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Circulating endothelial cells in coronary artery disease

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Abstract

Background: Endothelial damage and dysfunction play a crucial role in the pathophysiology of coronary artery disease (CAD). The quantification of circulating endothelial cells (CEC) in the peripheral blood is a novel method for assessing endothelial damage.

Aim: To evaluate the possible diagnostic use of single quantification of CEC in peripheral blood by flow cytometry in patients with CAD.

Methods: We examined 48 patients with CAD, including 23 patients with acute myocardial infarction (AMI) and 25 patients with stable angina (SA). The control group consisted of 20 healthy subjects without symptoms of CAD. The CEC count was evaluated by flow cytometry using antibodies against CD31, CD146, and CD45. Plasma biochemical markers of endothelial damage (von Willebrand Factor [vWF], thrombomodulin [TM]) were measured by ELISA. Serum concentrations of troponin I (TnI) and lipid parameters were also included in the statistical analysis.

Results: A significant increase in the CEC count was found in patients with AMI compared to the control group ($p < 0.05$) and SA patients ($p < 0.05$). However, no difference was found in the CEC count between patients with SA and the control group. Increased vWF activity was found in both groups of CAD patients compared to the control group (AMI: $p < 0.001$, SA: $p < 0.01$), and vWF activity was significantly higher in AMI patients compared to SA patients ($p < 0.001$). Thrombomodulin concentration did not differ significantly between any patient groups and the control group. The CEC count correlated positively with vWF activity ($r = 0.3852$, $p < 0.05$) and the atherogenic index TC/HDL-C ($r = 0.3844$, $p < 0.05$) in all patients with CAD (AMI + SA). The sensitivity of CEC count for the diagnosis of an acute coronary syndrome was lower than that of TnI level on admission (39% *vs* 69%).

Conclusions: We confirmed that CEC count in peripheral blood can be determined by flow cytometry in CAD patients with both AMI and SA. The CEC count in AMI was increased in comparison to healthy subjects and SA patients in one third of all cases. To determine whether CEC count could be used to improve the diagnosis of an acute coronary syndrome in patients with CAD, additional studies in larger patient groups would be required.

Key words: coronary artery disease, endothelium, circulating endothelial cells, von Willebrand factor

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INTRODUCTION

Endothelial dysfunction and damage play an important role in the development of cardiovascular disease [1–3]. Endothelial damage results in shedding of endothelial cells from the vessel wall. These shed cells are present in blood and have been named circulating endothelial cells (CEC) [1].

Endothelial cell shedding from the vessel wall results from apoptosis of these cells, subendothelial matrix proteolysis, and

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mechanical as well as drug-induced endothelial damage. In inflammatory conditions, this is related to the effects of cytokines, proteases, and complement-dependent neutrophil attack [4–6].

Circulating endothelial cells may also originate from bone marrow precursor cells. The precursor for vascular endothelial cells is the haemangioblast, located in the bone marrow stroma, which is also a precursor cell for haematopoietic cell lines. Upon action of angiogenic factors, haemangioblasts differentiate into multipotential angioblasts which are further transformed into endothelial progenitor cells. The latter migrate to the vascular zone, differentiate and populate the endothelium [7].

For the purpose of CEC determination, immunomagnetic isolation and flow cytometry techniques are used. Endothelial antigens (von Willebrand factor [vWF], CD31, CD146) are used for identification of CEC. A marker differentiating CEC and haematopoietic cell lines is the CD45 antigen, which is absent in cells of the endothelial line [8, 9].

The number of CEC in the peripheral blood of healthy subjects is small. Increased number of CEC was reported in conditions leading to endothelial damage including cardiovascular disease and inflammatory vascular disease [8, 9]. Laboratory markers of endothelial dysfunction and damage are increasingly used in the identification of early stages of cardiovascular disease. Among these novel markers, CEC present in the peripheral blood are of special interest [6, 8, 10, 11].

The aim of this study was to evaluate the possible diagnostic use of single quantification of CEC in peripheral blood by flow cytometry in patients with coronary artery disease (CAD). We also assessed relationships between the number of CEC and biochemical markers of endothelial damage, myocardial damage markers, and conventional risk factors for CAD.

METHODS

Study group

We studied 48 patients with CAD including 23 patients with a diagnosis of an acute myocardial infarction (AMI) on admission and 25 patients with stable angina pectoris (SA). The control group consisted of 20 healthy subjects without symptoms of CAD. The study was approved by the local Ethics Committee.

Laboratory testing

The CEC count was determined in EDTA venous blood by flow cytometry using a Dako flow cytometer. The absolute CEC numbers in 1 μL of blood were calculated using percentage CEC counts and absolute leukocyte numbers. Antibodies against CD31, CD146, and CD45 (BD Biosciences) were used to isolate CEC. Biochemical markers of endothelial damage, including vWF activity and thrombomodulin (TM) level, were determined in citrate venous blood plasma using ELISA method, Multiscan Ex microplatelet reader (Labsystems), and Asserachrom vWF (Roche Diagnostics Poland) and Thrombomodulin ELISA Kit (American Diagnostica Inc.) reagent kits. Serum total cholesterol (TC) and triglyceride (TG) levels were determined using an enzymatic method, and HDL cholesterol (HDL-C) level using a direct method. LDL cholesterol (LDL-C) level was calculated using the Friedewald formula. Troponin I (TnI) level was determined with the immunochemical method using the ARCHITECT STAT Troponina-I reagent kit and ARCHITECT c8000 System biochemical analyser (Abbott Laboratories). Sensitivity for CEC count and TnI level in patients with AMI was calculated as the ratio of values above the diagnostic threshold to all values in the study group. The diagnostic thresholds for the diagnosis of AMI were TnI level > 0.3 ng/mL and CEC count > 5/μL. Analyses were performed in blood samples collected 4–9 hours after the onset of chest pain.

Statistical analysis

Statistical analysis was performed using STATISTICA 8 software (StatSoft). Mann-Whitney U test was used to evaluate significance of differences in parameters between the study and control group. Correlations between parameters were tested using Spearman correlation coefficient. Differences in the sensitivity of diagnostic parameters and the prevalence of cardiovascular disease risk factors were tested using a two-fraction test. Parameters are presented as median values and quartiles (Q_{25}-Q_{75}). A p value < 0.05 was considered significant.

RESULTS

Clinical characteristics of patients with AMI, SA, and controls is shown in Table 1. Men comprised the majority of both AMI and SA patients, and the mean age of patients in the two groups did not differ significantly. The ST segment elevation myocardial infarction was diagnosed in most patients with AMI. The SA group included patients with CCS class II angina. Table 1 also shows myocardial necrosis marker levels, lipid parameters, and the prevalence of cardiovascular risk factors.

We showed increased CEC count in AMI patients compared to SA and control patients. The CEC count in SA patients did not differ significantly compared to the control group (Table 2).

Table 2 also shows endothelial damage marker levels (vWF and TM) in the study groups. We found increased vWF activity in AMI and SA patients compared to the control group. vWF activity was significantly higher in AMI patients compared to SA patients. The TM level in both groups of patients did not differ significantly in comparison to the control group.

We found a positive correlation between CEC count and vWF activity and atherogenic index TC/HDC-C in the whole group of patients with CAD (Fig. 1A, B).

The only atherogenic serum lipid abnormalities seen in both AMI and SA patients compared to the control group were increased TC/HDL-C (AMI: $p < 0.001$, SA: $p < 0.01$) and low HDL-C (AMI and SA: $p < 0.001$; Table 1).

Among patients with CAD, we found no significant differences in CEC count between patients with or without car-

Table 1. Clinical characteristics of patients with acute myocardial infarction (AMI), stable angina pectoris (SA) and control subjects

Parameter	AMI (n = 23)	SA (n = 25)	Controls (n = 20)
Gender: men/women	18 (78%)/5 (22%)	18 (72%)/7 (28%)	9 (45%)/11 (55%)
Age [years]	61 ± 12	63 ± 9	56 ± 14
STEMI/NSTEMI	19 (83%)/4 (17%)		
CCS class II angina		25 (100%)	
Troponin I [ng/mL]	1.027 (0.294–6.292)		
CK-MB [U/L]	37 (29–52)		
Body mass index > 30 kg/m^2	6 (23%)*	4 (16%)	0 (0%)
Hypertension	14 (61%)*	10 (40%)*	1 (5%)
Diabetes	4 (17%)	3 (12%)	0 (0%)
Cigarette smoking	11 (47%)	8 (32%)	4 (20%)
Cholesterol > 200 mg/dL	12 (52%)$^{\#}$	5 (20%) *	10 (50%)
Triglycerides > 200 mg/dL	2 (9%)	2 (8%)	0 (0%)
Total cholesterol [mg/dL]	202 (171–268)$^{\#}$	162 (144–193)*	226 (197–235)
LDL cholesterol [mg/dL]	137 (112–180)$^{\#}$	100 (89–119)*	140 (118–146)
HDL cholesterol [mg/dL]	42 (35–49)*	37 (34–43)*	61 (51–72)
Triglycerides [mg/dL]	92 (61–133)$^{\#}$	116 (100–151)	99 (84–130)
TC/HDL-C	5,3 (4.2–6.1)*$^{\#}$	4.3 (4–4.7)*	3.3 (2.7–4.1)

STEMI — ST segment elevation myocardial infarction; NSTEMI — non-ST segment elevation myocardial infarction; CCS — Canadian Cardiovascular Society; TC — total cholesterol; HDL-C — HDL cholesterol; CK-MB — creatinine kinase izoenzyme MB; *p < 0.05 *vs* control group; $^{\#}$p < 0.05 AMI group *vs* SA group

Table 2. Circulating endothelial cells (CEC) count, von Willebrand factor (vWF) activity, and thrombomodulin (TM) level in patients with acute myocardial infarction (AMI), stable angina pectoris (SA) and control subjects

Laboratory parameter	AMI (n = 23)	SA (n= 25)	Control group (n = 20)	P
CEC [count/μL]	4.4 (1.8–7.2)	2.4 (1.4–3.3)	2.4 (1.5–3.9)	< 0.05*; < 0.05$^{\#}$
vWF [%]	176 (146–195)	122 (108–131)	89 (69–116)	< 0.001*; < 0.01**; < 0.001$^{\#}$
TM [ng/mL]	2.98 (2.63–3.44)	3.02 (2.70–3.30)	2.80 (2.49–3.03)	NS*; NS**; NS$^{\#}$

*Difference between AMI and control group; **difference between SA and control group; $^{\#}$difference between AMI and SA; NS — not significant

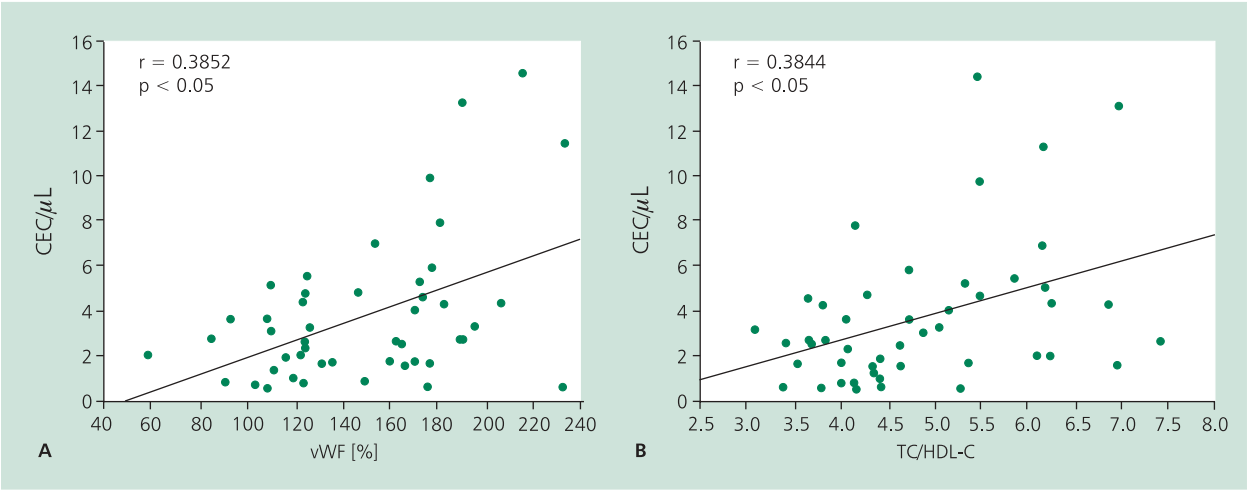

Figure 1. Correlation between circulating endothelial cells (CEC) count and von Willebrand factor (vWF) activity (**A**) and the atherogenic index TC/HDL-C (**B**) in patients with coronary artery disease (n = 48)

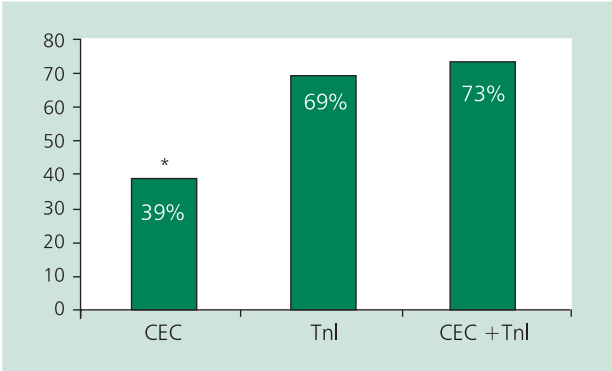

Figure 2. Sensitivity of circulating endothelial cells (CEC) count and troponin I (TnI) level in identifying patients with acute myocardial infarction (n = 23); *p < 0.05 for CEC *vs* TnI and CEC + TnI

diovascular risk factors (obesity, high blood pressure, diabetes, cigarette smoking).

In AMI patients, elevated CEC count was defined as > 5/μL. This threshold values corresponds to the highest values seen in SA patients and controls. With this diagnostic threshold, sensitivity of CEC count in the diagnosis of an acute coronary syndrome (ACS) was 39% (elevated CEC count in 9 of 23 AMI patients). Sensitivity of TnI level on admission was 69% (with the diagnostic threshold of 0.3 ng/mL). When an elevated value of at least one of these parameters (CEC count and/or TnI level) was taken into an account, sensitivity of the diagnosis of an ACS was increased to 73% (Fig. 2).

DISCUSSION

In our study group of patients with CAD, single quantification of CEC in peripheral blood by flow cytometry showed a relatively large CEC count only in patients with AMI but not in patients with SA. This finding suggests endothelial damage resulting in loss of its integrity during an ischaemic event. Previously published reports showed increased CEC count in the entire clinical spectrum of CAD except for SA [4, 6, 10, 12]. Literature data suggest that the most probable cause of increased presence of CEC in peripheral blood is coronary plaque rupture with endothelial cell shedding [6, 11].

Other endothelial damage markers evaluated in our study did not differentiate endothelial dysfunction and biochemical damage from more pronounced disruption leading to the loss of endothelial cell integrity during ischaemic events. A high vWF activity indicated endothelial dysfunction or damage in both groups of patients and only showed that these abnormalities are more pronounced in AMI than in SA. We found a positive correlation between vWF activity and CEC count in the whole group of patients with CAD which is in accordance with data presented by other authors [13–15].

The least useful endothelial damage marker was the TM level which did not differ significantly between any of the groups of patients with CAD and the control group. Normal TM level with increased vWF activity and CEC count was also reported in patients with chronic heart failure [16].

The usefulness of CEC count as a marker of endothelial damage related to plaque rupture suggests its possible use as a marker of an ACS. In our study, CEC count was less sensitive (39%) than TnI level (69%) when determined 4–9 hours from the onset of AMI. When an elevated value of any of these parameters (CEC count and/or TnI level) was taken into an account, sensitivity increased only slightly to 73%.

Quilici et al. [17] showed increased CEC count in 53% of patients with a non-ST segment ACS, and increased TnI level in 61.7% of these patients. In that study, increase in CEC count occurred prior to elevation of TnI level, and when an elevated value of at least one of these parameters (CEC count and/or TnI level) was taken into an account, sensitivity increased by 30%.

Increased CEC count in ACS may depend on both endothelial damage in the vicinity of ruptured plaque and on preservation at least minimal blood flow in the culprit vessel. This suggests that determination of CEC count might be diagnostically useful in the early stage of haemodynamic disturbances when other laboratory markers are still below the diagnostic threshold [17]. Determination of CEC count might help in the diagnosis of MI when its primary pathogenic mechanism is endothelial damage related to coronary plaque rupture.

In our study, we evaluated usefulness of CEC count in the diagnosis of an ACS in a group of patients with AMI. For clearer and more complete picture, similar data would be required regarding the value of CEC count as a marker o endothelial damage in patients with unstable angina.

Our study also showed that atherogenic lipid abnormalities coexisted with increased CEC count. In the overall group of patients with CAD, CEC count correlated with the atherogenic index TC/HDL-C. Increase in TC/HDL-C ratio was related to low HDL-C level ($p < 0.001$). It is possible that resultant reduced antiatherogenic action of this lipoprotein fraction increases endothelial cell susceptibility to damage in CAD [18]. Lee et al. [11] suggested that the damage of coronary and intracardiac endothelial cells induced by cardiovascular risk factors in patients with CAD predisposes to endothelial cell shedding during vessel occlusion, leading to increased CEC count in peripheral blood. Increased CEC count was reported in patients with primary and secondary pulmonary hypertension [19] and in patients with type 2 diabetes [20].

In our study, CEC count was determined using flow cytometry, while most studies published in the literature were performed using immunomagnetic isolation technique [4, 6, 10, 11]. Flow cytometry was previously used to determine CEC count in other vascular disease and neoplastic disease

[8, 21, 22]. More commonly used immunomagnetic isolation may be considered a gold standard in CEC determinations, but flow cytometry is more suitable for routine clinical use.

Increased CEC count in patients with AMI supports morphological endothelial damage resulting in loss of its integrity during ACS. Normal CEC count in patients with SA suggests that endothelial damage in these patients is limited to dysfunction and biochemical damage, as indicated by increased vWF activity. Increased CEC count in our patients with CAD was accompanied by atherogenic changes of serum lipoprotein profile.

CONCLUSIONS

We confirmed that CEC count in peripheral blood can be determined by flow cytometry in CAD patients with both AMI and SA. The CEC count in AMI was increased in comparison to healthy subjects and SA patients in one third of all cases. To determine whether CEC count could be used to improve the diagnosis of an ACS in patients with CAD, additional studies in appropriately large patient groups would be required.

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Krążące komórki śródbłonka w chorobie niedokrwiennej serca

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Streszczenie

Wstęp: W patofizjologii choroby niedokrwiennej serca kluczową rolę odgrywa uszkodzenie i dysfunkcja śródbłonka naczyń krwionośnych. Do nowych metod oceny uszkodzeń śródbłonkowych należy oznaczanie liczby krążących komórek śródbłonka (CEC) w krwi obwodowej.

Cel: Celem pracy była ocena diagnostycznej przydatności 1-krotnego oznaczania liczby CEC w krwi obwodowej metodą cytometrii przepływowej u pacjentów z chorobą niedokrwienną serca.

Metody: Badaniami objęto 48 osób z chorobą niedokrwienną serca: 23 pacjentów z ostrym zawałem serca (AMI) i 25 pacjentów ze stabilną chorobą wieńcową (SA). Grupę kontrolną stanowiło 20 zdrowych osób, które nie miały klinicznych objawów choroby wieńcowej. Liczbę CEC oznaczano metodą cytometrii przepływowej, używając przeciwciał przeciwko antygenom CD31, CD146, CD45. Stężenie biochemicznych markerów uszkodzenia śródbłonka (aktywność czynnika von Willebranda — vWF, stężenie trombomoduliny — TM) oznaczano w osoczu krwi metodą ELISA. W analizach statystycznych uwzględniono również stężenie troponiny I (TnI), stężenia parametrów lipidowych (cholesterolu całkowitego — TC, cholesterolu frakcji HDL — HDL-C, cholesterolu frakcji LDL i triglicerydów) oraz wartość wskaźnika aterogennego TC/HDL-C, wyliczonego jako iloraz stężenia TC do HDL-C.

Wyniki: Wykazano zwiększoną liczbę CEC w krwi obwodowej pacjentów z AMI w porównaniu z grupą kontrolną ($p < 0,05$) i pacjentów z SA ($p < 0,05$). W grupie chorych z SA liczba CEC nie różniła się istotnie statystycznie od wyników grupy kontrolnej. W obu grupach stwierdzono wyższą aktywność vWF niż w grupie kontrolnej (AMI: $p < 0,001$; SA: $p < 0,01$). Aktywność vWF była istotnie wyższa u pacjentów z AMI niż u osób z SA ($p < 0,001$). Stężenie TM w żadnej z badanych grup nie różniło się od wyników grupy kontrolnej. W grupie obejmującej wszystkich pacjentów z chorobą niedokrwienną serca (AMI + SA) liczba CEC korelowała dodatnio z aktywnością vWF ($r = 0,3852$; $p < 0,05$) oraz wartością wskaźnika aterogennego TC/HDL-C ($r = 0,3844$; $p < 0,05$). Czułość diagnostyczna CEC w diagnozowaniu ostrych zespołów wieńcowych wynosiła 39%, a czułość diagnostyczna TnI w momencie przyjęcia pacjentów do szpitala — 69%. Zwiększona liczba CEC u pacjentów z AMI potwierdza utratę integralności śródbłonka w czasie ostrych incydentów wieńcowych. Liczba CEC u pacjentów z SA wskazuje, że uszkodzenie śródbłonka w tej grupie jest ograniczone do dysfunkcji i uszkodzeń biochemicznych, co odzwierciedla zwiększona aktywność vWF. Zwiększonej liczbie CEC u badanych pacjentów towarzyszą zmiany aterogenne w profilu lipidowym surowicy krwi.

Wnioski: Wykazano możliwość oznaczania liczby CEC za pomocą cytometrii przepływowej u chorych z AMI lub z SA. Liczba CEC w AMI była podwyższona w porównaniu z osobami zdrowymi i pacjentami z SA w 1/3 przypadków. Należałoby sprawdzić na dużej i odpowiednio dobranej grupie klinicznej, w jakich sytuacjach oznaczanie CEC u chorych z ostrymi incydentami wieńcowymi mogłoby polepszyć dokładność rozpoznania.

Słowa kluczowe: choroba niedokrwienna serca, śródbłonek, krążące komórki śródbłonka, czynnik von Willebranda

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