International Journal of Medical and Pharmaceutical Sciences
Open access peer-reviewed Journal
Volume 2, Issue 11, Page 252-256.
https://doi.org/10.5281/zenodo.24565318

*Review Article*

# Conservative Management and Pharmacological Control of Bleeding in Traumatic Head Injury with Glanzmann Thrombasthenia; a Case Report

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Glanzmann thrombasthenia (GT) is a rare inherited platelet function disorder characterized by defective platelet aggregation despite normal platelet counts, predisposing affected individuals to recurrent mucocutaneous and occasionally life-threatening hemorrhage. Traumatic intracranial bleeding in patients with GT is exceptionally uncommon and poses significant therapeutic challenges because optimal management requires balancing effective hemostasis against unnecessary invasive intervention. We report the successful conservative management of traumatic subarachnoid hemorrhage following head injury in a middle-aged woman with previously diagnosed GT. The patient presented with traumatic intracranial hemorrhage associated with progressive anemia requiring intensive hemostatic support. Management consisted of prompt blood component replacement, repeated platelet transfusions, antifibrinolytic therapy with tranexamic acid, neurological observation, serial laboratory monitoring, and multidisciplinary supportive care without neurosurgical intervention. Progressive clinical stabilization and hematological recovery were achieved, allowing discharge without major neurological sequelae. This case highlights that carefully selected patients with traumatic intracranial hemorrhage and GT may be successfully managed using an individualized conservative strategy when neurological status remains stable and close hematological and radiological surveillance is maintained. Early recognition of the underlying platelet dysfunction, timely hemostatic optimization, and coordinated multidisciplinary management are essential to reduce bleeding-related morbidity and improve clinical outcomes in this rare but potentially life-threatening clinical scenario.

**Keywords:** Glanzmann thrombasthenia, Platelet function disorder, Traumatic subarachnoid hemorrhage, Head injury, Platelet transfusion, Tranexamic acid, Conservative management

## INTRODUCTION

Glanzmann thrombasthenia (GT) is a rare autosomal recessive inherited platelet function disorder caused by quantitative or qualitative defects of the platelet glycoprotein IIb/IIIa (integrin αIIbβ3) receptor, resulting in impaired platelet aggregation despite normal platelet counts and platelet morphology. The estimated prevalence is approximately one in one million individuals worldwide, although the disease occurs more frequently in populations with a high rate of consanguinity. Clinically, GT is characterized by

variable bleeding manifestations ranging from easy bruising, epistaxis, gingival bleeding, and menorrhagia to severe gastrointestinal, postoperative, and life-threatening hemorrhage, with considerable heterogeneity in bleeding severity even among patients with similar genetic variants [1-3].

The diagnosis of GT is established by demonstrating defective platelet aggregation with physiological agonists despite a normal platelet count and coagulation profile, followed by confirmation using flow cytometry and molecular analysis of the *ITGA2B* or *ITGB3* genes. Although advances in laboratory diagnostics have improved early recognition, the management of bleeding episodes remains challenging because the underlying defect cannot be corrected by conventional hemostatic mechanisms. Current treatment strategies are primarily directed toward controlling hemorrhage using local hemostatic measures, antifibrinolytic agents, platelet transfusions, and recombinant activated factor VII (rFVIIa) in selected patients, while hematopoietic stem cell transplantation remains the only definitive curative option in carefully selected cases [2,4,5].

Intracranial hemorrhage is an exceptionally uncommon but potentially fatal complication of GT, accounting for only a small proportion of all bleeding manifestations. Traumatic intracranial hemorrhage, particularly traumatic subarachnoid hemorrhage (tSAH), is even less frequently reported in the literature. Owing to the rarity of this presentation, standardized treatment protocols are lacking, and management is generally individualized according to neurological status, radiological findings, severity of bleeding, and availability of specialized hemostatic therapies. Published reports suggest that conservative treatment with intensive hematological support may be successful in neurologically stable patients, thereby avoiding the risks associated with neurosurgical intervention [6,7].

Platelet transfusion remains the cornerstone of therapy for major hemorrhage in GT; however, repeated transfusions increase the risk of alloimmunization against human leukocyte antigen (HLA) and glycoprotein IIb/IIIa antigens, which may subsequently result in platelet refractoriness and reduced therapeutic efficacy. Consequently, contemporary recommendations advocate judicious use of platelet concentrates, consideration of HLA-matched platelets when feasible, adjunctive administration of antifibrinolytic agents such as tranexamic acid, and the use of rFVIIa in patients with platelet refractoriness or when compatible platelet products are unavailable [5,8,9].

The present case is clinically significant because it describes the successful conservative management of traumatic subarachnoid hemorrhage in a middle-aged woman with known GT following a road traffic accident. Prompt recognition of the underlying platelet dysfunction, aggressive hemostatic optimization with platelet and red blood cell transfusions, antifibrinolytic therapy, and close multidisciplinary monitoring resulted in favorable clinical recovery without neurosurgical intervention. This case contributes to the limited evidence regarding the management of traumatic intracranial hemorrhage in GT and highlights the importance of individualized, multidisciplinary treatment strategies in achieving optimal outcomes.

## CASE PRESENTATION

A 45-year-old woman with a known history of Glanzmann thrombasthenia and hypothyroidism was admitted after a road traffic accident involving a skid and fall from a two-wheeler. She presented with a right temporal scalp laceration, facial bone fracture, and traumatic brain injury complicated by acute traumatic subarachnoid haemorrhage.On admission, she was hemodynamically stable, with a pulse rate of 83 beats/min, blood pressure of 110/70 mmHg, and respiratory rate of 16 breaths/min. Initial laboratory investigations revealed a hemoglobin level of 9.6 g/dL and a platelet count of approximately 1.59 lakh/µL. During hospitalization, her hemoglobin decreased to 6.8 g/dL, while platelet counts ranged from approximately 1.2–1.5 lakh/µL. C-reactive protein was elevated to 42 mg/L during the second and third days of admission, and serum urea increased from 34 mg/dL on admission to approximately 50 mg/dL before subsequently improving. In view of the traumatic bleeding and underlying Glanzmann thrombasthenia, the patient required intensive supportive and hemostatic management. She received a total of 4 units of packed red blood cells and approximately 14 units of platelet concentrates over the course of 4 days of hospitalization.

During the hospital stay, the patient received tranexamic acid along with supportive and symptomatic

therapy, including intravenous **INJ.LEVETIRACETAM 500 MG** twice daily, **INJ.TRANEXAMIC ACID** 500mg every eight hourly, **INJ.CEFUROXIME SODIUM** 1.5g twice daily, **INJ.PANTOPRAZOLE** 40mg once daily, **INJ.ONDANSETRON** TDS, **INJ.PARACETAMOL** 1g TDS**, TAB. CHYMORAL FORTE** every eighth hourly, and **T.BETAHISTINE** 16mg twice daily as clinically indicated. Hypertonic saline (8% NaCl) was also administered as part of her management. Hemoglobin, platelet count, renal parameters, inflammatory markers, and clinical bleeding manifestations were monitored throughout hospitalization. Following transfusion and supportive management, the patient's hemoglobin improved from a nadir of 6.8 g/dL to 9.3 g/dL at discharge, with a platelet count of approximately 1.4 lakh/μL. Her clinical condition subsequently stabilized, and she was discharged with appropriate follow-up advice. This case emphasizes the challenges in managing significant traumatic bleeding in a patient with Glanzmann thrombasthenia and highlights the importance of prompt hemostatic intervention, blood component support, and close hematological monitoring

## DISCUSSION

Glanzmann thrombasthenia (GT) is characterized by defective platelet aggregation due to abnormalities of the platelet αIIbβ3 receptor, despite a relatively preserved platelet count. Therefore, platelet count alone does not reflect the severity of bleeding in affected patients. Di Minno et al. [10] and Seligsohn [11] discussed the clinical significance and management challenges associated with this inherited platelet dysfunction. In the present case, significant traumatic intracranial hemorrhage occurred despite a platelet count ranging from approximately 1.2–1.59 lakh/μL, emphasizing the importance of recognizing the underlying platelet dysfunction in patients with GT who sustain head trauma.

Intracranial hemorrhage is an uncommon but potentially fatal complication of GT, and traumatic subarachnoid hemorrhage is particularly rare. Almesedin et al. [6] described cases of intracranial hemorrhage in patients with GT, while the emergency-management consensus recommendations [7] highlight the need for individualized treatment strategies. Published evidence is largely limited to case reports and small series, with management individualized according to neurological status, radiological findings, and severity of bleeding. In the present case, the absence of neurological deterioration and the patient's clinical stability supported a conservative approach, avoiding the additional bleeding risk associated with neurosurgical intervention.

Platelet transfusion remains an important treatment for major bleeding in GT because transfused platelets provide functional αIIbβ3 receptors. However, repeated platelet transfusions may lead to alloimmunization against HLA and platelet-specific antigens, resulting in platelet refractoriness and reduced transfusion efficacy. Seligsohn [11], Fiore et al. [12], and Huisman et al. [13] addressed the risks of alloimmunization and the potential role of matched platelet transfusions in patients with inherited platelet disorders. Therefore, platelet therapy should be used judiciously, with consideration of compatible or HLA-matched platelets when repeated transfusion is anticipated. In our patient, approximately 14 units of platelet concentrates were administered over four days, along with four units of packed red blood cells, resulting in improvement of hemoglobin from 6.8 g/dL to 9.3 g/dL at discharge.

Tranexamic acid was used as an adjunctive hemostatic therapy. The emergency-management consensus recommendations [7] and Di Minno et al. [10] support the use of antifibrinolytic agents as part of the multimodal management of bleeding in GT, as these agents may help stabilize the developing clot. Recombinant activated factor VII (rFVIIa) is another therapeutic option, particularly in patients with platelet refractoriness or significant anti-platelet antibodies. Poon et al. [14] reported on the use of rFVIIa and other hemostatic agents in GT, while Fiore et al. [15] evaluated its efficacy and safety in a systematic literature review. However, rFVIIa was not required in our patient because satisfactory hemostatic control was achieved with platelet transfusion and supportive therapy.

The favorable outcome in this case supports the possibility of conservative management in carefully selected, neurologically stable patients with GT and traumatic intracranial hemorrhage. Nevertheless, as a single case report, this experience cannot establish the

superiority of conservative treatment over surgical intervention. The successful outcome emphasizes the importance of early recognition, individualized hemostatic therapy, serial neurological and laboratory monitoring, and close multidisciplinary coordination in this rare clinical situation.

## CONCLUSION

Traumatic intracranial hemorrhage in patients with Glanzmann thrombasthenia is a rare but potentially life-threatening clinical event that requires rapid recognition and individualized management. This case demonstrates that conservative management with platelet transfusion, tranexamic acid, red blood cell replacement, close neurological observation, serial laboratory monitoring, and multidisciplinary supportive care can result in favorable clinical recovery in a neurologically stable patient. The case also highlights that a normal or near-normal platelet count does not exclude clinically significant bleeding in GT because the underlying defect is qualitative platelet dysfunction. Although platelet transfusion remains an important component of treatment for major hemorrhage, the potential for alloimmunization and platelet refractoriness necessitates judicious use and consideration of compatible platelet strategies when repeated transfusion is anticipated. Recombinant activated factor VII may provide an alternative or rescue strategy in selected patients with platelet refractoriness or clinically significant platelet antibodies. Further accumulation of well-documented cases and collaborative registry data is required to establish more standardized approaches to traumatic intracranial hemorrhage in Glanzmann thrombasthenia.

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**Cite:** Chythanya D. J., Iswarya Chandran, Alnon L. J., Pradeepananda Vaidya, Shaiju S. Dharan, Conservative Management and Pharmacological Control of Bleeding in Traumatic Head Injury with Glanzmann Thrombasthenia; a Case Report, Int. J. Med. Pharm. Sci., 2026, 2 (11), 252-256. https://doi.org/10.5281/zenodo.24565318