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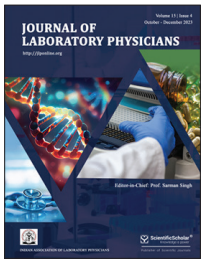

Original Article

Impact of platelet transfusion on outcome, clot dynamics, and platelet function in thrombocytopenic trauma patients

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ABSTRACT

Objectives: Of all trauma patients, 25% require a blood transfusion, of which 2–3% receive a massive transfusion. In severely injured bleeding trauma patients, early platelet administration has been shown to improve hemostasis and decrease mortality. The aim was to compare the changes after platelet transfusion on prothrombin time (PT) and activated partial thromboplastin time (aPTT), platelet function, and clot dynamics in thrombocytopenic trauma patients and to identify independent risk factors for in-hospital mortality.

Materials and Methods: Thrombocytopenic trauma patients who received platelets either with/without receipt of other blood components over two years were included in this prospective study. The pre-and post-transfusion platelet count, coagulation profile, clot dynamic analysis assessed by thromboelastography, and platelet function analysis assessed by flow cytometry were compared. The primary outcome was in-hospital mortality.

Statistical analysis: Data is summarized as mean±S.D or median (minima-maxima) for continuous variables and for categorical variables data is presented as frequency and percentages.

Results: Of the 45 thrombocytopenic trauma patients included in the study, 23 (51.1%) were refractory to platelet transfusion and 14 (31.1%) died. Significant differences were seen in alpha angle (P = 0.02) and maximum amplitude (P = 0.01), number of patients with coagulopathy (P = 0.007), percentage of patients with increased k-time (P = 0.03), and decreased alpha angle (P = 0.001) pre- and post-transfusion. The non-survivors had significantly lower post-transfusion PC (P < 0.001), increased pre- and post-transfusion PT (P = 0.007 and P = 0.01, respectively), and increased pre-and post-transfusion aPTT (P = 0.009 and P = 0.002, respectively). No significant differences were observed based on the thrombolelastography and platelet function parameters between survivors and non-survivors. Pre-transfusion aPTT and coagulopathy post-transfusion were independently associated with mortality (odds ratio [OR]: 9.4; 95% confidence interval [CI]: 1.6–54.3; P = 0.01 and OR: 12.6; 95% CI: 1.55–102.9; P = 0.01, respectively).

Conclusions: Prothrombin time coagulopathy status, clot kinetics, and clot strength improved after platelet transfusion. Pre-transfusion aPTT and coagulopathy post-transfusion are independent risk factors for death in thrombocytopenic trauma patients. The clinical significance of platelet function analysis and clot dynamics in thrombocytopenic trauma patients undergoing transfusion therapy is yet to be ascertained.

Keywords: Flowcytometry, Hemostasis, Refractoriness, Thromboelastography, Traumatology

INTRODUCTION

Globally, 4.4 million people die of injuries each year, which constitutes nearly 8% of all deaths.[1] Of all trauma patients, 25% require a blood transfusion, of which 2–3% receive a massive transfusion.[2] Thrombocytopenia after trauma is associated with increased morbidity and mortality, as described in the previous studies.[3,4] In severely injured bleeding trauma patients, an early platelet administration has been shown to improve hemostasis and decrease mortality.[5] Trauma-induced coagulopathy is also a well-researched entity that further contributes to mortality in one-third of trauma patients.[6] It can be identified using the conventional coagulation parameters, including prothrombin time (PT) and activated partial thromboplastin time (aPTT), D-dimer, fibrinogen, or with the advanced technologies of thromboelastography (TEG), and rotational thromboelastogram.[7] Only a few studies have assessed platelet function after injury, and an even lesser number of studies have analyzed the effect of platelet transfusion on platelet function and its association with outcomes in trauma patients.[8-11]

Therefore, this study aimed to compare the changes after platelet transfusion on PT and aPTT, platelet function, and clot dynamics in thrombocytopenic trauma patients receiving platelets and to identify independent risk factors for in-hospital mortality.

MATERIALS AND METHODS

The study was conducted in a core laboratory of an apex trauma center over 2 years (2016–2017) after approval from the Institute Ethics Committee (IECPG/97/2015). Thrombocytopenic (platelet count $<100 \times 10^3$ per cumm) trauma patients who were admitted and had received random donor platelets either with or without the receipt of other blood components were prospectively included in the study. Platelets were transfused first, followed by transfusion of red blood cells (RBC) and plasma units as per the patient's need. Patients who received platelets within a week after admission were included in the study. The case exclusion criteria included any history of recent blood transfusion post-injury, anticoagulant therapy, known platelet aggregation disorder, chronic hypertension or diabetes, cancer, sepsis, or incomplete/missing information. Specimens received in the laboratory with pre-analytical errors (overfilled/underfilled sample, clotted sample, hemolyzed samples, and wrong vacutainer) were rejected and excluded from the study.

Demographic data and clinical details, including age, sex, severity of injury based on injury severity score, pattern of injury, and length of hospital stay, were noted.[12] The pre-transfusion and post-transfusion (24-h after platelet transfusion) laboratory investigations, which included platelet count, coagulation profile, clot dynamic analysis by TEG, and platelet function analysis by flow cytometry, were recorded. The primary outcome of the study was in-hospital mortality. While the threshold for prophylactic platelet transfusion is as low as 10,000 per cubic millimeter in our institution, there is a higher threshold of less than 100×10^3 per cubic millimeter for platelet transfusions prior to invasive procedures or in trauma patients scheduled for neurosurgery.[13]

For complete blood count, the ethylenediaminetetraacetic acid (EDTA) sample was processed in a fully automated hematology analyzer, Sysmex XE 2100, manufactured by Sysmex Corporation, Kobe, Japan. Whole blood was collected from the patients in two 3.2% sodium citrate vials to extract platelet poor plasma (PPP) and platelet-rich plasma (PRP) for routine coagulation test (PT and aPTT) and platelet function analysis, respectively. Clot dynamic was assessed using citrated whole blood in a TEM-A automated Thromboelastometer (FramarBiomedica, Rome, Italy). The PPP was obtained from the vial used for TEG analysis by centrifugation of whole blood at 2000 g for 15 min at 4°C and analyzed using fully automated *in vitro* coagulation analyzer STA-COMPACT manufactured by DIAGNOSTICA STAGO 9, France. The PRP was obtained by centrifuging for 15 min at 210 g. The platelet-rich yellow supernatant was acquired without disturbing the layers of white and RBCs. The PRP was used immediately to assess platelet function (platelet activation and aggregation) by Accuri C6 flow cytometer, and the data were analyzed with Becton, Dickinson, and Company (USA) software.

Although light transmission aggregometry is considered the gold standard for evaluating platelet function, the unavailability of sensitive modalities for platelet function analysis in thrombocytopenic conditions necessitated the use of a flow cytometry-based methodology.[14] The method used is as follows: Platelets in PRP were washed with sequestering buffer by centrifuging them for 5 min at 2310 g and then suspended in 4-(2-hydroxyethyl)piperazine-1-ethanesulfonic acid (HEPES) solution to attain a final concentration of 50×10^3 per cumm.[15] The above suspension was divided into two aliquots of equal volume to label platelets in each with 15 µL CD31-Fluorescein isothiocyanate (FITC) and CD31-Phycoerythrin (PE) (BD Biosciences) for 15 mins at room temperature. A volume of 50 µL of washed platelets stained with CD31-PE was separated for the activation assay. Activated platelets were labeled with 15 µL platelet activation marker (PAC-1)-FITC after incubating for 1 min. The differently labeled washed platelet samples stained with CD31-FITC and CD31-PE were mixed in a ratio of 1:1, and 50 µL phenylalanyl-L-prolyl-L-arginine chloromethyl ketone was added. Samples were incubated at 37°C while shaking at 700 rpm for 15 min. A baseline value for platelet activation

and aggregation was noted (t = 0 min) to account for the changes due to manipulation during processing, if any. Following incubation, 15 µL of calcium chloride and 10 µL of adenosine diphosphate (ADP) (agonist) were added at 37°C while shaking at 1000 rpm. Cytofix buffer was added at 3 min to fix the samples and analyzed (t = 3 min) to provide optimal time for the platelets to aggregate. PAC-1 (activated GPIIb-IIIa)-FITC positive events were assessed for platelet activation, and double-colored events for CD31-FITC and CD31-PE were taken as aggregated platelets.[4] To analyze, a quadrant was set in a dot plot of respective channels on non-stimulated platelets. The appearance of double-colored events in the upper right quadrant (Q2) was quantified as per the percentage of the total amount of labeled events (Q1 + Q2 + Q4) before (t = 0 min) and 3 min (t = 3 min) after adding ADP.

Percentage of double-colored events = (Q2/[Q1+Q2+Q4]) ×100

Refractoriness was defined as <20% platelet recovery at 20–24 h post-transfusion.[13] Increased PT and aPTT were taken as values >16.5s and >36.5s, respectively. Coagulopathy was defined as PT and aPTT values more than 1.5 times the normal; control PT was 13.5s, and aPTT was 31.5s).[16] Coagulopathy with platelet dysfunction based on TEG parameters was defined as a value of k-time >3min and, alpha angle <55° and maximum amplitude <50 mm.[17]

Statistical analysis

Data were summarized as mean ± standard deviation or median (minima-maxima) for continuous variables. For categorical variables, data were presented as frequency and percentages. The continuous variables were analyzed between groups using the Student's t-test/Wilcoxon rank-sum test, whichever is applicable. Categorical variables were compared between the groups using Chi-Square/Fischer's exact test, as applicable. $P < 0.05$ was considered statistically significant. A step-wise logistic regression model was used to adjust for the other covariates to find the risk factors for in-hospital mortality.

RESULTS

Characteristics of the study population

A total of 45 thrombocytopenic trauma patients were included in the study. The demographic and clinical details are presented in Table 1. The mean age of the study population is 32.9 ± 11.1 years. The in-hospital mortality rate was 14 (31.1%). Eleven (78.5%) of the non-survivors had severe trauma. Of the total, 23 (51.1%) were refractory to platelet transfusion, and of all refractory patients, 10 (43.4%) died.

Table 1: Demographics and clinical details of the study population (n=45).

Variables	Values
Age (years)	32.9±11.1
Gender	
Male	42 (93.3)
Female	3 (6.67)
ISS	21±10.8
ISS	
<25	20 (45.45)
>25 (severe injury)	24 (54.54)
Pattern of injury	
Isolated head injury	16 (22.8)
Isolated abdominal injury	17 (24.2)
Isolated bone injury	17 (24.2)
Polytrauma	20 (28.5)
SBP (mm Hg)	111.8±22.7
GCS	12.7±3.6
GCS	
Mild	32 (72.7)
Moderate	7 (15.9)
Severe	5 (11.3)
Mean units of platelet transfused	5.02±2.7
Median units of fresh frozen plasma transfused	4 (0–8)
Median units of packed RBCs transfused	5 (0–10)
Refractoriness	
Present	23 (51.1)
Absent	22 (48.8)
Mortality rate	14 (31.1)

Continuous data are presented as mean±standard deviation or median (minimum, maximum); categorical data are presented as frequency (%). GCS: Glasgow coma scale, SBP: Systolic blood pressure, ISS: Injury severity score, RBC: Red blood cells

Comparison of laboratory parameters before and after platelet transfusion

Five patients received massive transfusions. Table 2 depicts the laboratory parameters before and after platelet transfusion. Significant differences were seen in values of platelets (63 [4–99] vs. 73 [4–234] $\times 10^3$ per cumm; $P = 0.01$), alpha angle (51.9 ± 19.7° vs. 63.7 ± 16.9°; $P = 0.02$) and maximum amplitude (44.1 ± 5.4mm vs. 53.9 ± 4.1mm; $P = 0.01$), number of patients with coagulopathy (18 [40.0%] vs. 8 [17.7%]; $P = 0.007$), patients with increased k-time (10 [58.8%] vs. 3 [17.6%]; $P = 0.03$), and decreased alpha angle (10 [58.8%] vs. 1 [5.8%]; $P = 0.001$). No significant difference between pre-and post-transfusion was observed in flow cytometry-based platelet function parameters.

Comparison of laboratory parameters between the survivors and the non-survivors

Table 3 demonstrates the comparison between the survivors and the non-survivors. The non-survivors had significantly

Table 2: Comparison of laboratory parameters pre- and post- platelet transfusion.

N	Variable	Normal reference interval	Pre-transfusion	Post-transfusion	*P*-value
45	Hemoglobin (g/dL)	15 ± 2 (male); 13.5 ± 2 (female)	9.5 ± 2.1	9.7 ± 2.0	0.65
	RBC count (per cumm)	5 ± 0.5 (male) 4.3 ± 0.5 (female)	3.2 ± 0.6	3.3 ± 1.1	0.59
	Hematocrit (%)		30.5 ± 6.9	30.4 ± 6.6	0.92
	Total leukocyte count (per cumm)	4000–10000	9032.8 ± 686.1	9548.2 ± 752.6	0.48
	<4000 (per cumm)	----	4 (8.8)	5 (11.1)	0.64
	4000–10000 (per cumm)		27 (60.0)	23 (51.1)	
	>10000 (per cumm)		14 (31.1)	17 (37.7)	
	Platelet count ($\times 10^3$ per cumm)	150–450	63 (4–99)	73 (4–234)	0.01
	<10000 (per cumm)	-----	3 (6.6)	2 (4.4)	0.7
	10000–50000 per cumm)		15 (33.3)	14 (31.1)	
	>50000 (per cumm)		27 (60.0)	29 (64.4)	
	Prothrombin time (seconds)	11–16	19.5 ± 8.4	17.2 ± 4.7	0.09
	Increased prothrombin time	----	32 (71.1)	22 (48.8)	0.01
	aPTT (seconds)	26–40	35.9 ± 17.3	32.1 ± 7.2	0.08
	Increased aPTT	----	19 (42.2)	12 (26.6)	0.08
	Coagulopathy				
	Present	-----	18 (40.0)	8 (17.7)	0.007
	Absent		27 (60.0)	37 (82.2)	
17	r-time (min)	2–8	4.3 ± 2.5	3.9 ± 2.4	0.63
	Increased r-time	-----	3 (17.6)	3 (17.6)	1.00
	k-time (min)	1–3	9.6 (0–17.8)	3.5 (0.3–12.8)	0.07
	Increased k-time	-----	10 (58.8)	3 (17.6)	0.03
	Alpha angle (◦)	55–78	51.9 ± 19.7	63.7 ± 16.9	0.02
	Decreased alpha angle	-----	10 (58.8)	1 (5.8)	0.001
	Maximum amplitude (mm)	51–69	44.1 ± 5.4	53.9 ± 4.1	0.01
	Decreased maximum amplitude	-----	12 (70)	10 (58.8)	0.15
	Coagulopathy with platelet dysfunction				
	Present	-----	13 (76.4)	11 (64.7)	0.31
	Absent		4 (23.5)	6 (35.2)	
45	Platelet Activation (%)	-----	8.9 (0–60.3)	7.4 (0–56.6)	0.98
	Platelet aggregation at t = 3 min (%)	-----	23.5 (0–61.3)	30.3 (0–65.4)	0.16

Continuous data are presented as mean ± standard deviation or median (minimum, maximum); categorical data are presented as frequency (%). RBC: Red blood cell, aPTT: Activated partial thromboplastin time

lower values of post-transfusion platelet count (73 [22–234] vs. 31.5 [4–102] $\times 10^3$ per cumm; $P < 0.001$), increased pre-transfusion and post-transfusion PT values (18 [11.7–26.3]s vs. 21 [14.5–70]s; $P = 0.007$ and 16 [12.5–20.4]s vs. 17.7 [13.4–40.1]s; $P = 0.01$, respectively). The values of pre-transfusion and post-transfusion aPTT were also significantly increased in the non-survivors than the survivors (33.8 [22.2–120]s vs. 45.6 [22.1–120]s; $P = 0.009$ and 31.6 [22.4–61.3]s vs. 34.1 [30–120]s; $P = 0.002$). However, no significant differences were observed based on the thrombolelastography and platelet function parameters between the survivors and the non-survivors.

Percentage of patients with increased pre-transfusion PT (>16.5s) and aPTT (>36.5s) were significantly higher in the non-survivors than the survivors (13 [92.8%] vs. 19 [61.2%]; $P = 0.03$ and 11 [78.5%] vs. 8 [25.8%]; $P = 0.001$, respectively).

The percentage of patients with coagulopathy before and after transfusion was also significantly higher in the non-survivors than the survivors (10 [71.4%] vs. 8 [25.8%]; $P = 0.004$ and 6 [42.6%] vs. 2 [6.4%]; $P = 0.003$, respectively).

In the step-wise multiple regression model, pre-transfusion aPTT value and coagulopathy after transfusion were independently associated with mortality (adjusted odds ratio [OR], 9.4; 95% confidence interval [CI], 1.6–54.3; $P = 0.01$ and adjusted OR, 12.6; 95% CI, 1.55–102.9; $P = 0.01$, respectively), as shown in Table 4.

DISCUSSION

The present study found that there was a significant improvement in the platelet counts post-transfusion. The

Table 3: Comparison between the survivors and the non-survivors.

Variable	Survivors (*n*=31)	Non-survivors (*n*=14)	*P*-value
Severe injury (ISS >25)	13 (43.3)	11 (78.5)	**0.02**
Hemoglobin (g/dL)			
Pre-transfusion	9.7±2.2	9.1±1.9	0.41
Post-transfusion	9.9±2.3	9.2±1.4	0.28
RBC count (per cumm)			
Pre-transfusion	3.3±0.6	3.0±0.6	0.29
Post-transfusion	3.4±1.4	3.1±0.4	0.37
Hematocrit (%)			
Pre-transfusion	31.4±7.3	28.4±5.8	0.18
Post-transfusion	31.1±7.4	28.7±4.3	0.26
Total leukocyte count (per cumm)			
Pre-transfusion	8300 (3100–23100)	7095 (3100 13100)	0.24
Post-transfusion	9220 (3600–29800)	8105 (3600–12700)	0.12
Platelet count ($\times 10^3$ per cumm)			
Pre-transfusion	63 (6–99)	38.5 (4–95)	0.08
Post-transfusion	73 (22–234)	31.5 (4–102)	**<0.001**
Platelet count (per cumm)			
Pre-transfusion			
<10000	1 (3.2)	2 (14.2)	0.19
10000–50000	9 (29.0)	6 (42.8)	
>50000	21 (67.7)	6 (42.8)	
Post-transfusion			
<10000	0 (0)	2 (14.2)	**0.009**
10000–50000	7 (22.5)	7 (50.0)	
>50000	24 (77.4)	5 (35.7)	
Refractory patients	13 (41.9)	10 (71.4)	0.06
Prothrombin time (seconds)			
Pre-transfusion	18 (11.7–26.3)	21 (14.5–70)	**0.007**
Post-transfusion	16 (12.5–20.4)	17.7 (13.4–40.1)	**0.01**
Patients with increased prothrombin time			
Pre-transfusion	19 (61.2)	13 (92.8)	**0.03**
Post-transfusion	13 (41.9)	9 (64.2)	0.16
aPTT (seconds)			
Pre-transfusion	33.8 (22.2–120)	45.6 (22.1–120)	**0.009**
Post-transfusion	31.6 (22.4–61.3)	34.1 (30–120)	**0.002**
Patients with increased aPTT			
Pre-transfusion	8 (25.8)	11 (78.5)	**0.001**
Post-transfusion	6 (19.35)	6 (42.8)	0.09
Patients with coagulopathy			
Pre-transfusion	8 (25.8)	10 (71.4)	**0.004**
Post-transfusion	2 (6.4)	6 (42.6)	**0.003**
r-time (min)			
Pre-transfusion	4.5 (0.5–8.7)	5.4 (0–10.3)	0.39
Post-transfusion	3.7 (0.7–10.5)	4.4 (2.2–7.3)	0.42
k-time (min)			
Pre-transfusion	3.3 (0.3–17.8)	3.1 (0–4.5)	1.00
Post-transfusion	1.8 (0.3–12.8)	1.4 (1–2)	0.73
Alpha angle (◦)			
Pre-transfusion	60.4 (27.4–87.03)	45.6 (0–51.0)	0.08
Post-transfusion	64.3±19.1	61.6±8.2	0.36
Maximum amplitude (mm)			
Pre-transfusion	45.2 (22.4–94.2)	32.8 (0–53.8)	0.25
Post-transfusion	54.3±18.4	52.6±13.7	0.95

(*Contd...*)

Table 3: (*Continued*)

Variable	Survivors (*n*=31)	Non-survivors (*n*=14)	*P*-value
Patients with coagulopathy and platelet dysfunction (TEG based)			
Pre-transfusion	9 (69.2)	4 (100)	0.20
Post-transfusion	8 (61.5)	3 (75.0)	0.62
Platelet activation (%)			
Pre-transfusion	14.9 (0–60.3)	3.2 (0–60.3)	0.23
Post-transfusion	9.1 (0–56.6)	4.4 (0–38.9)	0.10
Platelet aggregation (%)			
Pre-transfusion	19.6 (0–58.3)	24.1 (0–61.3)	0.24
Post-transfusion	30 (0–58)	33 (3–65)	0.06
Post-transfusion	27 (87.1)	13 (92.8)	0.56

Continuous data are presented as mean±standard deviation or median (minimum, maximum); categorical data are presented as frequency (%). RBC: Red blood cell, aPTT: Activated partial thromboplastin time, ISS: Injury severity score. Values in bold signify statistical significance.

Table 4: Multiple regression model to determine risk factors for in-hospital mortality.

Variable	Crude odds ratio	95% confidence interval	Adjusted odd ratio	95% confidence interval	*P*-value
ISS	4.7	1.1–20.7	---	---	---
Prothrombin time					
Pre-transfusion	8.2	0.9–71.0	---	---	---
Post-transfusion	2.4	0.6–9.1	---	---	---
Activated partial thromboplastin time					
Pre-transfusion	10.5	2.3–47.6	9.4	1.6-54.3	**0.01**
Post-transfusion	3.1	0.7–12.4	---	---	---
Coagulopathy					
Pre-transfusion	7.1	1.7–29.4	---	---	---
Post-transfusion	10.8	1.8–64.5	12.6	1.55-102.9	**0.01**

ISS: Injury severity score, Values in bold signify statistical significance.

clot kinetics and clot strength after platelet transfusion, suggested by alpha angle and maximum amplitude analyzed by TEG, also showed significant improvement. The findings can be attributed to the fresh frozen plasma, packed RBCs, and platelets transfused in the ratio of 1:1:1, a usual protocol followed in severely injured patients as a part of the early resuscitation strategy.[18] TEG aids in determining the type of blood product to be transfused in trauma patients.[19,20]

The rationale of platelet transfusion has remained focused on increasing the platelet counts and not platelet function. A previous study used a platelet function analyzer (100) and concluded that it is effective for supporting platelet transfusion decisions.[18] Another study suggests that a balanced transfusion strategy maintains normal hemostasis and has compared changes pre- and post-transfusion using thromboleastography in acutely bleeding patients.[21] Likewise, we also found improvement in TEG parameters post-transfusion. However, significant changes were not observed in flow cytometry-based parameters for platelet function. Further studies are required to determine the role of platelet transfusion in improving platelet function. The time period of assessment of platelet function of trauma patients requiring transfusion and the technique to be used also warrants further validation.

The mortality rate observed in the present study was less than one-third. The non-survivors had more severe thrombocytopenia, prolonged PT, and aPTT values after transfusion. The value of aPTT pre-transfusion was also significantly increased in the non-survivors. However, no significant differences were observed based on the thrombolelastography and flow cytometry-based parameters between the survivors and the non-survivors. Pre-transfusion aPTT value and post-transfusion coagulopathy were found to be independent risk factors for in-hospital mortality. The previous studies also suggest that early coagulopathy independently predicts mortality in trauma patients.[22-24] However, this study also highlights that coagulopathy persisting post-transfusion also predicts mortality. The coagulopathy associated with trauma could be either due to acute traumatic coagulopathy or iatrogenic due to hemodilution and depletion of factors during volume resuscitation. Patients with coagulopathy experience worse outcomes as compared to the ones with normal coagulation profiles.[25] The findings also suggest that clinically, the

conventional coagulation parameters continue to provide more information than the new and advanced methods in terms of survival of thrombocytopenic trauma patients and must be used for early identification of patients at risk of dying.

However, this study has certain limitations. First, the TEG could only be performed in a subset of the study population. Second, the refractoriness based on corrected count increment was not evaluated due to lack of relevant data on the body surface area of patients. In the future, studies should include results of D-dimer and fibrinogen for further correlation.

CONCLUSIONS

PT, coagulopathy status, clot kinetics, and clot strength improved in thrombocytopenic trauma patients after platelet transfusion. Pre-transfusion aPTT and coagulopathy persisting post-transfusion are independent risk factors for death in the settings of thrombocytopenic trauma patients. Early identification of the same using conventional parameters is recommended. The clinical significance of platelet function analysis in thrombocytopenic trauma patients undergoing transfusion therapy is yet to be ascertained.

Author contributions

TM: Concepts, design, definition of intellectual content, literature search, experimental studies, data acquisition, data analysis, statistical analysis, manuscript preparation, editing and review, AS: Concepts, design, definition of intellectual content, literature search, data analysis, statistical analysis, manuscript preparation, editing and review, VA: Design, definition of intellectual content, literature search, data acquisition, data analysis, statistical analysis, manuscript preparation, editing and review, AK: Data acquisition, data analysis, statistical analysis, manuscript preparation, editing and review, SP: Statistical analysis, manuscript preparation, editing and review, HP: Concepts, design, definition of intellectual content, manuscript preparation, editing and review.

Ethical approval

The Institutional Review Board at All India Institute of Medical Sciences, New Delhi approved the study; IECPG/97/2015 dated 2015.

Declaration of patient consent

Patient's consent not required as patients identity is not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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