Original article

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Effects of platelet and inflammatory system activation on outcomes in diabetic patients with ST segment elevation myocardial infarction treated with primary percutaneous coronary intervention

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Abstract

Background: Recurrent myocardial ischaemia and restenosis are more common in diabetic patients treated with primary percutaneous coronary intervention (PCI) due to an acute coronary syndrome (ACS) compared to patients without diabetes. Diabetes is also associated with increased residual platelet activity during dual antiplatelet treatment. In recent reports, platelet reactivity has been linked to outcomes after ACS. Appropriate platelet inhibition might lead to improved outcomes in this patient population. To this end, newest methods to evaluate platelet function may prove helpful.

Aim: To evaluate 6-month outcomes in diabetic patients treated with primary PCI due to ST segment elevation myocardial infarction (STEMI) in relation to platelet reactivity evaluated at discharge.

Methods: The study included 120 diabetic patients treated with primary PCI due to STEMI. Patients received loading doses of acetylsalicylic acid (ASA, 300 mg) and clopidogrel (600 mg) on admission, and later were treated with maintenance doses of 75 mg of ASA and clopidogrel. Blood for platelet aggregation testing was collected at discharge. We used whole blood impedance aggregometry using the Multiplate aggregometer with arachidonic acid (AA), adenosine diphosphate (ADP), collagen, and thrombin receptor peptide agonist (TRAP) as agonists. Six-month follow-up was based on telephone contact and hospital discharge summaries if hospitalisation occurred. The primary combined endpoint included recurrent ACS and restenosis.

Results: The primary combined endpoint occurred in 28 (23%) patients. Among patients with the primary endpoint, we found significantly higher platelet reactivity as evaluated by aggregation testing using AA and TRAP at discharge following the initial infarction compared to patients without the primary endpoint (area under aggregation curve 1137.4 ± 198.5 *vs* 833.5 ± 253.4; p = 0.013 for TRAP-induced aggregation, and 333.0 ± 263.8 *vs* 186.9 ± 105.4; p = 0.019 for AA-induced aggregation). We found no relationship between ADP- and collagen-induced aggregation at discharge and the occurrence of the primary endpoint.

Conclusions: Increased platelet reactivity evaluated by TRAP-induced aggregation is related to a higher rate of restenosis and recurrent ACS during a 6-month follow-up of diabetic STEMI patients treated with PCI.

Key words: diabetes, acute coronary syndrome, primary percutaneous coronary intervention

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INTRODUCTION

Despite use of stents during percutaneous coronary interventions (PCI), including antimitotic stens, restenosis in the target vessel remains a major problem [1]. This is most relevant in diabetic patients with acute coronary syndromes (ACS), in whom restenosis is more common than in other patient groups [2]. Recent studies suggested that increased restenosis rate may be related to the degree of platelet activation and inflammation [3]. Some data also suggested that platelets, thought to be involved mostly in thrombosis, may be also responsible for restenosis in the target vessel through stimulation of proliferation processes [4]. Animal studies showed that inhibition of platelet P-selectin by specific antibody administration reduces the degree of intimal proliferation in the vessel wall [3]. Platelet-leukocyte interactions at the sites of vascular injury may also be an important phenomenon affecting restenosis. Animal studies also showed that substances released by platelets exert a chemotactic effect on leukocytes, contributing to proliferation and restenosis [5].

Coronary angioplasty in the management of ACS increases survival but GRACE registry showed that a recurrent ischaemic event will occur during follow-up in every fifth patient with ACS, more commonly among diabetic patients [6]. These events may result from progression of atherosclerosis in other coronary vessels or restenosis in the target vessel [7]. One of the causes of recurrent ACS may be increased platelet activity despite chronic antiplatelet treatment. For some years now, attempts have been made to determine the most reliable test to assess platelet reactivity and a cut-off value identifying patients at risk of a recurrent ischaemic event that in the future might allow individualisation of antiplatelet drug dosage. Until now, success of these attempts has been modest, as no single reference method to assess platelet function has been established. Also, the cut-off values of increased platelet reactivity are difficult to replicate in other studies [8]. Even less is known about the relationship between the degree of platelet activation and occurrence of clinically overt restenosis in humans, as this issue has been only studied in animal models [9].

The purpose of our study was to evaluate the relationship between the degree of platelet activation and inflammation and the occurrence of restenosis and recurrent ACS during a 6-month follow-up in patients with diabetes type 2 and ST segment elevation myocardial infarction (STEMI) treated with primary PCI.

METHODS

Study inclusion criteria included STEMI treated with primary PCI with stent implantation and diabetes diagnosed before hospital admission. Exclusion criteria included cardiogenic shock, stenosis in a non-culprit coronary vessel scheduled for surgical revascularisation, platelet count below 100,000 or above 450,000 in mm^3, and allergy to salicylates or tienopyridines.

In all patients, platelet aggregation was tested at hospital discharge and blood samples were collected and stored for further determination of high sensitivity C-reactive protein (hsCRP). Combined primary endpoint included restenosis and recurrent ACS during a 6-month follow-up based on patient telephone contact and hospital discharge summaries if repeated hospitalisation occurred after the study inclusion.

The study was approved by the Ethics Committee of the Silesian Medical University. All patients gave written informed consent for their participation in the study.

Hospital management

Study patients were treated with primary PCI as per European Society of Cardiology (ESC) guidelines. All patients were given loading dose of 600 mg of clopidogrel and 300 mg of acetylsalicylic acid (ASA) before PCI and then received maintenance doses of 75 mg of clopidogrel and ASA, starting from the second day of hospitalisation, with discharge recommendation of clopidogrel administration for 12 months and indefinite continuation of treatment with ASA. In the cardiac catheterisation laboratory, coronary angiography was performed to evaluate coronary anatomy before PCI, and intravenous heparin was administered as per current standards. The use of glycoprotein IIb/IIIa inhibitors was left at the operator discretion. Patients who eventually had at least one stent implanted were eligible for the study. Bare metal stents (BMS) were used in all cases.

Platelet aggregation

To evaluate platelet aggregation, we used Multiplate impedance aggregometer (Dynabyte, Germany). Whole blood was collected to test tubes containing hirudin (25 μg/mL) as anticoagulant (Sarstedt, Germany). We used the following aggregation agonists: 0.5 mM arachidonic acid (AA), 6.4 μM adenosine diphosphate (ADP), 3.2 μg/mL collagen, and 32 μM thrombin receptor agonist peptide (TRAP). These reagents were supplied by the manufacturer of the aggregometer. Blood for testing was collected in the morning from fasting patients and assays were performed within 2 h after blood collection. Assessment of aggregation included evaluation of the area under curve (AUC) on the aggregation plot, the degree of aggregation (AGR), and aggregation velocity (V_{AGR}). Aggregation was determined twice in each sample and the mean value was calculated unless the two values differed by 10% or more. In the latter case, aggregation assays were repeated.

Evaluation of inflammatory activity

Determinations of hsCRP level were performed using the nefelometric method with Dade Behring (Germany) reagents and devices.

Statistical analysis

Statistical analysis was performed using STATISTICA software, version 9.0 (StatSoft, USA). For all patients, we calculated

arithmetic means and SD and tested for normal distributions of the evaluated parameters using Kolmogorov, Smirnov, Lillefors, and Shapiro-Wilk tests. Significance of the differences between groups was tested using the Student *t* test for unpaired samples for normally distributed variables and the Mann-Whitney U test for non-normally distributed variables. Correlations between variables were evaluated using a correlation matrix, with Pearson correlation coefficients calculated for normally distributed variables and Spearman correlation coefficients calculated for non-normally distributed variables. For dichotomous variables, significance of the differences between groups was tested using χ^2 test, χ^2 test with Yates correction or the exact Fisher test, depending on the sample size. A Phi coefficient was used to evaluate correlations between groups of dichotomous variables. A p value < 0.05 was considered significant.

RESULTS

The study included 120 patients. Detailed characteristics of the study group are shown in Table 1. Results of platelet aggregation testing at discharge are shown in Table 2 and Figures 1 and 2.

Table 1. Characteristics of the study group (n = 120)

Age [years]	61.4 ± 10.9
Men/women	90/30
Hypertension	87 (72.5%)
Hypercholesterolaemia	72 (60%)
Current smoking	66 (55%)
Positive family history	24 (20%)
Culprit vessel:	
LAD	57 (47.6%)
Cx	17 (14.1%)
RCA	42 (35%)
IM	4 (3.3%)
Number of implanted stents, mean (median)	1.2 (1)
Length of implanted stents, mean (median) [mm]	19.5 (18)
Diameter of implanted stents, mean (median) [mm]	3.2 (3)
Glycoprotein IIb/IIIa inhibitor use during PCI	34 (28.3%)
In-hospital death	0 (0%)
Death during follow-up	5 (4.1%)
LVEF at discharge [%]	43.1 ± 9.7
Statin	115 (95%)
Beta-blocker	112 (93%)
ACE inhibitor	98 (81%)
ASA 75 mg/d	120 (100%)
Clopidogrel 75 mg/d	120 (100%)
Insulin	36 (30%)
Biguanide	55 (45.8%)
Sulfonylurea	29 (24.2%)
hsCRP > 3 mg/L	84 (70%)
Peak troponin I [ng/mL]	22.5 ± 4.4
HbA1c [%]	7.1 ± 1.6
Duration of hospitalisation, mean (min–max) [days]	6.1 (3–16)
Length of follow up, min-max [months]	6–18

ACE — angiotensin-converting enzyme; ASA — acetylsalicylic acid; Cx — left circumflex artery; HbA1c — haemoglobin A1c; hsCRP — high-sensitivity C-reactive protein; IM — ramus intermedius; LAD — left anterior descending artery; LVEF — left ventricular ejection fraction; PCI — percutaneous coronary intervention; RCA — right coronary artery

Table 2. Results of platelet aggregation testing in the study group

Parameter	Mean ± SD
AA-AUC	203.9 ± 152.0
AA-AGR	46.7 ± 30.9
AA-V_{AGR}	6.3 ± 4.0
ADP-AUC	270.5 ± 157.9
ADP-AGR	49.4 ± 24.1
ADP-V_{AGR}	7.3 ± 3.4
COL-AUC	212.5 ± 136.5
COL-AGR	43.5 ± 24.8
COL-V_{AGR}	6.0 ± 2.8
TRAP-AUC	806.9 ± 267.7
TRAP-AGR	128.2 ± 41.7
TRAP-V_{AGR}	21.8 ± 7.7

AA — arachidonic acid; ADP — adenosine diphosphate; AGR — degree of aggregation; AUC — area under curve on the aggregation plot; COL — collagen; SD — standard deviation; TRAP — thrombin receptor peptide agonist; V_{AGR} — aggregation velocity

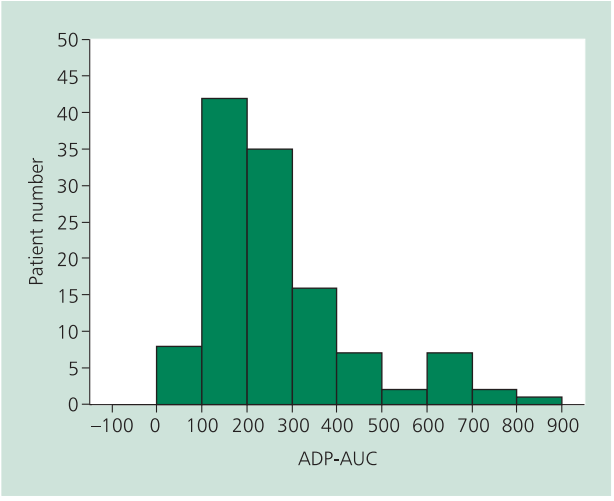

Figure 1. A histogram showing the distribution of ADP-AUC in the study group; ADP — adenosine diphosphate; AUC — area under curve on the aggregation plot

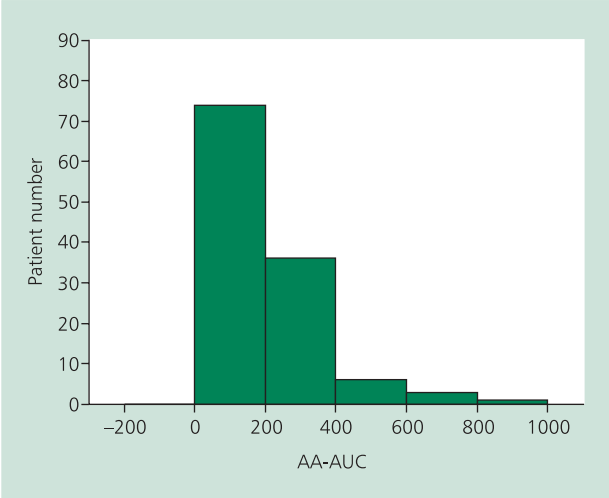

Figure 2. A histogram showing the distribution of AA-AUC in the study group; AA — arachidonic acid; AUC — area under curve on the aggregation plot

Incomplete response to ASA, defined as per the aggregometer manufacturer data as AA-AUC above 300, was found in 10 patients (8.3% of the overall study group). Incomplete response to clopidogrel was defined, again as per the aggregometer manufacturer data, as ADP-AUC above 500 and found in 20 patients (16.6% of the study group).

During follow-up, repeated hospitalisation was noted in 28 (23%) patients. Average time to rehospitalisation was 68.5 (range 25–123) days. In all patients, the cause of rehospitalisation was recurrent ACS, including STEMI due to in-stent thrombosis in 4 patients, non-STEMI (NSTEMI) due to restenosis in 11 patients, unstable angina due to restenosis in 7 patients, and NSTEMI related to a culprit lesion in a vessel other than the one stented during the index STEMI in 6 patients.

Among patients with the primary endpoint, we found significantly higher platelet reactivity as evidenced by aggregation testing using AA and TRAP at discharge following the initial infarction. Details of the aggregation testing are shown in Table 3. We found no significant relationship between incomplete response to clopidogrel and hsCRP level and the occurrence of the primary endpoint.

DISCUSSION

In this prospective clinical study, we found that platelet reactivity as assessed at discharge in diabetic patients with STEMI treated with primary PCI was related to the occurrence of recurrent ACS and clinically significant restenosis during a 6--month follow-up. In our study, to evaluate platelet aggregation we used simple whole impedance aggregometry method that does not require any sophisticated laboratory procedures. In contrast to the most commonly used optical aggregometry [10], it does not require any initial blood preparation prior to the assay, thus reducing the risk of laboratory errors. In addition, the device we used makes two simultaneous independent measurements, thus allowing estimation of reproducibility and providing good quality of the final result [11]. These advantages make this method useful as a point-of-care test in the cardiac catheterisation laboratory.

To evaluate response to antiplatelet drugs, most commonly performed aggregation testing involves the use of two agonists, AA to determine the response to ASA and ADP to determine the response to clopidogrel [8]. In our study protocol, we additionally used collagen and TRAP, as it may be speculated that additional platelet activation pathways, not

Table 3. Platelet aggregation as evaluated at discharge in patients with or without the primary endpoint

Parameter	Patients with the primary endpoint		Patients without the primary endpoint		Significance of the difference (p)
	Mean	SD	Mean	SD	
AA-AUC	333.0	263.8	186.9	105.4	0.019
AA-AGR	67.7	39.6	43.7	24.1	NS
AA-V_{AGR}	9.8	7.9	6.3	3.6	NS
ADP-AUC	303.2	179.6	288.2	154.8	NS
ADP-AGR	54.1	23.0	51.6	24.0	NS
ADP-V_{AGR}	8.0	4.4	7.7	3.6	NS
COL-AUC	211.4	184.4	236.3	153.9	NS
COL-AGR	42.4	34.6	47.3	28.3	NS
COL-V_{AGR}	5.0	3.4	6.4	3.4	NS
TRAP-AUC	1137.4	198.5	833.5	253.4	0.013
TRAP-AGR	179.8	34.5	131.2	40.9	0.014
TRAP-V_{AGR}	31.4	5.9	22.7	8.2	0.026

NS — non-significant; rest abbreviations as in Table 2

inhibited by ASA and clopidogrel, may be responsible for recurrent ischaemic events or restenosis. Increased response to TRAP (equivalent to increased response to thrombin) was associated with an increased risk of recurrent ischaemia during a 6-month follow-up. Our present findings are the first report on this subject in the literature.

In addition, in our study we evaluated platelet reactivity on average 6 days after the occurrence of ACS, i.e. when a new platelet pool had already appeared in the circulation, strongly inhibited by dual antiplatelet therapy. Using cut-off values reported in the literature [12] and data provided by the manufacturer of the aggregometer, incomplete response to ASA and clopidogrel was found at discharge in 8.3% and 16.6% of patients, respectively. These findings are consistent with the data from the literature, although they were obtained with a different type of aggregometer [13, 14]. Of note, such defined incomplete response to ASA and clopidogrel had no effect on the occurrence of recurrent ischaemic events and restenosis. These results are at variance with other reports in the literature, as incomplete response to antiplatelet drugs has been reported to be associated with worse outcomes [13, 14]. It should be remembered, however, that we evaluated platelet reactivity nearly a week after the occurrence of ACS, while in other studies platelet reactivity was usually tested before or just after coronary angioplasty. The cut-off values that were set arbitrarily by the manufacturer of the aggregometer may be another limitation. As this device has been introduced only recently, further studies are required to determine the most sensitive and specific cut-off values of the aggregation parameters that would predict recurrent ACS.

Platelets are continually released to the circulation and their blood pool is fully replaced within on average 7 days. Cyclooxygenase-1 inhibition by ASA and P2Y12 receptor blockade by a clopidogrel metabolite may homeostatically increase activity of other platelet activation pathways including thrombin-mediated activation. Indeed, activity of this pathway had an effect on recurrent ischaemic events and restenosis in conditions of a relatively efficient blockade by ASA and clopidogrel in our study. In contrast, such an association was not found for collagen-mediated platelet activation. A likely explanation is that this pathway is partially blocked by ASA [15].

Phase III studies are underway to evaluate an oral antiplatelet drug from a novel class of thrombin receptor antagonists, SCH 530348 (Schering-Plough) [16]. In this clinical trial, the novel drug is compared to placebo in patients with ACS treated with PCI and receiving concomitant treatment with ASA and clopidogrel. Based on our findings, additional benefits of blockade of this platelet activation pathway may be expected, particularly with no excess bleeding risk noted in phase II studies [17].

In our study, we did not find any relationship between elevated hsCRP level at discharge and recurrent ischaemia or restenosis in diabetic patients. Previous findings regarding this issue have been mixed, with some authors suggesting such an association [18] but others not being able to confirm this [19]. These inconsistent results may be related to different timing of hsCRP level measurements, as these were performed several days after ACS in our study compared to periprocedural testing in most other studies. It is also possible that the degree of systemic inflammation on average a week after the occurrence of ACS has no significant effect on recurrent infarction and restenosis, as it was noted regarding incomplete response to ASA and clopidogrel.

CONCLUSIONS

In diabetic patients with STEMI treated with PCI, increased platelet reactivity to thrombin as evaluated at discharge is related to a higher rate of restenosis and recurrent ACS during a 6-month follow-up.

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Conflict of interest: *none declared*

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Ocena wpływu pobudzenia płytek krwi i układu zapalnego na rokowanie u pacjentów z cukrzycą i zawałem serca z uniesieniem odcinka ST leczonych pierwotną angioplastyką wieńcową

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Streszczenie

Wstęp: U pacjentów z cukrzycą leczeni pierwotną angioplastyką wieńcową (PCI) z powodu ostrego zespołu wieńcowego (OZW) częściej występują nawroty niedokrwienia i restenozy w porównaniu z osobami bez cukrzycy. Cukrzyca wiąże się również z podwyższoną reaktywnością płytek krwi podczas stosowania podwójnej terapii przeciwpłytkowej. Najnowsze doniesienia łączą reaktywność płytek z rokowaniem po OZW. Odpowiednia blokada reaktywności płytek może pozwolić na poprawę rokowania w tej populacji pacjentów, a w osiągnięciu tego celu mogą się okazać pomocne najnowsze metody oceny funkcji płytek krwi.

Cel: Celem pracy była ocena 6-miesięcznego rokowania u pacjentów z cukrzycą leczonych pierwotną PCI z powodu zawału serca z uniesieniem odcinka ST (STEMI) w odniesieniu do reaktywności płytek krwi ocenianej przy wypisie.

Metody: Do badania włączono 120 pacjentów z cukrzycą leczonych za pomocą pierwotnej PCI z powodu STEMI. Chorzy otrzymali dawki nasycające kwasu acetylosalicylowego (ASA, 300 mg) i klopidogrelu (600 mg) przy przyjęciu do szpitala, a następnie stosowali przewlekle ASA i klopidogrel w dawkach 75 mg. Krew do wykonania agregacji była pobierana przy wypisie ze szpitala. Zastosowano metodę agregacji impedancyjnej we krwi pełnej za pomocą agregometru Multiplate, z użyciem jako agonistów kwasu arachidonowego (AA), dwufosforanu adenozyny (ADP), kolagenu i peptydowego agonistę receptora trombinowego (TRAP). Przeprowadzono 6-miesięczną obserwację odległą, wykorzystując kontakt telefoniczny i karty informacyjne z ewentualnych pobytów w szpitalu. Łączonym pierwotnym punktem końcowym badania były ponowny OZW lub restenoza.

Wyniki: Pierwotny punkt końcowy wystąpił u 28 (23%) pacjentów. U osób, u których wystąpił pierwotny punkt końcowy badania, wykazano istotną statystycznie wyższą reaktywność płytek krwi podczas agregacji wywołanej AA i TRAP, oznaczaną w dniu wypisu po pierwszym zawale w porównaniu z pacjentami, u których nie zaobserwowano pierwotnego punktu końcowego badania (pole pod krzywą agregacji 1137,4 ± 198,5 *v.* 833,5 ± 253,4; p = 0,013 dla agregacji wywołanej TRAP i 333,0 ± 263,8 *v.* 186,9 ± 105,4; p = 0,019 dla agregacji wywołanej AA). Nie stwierdzono związku między stopniem agregacji wywołanej ADP i kolagenem ocenianej przy wypisie a częstością wystąpienia pierwotnego punktu końcowego badania.

Wnioski: Podwyższona reaktywność płytek krwi oceniana za pomocą agregacji wywołanej TRAP wiąże się z częstszym występowaniem restenozy i ponownego zespołu wieńcowego w obserwacji 6-miesięcznej u pacjentów z cukrzycą leczonych z powodu STEMI za pomocą pierwotnej angioplastyki wieńcowej.

Słowa kluczowe: cukrzyca, ostry zespół wieńcowy, pierwotna angioplastyka wieńcowa

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