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Case Report

ROTEM-Guided Management of Postpartum Hemorrhage in Previously Unrecognized Glanzmann Thrombasthenia: A Case Report

Borislav Silaev[1], Olga Beznoshchenko[1*], Zulfiya Khodzhaeva[1], Elmira Mekhieva[1], Anastasia Ignatova[2], Tatiana Fedorova[1], Gennady Galstyan[1,3], Gennady Sukhikh[1]

[1]Department of Anesthesiology-Intensive Care, National Medical Research Center for Obstetrics, Gynecology and Perinatology named after Academician V.I. Kulakov, Moscow, Russia

[2]Laboratory of Cellular Hemostasis and Thrombosis, Dmitry Rogachev National Research Center of Pediatric Hematology, Oncology and Immunology, Moscow, Russia

[3]Department of Anesthesiology-Intensive Care, "National Medical Research Center of Hematology", Moscow, Russia

[*]**Corresponding author**: Olga S. Beznoshchenko, Department of Anesthesiology-Intensive Care, V.I. Kulakov National Medical Research Center for Obstetrics, Gynecology and Perinatology, Moscow, Russia

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Abstract

Postpartum hemorrhage (PPH) in patients with rare inherited platelet function disorders is difficult to diagnose promptly because specialized platelet testing is often unavailable in the acute setting. A 36-year-old multiparous woman with a longstanding prior diagnosis of von Willebrand disease (VWD) underwent emergency cesarean delivery at term. Despite an estimated intraoperative blood loss of 750 mL, she developed early postoperative wound oozing and progressive mucocutaneous bleeding manifestations with a marked hemoglobin decrease. Point-of-care rotational thromboelastometry (ROTEM) showed reduced clot firmness with a disproportionately low platelet contribution to clot strength (low EXTEM A5 with a low PLTEM), while clotting time and fibrinolysis parameters were within reference ranges. ROTEM-guided, goal-directed hemostatic therapy was initiated according to an A5-based obstetric bleeding algorithm. Subsequent platelet function testing (optical aggregometry) and flow cytometry demonstrated a severe αIIbβ3 (GPIIb/IIIa) defect, confirming Glanzmann thrombasthenia (GT). In this case, ROTEM provided rapid point-of-care evidence of markedly reduced platelet contribution to clot formation, enabling early targeted hemostatic management and prompting definitive diagnostic testing for GT. The case underscores how viscoelastic testing can guide therapy and accelerate the diagnosis of rare platelet disorders in acute obstetric hemorrhage.

Keywords: Postpartum Hemorrhage; Glanzmann Thrombasthenia; Rotational thromboelastometry; Platelet function; von Willebrand disease; Case Report

Introduction

Managing postpartum hemorrhage (PPH) presents a significant challenge in obstetrics, particularly when underlying hemostatic disorders are involved. The diagnosis and management of hereditary platelet function disorders rely largely on case reports and small series because these conditions are rare. Pregnant patients with inherited platelet dysfunction are of particular concern, as severe bleeding may affect both mother and neonate. Glanzmann thrombasthenia (GT) is a congenital platelet function disorder caused by deficiency or dysfunction of the platelet integrin αIIbβ3 (GPIIb/IIIa). While pregnancy often proceeds similarly to that in the general population, PPH is more frequent and may be severe, requiring transfusion support [1,2]. The lack of specific clinical features complicates diagnosis, and standard platelet aggregation testing or flow cytometry is typically not available during acute PPH. Therefore, detailed accounts of rapid diagnostic approaches and goal-directed treatment, such as this report, retain clinical relevance. This report describes the diagnostic evaluation and management of PPH in a woman with previously unrecognized GT who was admitted for delivery with a prior diagnosis of VWD.

Case Presentation

A 36-year-old woman with a prior diagnosis of VWD (type not specified) was admitted for planned operative delivery. The obstetric diagnosis at admission was term pregnancy (38 weeks' gestation), cephalic presentation, and a uterine scar after two previous cesarean deliveries.

Medical history

VWD was diagnosed at age 16. The patient reported a moderate bleeding history, including ecchymoses and mucosal bleeding (gingival bleeding and epistaxis). For menorrhagia, she previously received a plasma-derived factor VIII concentrate containing von Willebrand factor (vWF) (Wilate®). In 2016, she underwent surgery for endometriosis without bleeding complications; routine hemostasis tests were reportedly within reference ranges.

Obstetric history

2012: Term cesarean delivery with intraoperative cell salvage and concurrent right ovarian resection for an endometrioid cyst; no excessive maternal bleeding. The neonate developed bilateral grade 1 intraventricular hemorrhage (IVH) in the early neonatal period.

2017: Term repeat cesarean delivery with intraoperative cell salvage; estimated blood loss 800 mL. The neonate developed ecchymoses and bleeding from injection sites within 24 hours. The mother developed postpartum cutaneous bleeding manifestations and gross hematuria; hypocoagulability on native thromboelastography (TEG) was interpreted as VWD, and vWF-containing factor VIII replacement therapy was administered. She was discharged on postpartum day 7 in satisfactory condition.

2024 pregnancy: Followed locally and referred to the tertiary center after telemedicine consultation.

Presentation and perioperative course

On admission, the patient was in satisfactory condition. Petechiae and hematomas were present on the medial aspect of the left lower leg and on the abdomen. Laboratory data are summarized in Table 1.

During the first 24 hours of hospitalization, she developed a fever up to 38.0°C with chills and without upper respiratory symptoms (respiratory rate 17/min; blood pressure 121/78 mmHg; pulse 96/min). Emergency cesarean delivery was performed because of suspected uterine scar dehiscence after two prior cesarean deliveries. Spinal anesthesia was uneventful.

Estimated intraoperative blood loss was 750 mL. Oozing from puncture sites was noted intraoperatively; tranexamic acid (2 g IV) and vWF-containing factor VIII concentrate (Wilate®, 3600 IU) were administered. A term male neonate (3320 g, 51 cm) was delivered and had a "*blueberry muffin*" like purpuric rash at birth.

Within 2 hours postoperatively, the surgical dressing became saturated with blood, and abdominal petechiae/ecchymoses progressed (Figure 1A).

Point-of-care hemostasis assessment (ROTEM)

Point-of-care ROTEM was performed to evaluate the hemostatic profile. ROTEM revealed reduced clot amplitude at 5 minutes in FIBTEM (A5_FIBTEM <12 mm) and reduced clot firmness in EXTEM and INTEM, consistent with reduced overall clot strength. Clotting time parameters were within reference ranges and no hyperfibrinolysis was detected (Figure 2A-C). ROTEM results were interpreted using an A5-based obstetric bleeding algorithm (Figure 3).

At the time of evaluation, FVIII:C and vWF activity were elevated (Table 1), making clinically significant VWD-related deficiency unlikely in this acute setting. In accordance with the institutional ROTEM-guided algorithm, hemostatic therapy was initiated with 16 units of cryoprecipitate.

After cryoprecipitate transfusion, A5_FIBTEM increased to the target range (Figure 2F). However, the platelet contribution to clot strength remained markedly reduced: PLTEM = A5_EXTEM -A5_

FIBTEM = 3 mm, consistent with severe impairment of platelet-dependent clot firmness. Bleeding manifestations progressed (Figure 1B), and hemoglobin decreased while routine coagulation tests remained within reference ranges (Table 1). Based on the clinical picture and ROTEM pattern, a platelet function disorder was suspected. According to the algorithm, in the presence of clinically significant bleeding with A5_FIBTEM >12 mm and A5_EXTEM <35 mm, platelet transfusion was indicated.

Following transfusion of 1 unit of apheresis platelets (2×10^11 platelets), ROTEM continued to show low platelet contribution (PLTEM 5 mm; Figure 2G-I). Clinically, however, bleeding manifestations resolved. Hemoglobin decreased to 75 g/L, and 2 units of packed red blood cells were transfused for post-hemorrhagic anemia.

Definitive hemostasis testing

Blood smear evaluation showed normal platelet morphology. Optical aggregometry using ADP, epinephrine, collagen, and ristocetin demonstrated aggregation only with ristocetin (50%), a pattern consistent with GT. Flow cytometry confirmed markedly reduced αIIbβ3 (CD61) expression and reduced PAC-1 binding after activation (Table 2), establishing the diagnosis of GT.

Parameter (unit; reference range)	On admission	After transfusion of 16 units of cryoprecipitate	After transfusion of platelet concentrate
Hemoglobin (g/L; 110-139)	108	81	75
Hematocrit (L/L; 0.295-0.395)	0.331	0.246	0.237
Platelet count (×10^9/L; 118–340)	253	215	230
WBC (×10^9/L; 6.0-14.61)	10.7	13.4	11.7
Fibrinogen (g/L; 3.5-5.5)	3.61	4.10	3.75
INR (0.8-1.2)	0.98	0.97	0.94
APTT (s; 20-38)	26.6	25.7	24.6
FVIII:C (%; 50-150)	183.3	231.7	231.7
vWF:RCo (%; 48-202)	284.9	454.5	463.2
vWF:Ag (%)	Not measured	502.8	440.5

Table 1: Laboratory values.

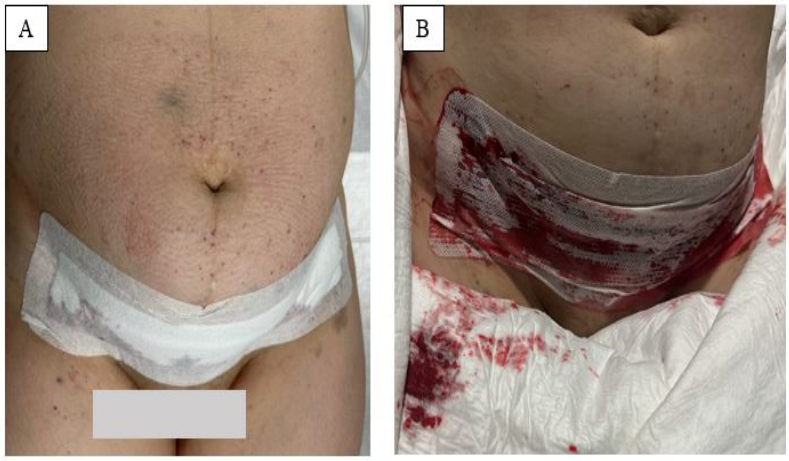

Figure 1: Postpartum bleeding manifestations in patient M. **(A)** Early postoperative wound oozing and abdominal petechiae/ecchymoses. **(B)** Progression of cutaneous bleeding manifestations prior to platelet transfusion.

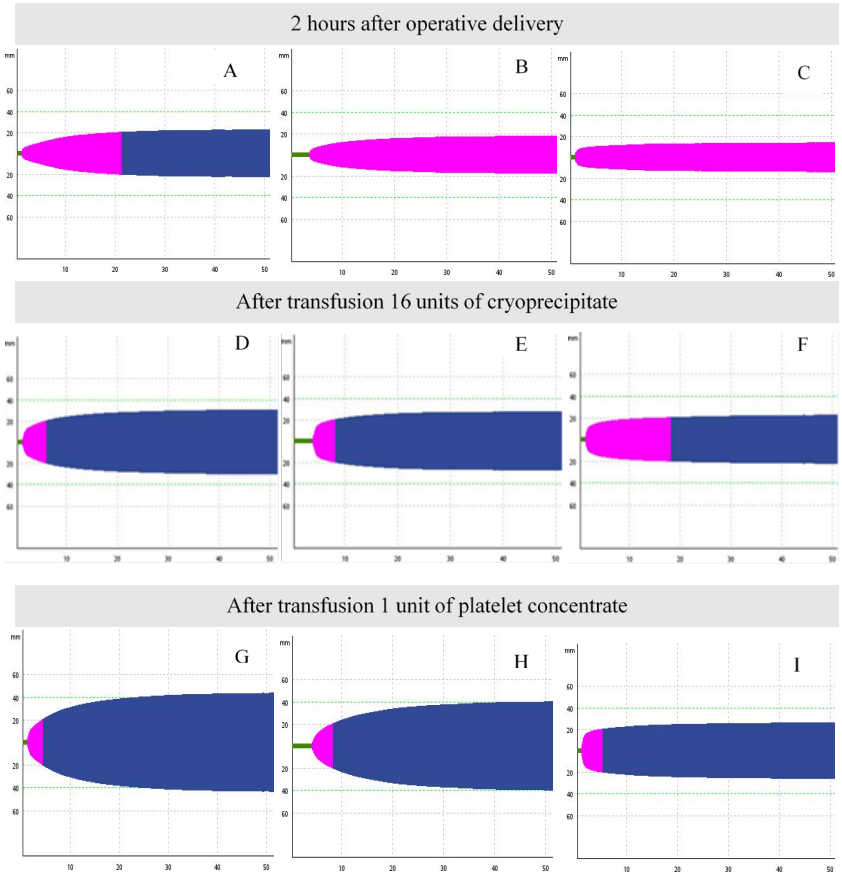

Figure 2: ROTEM tracings and parameters at three time points: **(A-C)** 2 hours after cesarean delivery; **(D-F)** after transfusion of 16 units of cryoprecipitate; **(G-I)** after transfusion of 1 unit of apheresis platelets.

Note: CT, clotting time (s); A5, clot amplitude at 5 minutes after CT (mm); ML, maximum lysis (%).

A. EXTEM: CT 62 (32-67) s; A5 12 (43–64) mm; ML 2 (0-18)%.

B. INTEM: CT 209 (102-240) s; A5 11 (41–63) mm; ML 1 (0-16)%.

C. FIBTEM: CT 55 (32-67) s; A5 10 (12-28) mm; ML 0 (0-3.6)%.

D. EXTEM: CT 68 (32-67) s; A5 20 (43-64) mm; ML 1 (0-18)%.

E. INTEM: CT 219 (102-240) s; A5 21 (41-63) mm; ML 0 (0-16)%.

F. FIBTEM: CT 67 (32-67) s; A5 17 (12-28) mm; ML 0 (0-3.6)%.

G. EXTEM: CT 61 (32-67) s; A5 25 (43-64) mm; ML 0 (0-18)%.

H. INTEM: CT 237 (102-240) s; A5 22 (41-63) mm; ML 0 (0-16)%.

I. FIBTEM: CT 67 (32-67) s; A5 20 (12-28) mm; ML 0 (0-3.6)%.

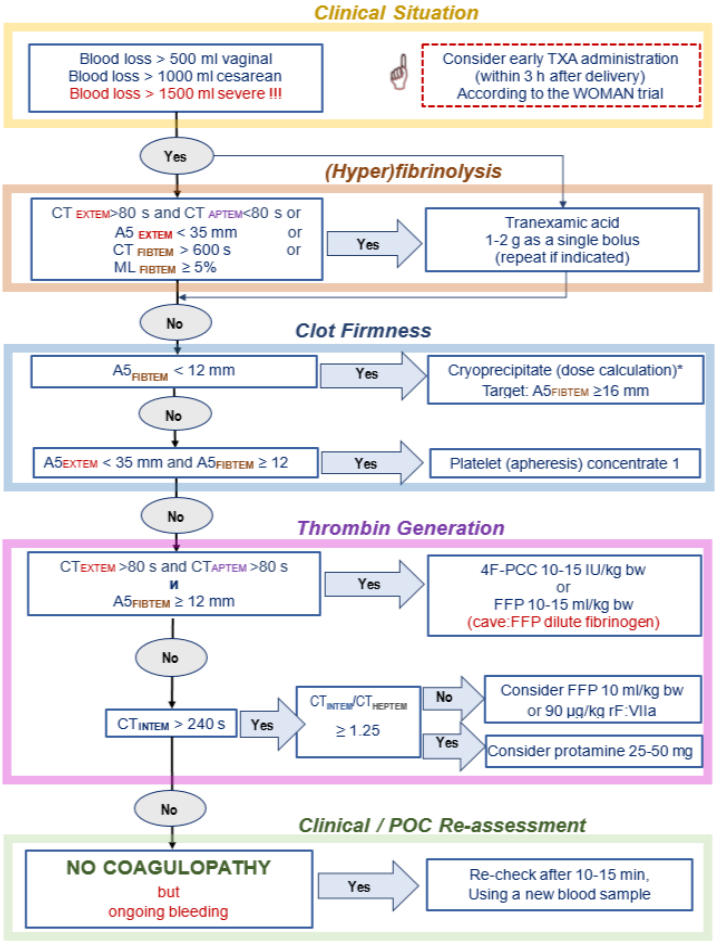

Figure 3: A5-based "*Obstetric Bleeding*" algorithm adapted from Görlinger et al. [5]. CT, clotting time (s); A5, amplitude at 5 minutes after CT (mm); ML, maximum lysis (%). 4F PCC, four-factor prothrombin complex concentrate; FFP, fresh frozen plasma. Cryoprecipitate calculation as shown in the adapted algorithm.

*Cryoprecipitate (units) = (target A5 FIBTEM - patient A5 FIBTEM) / 24 × body weight (kg).

Parameter	Control group	Result
Glycoprotein (GP) IIIa (CD61 antigen): *at rest, a.u.* *after activation, a.u.*	 78-118 208-302	 **2** **2**
PAC-1 binding (activated GP IIb/IIIa): *at rest, a.u.* *after activation, a.u.*	 2.5-5 31-132	 3.1 **5**
GP Ib (CD42b antigen): *at rest, a.u.* *after activation, a.u.*	 58-128 28-67	 114 44
Fluorescence of mepacrine-loaded dense granules: *at rest, a.u.* *after activation, a.u.*	 63-127 16-32	 106 28
P-selectin of α-granules (CD62p antigen) *at rest, a.u.* *after activation, a.u.*	 <5 80-140	 2.3 82
Procoagulant platelets (by annexin V) *at rest, %* *after activation, %*	 <2 2-30	 0.4 10.8
Note: Total αIIbβ3 expression was markedly reduced both at rest (CD61) and after activation (PAC-1). Dense granule content and release (mepacrine) and α-granule degranulation (CD62P) were within reference ranges; procoagulant activity (Annexin V) was within reference ranges.		

Table 2: Platelet flow cytometry (a.u., arbitrary units).

Hospital course and specialized management

For further management, the patient was transferred to the National Medical Research Center of Hematology (Ministry of Health of Russia). On admission, she was in serious condition due to ongoing bleeding, including a subaponeurotic hematoma at the surgical incision site, diffuse cutaneous bleeding manifestations, and active wound bleeding.

Additional sutures were placed. Hemostatic therapy included platelet transfusions and continuous intravenous recombinant activated factor VII (rFVIIa; eptacog alfa [activated]) at 20 µg/kg/h, resulting in decreased bleeding intensity.

Ultrasound revealed a resolving anterior abdominal wall intermuscular hematoma (anechoic band up to 12 mm). The uterus measured 100×68×10 mm, with the cavity dilated to 37 mm and containing mixed echogenic material; hematometra was diagnosed. On the following day, vacuum aspiration of the uterine cavity was performed under total intravenous anesthesia. Hemostasis was supported by a bolus dose of rFVIIa (7.2 mg; 120 µg/kg) and platelet transfusion. In total, 5 units of platelet concentrate were transfused during hospitalization. Continuous rFVIIa infusion at 20 µg/kg/h was continued with gradual tapering to discontinuation. The patient was discharged home in satisfactory condition 19 days later.

Genetic analysis and family history

Whole-exome sequencing in the newborn male identified a previously undescribed homozygous variant in ITGA2B (17-44385042-GAA-G), resulting in a frameshift and predicted truncation of the protein (p.Phe235LeufsTer20, NM_000419). This variant was absent from gnomAD v4.1.0 and was classified as likely pathogenic based on available evidence. Homozygous and compound heterozygous variants in ITGA2B are associated with GT (OMIM: 273800), whereas heterozygous variants of other types have been described in autosomal dominant platelet-type bleeding disorders with milder phenotypes (OMIM: 187800).

The patient's marriage was consanguineous (the husband is her mother's second cousin). The husband reported a bleeding history (epistaxis, gingival bleeding, ecchymoses) and had been evaluated in childhood for suspected VWD; current symptoms were mild. The couple's two daughters also had a bleeding history and had reportedly been diagnosed with GT in a pediatric hematology center (documentation unavailable).

Discussion

Hereditary disorders of hemostasis may present with similar mucocutaneous bleeding phenotypes. Both GT and VWD commonly manifest as petechiae, ecchymoses, mucosal bleeding, and menorrhagia [6]. Notably, VWD testing during pregnancy can be challenging because physiologic increases in FVIII and vWF may mask an underlying deficiency [7]. In this case, FVIII:C and vWF activity were elevated at presentation, making clinically significant deficiency less likely in the acute peripartum period, although certain VWD subtypes (e.g., type 2A) cannot be fully excluded without multimer analysis.

The diagnostic hallmark of GT is absent or markedly reduced platelet aggregation in response to multiple agonists, with preserved ristocetin-induced aggregation, along with reduced/abnormal αIIbβ3 expression or function on flow cytometry [4]. In the acute setting of PPH, however, platelet aggregometry and flow cytometry are usually unavailable, necessitating rapid bedside assessment tools.

Viscoelastic testing (TEG/ROTEM) has proven clinically useful in severe obstetric hemorrhage to support goal-directed transfusion and hemostatic therapy [12]. ROTEM does not diagnose VWD [13], but it can rapidly indicate whether reduced clot strength is driven predominantly by fibrinogen deficiency (FIBTEM) or by reduced platelet contribution (EXTEM vs FIBTEM). In this patient, ROTEM demonstrated persistently reduced platelet contribution to clot firmness despite correction of fibrinogen parameters, prompting platelet transfusion and targeted diagnostic workup.

Bleeding management in GT may include uterotonics, antifibrinolytics, platelet transfusion, and rFVIIa; however, platelet transfusion carries a risk of alloimmunization and platelet transfusion refractoriness [16]. rFVIIa can act as a bypassing agent to enhance thrombin generation on activated platelet surfaces despite αIIbβ3 dysfunction [17,18]. In patients with inherited bleeding disorders, careful adherence to patient blood management principles is essential to minimize unnecessary transfusion exposure.

Conclusion

Point-of-care ROTEM suggested a markedly reduced platelet contribution to clot formation as the primary driver of postpartum bleeding, which was subsequently confirmed as Glanzmann thrombasthenia by platelet function testing and flow cytometry. Thus, the application of a ROTEM-guided algorithm facilitated timely, goal-directed hemostatic management in this diagnostically complex case of a rare platelet disorder.

Declarations

Contributors: All authors contributed to planning, literature review and conduct of the review article. All authors have reviewed and agreed on the final manuscript.

Competing interests: None.

Patient consent for publication: Written informed consent was obtained from the patient.

Ethics approval and consent to participate: Ethical approval for this publication by National Medical Research Center for Obstetrics, Gynecology and Perinatology named after Academician V.I. Kulakov.

Availability of data and materials: Not applicable.

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References

1. Civaschi E, Klersy C, Melazzini F, Santoro C, Pujol-Moix N, et al. (2015). Analysis of 65 pregnancies in 34 women with five different forms of inherited platelet function disorders. Br J Haematol. 170: 559-563.
2. Noris P, Schlegel N, Klersy C, Heller PG, Civaschi E, et al. (2014). Analysis of 339 pregnancies in 181 women with 13 different forms of inherited thrombocytopenia. Haematologica. 99: 1387-1394.
3. Knight M, Callaghan WM, Berg C, Alexander S, Bouvier-Colle MH, et al. (2009). Trends in postpartum hemorrhage in high resource countries: a review and recommendations from the International Postpartum Hemorrhage Collaborative Group. BMC Pregnancy Childbirth. 9: 55.
4. Kumskova MA, Demina IA, Podoplelova NA, Balandina AN, Seregina EA, et al. (2015). Diagnosis of Glanzmann's thrombasthenia by assessment of plasma and platelet hemostatic parameters. Pediatric Hematology/Oncology and Immunopathology. 14: 17-24.
5. Görlinger K, Pérez-Ferrer A, Dirkmann D, Saner F, Maegele M, et al. (2019). The role of evidence-based algorithms for rotational thromboelastometry-guided bleeding management. Korean J Anesthesiol. 72: 297-322.
6. Blaauwgeers MW, Kruip MJHA, Beckers EAM, Coppens M, Eikenboom J, et al. (2020). Bleeding phenotype and diagnostic characterization of patients with congenital platelet defects. Am J Hematol. 95: 1142-1147.
7. Castaman G, James PD. (2019). Pregnancy and delivery in women with von Willebrand disease. Eur J Haematol. 103: 73-79.
8. Sabih A, Babiker HM. (2023). Von Willebrand Disease. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; NCBI Bookshelf ID: NBK459222.
9. Toogeh G, Sharifian R, Lak M, Safaee R, Artoni A, et al. (2004). Presentation and pattern of symptoms in 382 patients with Glanzmann thrombasthenia in Iran. Am J Hematol. 77: 198-199.
10. Nounou R, Spence D. (1993). Glanzmann's thrombasthenia with mild von Willebrand's disease. J Clin Pathol. 46: 1134-1136.

11. Botero JP, Lee K, Branchford BR, Bray PF, Freson K, et al. (2020). Glanzmann thrombasthenia: genetic basis and clinical correlates. Haematologica. 105: 888-894.

12. Snegovskikh D, Souza D, Walton Z, Dai F, Rachler R, Garay A, et al. (2018). Point-of-care viscoelastic testing improves the outcome of pregnancies complicated by severe postpartum hemorrhage. J Clin Anesth. 44: 50-56.

13. Carll T, Wool GD. (2020). Basic principles of viscoelastic testing. Transfusion. 60: S1-S9.

14. Matsunaga S, Takai Y, Nakamura E, Era S, Ono Y, et al. (2017). The clinical efficacy of fibrinogen concentrate in massive obstetric haemorrhage with hypofibrinogenaemia. Sci Rep. 7: 46749.

15. Lim HJ, Jang H, Lee N, Jeong E, Park Y, et al. (2024). Prediction of mid-term platelet transfusion in stable trauma patients using rotational thromboelastometry. Ann Lab Med. 44: 74-81.

16. Priyanka A, Arulprakasam S, Rudingwa P. (2023). Emergency cesarean section in glanzmann thrombasthenia: Anaesthetic management without recombinant factor VIIa. Saudi J Anaesth. 17: 407-409.

17. Poon MC. (2021). The use of recombinant activated factor VII in patients with Glanzmann's thrombasthenia. Thromb Haemost. 121: 332-340.

18. Saultier P, Grino M, Falaise C, Voisin S, Lavenu-Bombled C, et al. (2025). Efficacy and safety of recombinant activated factor VII in Glanzmann thrombasthenia: A systematic literature review. Haemophilia. 31: 7-15.