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Platelet transfusion refractoriness in patients with acute myeloid leukemia - the role of anti-platelet antibodies

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Улога антитромбоцитних антитела у развоју рефрактарности на трансфузије тромбоцита код болесника са акутном мијелоидном леукемијом

SUMMARY

Introduction/Objective Platelet transfusion refractoriness represents a common issue in the treatment course of acute myeloid leukemia (AML) patients who require chronic transfusion support. Antibodies to the human platelet antigens could cause immune-mediated transfusion refractoriness. The main goal of this study was to examine the presence and the quantity of antibodies to GP IIb/IIIa, GP Ib/IX and GP Ia/IIa in AML patients refractory to platelet transfusion in comparison to healthy male subjects.

Methods This prospective study involved 22 adult AML patients who demonstrated resistance to platelet transfusion. Antibody titers for the following platelet antigens: GP IIb/IIIa, GP Ib/IX and GP Ia/IIa were measured in these patients before and two hours after platelet transfusion as well as in healthy male controls.

Results The antibodies to platelets antigens taken before the platelet transfusion were significantly increased in all patients compared with healthy untransfused controls ($p = 0.001$). In all three groups of antibodies, the titers were significantly higher after platelet transfusion ($p = 0.001$) than before. There was no statistically significant difference between males and females, platelet sources (buffy-coat *vs.* apheresis-derived) in the examined initial antibody's levels, as well as in the antibody titers increment after the transfusion. No significant correlation between platelet count increment (absolute numbers) and initial levels of anti-platelet antibodies was found.

Conclusion Platelet transfusion refractoriness in AML patients could be caused by the antibodies to platelet antigens, since their levels are significantly higher in examined patients than in healthy male untransfused controls.

Keywords: platelet transfusion refractoriness; acute myeloid leukemia; humane platelet antigens; AML; HPA

САЖЕТАК

Увод/Циљ Рефрактарност на трансфузије тромбоцита представља чест проблем у лечењу болесника са дијагнозом акутне мијелоидне леукемије (*AML* болесници), код којих је неопходна дуготрајна трансфузиона потпора. Основни циљ овог истраживања био је испитивање присуства и титра антитела на хумане тромбоцитне антигене *GP IIb/IIIa*, *GP Ib/IX* и *GP Ia/IIa* код *AML* болесника рефрактарних на трансфузије тромбоцита у поређењу са здравим мушким, претходно нетрансфундованим, контролама.

Методе Спроведена је проспективна студија којом је укључено 22 пунолетна *AML* болесника код којих је доказана рефрактарност на трансфузије тромбоцита. Титар антитела за следеће тромбоцитне антигене: *GP IIb/IIIa*, *GP Ib/IX* и *GP Ia/IIa* процењиван је применом ензимског имуноесеја код ових пацијената пре и два сата након трансфузије тромбоцита, као и код здравих мушких контрола.

Резултати Антитела на тромбоцитне антигене из крви узорковане пре трансфузије тромбоцита су била статистички значајно виша код свих болесника у односу на здраве нетрансфундоване контроле (p = 0,001). У све три групе антитела титар је био значајно виши након трансфузије тромбоцитима (p = 0,001). Није пронађена статистички значајна разлика између полова, извора тромбоцита (*buffy-coat* / аферезни) у иницијалном титру антитела, као ни у порасту титра антитела након трансфузије тромбоцита (p > 0,05). Није пронађена статистички значајна корелација између пораста броја тромбоцита (изажено у апсолутним бројевима) и иницијалнога нивоа анти-тромбоцитних антитела.

Закључак Рефрактарност на трансфузије тромбоцитима код *AML* болесника може бити посредована антителима на тромбоцитне антигене имајући у виду да је титар ових антитела значајно виши код испитиваних болесника него код здравих, нетрансфундованих контрола.

Кључне речи: рефрактарност на трансфузије тромбоцитима; актуна мијелоидна леукемија; хумани тромбоцитни антигени; *AML*; *HPA*

INTRODUCTION

Treating acute myeloid leukemia (AML) often involves high-intensity chemotherapy treatment, which results in bone marrow aplasia and (pan)cytopenia. Moreover, cytopenia in AML patients could be a manifestation of the disease itself as well [1]. Consequently, repeated allogeneic transfusions of red blood cells and platelets are frequently required. Prophylactic transfusion of platelets is recommended

to maintain platelet count (PC) above 10×10^9/L [2]. However, an adequate post-transfusion PC increment cannot always be achieved.

A PC increment of less than 10×10^9/L after administration of an aphaeresis unit (or 1.75×10^9/L per random donor platelet concentrate) is indicative of refractoriness [3]. Platelet refractoriness may be the result of immune and non-immune factors. Non-immune causes of refractoriness are more common, and they are usually associated with splenomegaly, infection (sepsis), fever, administration of drugs, disseminated intravascular coagulation, and bleeding [4]. On the other hand, immune-mediated refractoriness is caused by antibodies to human leucocyte antigens (HLA) and human platelet antigens (HPA). Previous transfusions or pregnancy is considered a risk factor for such alloimmunization. While HLAs are widely present in human tissues, HPAs are platelet-specific [5]. HLA class I antigens are present on the platelet membrane. These molecules are known for their very high polymorphism, which makes them extremely immunogenic [6]. For that reason, HLA-matched platelets, antigen-restricted platelets, or cross-matched platelets are increasingly used, especially among those patients who require chronic transfusions [7]. On the other hand, HPA polymorphisms occur mostly due to single amino acid changes in glycoproteins (GPs) [8]. Numerous platelet-specific antigens have been characterized, and those that are considered the most common polymorphic are GPIa, GPIb, GPIIb, GPIIIa, and CD109 [8, 9]. However, their role in transfusion refractoriness is still controversial [4]. It was observed that leukoreduction does not affect the incidence of HPA [10]

The goal of this study was to assess the presence and levels of antibodies to GP IIb/IIIa, GP Ib/IX and GP Ia/Iia, which carry major HPA epitopes, in AML patients refractory to platelet transfusion in comparison to healthy male subjects.

METHODS

This prospective study, conducted from 2012 to 2015, included adult patients diagnosed with acute myeloid leukemia in the Clinical Center of Serbia, who were platelet transfusion-refractory. All patients received intensive chemotherapy regimens, which resulted in chemotherapy-induced thrombocytopenia (platelet count $< 100 \times 10^9$/L) and required platelet transfusions. A transfusion-related allergic reaction (e.g., chills, fever, and skin manifestations such as erythema and urticaria) was observed in all included patients, but it was not used as an inclusion criterion. Refractoriness was defined as a posttransfusion platelet count increment equal to or lower than 10×10^9/L. Due to the lack of precise data on transfused platelet dose and body surface area, platelet count increment was used instead of corrected count increment. Platelet transfusion refractoriness accompanied with PC $< 10 \times 10^9$/L is life-threatening condition and the main reason for our investigation was to find what is cause for that and could this condition be prevented. All of the patients have received multiple platelet transfusions before the inclusion in the study (more than five). Seven patients were transfused with buffy coat-derived platelet concentrates since apheresis-derived platelet concentrates were not available, and fifteen patients were

transfused with apheresis-derived platelet concentrates. Apheresis units minimize donor exposure and have been shown to allow for more prolonged survival of platelets compared to pooled platelets [11]. Age, sex, and history of pregnancies and labor in female sub-cohort were collected. Patients with potential non-immune causes of refractoriness (splenomegaly, fever/sepsis, disseminated intravascular coagulation or other consumptive coagulopathy at the time of screening) were excluded from this study. The control group consisted of 22 untransfused healthy male donors. Female donors were not included in order to avoid confounding by pregnancy-related alloimmunization. Other AML patients who did not meet the platelet transfusion refractoriness criteria were not used as controls because of the previous exposition to platelet transfusions.

Antibody titers were measured by enzyme-linked immunosorbent assay (ELISA) before (up to 30 minutes before) and two hours after platelet transfusion for the following platelet antigens: GP IIb/IIIa, GP Ib/IX, and GP Ia/IIa. Post-transfusion samples were analysed to evaluate potential immediate changes in circulating antibody levels following platelet exposure. The anti-platelet antibodies were measured by using a PakAuto assay®, and the results are expressed as optical density (OD) values measured at 405 nm. To determine the presence of antibodies, the OD ratio (sample OD divided by the mean OD of the negative control) was used. In our study, a sample was considered positive if the OD value was equal to or greater than twice the mean OD of the negative control. The procedure was conducted in accordance with the manufacturer's instructions.

SPSS version 23 (IBM, Armonk, New York, USA) was used for the statistical analyses. The methods of descriptive and inferential statistics were used. For continuous variables with normal distribution mean ± standard deviation (SD) was used, while for those which did not follow normal distribution median and interquartile range were used. The frequencies are presented as relative (percentages) and absolute numbers. Histogram and Shapiro–Wilk test were used to assess the normality. The Wilcoxon test was used for numerical paired variables, and the Mann–Whitney U test was used for numerical independent variables, with the level of significance of 5%. The Spearman rank-order correlation coefficient was used to examine the correlation between platelet count increment and initial antibody levels.

Ethics: The study protocol received ethical approval from the Ethics Committee of the University Clinical Center of Serbia (No. 1435/10, September 08, 2011)

RESULTS

During the study period 22 adult patients were included in study (9 (40.91%) males). Mean age of the patients was 52.1 ± 10.8. Patients' demographic and clinical data are shown in Table 1.

The initial antibody titers (before transfusion) to platelets antigens (GP IIb/IIIa, GP Ib/IX, and GP Ia/IIa) were significantly higher in all patients compared with healthy untransfused donors (Wilcoxon matched-pairs signed-rank test: $Z = -3.408$, $p = 0.001$).

Antibody titer values before and after platelet transfusion for the following platelet antigens: GP IIb/IIIa, GP Ib/IX, and GP Ia/IIa are shown in Table 2. In all three groups of antibodies, the titers were significantly higher after platelet transfusion (0.180 *vs.* 0.369, 0.180 *vs.* 0.325, 0.145 *vs.* 0.320, respectively, $p = 0.001$).

No significant differences were observed in baseline antibody levels between male and female patients (anti-GP IIb/IIIa: $p = 0.624$, anti-GP Ib/IX: $p = 0.540$, anti-GP Ia/IIa: $p = 0.713$). Similarly, no differences were found between patients receiving buffy coat-derived *vs.* apheresis-derived platelet concentrates (anti-GP IIb/IIIa: $p = 0.346$, anti-GP Ib/IX: $p = 0.637$, anti-GP Ia/IIa: $p = 0.953$) (Tables 3, 4, and 5).

Antibody titers changes were measured for all three groups and compared across sexes (anti-GP IIb/IIIa: $p = 0.462$, anti-GP Ib/IX: $p = 0.129$, anti-GP Ia/IIa: $p = 0.902$), different types of transfusion products (anti-GP IIb/IIIa: $p = 0.906$, anti-GP Ib/IX: $p = 0.724$, anti-GP Ia/IIa: $p = 0.859$) with no differences found between the groups (Tables 3, 4, and 5).

No significant correlation between platelet count increment (median 10×10^9/L; IQR 5–10) and initial levels of anti-platelet antibodies was found (anti-GP IIb/IIIa Ab: $p = 0.254$; anti-GP Ib/IX Ab: $p = 0.726$; anti-GP Ia/IIa Ab: $p = 0.383$) (Table 6).

DISCUSSION

Platelet transfusion is an inevitable part of the treatment course of acute myeloid leukemia. Patients with acute myeloid leukemia often require chronic transfusion support, which presents an obstacle in the form of alloimmunization and consequential transfusion refractoriness. The presence of platelet refractoriness complicates the management of AML patients [12]. Additionally, AML patients with platelet refractoriness have shorter overall survival than those without [13]. Platelet-specific antibodies are common in patients with a history of possible previous immunization, particularly if patients were refractory to platelet transfusions [14]. In our study we engaged healthy untransfused male donors as controls in order to avoid previous immunization caused by previous transfusions (frequent in AML patients) or pregnancies. Our results have shown that in all of our transfusion-refractory patients, levels of antiplatelet antibodies were significantly higher than those measured in controls. The prevalence of baseline autoantibodies was higher than in some previous studies [15, 16]. The reason for such a high prevalence of anti-HPA antibodies could be explained by the strict inclusion criteria. Moreover, acute leukemia patients are found to be all immunized more frequently than patients with other malignant hematological diagnoses [17]. On the other hand, this could be an explanation for the post-transfusion low platelet increment in our patients, bearing in mind that previous studies have shown that in post-transfusion purpura, antigen-negative transfusion recipients are sensitized against antigen-positive donor platelets. which led to accelerated clearance of both donor and recipient platelets [18]. Interestingly, antibodies' titers were observed to be higher after the subsequent transfusion in all

subjects. It should be noted that the observed increase in antibody levels 2 hours post-transfusion likely reflects redistribution or immediate immune complex formation, rather than de novo antibody production, which requires several days. However, our study did not observe any significant correlation between the increment in platelet count and the initial levels of anti-platelet antibodies.

Regarding post-transfusion platelet increment, previous studies have shown that single-donor platelets were not superior to pooled-donor platelets [18]. Moreover, in our study, it has been found that the level of examined antiplatelet antibodies was not significantly different between patients who received buffy coat-derived and apheresis-derived platelets.

Comont et al. [19] showed that platelet transfusion refractoriness is more common among female subjects. In our study there was no difference in titers between sexes.

We are aware of the limitations of this study. First and foremost, the small number of participants in the study may limit the accuracy of correlations and conclusions. Nevertheless, the observed differences compared with the control group and the effects of transfusion on antibody titers warrant further investigation in larger cohorts, and would potentially inform better transfusion protocol. Moreover, we have not taken into account the presence of anti-HLA antibodies, which could be an important component of the anti-platelet alloreactivity, and it is often noted that those who develop anti-HPA antibodies develop anti-HLA antibodies at the same time [20]. Additionally, we engaged male only control in order to avoid potential alloimmunization in control group. Finally, clinically relevant factors associated to platelet transfusion like storage duration (age) of platelet concentrates were not taken into account [21].

CONCLUSION

We showed the presence of antiplatelet antibodies in all examined platelet transfusion-refractory patients, which support the hypothesis that anti-platelet antibodies may contribute in platelet transfusion refractoriness. However, no significant connection was found between antibody titers and subjects' sex and source of platelets. Future studies should include larger cohorts, assessment of anti-HLA antibodies, and genetic analysis of HPA polymorphisms to further clarify immune causes of platelet transfusion refractoriness

Conflict of interest: None declared.

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Table 1. Patients' characteristics

Characteristics		Total (N = 22 pts)
Sex N (%)	Male	9 (40.91)
	Female	13 (59.09)
Age mean ± SD		52.1 ± 10.8
Platelet source N (%)	Buffy coat-derived	7 (31.82)
	Apheresis-derived	15 (68.18)
Pregnancy* N (%)		11 (84.61)
PC increment median (IQR)		10 (5–10)
PC increment N (%)	$< 10 \times 10^9$/L	9 (40.91)
	10×10^9/L	13 (59.09)

N – number; pts – patients; IQR – interquartile range; SD – standard deviation; PC – platelet count

*applies only to women subjects

Table 2. Antibody titers before and after platelet transfusion

Target antigens	Time point	Total (N = 22 pts)	p*
GP IIb/IIIa median (IQR)	before	0.180 (0.121–0.268)	**0.001**
	after	0.369 (0.268–0.421)	
GP Ib/IX median (IQR)	before	0.180 (0.123–0.239)	**0.001**
	after	0.325 (0.260–0.370)	
GP Ia/IIa median (IQR)	before	0.145 (0.114–0.227)	**0.001**
	after	0.320 (0.248–0.397)	

N – number; pts – patients; GP – glycoprotein;

*Wilcoxon test; level of significance of 5%

Table 3. Anti-GP IIb/IIIa titers before and antibody titers' increment after platelet transfusion – differences among sexes, platelet source and levels of platelet increment

Parameters		Anti-GP IIb/IIIa Ab initial levels		Anti-GP IIb/IIIa Ab increment after-before PT	
		Median	p*	Median	p*
Sex	Male	0.180	0.624	0.189	0.462
	Female	0.182		0.147	
Platelet source	Buffy coat-derived	0.187	0.346	0.168	0.906
	Apheresis-derived	0.180		0.162	

GP – glycoprotein; Ab – antibody; PT – platelet transfusion;

*Mann–Whitney U test; level of significance of 5%

Table 4. Anti-GP Ib/IX titers before and antibody titers' increment after platelet transfusion - differences among sexes, platelet source and levels of platelet increment

Parameters		Anti-GP Ib/IX Ab initial levels		Anti-GP Ib/IX Ab increment after-before PT	
		Median	p*	Median	p*
Sex	Male	0.179	0.540	0.153	0.129
	Female	0.180		0.106	
Platelet source	Buffy coat-derived	0.179	0.637	0.106	0.724
	Apheresis-derived	0.180		0.140	

GP – glycoprotein; Ab – antibody; PT – platelet transfusion

*Mann–Whitney U test; level of significance of 5%

Table 5. Anti-GP Ia/IIa titers before and antibody titers' increment after platelet transfusion - differences among sexes, platelet source and levels of platelet increment

Parameters		Anti-GP Ia/IIa Ab initial levels		Anti-GP Ia/IIa Ab increment after-before PT	
		Median	p*	Median	p*
Sex	Male	0.210	0.713	0.119	0.902
	Female	0.130		0.150	
Platelet source	Buffy coat-derived	0.137	0.953	0.134	0.859
	Apheresis-derived	0.205		0.150	

GP – glycoprotein; Ab – antibody; PT – platelet transfusion

*Mann–Whitney U test; level of significance of 5%

Table 6. Correlation between platelet count increment and initial levels of anti-platelet antibodies (Spearman rank-order correlation coefficient)

Parameters	Correlation coefficient	p
Anti-GP IIb/IIIa Ab initial levels	0.314	0.254
Anti-GP Ib/IX Ab initial levels	0.099	0.726
Anti-GP Ia/IIa Ab initial levels	0.243	0.383

GP – glycoprotein; Ab – antibody

