Early fibrinogen replacement based on shock index and lactate levels in massive postpartum hemorrhage: a retrospective cohort study

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Background - Massive postpartum hemorrhage (PPH) remains a major cause of maternal morbidity and mortality worldwide. Timely fibrinogen replacement is critical in hemostatic resuscitation, yet laboratory delays may hinder early intervention. This study evaluates the clinical outcomes of early fibrinogen concentrate administration in massive PPH using a protocol based on shock index and serum lactate levels rather than laboratory-confirmed hypofibrinogenemia.

Materials and methods - This retrospective cohort study included 103 PPH patients treated at a tertiary hospital in Istanbul, Turkey, between 2016 and 2020. Patients were divided into four groups based on fibrinogen dose: Group I (<2 g), Group II (2-4 g), Group III (>4 g), and Group IV (non-massive PPH, no fibrinogen). A predefined protocol guided early fibrinogen administration based on clinical indicators. Fibrinogen was administered without awaiting lab confirmation.

Results - Group III had the highest estimated blood loss (2,600±500 mL) and Group IV the lowest (600±150 mL; p<0.001). ICU admission was significantly lower in Group III (23.8%) than in Group I (62.1%; p=0.020). Group III patients also had fewer secondary surgical interventions and reduced transfusion requirements compared to Groups I and II. No thromboembolic events or mortality were observed in any group.

Discussion - Early fibrinogen replacement based on shock index and lactate levels appears feasible and beneficial in managing massive PPH. This approach was associated with improved hemostatic control, reduced ICU admissions, and fewer surgical interventions. Prospective studies are warranted to further assess this strategy's safety, efficacy, and cost-effectiveness.

Keywords: *fibrinogen concentrate, postpartum hemorrhage, blood coagulation disorders, hemostatic agents.*

INTRODUCTION

Postpartum hemorrhage (PPH) remains one of the leading causes of maternal morbidity and mortality worldwide[1]. According to the Turkish National Maternal Mortality Study, PPH accounts for approximately 15.7% of maternal deaths and more than 50% of postpartum-related fatalities in Turkey[2]. The American College of Obstetricians and

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Gynecologists (ACOG) defines PPH as cumulative blood loss >1,000 mL or bleeding associated with signs of hypovolemia within the first 24 hours postpartum[3].

Massive PPH is characterized by blood loss exceeding 1000 mL, often leading to hemodynamic instability and coagulopathy due to impaired coagulation factor consumption[4]. This condition, coupled with hypovolemic shock, necessitates rapid intervention. Conventional resuscitation methods, including whole blood or erythrocyte suspension (ES) transfusion and fresh frozen plasma (FFP) administration, may be insufficient due to FFP's low fibrinogen concentration[5]. Furthermore, large-volume FFP transfusion increases the risk of transfusion-related acute lung injury (TRALI), a serious and potentially fatal complication[6].

TRALI is a non-cardiogenic pulmonary edema caused by donor-derived leukocyte antibodies or proinflammatory mediators present in transfused plasma[7]. It is characterized by acute respiratory distress syndrome (ARDS)-like symptoms, including severe hypoxemia, pulmonary infiltrates, and respiratory failure developing within six hours of transfusion. The incidence of TRALI is higher with FFP and platelet transfusions compared to red blood cell transfusions, due to the higher plasma content[8]. This risk underscores the importance of fibrinogen concentrate as a viable alternative, as it minimizes plasma transfusion exposure and its associated complications[9].

Fibrinogen plays a critical role in hemostasis, with plasma levels typically ranging from 200 to 450 mg/dL[10]. During pregnancy, fibrinogen levels rise to an average of 500 mg/dL[11]. A fibrinogen concentration below 200 mg/dL is a strong predictor of coagulopathy in PPH[12]. Replacement therapy can be performed using cryoprecipitate or fibrinogen concentrate. Cryoprecipitate requires thawing and carries a risk of viral or bacterial transmission[13]. In contrast, fibrinogen concentrate is a ready-to-use, lyophilized plasma-derived product that undergoes viral inactivation and does not require thawing[14].

The shock index, calculated by dividing the heart rate by the systolic blood pressure, is an early indicator of hemodynamic deterioration in patients with major PPH as well as in non-pregnant patients[15]. A shock index ≥1.5 in the presence of acute bleeding (heart rate >120/min, systolic blood pressure <90 mmHg) suggests a blood volume loss of approximately 40-50% and indicates significant coagulopathy[16].

Lactic acid accumulation and acute blood loss cause hypoperfusion that may lead to tissue ischemia. This is the basic mechanism of acidosis. Serum lactate levels above 2 mmol/L are considered abnormal, while levels exceeding 4 mmol/L are associated with significantly increased mortality if they do not normalize within 48 hours[17]. In our institution, serum lactate levels are reported within 10-15 minutes of admission and are therefore available prior to coagulation test results.

The clinical decision to initiate fibrinogen replacement therapy in this study was not solely based on laboratory-confirmed hypofibrinogenemia, but rather on predefined criteria involving shock index and lactate levels at admission. These parameters were used to stratify patients into risk groups and guide the initial fibrinogen dose. Although this constitutes a standardized institutional protocol, the retrospective nature of the study and the absence of randomization should be taken into account when interpreting causality.

Traditionally, fibrinogen replacement therapy is initiated based on laboratory-confirmed hypofibrinogenemia; however, waiting for coagulation test results can delay effective treatment. In our institution, fibrinogen concentrate is administered without waiting for laboratory results, based on shock index and serum lactate levels, in conjunction with surgical hemostasis and blood product replacement. This approach allows for faster hemostatic intervention in cases of massive PPH, where delayed treatment can lead to worsened outcomes.

This study evaluates fibrinogen concentrate use and its outcomes in massive PPH cases with coagulopathy. Furthermore, by including a control group of non-massive PPH patients who did not require fibrinogen therapy, this study also explores whether fibrinogen administration should be targeted based on bleeding severity and coagulation status rather than applied indiscriminately.

MATERIALS AND METHODS

This retrospective cohort study was conducted at a tertiary care hospital in Istanbul, Turkey, and included consecutive, non-selected patients diagnosed with postpartum hemorrhage (PPH) between January 1, 2016 and December 31, 2020. A total of 103 eligible patients

aged 19-42 years were included in the analysis. The study was approved by the Clinical Research Ethics Committee of Istanbul Medeniyet University Göztepe Training and Research Hospital (Date: 22.07.2020; Decision No: 2020/0483) and conducted in accordance with the Declaration of Helsinki. Due to the retrospective nature of the study, the requirement for written informed consent was waived.

Patients were categorized into four groups based on the amount of fibrinogen replacement therapy they received: Group I (<2 g fibrinogen, No.=29), Group II (2-4 g fibrinogen, No.=27), Group III (>4 g fibrinogen, No.=21), and Group IV (non-massive PPH, no fibrinogen therapy, No.=26). Patients in Groups I to III were considered to have massive PPH, defined as postpartum blood loss exceeding 1,000 mL along with clinical signs of hemodynamic instability and ongoing bleeding beyond the initial 1,000 mL. Eligibility for inclusion also required evidence of coagulopathy, defined as an INR >1.5, serum fibrinogen level <200 mg/dL, or platelet count <100×10^9/L. Patients in Group IV experienced blood loss <1,000 mL and did not require fibrinogen administration. Exclusion criteria included known coagulation disorders, pre-existing liver disease, ongoing sepsis, amniotic fluid embolism, or cases where PPH was managed successfully without fibrinogen therapy.

Due to a 40-50 minute delay in receiving laboratory test results, the initial decision to administer fibrinogen concentrate was based on clinical parameters. Shock index (heart rate divided by systolic blood pressure) and serum lactate levels were used as rapid hemodynamic markers for early intervention. Group I patients had a shock index of 0.8-1.5 and serum lactate <2 mmol/L, and received <2 g fibrinogen. Group II had a shock index ≥1.5 and lactate between 2-4 mmol/L, and received 2-4 g fibrinogen. Group III had a shock index ≥1.5 and lactate >4 mmol/L, and received >4 g fibrinogen. Once laboratory test results became available, additional doses of fibrinogen were calculated using the formula:

Required fibrinogen dose (g) = (200 mg/dL - baseline fibrinogen level)/35.

The target was to increase fibrinogen concentration to above 200 mg/dL to support hemostasis.

All patients were managed according to a standardized postpartum hemorrhage (PPH) protocol that included simultaneous implementation of surgical hemostasis (such as uterine massage, bimanual compression, intrauterine balloon tamponade, uterotonic agents, and, when necessary, surgical procedures including uterine artery ligation or hysterectomy), blood product replacement (erythrocyte suspension, fresh frozen plasma, platelets, and apheresis), and fibrinogen concentrate administration, which was initiated concurrently based on bleeding severity and clinical categorization at admission (**Figure 1**).

Clinical and laboratory parameters recorded for all patients included hemoglobin, hematocrit, platelet count, serum lactate, PT-INR, and fibrinogen levels at admission and post-treatment. Data on the volume of transfused blood products, total fibrinogen administered, need for ICU admission, duration of ICU stay, requirement for secondary surgery, and any complications were also collected. Categorical variables were compared using the chi-square test, while continuous variables were analyzed using Welch's t-test. A p-value of <0.05 was considered statistically significant. All analyses were performed using IBM SPSS Statistics for Windows, Version 22.0 (IBM Corp., Armonk, NY, USA).

RESULTS

A total of 103 patients with postpartum hemorrhage were included in the study, of whom 77 experienced massive PPH requiring fibrinogen concentrate, while 26 had non-massive PPH and did not receive fibrinogen therapy. Patients were categorized into four groups based on fibrinogen replacement therapy: Group I (<2 g fibrinogen, No.=29), Group II (2-4 g fibrinogen, No.=27), Group III (>4 g fibrinogen, No.=21), and Group IV (non-massive PPH, no fibrinogen, No.=26). Fibrinogen concentrate was administered at the time of admission based on clinical parameters, without waiting for laboratory confirmation, allowing early intervention. There was no significant difference between the four groups regarding maternal age, type of delivery, or obstetric complications such as preeclampsia, uterine atony, placental abruption, and placenta previa (p>0.05 for all). Vaginal delivery rates were highest in Group IV (51%) compared to Group I (24.1%), Group II (33.3%), and Group III (42.9%). The demographic characteristics of the study population are summarized in **Table I**.

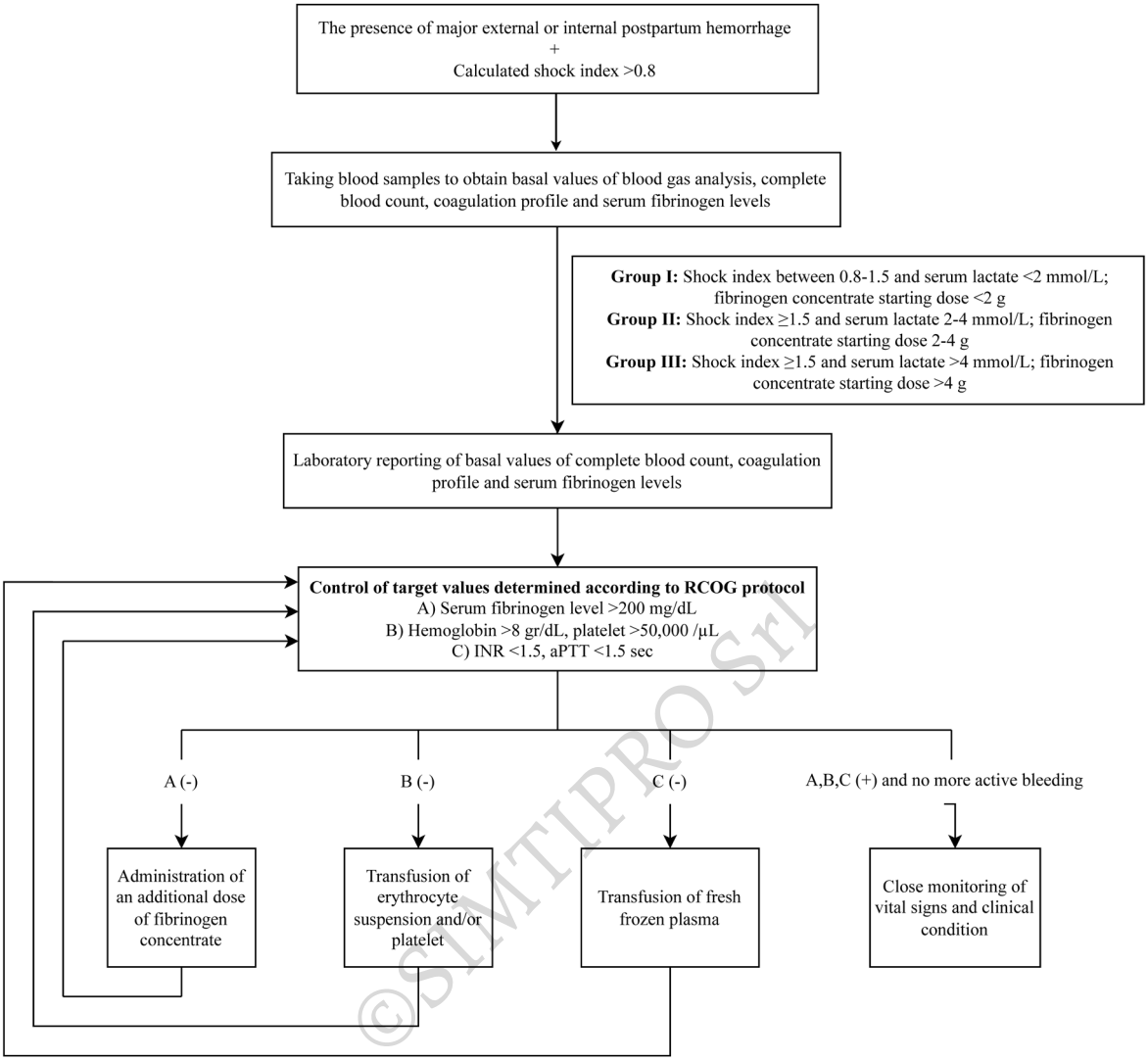

Figure 1 - Algorithm for simultaneous administration of fibrinogen concentrate and blood product replacement with surgical management in patients with major postpartum hemorrhage

At the time of admission, serum fibrinogen levels were significantly lower in Group III (126.71±56.91 mg/dL) compared to Group IV (325.42±42.77 mg/dL, p=0.001). Shock index values were highest in Group III (2.48±0.59) and lowest in Group IV (1.32±0.28, p=0.020). Lactate levels were also significantly elevated in Group III (6.49±2.25 mmol/L) compared to Group IV (1.10±0.29 mmol/L, p=0.001). PT-INR was significantly higher in Group III (p=0.005), whereas hemoglobin, hematocrit, and platelet count did not differ significantly between the groups. Patients in Group IV had the highest platelet count (185.3±50.8×10³/µL), whereas Group III had the lowest (90.28±42.25×10³/µL). The basal laboratory values of patients at the time of admission are shown in **Table II**.

The need for erythrocyte suspension (ES), fresh frozen plasma (FFP), and platelet transfusion was significantly higher in Group III compared to Group I, II, and IV (p=0.001 for all comparisons). Patients in Group III received a mean of 14.14±9.70 ES units, whereas Group I and II required 5.17±4.24 and 7.96±3.89 ES units, respectively. Patients in Group IV required minimal transfusion support, with an average of 0.5±0.3 ES units and 0.2±0.1 FFP units. Platelet transfusion was highest in Group III (8.14±5.42 units) and lowest in Group IV (0.0 units). The distribution of

blood product transfusion requirements among groups is detailed in **Table III**.

ICU admission rates were significantly higher in Group I (62.1%) compared to Group IV (5.8%, p=0.020). The need for ICU admission was also lower in Group III (23.8%) compared to Groups I and II. The total number of ICU hospitalization days was longest in Group III (2.42±3.17 days), but this difference was not statistically significant. The requirement

Table I - *Demographic characteristics of the patients*

Variable	Group I (No.=29, 37.6%)	Group II (No.=27, 35.2%)	Group III (No.=21, 27.2%)	Group IV (No.=26, 25.0%)	p value
Age (Mean±SD)	28.9±3.96	29.55±4.61	31.19±5.77	30.12±4.38	NS
Type of delivery					NS
Vaginal delivery (%)	24.1	33.3	42.9	51.0	
Cesarean section (%)	75.9	66.7	57.1	49.0	
Uterine atony (%)	58.6	59.3	38.1	30.0	NS
Preeclampsia (%)	13.8	29.6	42.9	10.2	NS
Placental abruption (%)	13.9	25.9	38.2	9.5	NS
Placenta previa (%)	24.1	18.5	0.0	4.5	NS
Uterine rupture (%)	6.9	11.1	23.8	0.0	NS

Group I includes patients receiving <2 g fibrinogen concentrate, Group II includes patients receiving 2-4 g fibrinogen concentrate, and Group III includes patients receiving >4 g fibrinogen concentrate. Group IV consists of non-massive PPH patients who did not require fibrinogen therapy.
SD: standard deviation; NS: not statistically significant.

Table II - *Basal laboratory values of postpartum hemorrhage patients at the time of admission*

Variable	Group I (No.=29)	Group II (No.=27)	Group III (No.=21)	Group IV (No.=26)	p value
Shock Index	1.91±0.62	2.14±0.37	2.48±0.59	1.32±0.28	0.020*
Hb (g/dL)	8.14±1.68	7.71±1.94	7.46±1.88	10.2±1.75	NS
Hct (%)	24.38±4.93	23.22±5.72	22.23±5.42	31.1±4.21	NS
Plt (10^3/µL)	119.13±66.94	101.13±44.09	90.28±42.25	185.3±50.8	NS
PT-INR	1.12±0.64	1.26±0.54	1.82±1.02	1.00±0.15	0.005*
Lactate (mmol/L)	1.46±0.31	2.59±1.91	6.49±2.25	1.10±0.29	0.001*
Serum fibrinogen (mg/dL)	220.31±105.8	163.07±71.72	126.71±56.91	325.42±42.77	0.001*

Values are presented as mean ± standard deviation (Mean±SD). Group I includes patients receiving <2 g fibrinogen concentrate, Group II includes patients receiving 2-4 g fibrinogen concentrate, and Group III includes patients receiving >4 g fibrinogen concentrate. Group IV consists of non-massive PPH patients who did not require fibrinogen therapy. PT-INR: prothrombin time-international normalized ratio; NS: not statistically significant; *p <0.05 CI (confidence interval).

Table III - *Distribution of patients' need for blood product replacement between groups*

Variable	Group I (No.=29)	Group II (No.=27)	Group III (No.=21)	Group IV (No.=26)	p value
Erythrocyte suspension (unit)	5.17±4.24	7.96±3.89	14.14±9.70	0.5±0.3	0.001*
Fresh frozen plasma (unit)	6.17±5.77	6.89±4.23	11.9±5.9	0.2±0.1	0.001*
Platelet transfusion (unit)	2.17±3.14	2.29±3.14	8.14±5.42	0.0	0.001*
Apheresis (unit)	1.13±1.43	1.18±1.08	2.52±2.67	0.0	0.014*

Group I includes patients receiving <2 g fibrinogen concentrate, Group II includes patients receiving 2-4 g fibrinogen concentrate, and Group III includes patients receiving >4 g fibrinogen concentrate. Group IV consists of non-massive PPH patients who did not require fibrinogen therapy. *p <0.05 CI (confidence interval).

for secondary surgical intervention was highest in Group I (89.7%), followed by Group II (85.2%) and Group III (42.9%, p=0.001). Patients in Group IV had the lowest reoperation rate (3.9%). The need for ICU admission, ICU hospitalization days, and secondary surgical interventions between groups are displayed in **Table IV**.

Table IV - *Distribution of patients' need for ICU admission, total number of ICU hospitalization days, and secondary surgery needs between groups*

Variable	Group I (No.=29)	Group II (No.=27)	Group III (No.=21)	Group IV (No.=26)	p value
Need for ICU admission (%)	62.1	37.0	23.8	5.8	0.020*
Total number of ICU hospitalization days (Mean±SD)	0.62±2.38	1.00±3.45	2.42±3.17	0.00±0.00	NS
Secondary surgery needs (%)	89.7	85.2	42.9	3.9	0.001*

Group I includes patients receiving <2 g fibrinogen concentrate, Group II includes patients receiving 2-4 g fibrinogen concentrate, and Group III includes patients receiving >4 g fibrinogen concentrate. Group IV consists of non-massive PPH patients who did not require fibrinogen therapy.
ICU: intensive care unit; NS: not statistically significant; *p <0.05 CI (confidence interval).

No cases of pulmonary embolism, deep vein thrombosis, or disseminated intravascular coagulation (DIC) were recorded in any group. Minor complications included acute renal failure in one patient (1.29%, Group I), acute tubular necrosis in two patients (2.59%, Group II), and temporary vision loss in one patient (1.29%, Group III). Subdural hematoma, low oxygen saturation, and right forearm basilic vein thrombosis each occurred in one patient (1.29%). All complications resolved with supportive treatment, and no patients required long-term medical intervention. No mortality was observed in any group. Complications and mortality rates among the study population are presented in **Table V**.

Table V - *Complications after fibrinogen concentrate application, number of patients developing complications and mortality rate*

Complication	Number of patients No. (%)	Clinical course	Sequelae
Acute renal failure	1 (1.29%)	CRF	CRF-still on dialysis
Right forearm basilic vein thrombosis	1 (1.29%)	Cure	None
Acute tubular necrosis	2 (2.59%)	Cure	None
Subdural hematoma	1 (1.29%)	Cure	None
Temporary loss of vision	1 (1.29%)	Cure	None
Low oxygen saturation	2 (2.59%)	Cure	None
Mortality	0 (0%)	-	-

DISCUSSION

The management of massive postpartum hemorrhage (PPH) requires a combination of surgical interventions, blood transfusion, and targeted coagulation therapy. Early correction of hypofibrinogenemia has been increasingly recognized as a key strategy to improve maternal outcomes[1]. The use of fibrinogen concentrate in major PPH has gained increasing interest, with studies suggesting its efficacy in rapidly correcting hypofibrinogenemia[2,3]. The results of this study demonstrate that fibrinogen replacement therapy initiated based on clinical parameters rather than laboratory confirmation was associated with favorable maternal outcomes. By utilizing shock index and serum lactate levels as primary indicators for early intervention, we were able to administer fibrinogen concentrate in a timely manner, reducing the need for ICU admission and secondary surgical interventions.

Our findings indicate that patients receiving more than 4 g of fibrinogen replacement had significantly lower ICU admission rates and reduced need for secondary surgical interventions. This effect is likely attributed to fibrinogen's critical role in hemostatic mechanisms. While vasoconstriction and platelet aggregation contribute to initial hemostasis, fibrinogen is essential for clot stability[4]. Without adequate fibrinogen, platelets fail to form a stable clot, leading to ineffective hemostasis and increased bleeding risk[5]. Fibrinogen acts as a structural

bridge between platelets, facilitating the formation of a stable fibrin network, which is critical in controlling massive bleeding[6,18]. Inadequate fibrinogen replacement leads to accelerated platelet consumption, worsening coagulopathy, and increasing the risk of disseminated intravascular coagulation (DIC)[7,18]. Therefore, maintaining fibrinogen levels above 250 mg/dL is crucial in patients with ongoing active bleeding.

Additionally, we predict that patients who receive early fibrinogen replacement and maintain levels above 200 mg/dL will have reduced erythrocyte suspension (ES), fresh frozen plasma (FFP), and platelet transfusion requirements. This is because fibrinogen is the most essential factor for clot stability, and in its deficiency, platelets fail to form a solid clot and are rapidly consumed, accelerating the progression toward consumptive coagulopathy[18]. Given this, early fibrinogen replacement not only prevents excessive platelet consumption but also reduces the likelihood of requiring extensive blood product transfusions.

This study differs from previous trials by implementing a standardized early fibrinogen replacement protocol that was based on predefined clinical criteria (shock index and serum lactate levels) rather than relying solely on laboratory-confirmed hypofibrinogenemia. In emergency settings such as massive PPH, waiting for laboratory results can delay timely intervention. Instead, our protocol enabled the prompt administration of fibrinogen concentrate at admission, followed by dose adjustments based on laboratory data. The results suggest that this approach stabilized hemostasis more efficiently, reduced transfusion requirements, and minimized the need for surgical interventions, supporting the use of empirical fibrinogen therapy in severe PPH cases.

Our findings align with prior observational studies suggesting that fibrinogen concentrate administration in massive PPH patients reduces ICU admission and the need for secondary surgical intervention[9,10]. However, the retrospective nature of this study limits causal inference. While our results indicate that early fibrinogen administration may improve maternal outcomes, randomized controlled trials (RCTs), such as the FIB-PPH trial, have suggested that fibrinogen concentrate does not significantly reduce transfusion requirements compared to standard treatment[11]. Future RCTs should further evaluate the optimal timing of fibrinogen administration and whether clinical decision-making based on shock index and lactate levels provides superior outcomes compared to laboratory-based treatment protocols.

Previous studies have reported mixed results regarding the efficacy of fibrinogen concentrate in reducing transfusion requirements. A systematic review found that fibrinogen concentrate did not significantly reduce the need for erythrocyte transfusion in PPH patients, but it was associated with lower ICU admission rates[12]. Our findings suggest that initiating fibrinogen therapy without waiting for laboratory confirmation reduces the need for secondary surgery, possibly by enabling earlier and more effective hemostasis[13]. The absence of severe thromboembolic events in our study is consistent with prior literature, indicating a low risk of hypercoagulability with fibrinogen concentrate use[14]. Furthermore, the inclusion of a non-massive PPH control group allowed for a more precise comparison, confirming that patients with lower blood loss and stable coagulation parameters do not benefit from fibrinogen administration.

Current guidelines recommend viscoelastic hemostatic tests such as rotational thromboelastometry (ROTEM) and thromboelastography (TEG) to guide fibrinogen replacement therapy[15-17]. These tests provide real-time assessment of coagulation dynamics and facilitate targeted fibrinogen replacement strategies. However, our study did not incorporate viscoelastic testing, which is a limitation. Future studies should integrate ROTEM/TEG-based algorithms to optimize fibrinogen administration and assess their impact on clinical outcomes in massive postpartum hemorrhage. Additionally, further research is required to determine whether fibrinogen concentrate should be administered empirically in severe postpartum hemorrhage cases or if ROTEM/TEG-based thresholds should guide its use. Given that fibrinogen is the first coagulation factor to reach critically low levels in massive obstetric hemorrhage, the ability to assess fibrinogen function in real-time could significantly impact patient management and reduce unnecessary clotting factor administration[19,20].

One of the primary concerns regarding fibrinogen concentrate administration is the potential risk of thromboembolic events due to hypercoagulability. Our study did not observe any cases of deep vein thrombosis

or pulmonary embolism, which is consistent with previous reports indicating a low risk of thrombosis with fibrinogen replacement in postpartum hemorrhage[21]. However, the relatively short follow-up period may limit the detection of delayed thrombotic events. Future prospective studies should include long-term monitoring for thromboembolic complications, particularly in high-risk obstetric populations. As fibrinogen concentrate is a purified clotting factor, the theoretical risk of excessive fibrin deposition leading to thrombosis remains a concern, particularly in patients with preeclampsia, gestational diabetes, or thrombophilia.

While large doses of fibrinogen concentrate theoretically increase the risk of thrombosis, no significant thromboembolic complications were observed in our study population. This aligns with prior research suggesting that fibrinogen concentrate may be a safe alternative to fresh frozen plasma in patients with massive hemorrhage[22]. Additionally, fibrinogen concentrate offers practical advantages over fresh frozen plasma, including its rapid availability, elimination of the need for thawing, and reduced risk of transfusion-related complications such as transfusion-related acute lung injury (TRALI)[23]. In our study, non-massive postpartum hemorrhage patients did not receive fresh frozen plasma, whereas massive postpartum hemorrhage patients who required fibrinogen concentrate received significantly more fresh frozen plasma and erythrocyte transfusions, suggesting that fibrinogen concentrate may serve a critical role in reducing overall transfusion requirements in the setting of severe postpartum hemorrhage.

Despite these promising findings, the study has several limitations. First, its retrospective design and lack of randomization may introduce selection bias, limiting the ability to infer causality. Second, although the clinical protocol for fibrinogen administration was predefined, individual clinician decisions may have influenced the timing and dosing. Third, the absence of ROTEM or TEG monitoring may have limited the precision of coagulation assessment.

CONCLUSIONS

Early fibrinogen replacement based on clinical indicators such as shock index and serum lactate level appears to be a safe and effective approach for managing massive postpartum hemorrhage. This strategy was associated with reduced ICU admissions and surgical interventions without increasing thromboembolic risk. Fibrinogen concentrate enables rapid and targeted correction of hypofibrinogenemia and may improve maternal outcomes. However, larger prospective trials are warranted to confirm these results and optimize dosing protocols.

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AUTHOR CONTRIBUTIONS

MK and EGÖ contributed to the conceptualization and methodology of the study. MK, EGÖ, and AK were responsible for study design and project administration. EGÖ and SÖ performed data curation, while MK and EGÖ conducted the formal analysis. SÖ, SS, and MP contributed to investigation and data collection. MK and EGÖ prepared the original draft of the manuscript, and all authors (MK, EGÖ, SÖ, SS, MP) participated in reviewing and editing. AK supervised the entire study and provided final validation. All authors reviewed and approved the final version of the manuscript.

The Authors declare no conflicts of interest.

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