship between white blood cell count (WBCc) and infarct size assessed by cardiovascular magnetic resonance imaging (CMR) in patients undergoing primary percutaneous coronary intervention (PCI) for ST-elevation myocardial infarction (STEMI). In 198 patients undergoing primary PCI for STEMI, WBCc was measured upon arrival and CMR was performed a median of 7 days after the index event. Infarct size was measured on delayed enhancement imaging and the area at risk (AAR) was quantified on T2-weighted images. Baseline characteristics were not significantly different between the high WBCc group (>11,000/mm^3, n = 91) and low WBCc group (≤11,000/mm^3, n = 107). The median infarct size was larger in the high WBCc group than in the low WBCc group [22.0 % (16.7–33.9) vs. 14.7 % (8.5–24.7), $p < 0.01$]. Compared with the low WBCc group, the high WBCc group had a greater extent of AAR and a smaller myocardial salvage index [MSI = (AAR−infarct size)/AAR × 100]. The major adverse cardiovascular events (MACE) including cardiac death, nonfatal reinfarction, and rehospitalization for congestive heart failure at 12-month occurred more frequently in the high WBCc group (12.1 vs. 0.9 %, $p < 0.01$). In multivariate analysis, high WBCc significantly increased the risk of a large infarct (OR 3.04 95 % CI 1.65–5.61, $p < 0.01$), a low MSI (OR 2.08, 95 % CI 1.13–3.86, $p = 0.02$), and 1-year MACE (OR 16.0, 95 % CI 1.89–134.5, $p = 0.01$). In patients undergoing primary PCI for STEMI, an elevated baseline WBCc is associated with less salvaged myocardium, larger infarct size and poorer clinical outcomes.

Keywords Myocardial infarction · White blood cell · Magnetic resonance imaging

Introduction

In patients with an acute myocardial infarction (AMI), elevated white blood cell count (WBCc) at presentation is associated with a worse angiographic appearance of the culprit lesions and an increased risk of adverse clinical outcomes [1]. A recent observational study showed that an elevated baseline WBCc is an independent predictor of mortality, major bleeding, and infarct size as assessed by cardiac enzyme levels [2]. Although previous studies found a strong relationship between high WBCc, infarct size, and subsequent adverse clinical outcomes [1, 3, 4], the causality and pathological mechanisms underlying these associations are still unknown.

In the setting of AMI, cardiovascular magnetic resonance imaging (CMR) is useful in the accurate assessment of myocardial edema, infarcted tissue, microvascular obstruction (MVO), and myocardial hemorrhage [5]. In

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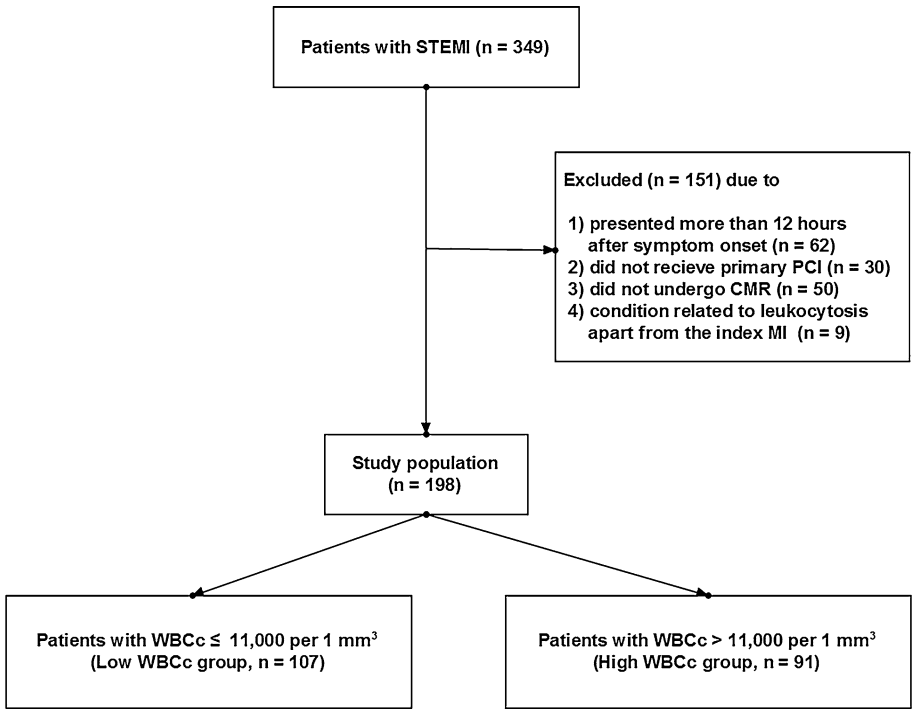

Fig. 1 Study subjects. Schematic of selection of study cohort

addition, CMR allows for quantification of the extent of salvaged myocardium, which is defined as the myocardium at risk for irreversible injury as indicated by the presence of acute myocardial edema by T2-weighted image (T2W) but negative for delayed enhancement image, after primary percutaneous coronary intervention (PCI) [6]. Although 1 previous CMR study reported that neutrophil count independently predicted large infarctions, other valuable information such as myocardial salvage or MVO was not assessed [7].

In the present study, we aimed to assess the relationship between WBCc at presentation, myocardial salvage, and infarct size as determined by CMR in patients undergoing primary PCI for ST-elevation myocardial infarction (STEMI). Furthermore, we evaluated the impact of higher WBCc on clinical outcomes.

Methods

Study population

From January 2006 to November 2009, a total of 349 STEMI patients presented to Samsung Medical Center, Seoul, Korea. Patients were eligible if they had (1) chest pain for less than 12 h after symptom onset; (2) ST-segment elevation of more than 1 mm in two or more contiguous leads or a presumably new-onset left bundle branch block on electrocardiogram; and (3) had undergone successful primary PCI and CMR. All patients met the following criteria: no contraindication to CMR, no condition related to leukocytosis apart from the index MI, and no prior history of MI. In total, 198 patients were enrolled in this study and followed prospectively (Fig. 1). Baseline clinical data including past medical history, the presence of risk factors, medications, angiographic/procedural data, and clinical outcomes were recorded prospectively by the research coordinators of the dedicated registry. This study was approved by the local institutional review board. Informed consent was obtained from all subjects.

Blood samples

Blood samples were taken for routine WBC measurement on admission, before primary PCI (Sysmex XE-2100; TOA Medical Electronics Co., Kobe, Japan).

Percutaneous coronary intervention

All patients were given a loading dose of aspirin (300 mg) and clopidogrel (300 or 600 mg) before PCI. Coronary angiography and stent implantation were performed using standard interventional techniques [8]. The decision to use glycoprotein IIb/IIIa receptor inhibitors was made by individual operators. All baseline and procedural cine coronary angiograms were reviewed and analyzed quantitatively at the angiographic laboratory of our institution. Myocardial blush grade (MBG) was evaluated using the final angiogram, as described previously [9]. After PCI, aspirin (100–200 mg daily) was continued indefinitely and

clopidogrel (75 mg daily) for at least 1 year was recommended.

CMR protocol

CMR was performed using a 1.5-T scanner (Achieva, Philips Medical Systems, Best, Netherlands) with a SENSE cardiac coil according to our laboratory protocol [10]. Images were acquired using electrocardiographic gating and expiratory breath holds. The CMR protocol consisted of cine, T2W, first-pass perfusion, and late-gadolinium enhancement (LGE) imaging. Cine imaging was carried out based on balanced steady-state free precession sequences along the long and short axes from the apex to the base of the left ventricle (LV). Next, T2Ws were acquired in the cardiac short-axis direction using a dark-blood T2W inversion-recovery fast-spin echo sequence. First-pass perfusion imaging was obtained with the T1-weighted dynamic sequence (turbo field echo with SENSE, repetition time/echo time, 2.6/1.3 ms) after intravenous infusion of gadolinium-diethylenetriamine pentaacetic acid (Gd-DTPA, Magnevist, Bayer Schering Pharma, Berlin, Germany; 0.15 mmol/kg body weight in total amount at 3 mL/sec). The slice thickness was 6 mm with a field-of-view of 40 cm × 40 cm and an image matrix of 128 × 128. Images of 4 locations for every 2 heart beats were acquired for 40 phases. LGE and the extent of MVO were assessed 5, 10, and 15 min after Gd-DTPA administration in contiguous 10–12 slices of 6 mm thickness with a 4-mm interslice gap by use of a multi-shot turbo field echo breath-hold sequence with a non-selective inversion (typical repetition time/time to echo, 4.6/1.4 ms). The field-of-view and image matrix were 35 cm × 35 cm and 256 × 256, respectively. The inversion delay time was varied in a range of 200–300 ms. A Look-Locker sequence was used to determine optimal inversion time.

CMR analysis

The CMR images were analyzed using validated software (ARGUS, Siemens Medical System, Erlangen, Germany) at our MRI core laboratory by two experienced radiologists who were blinded to the clinical information of the patient. After acquiring the short-axis images at the end of diastole and the end of systole, endocardial borders were traced manually. LV end-diastolic volume, end-systolic volume, and ejection fraction were calculated using the Simpson rule. The infarct volume was quantified from the sum of the area with LGE within each segment of the short-axis images multiplied by the slice thickness to cover the entire LV. The infarct area was traced by the visual border detection using manual drawing method using commercialized analysis software. Interobserver and intraobserver variability of infarct size in our laboratory (intraclass correlation coefficient) is 0.82 and 0.87 as previously reported [11]. The extent of MVO was calculated in the same manner. The endocardial and epicardial borders were planimetered to calculate myocardial area and summed to calculate LV myocardial volume. The percent infarct volume was expressed as percentage of LV myocardial volume. T2Ws were used to determine the presence of myocardial hemorrhage [12]. The area at risk (AAR) was quantified on T2Ws by using a similar algorithm as above and expressed as percentage of LV myocardial volume. Myocardial salvage index (MSI) was computed as follows: MSI = (AAR−infarct size)/AAR × 100 [6]. The infarct transmurality of each segment was calculated by dividing the LGE area by the total area of the affected myocardium in each segment. The transmural extent of infarction was expressed as the sum of segments with >75 % of infarct transmurality.

End points

The primary objective was to compare myocardial infarct size assessed by CMR according to baseline WBCc (WBCc > 11,000 vs. ≤11,000 per 1 mm^3). The cutoff value of WBCc was determined in reference to previous studies [2, 13]. The secondary objectives included (1) AAR, MSI, extent of MVO, number of segments with >75 % of infarct transmurality, and the presence of myocardial hemorrhage as assessed by CMR, (2) the incidence of stent thrombosis and the composite of major adverse cardiovascular events (MACE), including cardiac death, nonfatal reinfarction, and rehospitalization for congestive heart failure at the 12-month follow-up. Stent thrombosis was assessed based on the definitions of the Academic Research Consortium [14]. All deaths were considered cardiac unless a definite non-cardiac cause could be established. Reinfarction was defined as elevated cardiac enzymes (troponin or MB fraction of creatine kinase, CK-MB) greater than the upper limit of the normal value with ischemic symptoms or electrocardiography findings indicative of ischemia that were not related to the index procedure. Rehospitalization for congestive heart failure was defined as hospitalization due to exacerbation of congestive heart failure occurring after discharge.

Statistical analysis

Continuous variables are expressed as the mean ± SD or the median and interquartile range and were compared using the independent *t* test or Wilcoxon rank sum test. Categorical variables were compared with Pearson's Chi square or Fisher's exact tests. Multivariate logistic regression analysis was performed with a stepwise,

Table 1 Baseline characteristics of the study patients

Variable	WBC > 11,000 (n = 91)	WBC ≤ 11,000 (n = 107)	*p* value
Age (y)	56 (47–66)	58 (51-68)	0.07
Age ≥65 years	26 (28.6)	36 (33.6)	0.44
Male	83 (91.2)	89 (83.2)	0.10
Body mass index (kg/m^2)	24.0 ± 2.9	24.8 ± 2.8	0.04
Current smoker	66 (72.5)	52 (48.6)	<0.01
Diabetes mellitus	20 (22.0)	26 (24.3)	0.70
Hypertension	26 (28.6)	38 (35.5)	0.30
Dyslipidemia	20 (22.0)	26 (24.3)	0.70
Chronic renal failure	4 (4.4)	7 (6.6)	0.50
Previous PCI	5 (5.5)	2 (1.9)	0.17
Killip class ≥2	16 (17.6)	13 (12.1)	0.28
Anterior infarction	54 (59.3)	58 (54.2)	0.47
LVEF < 40 %	16 (17.6)	8 (7.5)	0.03
Glucose	142 (123–183)	128 (111–159)	0.03
hsCRP (mg/L)	0.16 (0.08–0.62)	0.13 (0.07–0.41)	0.10
NT-proBNP (pg/mL)	115 (43–456)	102 (33–349)	0.26
Peak CK-MB (ng/mL)	225 (106–317)	146 (56–257)	<0.01
Door-to-balloon time (min)	86 (69–108)	87 (63–121)	0.87
Symptom-to-balloon time (min)	280 (171–560)	265 (152–399)	0.12
600 mg loading dose of clopidogrel	50 (54.9)	67 (62.6)	0.27
Medication after PCI			
Aspirin	91 (100)	106 (99.1)	>0.99
Clopidogrel	88 (96.7)	103 (96.3)	>0.99
Beta-blocker	77 (84.6)	94 (87.9)	0.51
Statins	85 (93.4)	100 (93.5)	0.99
ARB or ACEI	53 (58.2)	74 (69.2)	0.11

Continuous variables are expressed as median (interquartile range) or mean ± SD, categorical variables as n (%)

PCI percutaneous coronary intervention, *LVEF* left ventricular ejection fraction, *ARB* angiotensin II receptor blocker, *ACE* angiotensin converting enzyme inhibitor

backward selection process to determine the independent predictors of a large infarct (percent infarct volume > median infarct size in the present study), a low MSI (MSI < median MSI in the present study), and MACE. Age, sex, diabetes, high baseline WBCc, anterior myocardial infarction, aspiration thrombectomy during PCI, angiographic no-reflow, and a loading dose of 600 mg of clopidogrel were included in the multivariate logistic regression model. The criteria for the entry and removal of variables were set at 0.05 and 0.10, respectively. A value of $p < 0.05$ in the two-tailed test was considered significant. Statistical analysis was performed with the SPSS 17.0 statistical package (SPSS Inc., Chicago, Illinois).

Results

Patient characteristics

The clinical characteristics of the patients stratified by baseline WBCc are shown in Table 1. Compared to patients with WBCc ≤ 11,000 per 1 mm^3 (low WBCc group), those with WBCc > 11,000 per 1 mm^3 (high WBCc group) were more likely to be smokers, have a lower body mass index, a higher glucose level at admission, and a higher peak CK-MB level. The incidence of an LV ejection fraction of less than 40 % was significantly higher in the high WBCc group than in the low WBCc group. Other baseline clinical characteristics were not different according to WBCc.

Angiographic and procedural data

Angiographic and procedural variables stratified by baseline WBCc are shown in Table 2. There were no statistically significant differences between the two groups except for stent length. The most frequent culprit vessel was the left anterior descending artery in both groups. The baseline TIMI flow grade was 0 or 1 in most patients.

CMR analysis

The results of CMR are presented in Table 3. Figure 2 shows a representative CMR of a reperfused anterior STEMI with high WBCc versus low WBCc. CMR was performed at a median of 7 days after the index event [interquartile range (IQR), 4–15 days]. There was no difference in the interval from procedure to CMR between the groups [7 days (IQR, 4–15) in the high WBCc group versus 7 days (IQR, 3–16) in the low WBCc group, $p = 0.98$]. LV ejection fraction was significantly lower in the high WBCc group than in the low WBCc group. Patients with high WBCc had a significantly larger AAR than those with low WBCc. The median infarct size was significantly larger in the high WBCc group compared with the low WBCc group. Moreover, MSI was significantly lower in the high WBCc group than the low WBCc group. The extent of MVO and the number of segments with a >75 % infarct transmurality were significantly greater in the high WBCc group compared with the low WBCc group. Myocardial hemorrhage was detected more frequently in the high WBCc group than in the low WBCc group. In addition, WBCc was related to infarct size by univariate linear regression analysis. (β ± SE 0.841 ± 0.269, $p \leq 0.01$).

Table 2 Angiographic and procedural findings

Variable	WBC > 11,000 (n = 91)	WBC ≤ 11,000 (n = 107)	*p* value
Culprit vessel			0.28
Left anterior descending artery	54 (59.3)	58 (54.2)	
Left circumflex artery	7 (7.7)	16 (15.0)	
Right coronary artery	30 (33.0)	33 (30.8)	
Number of diseased vessels			0.75
1	51 (56.0)	60 (56.1)	
2	25 (27.5)	33 (30.8)	
3	15 (16.5)	14 (13.1)	
Baseline TIMI flow grade			0.60
0/1	77 (84.6)	81 (77.6)	
2	5 (5.5)	10 (9.3)	
3	9 (9.9)	14 (13.1)	
Final TIMI flow grade			0.44
0/1	2 (2.4)	1 (0.9)	
2	3 (3.7)	5 (4.3)	
3	76 (93.8)	111 (94.9)	
Angiographic no-reflow	10 (11.0)	11 (10.3)	0.87
Final MBG 2 or 3	54 (59.3)	75 (70.1)	0.11
Thrombus aspiration	32 (35.2)	31 (29.0)	0.35
Glycoprotein IIb/IIIa inhibitor	18 (19.8)	20 (18.7)	0.87
Type of stents			0.55
No stenting	7 (7.7)	6 (5.6)	
Bare-metal stents	12 (13.2)	10 (9.3)	
Drug-eluting stents	72 (79.1)	91 (85.0)	
Stent diameter (mm)	3.3 ± 0.4	3.3 ± 0.4	0.73
Stent length (mm)	23.2 ± 5.8	24.9 ± 6.1	0.04

TIMI thrombolysis in myocardial infarction, *MBG* myocardial blush grade

Table 3 Results of CMR

Variable	WBC > 11,000 (n = 91)	WBC ≤ 11,000 (n = 107)	*p* value
LVEDV (mL)	137 (106–157)	124 (105–143)	0.09
LVESV (mL)	73 (48–95)	58 (44–72)	<0.01
LV mass (g)	131 (109–146)	123 (100–143)	0.24
LV ejection fraction (%)	46 (38–54)	54 (45–62)	<0.01
Infarct size (% of LV)	22.0 (16.7–33.9)	14.7 (8.5–24.7)	<0.01
AAR (% of LV)	63.3 (47.6–76.5)	52.3 (39.3–70.4)	0.01
Myocardial salvage index	36.7 (23.5–52.4)	47.7 (29.6–60.7)	0.01
Hemorrhagic infarction, n (%)	59 (64.8)	49 (45.8)	<0.01
MVO area (% of LV)	1.3 (0–4.2)	0.9 (0–2.4)	0.03
Number of segments with >75 % of infarct transmurality	5 (3–7)	3 (2–5)	<0.01

LVEDV left ventricular end-diastolic volume, *LVESV* left ventricular end-systolic volume, *AAR* area at risk, *MVO* microvascular obstruction

Multivariate analysis showed that the independent predictors of a large infarct (>18.6 % of median infarct size) were an elevated baseline WBCc, aspiration thrombectomy during PCI, and angiographic no-reflow [odds ratio (OR) 3.04, 95 % confidence interval (CI) 1.65–5.61, $p < 0.01$; OR 0.20, 95 % CI 0.23–0.89, $p = 0.02$; OR 3.54, 95 % CI 1.17–10.7, $p = 0.03$, respectively]. Moreover, high baseline WBCc, anterior myocardial infarction, angiographic no-reflow, and a loading dose of 600 mg of clopidogrel were the independent predictors of a low MSI (<42.2 of median MSI) in multivariate analysis (OR 2.08, 95 % CI 1.13–3.86, $p = 0.02$; OR 2.54, 95 % CI 1.32–4.89, $p < 0.01$; OR 3.03, 95 % CI 1.04–8.83, $p = 0.04$; OR 0.40, 95 % CI 0.21–0.75, $p = 0.04$, respectively).

Clinical outcomes

At the 12-month follow-up, there had been four cardiac deaths (4.4 %) in the high WBCc group and none in the low WBCc group ($p = 0.08$). Nonfatal reinfarction and rehospitalization for congestive heart failure occurred in a similar pattern between the 2 groups (5.5 vs. 0.9 %, $p = 0.16$, and 3.3 vs. 0 %, $p = 0.15$, respectively). The composite MACE rates at the 12-month follow-up were higher in the high WBCc group than in the low WBCc group (12.1 vs. 0.9 %, $p < 0.01$). Multivariate analysis showed that high WBCc was an independent predictor of a 1-year MACE (OR 16.0, 95 % CI 1.89–134.5, $p = 0.01$).

Myocardial blush grade and CMR findings

Since we evaluated MBG after PCI, we analyzed post PCI MBG (MBG 0/1 versus MBG 2/3) and the correlation with WBCc and CMR findings. WBCc was not different between MBG 0/1 group and MBG 2/3 group [10,970/m^3 (8,485–13,575) vs. 10,380/m^3 (8,640-12,530), $p = 0.23$). The rates of WBCc ≥ 11,000/m^3 was numerically higher in MBG 0/1 group than in MBG 2/3 group, but it was not statistically significant (53.6 vs. 41.9 %, $p = 0.11$). The median infarct size [22.0 % (16.7–33.9) vs. 17.0 %

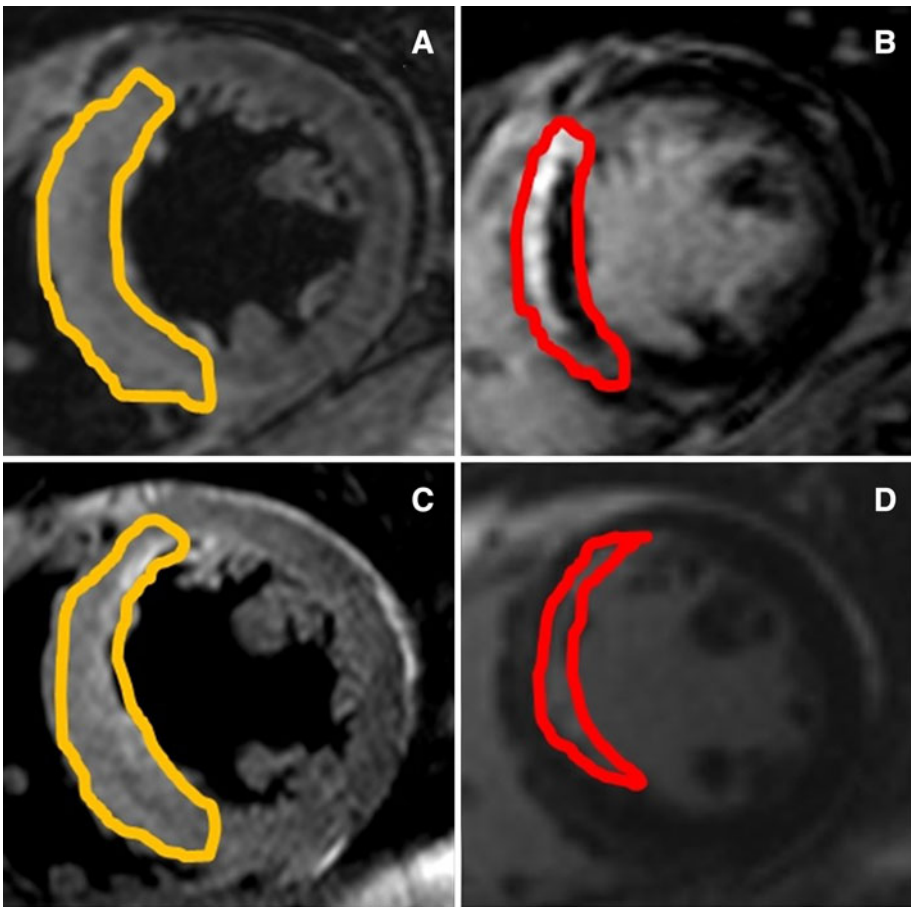

Fig. 2 A representative CMR of a reperfused anterior ST-elevation myocardial infarction with high WBCc (**a**, **b**) versus low WBCc (**c**, **d**); short-axis slices of a T2-weighted image (**a**, **c**) and the corresponding late-gadolinium enhancement image (**b**, **d**). In these cases, the extent of area at risk and of infarct size were 46.5 versus 28.2 % and 34.1 versus 15.0 %, respectively, yielding myocardial salvage index of 26.7 versus 46.8

[9.1–26.9], $p = 0.01$) and MVO area [1.3 % (0–4.2) vs. 0.8 % (0–2.4), $p = 0.04$] were larger in MBG 0/1 group than in MBG 2/3 group, respectively. Compared with MBG 2/3 group, the MBG 0/1 group showed tendencies of a greater extent of AAR [63.3 % (47.6–76.5) vs. 53.0 % (40.2–72.7), $p = 0.06$] and a smaller MSI [36.7 (23.5–52.4) vs. 47.0 (10.2–72.7), $p = 0.06$]. Number of segments with ≥75 % of infarct transmurality was greater in MBG 0/1 group than in MBG 2/3 group (5 [3–7] versus 4 [2–5], $p = 0.03$). However, the MACE rates did not differ according to the MBG (7.2 vs. 5.4 %, $p = 0.76$).

Discussion

The salient findings of this study are as follows: (1) an elevated baseline WBCc in patients with STEMI who undergo primary PCI is associated with a larger extent of myocardial edema (AAR), less myocardial salvage, and larger infarct size as assessed by CMR; (2) patients with an elevated baseline WBCc have poor midterm clinical outcomes compared to those without leukocytosis in the setting of STEMI.

WBC count, myocardial salvage, and final infarct size

The results of the present study correspond well with those of earlier studies that established an association between elevated WBCc and infarct size as assessed by cardiac enzymes[1, 15, 16], single photon emission computed tomography [17], and CMR [7]; however, the causality and pathological mechanisms underlying these associations have not been fully elucidated. In the setting of AMI, CMR can identify the pathological consequences of reperfusion strategies in vivo and provide more information such as infarct related myocardial edema (so-called AAR) by T2W, acute irreversible infarcted myocardium by LGE image, and the extent of salvaged myocardium [18–20]. In the present study, elevated WBCc at the time of presentation of STEMI was associated with a larger AAR assessed by T2W, which suggests that a greater extent of ischemia-induced inflammation promotes leukocytosis. On the other hand, salvaged myocardium was also significantly reduced in the high WBCc group. These findings support the contention that leukocytosis may play a significant role in infarct expansion. After an AMI, the release of chemoattractants draws neutrophils into the infarct zone during the

first 6 h of myocardial reperfusion, and during the next 24 h they migrate into the myocardial tissue [21]. Neutrophil infiltration is regulated through a complex sequence of molecular steps involving the selectins and the integrins, which mediate leukocyte rolling and adhesion to the endothelium [22]. These neutrophils cause proteolytic and oxidative damage to the endothelial cells, plug the microvasculature, and induce hypercoagulability and may promote infarct expansion [21–23]. In patients with STEMIs undergoing primary PCI, a high neutrophil count at presentation is associated with more severe microvascular dysfunction after primary PCI [24]. Of note, our study showed that the extent of MVO and hemorrhagic infarction was significantly greater in patients with high WBCc. Therefore, our findings suggest that a higher WBCc is not only a consequence of wider myocardial damage, but also directly responsible for increased infarct size by inducing infarct expansion. However, it should be noted that these are hypothesis-generating findings. Experimental data have demonstrated that reductions in infarct size are observed after the depletion or pharmacologic inhibition of neutrophils, supporting the primary role of these cells in myocardial and microvascular injury [21, 22].

WBC count and clinical outcomes

Prior studies have reported conflicting findings regarding the association between WBCc and mortality in patients with STEMI undergoing primary PCI [16, 25–27]. A recent large-scale study showed that an elevated baseline WBCc is an independent predictor of infarct size, as assessed by peak CK-MB level, and of 1-year cardiac mortality, noncardiac mortality, and major bleeding in STEMI patients treated by primary PCI [2]. Another study using CMR to assess infarct size in STEMI patients reported that neutrophil count independently predicts large infarctions and MACE [7]. The findings of the present study are consistent with these recent studies. Collectively, wider myocardial damage and infarct expansion induced by elevated baseline WBCc might lead to poor clinical outcomes.

Myocardial blush grade and CMR findings

Successful microcirculatory reperfusion, defined as MBG 2 or 3, is associated with smaller enzymatic infarct size [28]. Recent studies comparing infarct size measured by CMR in AMI patients showed that MBG 2/3 was associated with reduction of infarct size and MVO [29] or infarct transmurality [30]. In the present study, MBG 2/3 was achieved in 65.2 % of patients and associated with smaller infarct size, less MVO and lower infarct transmurality. The results of our study correspond with prior studies. These data might provide the mechanistic link between MBG and mortality [31].

Study limitations

There are several limitations to this study. First, this was a nonrandomized, observational study, which may have significantly affected the results due to confounding factors. Second, information on the WBC subtypes was not available. Nevertheless, a prior study suggested that total WBCc is correlated better with long-term prognosis than WBC differential count [32]. Third, serial data on WBCc were not available. It has been reported that neutrophil count at 12 h after revascularization independently predicted large infarctions. Although serial measurements can increase the predictive power of WBCc, it is more practical and feasible to measure WBCc on admission. Fourth, despite that there was no difference in the interval from procedure to CMR between the groups, but infarct size may vary significantly if measured 3–45 days post PCI. Lastly, low event rate in our study population and the wide C.I suggest multivariate model over fitting and does not provide convincing evidence toward poorer outcomes.

Conclusions

The present study shows that an elevated baseline WBCc in patients with STEMI undergoing primary PCI is associated with less myocardial salvage and larger infarct size as assessed by CMR and higher 1-year MACE rates. WBCc on admission is a marker of infarct severity, and furthermore, can aggravate infarcts per se. The potential therapeutic implications of these conclusions deserve further investigation.

Conflict of interest None.

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