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A case of severe intracranial hemorrhage following transfusion in a patient with β-thalassemia major

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Abstract

Summary of History: An 11-year-old girl with a 9-year history of β-thalassemia major (β-TM) managed with irregular transfusions and iron chelation therapy, complicated by hepatic dysfunction, hypersplenism, and cholelithiasis. Two days prior to admission, she received platelet transfusion and Intravenous Immunoglobulin (IVIG) due to a platelet count of 10×10^9/L. Intracranial Hemorrhage (ICH) occurred post-transfusion.

Symptoms and signs: Dizziness, fatigue, pallor of skin and mucous membranes, mild scleral icterus, abdominal distension with shifting dullness. Laboratory tests showed Hb 74 g/L, PLT 64×10^9/L, elevated liver enzymes and bilirubin.

Diagnostic methods: Emergency head CT revealed right frontal intracerebral hemorrhage (43.6 ml), frontotemporal subdural hematoma (13.8 ml) with subarachnoid hemorrhage, and midline shift of 7 mm. Initial coagulation screening was abnormal.

Treatment methods: Initial right frontal hematoma burr hole drainage evacuated approximately 30 ml of dark red hematoma. Immediate post-op CT showed good catheter placement with reduction of frontal hematoma by 13.4 ml, but a new right frontal epidural hematoma (~22 ml) was noted. Within 48 hours, a second surgery was performed for epidural and frontal hematoma evacuation, large decompressive craniectomy, and subdural drainage, with intraoperative transfusion of 2 units of RBCs and 100 ml of Fresh Frozen Plasma (FFP). Postoperative management included ICU sedation, analgesia, and mechanical ventilation.

Clinical outcome: Postoperative CT showed gradual hematoma absorption and midline structure normalization. The child's consciousness, speech, and limb muscle strength progressively recovered. She was transferred to a general ward for continued rehabilitation and was eventually discharged.

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Keywords: β-Thalassemia Major; Intracranial hemorrhage; Post-transfusion complication.

Introduction

β-Thalassemia Major (β-TM), caused by defects in the β-globin gene, leads to severe anemia, iron overload, and multi-organ damage, and is prevalent among children in the Mediterranean, Middle East, and South Asia. Post-transfusion thrombocytopenia and coagulation disorders can trigger Intracranial Hemorrhage (ICH), which carries a high mortality rate [1]. This report describes an 11-year-old girl who developed sudden ICH following transfusion and was successfully rescued through two-stage surgery, highlighting the importance of standardized transfusion practices and early recognition of bleeding signs.

Summary of history

General information

An 11-year-old female, resident of Guizhou, was admitted via the emergency department with a chief complaint of "dizziness and fatigue for 2 days." She was diagnosed with β-TM 9 years ago and subsequently received transfusions at irregular intervals of 3-8 weeks, with the last transfusion 10 days before admission. Long-term oral Deferasirox (DFX) for iron chelation was used, with the dose gradually increased from 0.25 g/d to 0.625 g/d, intermittently combined with subcutaneous Deferoxamine (DFO) infusion. Past medical history included hepatic dysfunction, hypersplenism, and cholelithiasis. There was no history of head trauma or epilepsy.

Family history: Mother is a β-thalassemia trait carrier; father refused testing. Consanguinity and similar bleeding history were denied. No surgical or high-risk sexual behavior history. Forty-eight hours before admission, she received 1 therapeutic dose of type-matched apheresis platelets and 20 g of Intravenous Immunoglobulin (IVIG) at an outside hospital due to a platelet count of 10×10^9/L, and was transferred without repeat blood tests.

Transfusion therapy: With hemoglobin at 74 g/L, meeting transfusion criteria, she received 3 units and 2 units of leukocyte-reduced suspended red blood cells on hospital days 1 and 2, respectively. The first transfusion was uneventful. Two hours after completion of the second transfusion, her condition deteriorated abruptly. She developed headache and vomiting, partially relieved by oxygen. Twenty minutes later, she suddenly lost consciousness, became stuporous, with unequal pupils (left 5.0 mm, right 6.0 mm) and sluggish light reflexes. Acute cerebrovascular accident was highly suspected, and a multidisciplinary rescue protocol was initiated immediately.

Examinations

Laboratory (11-25): White blood cells (WBC) 3.28×10^9/L, hemoglobin (Hb) 74 g/L, platelets (PLT) 64×10^9/L; alanine aminotransferase (ALT) 87 U/L (↑2.9x), total bilirubin (TBil) 32.2 μmol/L (↑1.4x), albumin (ALB) 33.9 g/L (↓); fibrinogen (Fbg) 1.72 g/L (↓0.28), prothrombin time (PT) 15.0 s (↑1.07x), NT-proBNP 6,690 pg/mL (↑53x) (Table 1).

Imaging: Non-contrast head CT showed right frontal intracerebral hemorrhage (43.6 ml), frontotemporal subdural hematoma (13.8 ml) with subarachnoid hemorrhage, and midline shift of 7 mm. Chest CT showed patchy shadows in both lungs. Cardiac ultrasound: Left Ventricular Ejection Fraction (LVEF) 58%, with a small pericardial effusion.

Table 1: Laboratory test results.

Parameter	Nov 24	Nov 26	Nov 28	Reference range
WBC ($\times10^9$/L)	3.28	—	12.68	4.3-11.3
RBC ($\times10^{12}$/L)	—	—	3.8	3.8-5.1
Hemoglobin (g/L)	74	113	108	115-150
Hematocrit (%)	21.5	32.7	32.7	37-47
MCV (fL)	81.4	83.8	—	82-100
MCHC (g/L)	—	—	—	320-360
Neutrophils (%)	—	78.4	—	32-71
Lymphocytes (%)	—	16.7	—	22-57
Platelets ($\times10^9$/L)	64	69	127	125-350
Uric Acid (μmol/L)	410	389	—	155-357
ALT (U/L)	87	74	—	7-30
Total Bilirubin (μmol/L)	32.23	33.11	—	0-23
Direct Bilirubin (μmol/L)	14.68	17.12	—	0-8
Indirect Bilirubin (μmol/L)	17.55	15.99	—	0-11
Albumin (g/L)	33.9	31.9	—	35-54
AST (U/L)	57	57	—	13-35
Serum Calcium (mmol/L)	—	2.06	—	2.10-2.80
hs-CRP (mg/L)	—	6.66	—	≤4
NT-proBNP (pg/mL)	—	6690.44	781.14	≤125
Fibrinogen (g/L)	1.72	—	—	2-4
PT (s)	—	15.0	—	10-14
Coagulation Function	—	—	Normal	—
HBsAb	Positive	—	—	Positive/ Negative
HBcAb	Positive	—	—	Positive/ Negative

Diagnosis and differential diagnosis

Primary diagnoses

(1) β-Thalassemia major with post-transfusion acute intracranial hemorrhage (ICH) (right frontal lobe + subdural + subarachnoid);

(2) Dilutional coagulopathy;

(3) Chronic iron overload (serum ferritin >3,000 ng/mL);

(4) Bilateral pulmonary infection.

Basis for diagnosis: Genetic confirmation of β-TM; sudden neurological deterioration; quantified hemorrhage on imaging; abnormal coagulation indices; platelet count improved but remained low.

Differential diagnoses: Cerebral Venous Sinus Thrombosis (CVST) with hemorrhage: CT venography showed no filling defects, not supporting [2].

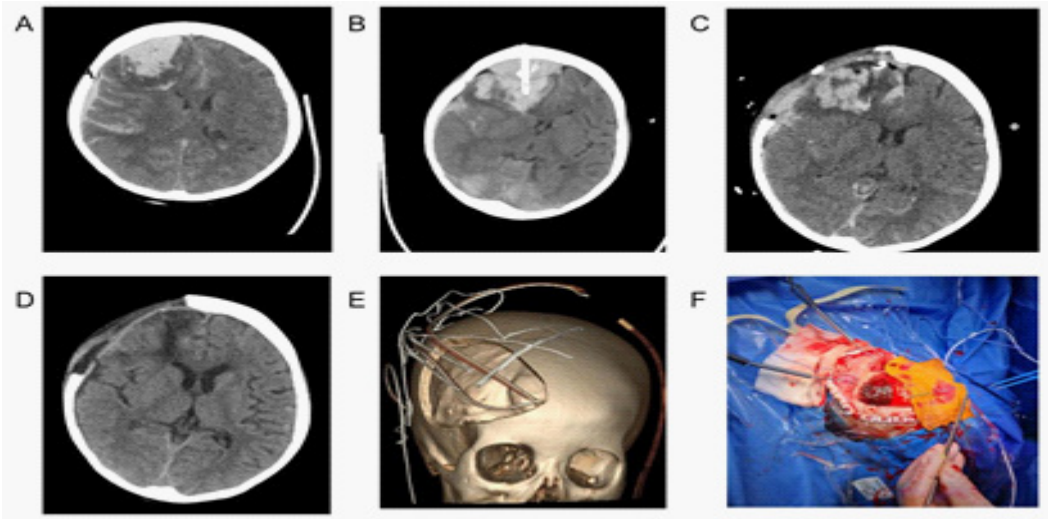

Figure 1: Initial head CT at symptom onset: **(A)** Right frontal and basal ganglia parenchymal hemorrhage, hematoma volume ~43.6 mL. **(B)** Immediate post-emergency burr hole drainage CT. **(C)** Follow-up CT at 2 weeks post-op: Gradual hematoma absorption and midline structure recovery. **(D)** 3D reconstruction CT after decompressive craniectomy, showing skull defect area. **(E)** Intraoperative photograph: Epidural hematoma visible.

Ruptured vascular malformation: CTA showed no obvious vascular malformation nidus.

Traumatic hemorrhage: No trauma history; imaging showed no fractures or contrecoup injuries.

Treatment

Preoperative correction: Infusion of Fresh Frozen Plasma (FFP) 10 mL/kg and fibrinogen concentrate 2 g to achieve PT 2 g/L; piperacillin-tazobactam for anti-infection; intravenous furosemide 1 mg/kg for volume reduction, reducing NT-proBNP to 3200 pg/mL.

First surgery (Hospital day 3): Right frontal hematoma burr hole drainage under local anesthesia with sedation, evacuating approximately 30 ml of dark red clots. Immediate post-op CT suggested a small increase in bleeding. Due to persistent low platelets, aggressive correction was performed.

Second surgery (Hospital day 5): Under general anesthesia, epidural hematoma and frontal hematoma evacuation, large decompressive craniectomy, and subdural drainage were performed. Intraoperative blood loss was approximately 500 ml, requiring transfusion of 2 units of RBCs and 100 ml of FFP. The bone flap was preserved at -80°C (Figure 1).

Postoperative management: Goal-directed sedation and analgesia (dexmedetomidine + sufentanil), mechanical ventilation; Intracranial Pressure (ICP) control

Rehabilitation: Bedside passive limb training began on postoperative day 3. On postoperative day 10, she was transferred to the rehabilitation department for speech, motor training, and intermittent transfusions. On postoperative day 17, she was conscious, answered questions appropriately (though with low volume and hoarseness, showing improvement), and could follow simple commands (nodding, handshake, leg lifting). Limb muscle strength was approximately grade 4+.

Treatment outcome, follow-up, and prognosis

CT 24 hours after craniectomy showed approximately 60% reduction in hematoma volume and normalized midline. She stayed in the ICU for 13 days, was successfully extubated, and NT-proBNP decreased to 781 pg/mL. Subsequent imaging showed gradual absorption of the residual intracerebral hematoma and normalization of the midline structures. With active rehabilitation, her consciousness, limb muscle strength, and language function improved, and she was eventually transferred to a general ward. After 28 days in the rehabilitation ward involving active swallowing training, speech therapy, and hemiplegic limb training, she was discharged in improved condition.

Discussion/Conclusion

Intracranial hemorrhage in children with β-Thalassemia Major (β-TM) is a critically underrated emergency at the intersection of hematology and neurosurgery [3]. This case of an 11-year-old girl who suffered massive multi-compartment cerebral hemorrhage despite her platelet count having recovered from 10×10^9/L to 64×10^9/L highlights the limitations of the traditional "platelet threshold" mindset in this population [4]. Her bleeding risk was driven by a combination of volume overload, dilutional coagulopathy, and synthetic defects from chronic hepatic fibrosis, rather than isolated platelet deficiency. During management, integrating coagulation parameters and NT-proBNP into the pre-transfusion assessment, and prioritizing correction of coagulation with FFP and fibrinogen concentrate upon finding fibrinogen below 2 g/L, prolonged PT, and a 53-fold elevation in BNP, followed by staged intracranial hematoma evacuation surgery, was crucial. A damage-control strategy was employed during frontal hematoma evacuation, focusing on removing the central clot core. This comprehensive approach considering bleeding risk throughout the process ultimately prevented uncontrolled intraoperative hemorrhage and secondary brain injury [5].

By quantifying volume and coagulation parameters, this report adds clinical data for the "non-thrombotic, non-severely thrombocytopenic" intermediate phenotype of post-transfusion ICH in β-TM. It suggests that in future similar cases, simultaneously incorporating volume status, coagulation function, and iron load into the decision-making process is essential to truly reduce the incidence and mortality/disability rates of intracranial hemorrhage.

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