# Supplemental File

**Supplementary Table S1. Papers from which clinical vignettes were sourced**

| Full reference | Disorder / Clinical Domain |
|---|---|
| Slob J, Loer SA, Koopman SSHA, van der Vlies CH. Can viscoelastic haemostatic tests in burn patients help to predict outcome? – A case report and narrative review of the literature. Burns Open. 2024;8(4):100354. doi:https://doi.org/10.1016/j.burnso.2024.06.005 | Bleeding & Clotting / Trauma, Critical Care |
| Asma Ladib, Jihen Ladib, Fethi Jebali, Asma Korbi, Imen Ghaddab, Sassi M. Optimizing the anesthetic management of factor XI deficiency in obstetrics: A case report using ROTEM. International Journal of Surgery Case Reports. 2025;133:111566-111566. doi:https://doi.org/10.1016/j.ijscr.2025.111566 | Bleeding / Obstetrics |
| Grigorios Karampas, Konstantinos Karkalemis, Bagiasta A, et al. Severe Hereditary Hypofibrinogenemia in Pregnancy: A Case Report of a Novel Obstetrical Management with Thromboelastometry Guided Fibrinogen Supplementation. Diagnostics. 2025;15(13):1671-1671. doi:https://doi.org/10.3390/diagnostics15131671 | Bleeding / Obstetrics |
| Bhanwar M, Siddiqua N, Nayer J. Balancing bleeding and thrombosis in chronic liver disease: ROTEM-guided management of life-threatening lower GI bleed. JEM Reports. 2025;5(1):100200. doi:https://doi.org/10.1016/j.jemrpt.2025.100200 | Bleeding & Clotting / Gastroenterology |
| Introna M, Broggi M, Paolo Ferroli, et al. Perioperative ROTEM® evaluation in a patient affected by severe VII factor deficiency undergoing microvascular decompression craniotomy for hemifacial spasm. Journal of Clinical Monitoring and Computing. 2024;38(6):1431-1435. doi:https://doi.org/10.1007/s10877-024-01183-w | Bleeding / Neurosurgery |
| Iarossi M, Caroline, Véronique Hamoir, Geoffroy Vanderweerden, Hermans C. Hypofibrinogenemia Following an Anaphylactic Shock Caused by a Neuromuscular Blocking Agent—Case Report. Case Reports in Critical Care. 2025;2025(1):6954832-6954832. doi:https://doi.org/10.1155/crcc/6954832 | Bleeding / Anaphylaxis |
| Zhou Y, Gelson W, Brais R, Besser M. Paraneoplastic Hyperfibrinolysis in Oesophageal Adenocarcinoma: A Case Report. *Cureus*. Published online August 25, 2025. doi:https://doi.org/10.7759/cureus.90922 | Bleeding / Oncology |

 

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| Wakabayashi M, Tokinaga Y, Kawamata T. Thromboelastography-Guided Anesthetic Management for Cesarean Section in a Patient With Antiphospholipid Syndrome: A Case Report. *Cureus*. Published online August 11, 2025. doi:https://doi.org/10.7759/cureus.89829 | Clotting / Obstetrics |
| Miyahara S, Shohei Mitsumata, Kaneda S, et al. Perioperative Management of Single Lung Transplantation for a Chronic Obstructive Lung Disease Patient with Congenital Alpha$_2$-Plasmin Inhibitor Deficiency. *Surgical Case Reports*. 2025;11(1):n/a-n/a. doi:https://doi.org/10.70352/scrj.cr.25-0230 | Bleeding / Thoracic Surgery |
| Martinez-Lopez PR, Barroso-Gonzalez A. Anesthetic Implications of Factor XI Deficiency: A Clinical Case Study and Review of Literature. *Cureus*. Published online October 28, 2024. doi:https://doi.org/10.7759/cureus.72594 | Bleeding / Anesthesiology |

**Supplementary Table S2. Prompts and LLM outputs, organized by clinical vignette (in vignette order). Assessments of each LLM's performance are summarized on the right**

| No. | Reference | Clinical Vignette | ChatGPT | Deepseek | Gemini | Assessment of LLM Performance |
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| 1 | Slob J et al. (Case on outcome of burn patients) | A 77-year-old patient was referred with 16% total body surface area flame burns involving the chest, abdomen, neck, and upper arms, predominantly full-thickness. | Predominantly hypercoagulable, to avoid procoagulant transfusions on ROTEM alone and treat as a high-VTE-risk burns patient. | Life-threatening burn coagulopathy managed with immediate tranexamic acid plus urgent fibrinogen replacement. | Hypercoagulable pattern. Management is to avoid pro-hemostatic products in the absence of bleeding and prioritize early VTE prophylaxis. | ChatGPT: Accurate assessment but partial fabrication<br>Deepseek: Dangerously misread ROTEM leading to unsafe management plan.<br>Gemini: Accurate interpretation and management |
| 2. | Asma L et al. (Case on factor XI deficiency) | A 28-year-old primigravid woman with an incidentally discovered moderate FXI deficiency (52.6 %) during routine preoperative evaluation for suspected macrosomia | No global hypocoagulable phenotype. No routine prophylactic factor replacement is indicated. Consider TXA at delivery. | Isolated intrinsic-pathway delay. To avoid routine prophylactic factor replacement and proceed with planned delivery with blood and TXA available. | Isolated intrinsic-pathway initiation delay. To avoid routine factor replacement and manage conservatively with prophylactic TXA. | ChatGPT: Accurate interpretation and management<br>Deepseek: Missed early hypocoagulability and over-escalated management.<br>Gemini: Accurate interpretation and management |
| 3. | Grigorios K et al. (Case on hypofibrinogenemia pregnancy) | A 37-year-old gravida 4, para 1 woman with a BMI of 20.3 kg/m2 presented to our institution for an outpatient appointment during her fourth pregnancy. | Preserved hemostasis, managed with obstetric planning with VHA-guided peripartum replacement if needed. Avoid neuraxial anesthesia until fibrinogen is corrected. | Fibrinogen deficiency, managed with peripartum planning with fibrinogen replacement guided by labs and ROTEM, and neuraxial anesthesia only after correction. | Severe hypofibrinogenemia managed with individualized, ROTEM-guided fibrinogen replacement and proactive peripartum planning. | ChatGPT: Overcalled prophylaxis, fabricated citations<br>Deepseek: Over-escalated management with misprioritized assay values.<br>Gemini: Hallucinated FIBTEM CT reference. |
| 4. | Bhanwar M et al. (Case on Chronic Liver Disease) | A 28-year-old male with a known history of chronic liver disease presented to the emergency department with complaints of bleeding per rectum for 3 days, associated with passage of clots and | Hypofibrinogenemia with intact platelet contribution managed with ROTEM-guided fibrinogen replacement and simultaneous resuscitation. | Liver disease related coagulopathy, managed with ROTEM-guided FFP and resuscitation. | Rebalanced hemostasis in chronic liver disease, managed with hemodynamic stabilization, ROTEM-guided transfusion, vasoactive therapy, prophylactic | ChatGPT: Under-called factor deficiency causing misdirected management toward fibrinogen<br>Deepseek: Appropriate assessment and FFP-centered management, but mislabelled platelet dysfunction<br>Gemini: Misclassified EXTEM CT with missing factor deficiency leading to inadequate management. |

 

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| | | frank red blood. He also reported abdominal pain and progressive distension for 5 days. | | | antibiotics, and urgent interventional source control. | |
| 5. | Introna M et al. (Case on factor VII deficiency) | A 79-year-old male with a history of miastenia gravis previously treated with thymectomy and a recent femoral aneurysm repair, after which he received chronic antiplatelet therapy | Preserved global hemostasis managed by holding P2Y12/aspirin, ROTEM- and platelet-function–guided therapy, and platelet transfusion. | Coagulation factor deficiency managed with preoperative correction with PCC/FFP, empirical vitamin K, withholding antiplatelets, and ROTEM-guided intraoperative hemostatic support. | Preserved global hemostasis managed with perioperative assessment and antiplatelet dysfunction with platelet function testing, blood pressure control, and myasthenia-aware anesthetic planning. | ChatGPT: Missed factor deficiency due to over-reliance of ROTEM reliability.<br>Deepseek: Missed isolated FVII deficiency resulting in non-specific management.<br>Gemini: Treated platelet dysfunction over FVII deficiency. |
| 6. | Iarossi M et al. (Case on anaphylaxis) | A 47-year-old male with a history of untreated allergic asthma was admitted to the hospital for a scheduled arthroscopy of the temporomandibular joint under general anesthesia | Hypocoagulability consistent with acute anaphylaxis managed with anaphylaxis treatment and ROTEM-guided fibrinogen. | Suggestive of acute anaphylaxis managed with anaphylaxis treatment, ROTEM-guided fibrinogen replacement and supportive resuscitation. | Anaphylaxis–associated heparin-like coagulopathy managed with anaphylaxis resuscitation and conservative ROTEM-guided fibrinogen. | ChatGPT: Conservative management plan unbacked by evidence<br>Deepseek: Overgeneralized management plan and over-interpreted VHA parameters<br>Gemini: Mistakes in interpretation and wrongly withheld treatment |
| 7. | Zhou Y et al. (Case on oesophageal CA) | A male patient in his 70s with a history of oesophagectomy for oesophagogastric junction adenocarcinoma who presented with dysphagia, epigastric pain, and spontaneous bruising. | DIC-spectrum consumptive coagulopathy with severe hyperfibrinolysis managed by stabilizing hemodynamics and give fibrinogen replacement while treating underlying cause. | DIC-spectrum consumptive coagulopathy managed with urgent hemostatic stabilization while treating underlying cause. | DIC with a hyperfibrinolytic phenotype managed with hemostatic stabilization, fibrinogen replacement, while treating underlying cause. | ChatGPT: Over-anchoring on DIC led to management overreach<br>Deepseek: Premature management of broader hemostatic steps led to biased downstream recommendations.<br>Gemini: Over-anchored on systemic labels instead of urgent source workup, with slight over-attribution to fibrinogen. |
| 8. | Wakabayashi M et al. (Case on antiphospholipid syndrome) | A 25-year-old Japanese woman with mixed connective tissue disease and APS was treated with prednisolone, tacrolimus, and azathioprine. | Residual heparin effect, to avoid neuraxial anesthesia with coordination for perioperative anticoagulation and postpartum thromboprophylaxis. | No evidence of clinically significant residual heparin effect, to proceed with neuraxial anesthesia and restart postpartum thromboprophylaxis per APS protocols. | Residual heparin effect plus non heparin-mediated initiation delay, to use general anesthesia with rapid sequence induction and avoid routine protamine reversal.. | ChatGPT: Over-escalated management with peri-hemorrhage products. Missed VHA parameters.<br>Deepseek: Wrongly dismissed heparin effect leading to erroneous management plan.<br>Gemini: Overall accurate but FFP recommendation was beyond required management. |
| 9. | Miyahara S et al. (Case on lung transplant) | A 46-year-old female affected by chronic obstructive pulmonary disease with congenital α2-PI deficiency was referred to our hospital for cadaveric lung transplantation. | Hyperfibrinolysis risk managed with tranexamic acid prophylaxis and FIBTEM-guided fibrinogen replacement. | Severe hyperfibrinolysis managed with high-dose tranexamic acid with hematology follow-up for perioperative planning. | Severely low FIBTEM improved with fibrinogen replacement, to start aggressive antifibrinolysis, fibrinogen replacement and ROTEM monitoring. | ChatGPT: Overemphasis on FIBTEM which added bias to management.<br>Deepseek: Linked EXTEM CT to α2-antiplasmin deficiency resulting in over-specific post-op plan.<br>Gemini: Overspecific fibrinogen, leading to semi-appropriate management. |
| 10. | Martinez-Lopez PR et al. (Case on factor XI deficiency) | A 25-year-old woman with no significant medical history except for a known factor XI deficiency (40%) presented to the emergency department with a diagnosis of five-week ectopic pregnancy. | Mild factor XI deficiency, for urgent hemorrhage control with FFP, standard resuscitation, operative intervention, and lab-guided transfusions. | Factor XI deficiency is less relevant, for urgent hemorrhage control, first-line fibrinogen replacement, FFP for factor XI support, and TXA for adjunctive hemostasis. | Pregnancy-related hyperfibrinogenemia, managed with urgent surgical control of ectopic pregnancy, FXI replacement, tranexamic acid, and fluids and PRBCs as needed. | ChatGPT: Overall accurate but understated initiation delay.<br>Deepseek: Poor interpretation which led to highly inappropriate management plan.<br>Gemini: Overall accurate but FIBTEM was over-interpreted. |

 

**Supplementary Table S3. Raw LLM Outputs (verbatim)**

| **Clinical Vignette 1: Can Viscoelastic haemostatic tests in burn patients help to predict outcome? - A case report and narrative review of the literature** | |
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| **ChatGPT** | Interpretation<br>**1) FIBTEM (fibrin-based clot strength)**<br>• **CT 61 s** (within ref 46–84)<br>• **A5 25 mm, A10 27 mm, A20 29 mm, MCF 31 mm** — all **above the reference ranges** shown on the report<br>➡️ **Markedly increased fibrin clot contribution** → most consistent with **hyperfibrinogenaemia / acute-phase response** (very plausible in burns/inflammation), i.e. **hypercoagulable tendency driven by fibrin(ogen)**.<br>**2) EXTEM (extrinsic pathway / overall clot including platelets)**<br>• **CT 82 s** (slightly **prolonged** vs ref 50–80)<br>• **CFT 52 s** (normal), **A10 62 / MCF 68 mm** (normal-high)<br>• **ML 2%, LI30 100%, LI45 98%**<br>➡️ Clot strength is **adequate**; **no hyperfibrinolysis**. Borderline CT prolongation is mild and, in context of strong clot firmness, is **not a bleeding phenotype**.<br>**3) INTEM (intrinsic pathway)**<br>• **CT 127 s** (**shorter** than the reference range printed), **CFT 59 s** (slightly short), **MCF 65 mm** (normal)<br>• Lysis indices normal<br>➡️ **Fast initiation/propagation** overall, again supporting a **hypercoagulable tendency**, not factor deficiency.<br>**4) APTEM (fibrinolysis check)**<br>• Parameters broadly similar firmness to EXTEM, **ML 1%**, normal lysis indices<br>➡️ **No evidence of hyperfibrinolysis** (APTEM not "rescuing" a lysis problem).<br><br>**Bottom line**<br>This ROTEM pattern is **not burn-induced hypocoagulable bleeding coagulopathy**. Instead it suggests **hypercoagulability**, driven largely by **high fibrin(ogen) contribution** (high FIBTEM A10/MCF), with **no fibrinolysis**. |

 

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| | ## Suggested Management<br>**If the patient is not actively bleeding**<br>• **Do NOT give fibrinogen/cryoprecipitate or platelets** based on this ROTEM (clot strength is already high).<br>• Treat as a **high VTE-risk burns patient**:<br>  ○ **Early pharmacologic thromboprophylaxis** (e.g., LMWH) *when clinically safe* + mechanical prophylaxis.<br>  ○ Maintain adequate hydration, avoid unnecessary procoagulant products.<br>**If going for debridement/grafting (peri-op plan)**<br>• **Repeat ROTEM intra-op and post-op** (burn surgery + fluids can shift CT/MCF).<br>• Transfuse/target therapy **only if bleeding with ROTEM phenotype**, e.g.:<br>  ○ **EXTEM CT clearly prolonged** with bleeding → consider **coagulation factors** (institution-dependent: PCC vs plasma).<br>  ○ **EXTEM A10/MCF low** with **normal FIBTEM** → **platelet issue** → platelets.<br>  ○ **FIBTEM A10/MCF low** → **fibrinogen replacement** (cryo/fibrinogen concentrate).<br>  ○ **High ML / falling LI** and **APTEM improves vs EXTEM** → **hyperfibrinolysis** → **TXA**. |
| **DeepSeek** | ## Interpretation<br>The patient demonstrates a coagulopathy consistent with **severe burn injury**, characterized primarily by **hyperfibrinolysis and fibrinogen depletion**.<br>**Key Findings:**<br>1. **Hyperfibrinolysis (Primary Issue):**<br>  ○ **EXTEM & INTEM:** Show a pattern of **normal to accelerated clot initiation (CT)** but **premature and complete clot lysis**.<br>  ○ **Evidence:** The maximum clot firmness (**MCF**) is significantly decreased or unmeasurable (*), and the maximum lysis (**ML**) is 100% in both EXTEM and INTEM. This means the clot forms but then dissolves entirely within the test period. |

 

- **Confirmation:** The **APTEM** test (which contains an antifibrinolytic agent, aprotinin) shows **correction** of this lysis pattern. The ML is now 0% (or very low), and the MCF improves. This confirms that the coagulopathy is driven by excessive fibrinolysis.

2. **Fibrinogen Deficiency:**
   - **FIBTEM:** This test isolates the contribution of fibrinogen to clot strength.
   - **Evidence:** The FIBTEM MCF is very low (*), and the amplitude values (A5-A30) are severely reduced. This indicates a **critical deficiency of functional fibrinogen**, which is a hallmark of burn shock and consumption.
3. **Overall Pattern:** This is a **combined coagulopathy**:
   - **Consumptive:** Fibrinogen and other factors are being consumed.
   - **Hyperfibrinolytic:** The body's clot-breaking system is overactive.
   - This is a classic and life-threatening complication of major thermal injury.

## Suggested Management

Management must address both the burn shock and the acute coagulopathy simultaneously.

**1. Immediate Resuscitation & Monitoring:**

- **Fluid Resuscitation:** Adhere to the **Parkland formula** (4 mL/kg/%TBSA) for the first 24 hours, with half given in the first 8 hours post-burn. Use balanced crystalloids (e.g., Lactated Ringer's). **Monitor urine output (target 0.5-1 mL/kg/hr)** closely, as the patient's age and hypertension increase the risk of fluid overload and organ dysfunction.
- **Hemodynamic Monitoring:** Consider invasive arterial and central venous pressure monitoring given the patient's age, extent of injury, and developing coagulopathy.
- **Airway:** Given the neck burns, maintain a high index of suspicion for airway compromise and have a low threshold for early intubation.

**2. Targeted Coagulation Management (ROTEM-Guided):**

- **Antifibrinolytic Therapy:** Administer **Tranexamic Acid (TXA)** immediately. A typical regimen is 1g IV bolus over 10 minutes, followed by a 1g infusion over 8 hours. This directly counters the hyperfibrinolysis seen on ROTEM.
- **Fibrinogen Replacement:** This is the **highest priority** based on the FIBTEM.

 

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| | ○ Administer **Fibrinogen Concentrate** (e.g., 50-100 mg/kg) or **Cryoprecipitate** (target fibrinogen level > 1.5-2.0 g/L).<br>○ This will directly improve clot strength and stability.<br>• **Clotting Factors:** Consider **Prothrombin Complex Concentrate (PCC)** or **Fresh Frozen Plasma (FFP)** if there is evidence of multiple factor deficiencies (prolonged CT). However, based on the normal CTs, this is less urgent than fibrinogen and antifibrinolysis. FFP may be required as part of ongoing resuscitation.<br>• **Platelets:** The **INTEM/EXTEM** amplitudes are low, but this is likely due more to fibrinogen deficiency than thrombocytopenia. Check a platelet count. Transfuse platelets only if the count is very low (<50,000-100,000/µL) or there is active microvascular bleeding.<br>**3. Comprehensive Burn & Medical Care:**<br>• **Wound Management:** Early surgical consultation for **escharotomy** (if full-thickness circumferential burns are present on chest/arms causing restriction) and planning for **early excision and grafting**.<br>• **Analgesia & Sedation:** Aggressive pain management is essential. Consider the psychiatric history (bipolar disorder) when choosing agents.<br>• **Infection Prophylaxis:** Start broad-spectrum antibiotics for documented infections only, not for prophylaxis. Tetanus prophylaxis is mandatory.<br>• **Comorbidity Management:**<br>○ **Thyroid (Hashimoto's):** Continue or initiate thyroid hormone replacement as needed; stress can affect requirements.<br>○ **Hypertension:** Antihypertensives may need to be held during the acute resuscitation phase but reinstated as the patient stabilizes.<br>○ **Psychiatric:** Collaborate with psychiatry for management of bipolar disorder, as the stress of injury can trigger episodes.<br>**4. Monitoring:**<br>• Repeat **ROTEM** 1-2 hours after initiating therapy (TXA, fibrinogen) to assess response.<br>• Frequent monitoring of standard labs: CBC, PT/aPTT, fibrinogen, electrolytes, lactate, and base deficit to guide overall resuscitation.<br>This elderly patient with 16% TBSA full-thickness burns is developing a **severe, hyperfibrinolytic coagulopathy with profound fibrinogen deficiency**, as clearly demonstrated by ROTEM. Immediate management must be two-pronged: **1) aggressive burn resuscitation** and **2) targeted hemostatic therapy** with **tranexamic acid and** |

 

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| | **fibrinogen replacement**. Failure to correct this coagulopathy will lead to uncontrolled hemorrhage, worsening shock, and increased mortality. Close monitoring and a multidisciplinary approach (burn surgery, hematology, critical care, psychiatry) are crucial. |
| **Gemini** | ## Interpretation<br>The patient's ROTEM profile indicates a **hypercoagulable state**, characterized by significantly increased fibrinogen activity and accelerated clot initiation in the intrinsic pathway.<br>• **FIBTEM (Fibrinogen contribution):** This is the most striking result. All amplitude values are significantly elevated (A5: 25mm, A10: 27mm, A20: 29mm) with a Maximum Clot Firmness (**MCF**) of **31mm** (Reference: 6–21mm). This indicates **hyperfibrinogenemia**, which is common in major burns as fibrinogen acts as an acute-phase reactant.<br>• **INTEM (Intrinsic pathway):** The Clotting Time (**CT**) is shortened at **127s** (Reference: 161–204s). The Clot Formation Time (**CFT**) is also shortened at **59s** (Reference: 62–130s). This suggests a "primed" or hyperactive intrinsic coagulation system.<br>• **EXTEM (Extrinsic pathway):** The **CT** is slightly prolonged at **82s** (Reference: 50–80s) , but the initial clot strength (A5 and A10) is high, and the **MCF** is within the high-normal range at **68mm**.<br>• **Fibrinolysis:** There is **no evidence of hyperfibrinolysis**. The Maximum Lysis (**ML**) is very low across all tests (0–2%) , and the **APTEM** (which uses aprotinin to inhibit fibrinolysis) mirrors the EXTEM results, confirming the clot is stable.<br><br>## Suggested Management<br>Given the hypercoagulable nature of these results and the clinical context of full-thickness burns in an elderly patient, management should focus on the following:<br>**1. Thromboembolism (VTE) Prophylaxis**<br>The patient is at high risk for Deep Vein Thrombosis (DVT) and Pulmonary Embolism (PE) due to the combination of:<br>• Advanced age (77 years).<br>• Major thermal injury (SIRS response).<br>• Confirmed hypercoagulability (high FIBTEM MCF and shortened INTEM CT). |

 

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| | • **Action:** Initiate pharmacological VTE prophylaxis (e.g., Low Molecular Weight Heparin) as soon as it is surgically safe.<br>**2. Fluid Resuscitation and Monitoring**<br>Standard burn management (e.g., Parkland formula) is essential for a 16% BSA burn, especially given the patient's history of arterial hypertension.<br>• **Action:** Monitor urine output and hemodynamic status closely to avoid over-resuscitation, which could exacerbate edema in the neck (airway risk) and chest burns.<br>**3. Avoid Pro-hemostatic Agents**<br>The ROTEM shows no signs of coagulopathy or bleeding risk; in fact, the clot is exceptionally firm.<br>• **Action:** Do **not** administer Tranexamic Acid (TXA), fibrinogen concentrate, or fresh frozen plasma unless active, significant surgical bleeding occurs.<br>**4. Surgical Intervention**<br>The physician estimated these as full-thickness burns.<br>• **Action:** Early escharotomy or excision and grafting may be necessary. Given the burns to the neck and chest, maintain a high suspicion for airway compromise or restricted chest wall excursion.<br>**5. Management of Comorbidities**<br>• **Psychiatric:** Monitor bipolar disorder symptoms, as the stress of a major burn and hospital environment can trigger episodes.<br>• **Endocrine:** The patient has Hashimoto's thyroiditis. Monitor thyroid function tests, as "Euthyroid Sick Syndrome" is common in severe burns and may complicate recovery. |
| **Clinical Vignette 2: Optimizing the anesthetic management of factor XI deficiency in obstetrics: A case report using ROTEM** | |
| **ChatGPT** | Interpretation<br>ROTEM interpretation (pre-delivery / pre-op)<br>From the trace provided:<br>EXTEM (tissue factor / overall clotting + platelets)<br>• CT 71 s (normal 34–79), CFT 50 s (40–70)<br>• A10 61 mm (56–68), MCF 72 mm (66–76)<br>➡️ Normal initiation and normal clot strength (platelet + fibrin contribution looks adequate). |

 

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| | FIBTEM (functional fibrinogen contribution)<br>• CT 62 s (47–64)<br>• A10 21 mm (13–24), MCF 24 mm (14–27)<br>➡ Fibrinogen/fibrin-based clot firmness is normal (no suggestion of hypofibrinogenemia).<br>INTEM (intrinsic pathway / sensitive to FXI deficiency)<br>• CT 236 s (122–220) mildly prolonged, CFT 82 s (47–82) at upper limit<br>• A10 54 mm (54–67), MCF 65 mm (63–75)<br>➡ Slight delay in intrinsic pathway clot initiation, consistent with FXI deficiency but downstream clot propagation/strength is preserved.<br>APTEM (antifibrinolytic challenge)<br>• Values and curve are strong (MCF ~68 mm) and not showing excess breakdown compared with EXTEM.<br>➡ No evidence here of clinically significant hyperfibrinolysis.<br>Bottom line<br>This ROTEM is overall reassuring: normal clot strength (EXTEM/ FIBTEM) with only a mild intrinsic initiation delay (INTEM CT). In a primigravid patient with minimal bleeding history (ISTH-BAT 1) and FXI ~53% (mild–moderate deficiency), this pattern supports a plan of no routine factor replacement, but having targeted therapy ready.<br>**Suggested management**<br>1) Pre-delivery planning<br>• Multidisciplinary: obstetrics + anesthesia + hematology.<br>• Check/confirm: CBC/platelets, fibrinogen, PT/INR, aPTT, group & screen/crossmatch.<br>• Neuraxial anesthesia: with FXI ~53% and reassuring ROTEM, neuraxial is often acceptable in low-bleeding-history patients, but follow your institution's threshold and anesthesia policy.<br><br>2) Prophylaxis at delivery / C-section<br>Given the low bleeding history + reassuring EXTEM/FIBTEM:<br>• Avoid routine FXI replacement (FFP/FXI concentrate) up front.<br>• Consider tranexamic acid (TXA) prophylaxis (common approach in FXI deficiency and also supported in obstetric hemorrhage care): |

 

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| | • e.g., 1 g IV after cord clamp (or at delivery), then consider continued TXA postpartum (oral/IV) for several days if your unit uses this strategy, balancing thrombosis risk and VTE prophylaxis needs.<br>3) If bleeding occurs (treat-to-target, ROTEM-guided)<br>Manage obstetric causes first (uterine atony, trauma, retained products), then hemostatic support:<br>A. If bleeding with prolonged INTEM CT and "coagulation factor" concern<br>• FFP is the pragmatic replacement for FXI (especially where FXI concentrate is avoided/limited in pregnancy due to thrombosis risk).<br>• Typical dosing: ~10–15 mL/kg, reassess clinically ± repeat ROTEM/aPTT.<br>B. If fibrinogen becomes the issue<br>• Use FIBTEM A5/A10/MCF:<br>• If low, give cryoprecipitate or fibrinogen concentrate per local thresholds.<br>C. If platelet contribution falls<br>• If EXTEM A10/MCF drop with relatively preserved FIBTEM, consider platelet transfusion (plus evaluate thrombocytopenia/consumption).<br><br>D. If hyperfibrinolysis suspected<br>• Treat with TXA (if not already given) and confirm with EXTEM vs APTEM patterns.<br>4) Postpartum<br>• Watch for secondary postpartum hemorrhage (FXI deficiency can present with delayed bleeding).<br>• If you use postpartum TXA, give clear stop rules (bleeding stops, VTE risk, renal impairment).<br>• Standard VTE prophylaxis should be individualized (pregnancy is prothrombotic; adding TXA/FFP can shift balance).<br><br>ROTEM shows preserved clot firmness (normal EXTEM/FIBTEM) with only a mild intrinsic pathway delay (slightly prolonged INTEM CT), so in a low-bleeding-hx woman with FXI ~53%, avoid routine replacement, consider TXA prophylaxis, and keep FFP available for ROTEM-/bleeding-triggered escalation |

 

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| **DeepSeek** | ## Interpretation<br>The patient has a known **Factor XI (FXI) deficiency** (52.6%, moderate). ROTEM findings are classic for this condition:<br>1. **INTEM CT is significantly prolonged (236 sec; normal range 122-220 sec).** The INTEM assay activates the intrinsic pathway (involving factors XII, XI, IX, VIII). The isolated prolongation here directly reflects the FXI deficiency.<br>2. **EXTEM CT is normal (71 sec; normal range 34-79 sec).** The EXTEM activates the extrinsic pathway (via tissue factor). This pathway bypasses FXI, so clotting time is normal.<br>3. **All other parameters are normal:**<br>○ **Clot Firmness (A5, A10, MCF) in INTEM, EXTEM, and FIBTEM:** These values are all within or above the normal reference ranges. This indicates that once clotting is initiated (despite the delay in INTEM), the clot forms with normal strength. This reflects adequate **fibrinogen levels** (normal FIBTEM) and normal platelet function.<br>○ **APTEM** (performed with aprotinin to inhibit fibrinolysis) shows parameters similar to EXTEM, ruling out significant hyperfibrinolysis.<br>**Conclusion:** The ROTEM profile is characteristic of an **isolated intrinsic pathway coagulation defect**, perfectly consistent with the patient's moderate FXI deficiency. Importantly, **overall clot strength is preserved**. This correlates with her minimal bleeding history (ISTH-BAT score: 1).<br><br>## Suggested Management<br>Management of FXI deficiency in pregnancy is highly individualized, based on bleeding history, factor level, and the mode of delivery. Given her **moderate level (52.6%) and excellent clinical phenotype (minimal bleeding)**, the approach is generally conservative.<br>**1. Prior to Delivery (Multidisciplinary Planning)**<br>• **Team:** Involve Hematology, Anesthesia (Obstetric and possibly Regional), and Obstetrics.<br>• **Type & Crossmatch:** Ensure blood products (packed red blood cells, fresh frozen plasma) are available on standby. |

 

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| | • **Consider Prophylaxis:** For a patient with a level >50% and no significant bleeding history, **routine prophylactic FXI concentrate or plasma is often NOT required for vaginal delivery.** However, it should be available rapidly if needed.<br>• **Tranexamic Acid:** Have a supply ready for use (e.g., 1g IV at delivery, followed by oral post-partum).<br>**2. Anesthesia Considerations**<br>• **Neuraxial Anesthesia (Epidural/Spinal):** This is the main risk vs. benefit discussion.<br>  ○ **Risk:** The prolonged INTEM CT indicates a potential risk for spinal hematoma, a rare but serious complication.<br>  ○ **Benefit:** Normal EXTEM and normal clot strength suggest overall hemostatic competence is good.<br>  ○ **Common Practice:** Many centers would consider neuraxial anesthesia **acceptable** in a patient with FXI >50% and no bleeding history, especially with a normal EXTEM CT. However, a final decision requires a detailed risk-benefit discussion between the patient, hematologist, and anesthesiologist. A cautious approach might favor IV analgesia or general anesthesia for C-section if needed.<br>**3. Management by Mode of Delivery**<br>• **Vaginal Delivery:**<br>  ○ **Prophylaxis:** Likely not needed. **Tranexamic Acid** (1g IV at delivery, then 1g orally every 8 hours for 24-48 hours) is a reasonable first-line strategy to stabilize any clot formed.<br>  ○ **Monitoring:** Vigilant monitoring for postpartum hemorrhage (PPH).<br>• **Cesarean Delivery:**<br>  ○ **Higher bleeding risk** due to surgery. Prophylaxis is more commonly used.<br>  ○ **Options:**<br>  ▪ **FXI Concentrate (preferred):** Dose to achieve a level of ~70% at the time of incision. This allows for safer neuraxial anesthesia if levels are corrected beforehand.<br>  ▪ **Tranexamic Acid:** 1g IV at incision, repeated if needed.<br>  ▪ **Fresh Frozen Plasma (FFP):** Less preferred due to volume and transfusion risks, but an option if concentrate is unavailable.<br>  ○ **Post-op:** Continue tranexamic acid orally for 3-5 days.<br>**4. Postpartum Management**<br>• **Duration of Risk:** The risk of delayed PPH (up to 3-4 weeks) is increased in FXI deficiency due to abnormal wound healing and fibrinolysis at the placental site. |

 

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| | • **Strategy: Extended tranexamic acid prophylaxis** (e.g., 1g orally three times daily for **7-14 days**) is strongly recommended regardless of delivery mode.<br>• **Newborn:** Inform pediatricians. FXI deficiency is autosomal; the baby has a 50% chance of inheritance but testing is not urgent in the newborn period unless bleeding occurs. Avoid traumatic delivery and intramuscular injections (e.g., vitamin K orally if possible).<br>**Summary of Recommendations for This Patient:**<br>1. **Interpretation:** ROTEM confirms an isolated intrinsic pathway defect (prolonged INTEM CT) with preserved clot strength, consistent with her moderate FXI deficiency and mild phenotype.<br>2. **Prophylaxis for Vaginal Delivery:** Likely **not required**. Have **tranexamic acid** available for therapeutic use if bleeding occurs.<br>3. **Neuraxial Anesthesia:** Possible but requires high-level discussion. The normal EXTEM supports its safety, but the final decision is clinical.<br>4. **Postpartum: Mandatory extended tranexamic acid course** (e.g., 7-14 days) to prevent delayed hemorrhage.<br>5. **Communicate:** Ensure all managing teams (OB, anesthesia, nursing) are aware of the diagnosis and the specific plan. |
| **Gemini** | ## Interpretation<br>The Rotational Thromboelastometry (ROTEM) results provided reflect the patient's known moderate Factor XI (FXI) deficiency. Below is the interpretation of the traces and the recommended management plan based on her clinical profile.<br><br>**ROTEM Interpretation**<br>The traces show an isolated defect in the intrinsic pathway, which is the classic "signature" for Factor XI deficiency in viscoelastic testing.<br>• **INTEM (Intrinsic Pathway):** The **Clotting Time (CT)** is significantly prolonged at **238 seconds** (Reference range: 122–221 s) . This confirms a delay in the initiation of clot formation via the intrinsic pathway.<br>• **EXTEM (Extrinsic Pathway):** The CT is **71 seconds** , which is within the normal reference range (34–79 s). This indicates that the extrinsic initiation of coagulation is intact. |

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| | • **FIBTEM (Fibrinogen Contribution):** The **Maximum Clot Firmness (MCF)** is **24 mm** , which is normal (Ref: 14–27 mm). This confirms that fibrinogen levels and fibrin polymerization are adequate.<br>• **Clot Strength:** The **EXTEM MCF** of **72 mm** and **INTEM MCF** of **65 mm** are both within normal limits, suggesting that once the clot eventually forms, its ultimate strength (driven by platelets and fibrin) is robust.<br><br>**Clinical Context & Risk Assessment**<br>• **FXI Level (52.6%):** In FXI deficiency, plasma levels do not always correlate well with the risk of bleeding. However, levels above 50% are generally considered "mild" or "moderate" and often do not lead to spontaneous hemorrhage.<br>• **Bleeding History:** A low **ISTH-BAT score of 1** is a strong negative predictor for surgical bleeding. Patients with FXI deficiency are best managed based on their historical bleeding phenotype rather than their factor level alone.<br>• **Obstetric Risk:** The suspected **macrosomia** increases the risk of postpartum hemorrhage (PPH) due to potential uterine atony, regardless of the coagulation status.<br><br>## Suggested Management<br>Given the patient's minimal bleeding history and moderate FXI levels, a conservative but vigilant approach is recommended:<br>**1. Delivery Planning**<br>• **Tranexamic Acid (TXA):** Administer TXA (e.g., 1g IV) at the start of active labor or prior to Cesarean section, and continue for 24–48 hours postpartum. TXA is highly effective in FXI deficiency because these patients often have impaired resistance to fibrinolysis.<br>• **Factor Replacement:** Routine prophylaxis with Fresh Frozen Plasma (FFP) or FXI concentrate is **not** typically required for a patient with a level > 50% and a negative bleeding history. However, cross-matched FFP should be available in the building as a precaution.<br>• **Uterotonic Agents:** Proactive third-stage management is essential due to the risk of macrosomia-related atony.<br>**2. Neuraxial Anesthesia** |

 

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| | • Epidural or spinal anesthesia is generally considered safe when FXI levels are > 50% and the patient has no history of abnormal bleeding. The normal FIBTEM and EXTEM results provide additional reassurance regarding her ability to form a stable clot once initiated.<br>**3. Postpartum Vigilance**<br>• FXI deficiency is associated with **delayed PPH**. The patient should be monitored closely for several days, and TXA may be extended if there is any concern regarding the stability of the uterine contraction or lochia flow. |
| **Clinical Vignette 3: Severe Hereditary Hypofibrinogenemia in Pregnancy: A Case Report of a Novel Obstetrical Management with Thromboelastometry Guided Fibrinogen Supplementation** | |
| **ChatGPT** | Interpretation<br>**FIBTEM (functional fibrin-based clot)**<br>Key pattern across pregnancy + postpartum:<br>• **Clauss fibrinogen is profoundly low throughout** (≈0.63–1.29 g/L; ref 1.8–4.5 g/L).<br>• **FIBTEM CT is repeatedly prolonged** (often ~77–128 s; ref 38–62 s) → slower *initiation* in the fibrin-only channel.<br>• **Despite that, fibrin clot firmness is preserved**:<br>  ○ **A10/A20 and MCF are mostly within the lab's reference range** (A10 ~8–15 mm; MCF ~10–21 mm; ref MCF 9–25 mm).<br>  ○ Interpretation: *mechanically adequate fibrin clot strength*, even though the lab fibrinogen number looks very low. In congenital fibrinogen disorders this discordance can happen (assay/variant effects, and viscoelastic tests reflecting overall functional clot behavior).<br>• **No hyperfibrinolysis signal** (your "LI30/LI60" columns sit at ~100% throughout; clinically: clot breakdown is not excessive).<br>**Bottom line (FIBTEM):** delayed fibrin clot initiation, but **final fibrin clot strength is acceptable** and stable. |
| | **NATEM (native whole-blood clotting, no activator)**<br>Across pregnancy + postpartum:<br>• **CT and CFT largely within reference** (CT mostly 444–717 s in pregnancy; ref 300–999; CFT mostly 124–198 s; ref 150–700). |

 

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| | • **α-angle normal** (≈54–66; ref 30–70) → propagation kinetics acceptable.<br>• **A10/A20/MCF normal** (MCF ≈52–63; ref 40–65) → **overall clot strength is normal**.<br>• **No fibrinolysis signal** (LI values remain high).<br>**Bottom line (NATEM):** global hemostasis appears **preserved**, with no suggestion of hypocoagulability or hyperfibrinolysis. |
| | **Clinical synthesis**<br>This looks like a **congenital fibrinogen disorder** (your "SHH" history + persistently low fibrinogen) with:<br>• **No bleeding phenotype** clinically, and<br>• **ROTEM showing preserved clot strength** (both fibrin contribution and whole-blood firmness).<br>However, pregnancy/delivery is still **high-stakes** in congenital fibrinogen disorders (miscarriage, bleeding, and also thrombosis—especially around replacement therapy).<br>## Suggested Management<br>**A. Antenatal (now → delivery)**<br>• **Multidisciplinary plan**: obstetrics + hematology + anesthesia; document thresholds and product availability.<br>• **Monitoring**: at least **monthly fibrinogen checks** (and repeat ROTEM if symptoms/bleeding or near delivery). A recent review/consensus approach commonly targets maintaining fibrinogen around **0.5–1.0 g/L** in pregnancy to reduce miscarriage/bleeding risk, with higher targets peripartum.<br>• **Avoid routine prophylactic replacement if clinically stable** *unless* there is bleeding, placental issues, planned invasive procedures, or prior obstetric complications strongly suggest benefit—because replacement can also increase thrombosis risk.<br>**B. Delivery planning (vaginal vs C-section)**<br>Because her **Clauss fibrinogen is <1.0 g/L** most of the time, I'd plan as if she is at **meaningful hemorrhage risk**, even though ROTEM looks reassuring.<br>Commonly used targets (pragmatic, not perfect evidence):<br>• **Peripartum/labor:** keep fibrinogen **>1.5 g/L (150 mg/dL)**<br>• Some case-based guidance aims **≥2.0 g/L (200 mg/dL) at delivery** to reduce bleeding risk.<br>**Practical approach** |

 

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| | 1. **On admission in labor / pre-op for planned delivery**<br>  ○ Check: Hb, platelets, PT/aPTT, fibrinogen, and **ROTEM (include FIBTEM; ideally also EXTEM/INTEM)**.<br>2. **Prophylactic fibrinogen concentrate** (preferred over cryo/FFP when available) immediately before planned delivery / at early labor **to reach target fibrinogen** (e.g., >1.5–2.0 g/L) **and/or a reassuring FIBTEM MCF**.<br>  ○ ROTEM-based dosing can be guided by the relationship between target MCF and current MCF (dose formulas exist).<br>3. **Have products physically available**: fibrinogen concentrate (first line), TXA, uterotonics, blood components.<br>**C. Neuraxial anesthesia (epidural/spinal)**<br>Given the very low Clauss fibrinogen, I would **not** site neuraxial anesthesia *unless* you first **raise fibrinogen to a safer target and confirm stability**.<br>• Case experience in congenital fibrinogen deficiency has used **≥250 mg/dL (2.5 g/L)** around epidural catheter *removal* with ongoing monitoring.<br>• In practice: **coordinate with anesthesia + hematology**, give fibrinogen concentrate, then confirm with repeat fibrinogen/ROTEM before placement and again before catheter removal.<br>**D. If bleeding / postpartum hemorrhage (PPH) occurs**<br>Treat obstetric cause + hemostasis in parallel:<br>• **TXA 1 g IV as soon as PPH is diagnosed**, ideally **within 3 hours**; consider a second dose per protocol if bleeding continues.<br>• **ROTEM-guided escalation**<br>  ○ If **FIBTEM MCF drops** (or A5/A10 low): give **fibrinogen concentrate** promptly (this is a common ROTEM-PPH algorithm cornerstone).<br>  ○ If **CT becomes prolonged with bleeding** (suggesting factor depletion): consider plasma/PCC per institutional algorithm and the rest of the ROTEM picture (ideally EXTEM/INTEM).<br>  ○ Platelets and RBC transfusion per counts/clinical loss.<br>**E. Postpartum (bleeding + thrombosis balance)**<br>• Continue **fibrinogen/ROTEM surveillance for several days**, especially if replacement was given or if there was operative delivery/PPH.<br>• **VTE prophylaxis**: congenital fibrinogen disorders can carry thrombosis risk, and risk may rise with replacement; decide LMWH postpartum based on overall risk and hemostatic stability (hematology-led). |

 

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| | **One-line summary**<br>**ROTEM suggests preserved functional clot strength (FIBTEM + NATEM reassuring), but the persistently very low fibrinogen means delivery should be managed as high-risk with a pre-agreed plan: correct fibrinogen to a peripartum target (≥1.5–2.0 g/L; higher if neuraxial), use ROTEM-guided fibrinogen therapy, and treat any PPH early with TXA + targeted factor replacement.** |
| **DeepSeek** | ## Interpretation<br>Based on the provided case and laboratory data, here is an interpretation and management suggestion for this patient with suspected **Symptomatic Hereditary Hypofibrinogenemia (SHH)**. |
| | **1. Interpretation of FIBTEM and NATEM Assays**<br>**Overall Pattern:**<br>Both assays consistently show **severely reduced fibrinogen levels** (all values between 63.2 and 128.9 mg/dL, against a normal range of 180–450 mg/dL). This is the hallmark of **hypofibrinogenemia**. Despite the low fibrinogen concentration, the *quality* of the fibrin clot appears to be relatively preserved, which is typical in quantitative (rather than qualitative) fibrinogen disorders.<br>**FIBTEM (assesses fibrin-based clot formation):**<br>• **Fibrinogen Level:** Persistently low throughout pregnancy and postpartum.<br>• **Clotting Time (CT):** Prolonged in nearly all readings (range 77–128 sec, normal 38–62 sec), indicating delayed initiation of fibrin clot formation due to low fibrinogen.<br>• **Clot Firmness (A10, A20, MCF):** These values are generally **within or even above the normal reference range** despite low fibrinogen. This suggests that once clot formation begins, the fibrin network that does form has **normal strength and stability**. This is a key finding distinguishing hypofibrinogenemia from dysfibrinogenemia (where clot quality is impaired).<br>• **Lysis Indices (LI30, LI60):** Consistently at 100% (or 98-100%), indicating **no significant fibrinolysis**. The clot, once formed, is stable.<br>**NATEM (non-activated assay, more sensitive to overall coagulation kinetics):** |

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| | • **Clotting Time (CT):** Variably prolonged (range 243–717 sec, normal 300–999 sec), though many values fall within the very wide normal range for this slower test. The trend suggests a slower initiation phase.<br>• **Clot Firmness Parameters (A10, A20, MCF, Alpha-angle):** These are consistently **within or above the normal range**. This corroborates the FIBTEM finding of **adequate clot quality and strength** once formed.<br>• **Lysis Indices:** Again show **complete clot stability** (97–100%), ruling out hyperfibrinolysis as a concern.<br>**Summary of Interpretation:**<br>The patient has a **quantitative fibrinogen deficiency (Hypofibrinogenemia)**. The clotting process is delayed in starting due to low fibrinogen substrate, but the clot that eventually forms is structurally sound and resistant to premature breakdown. This pattern is classic for hereditary hypofibrinogenemia and explains her history of early pregnancy losses (likely due to inadequate decidual-placental fibrin deposition) without a history of severe surgical or traumatic bleeding.<br>## Suggested Management<br>The primary goal is to **prevent peripartum and postpartum hemorrhage**, which is the major risk for such patients. Management should be proactive and multidisciplinary (Obstetrics, Hematology, Anesthesia).<br>**Antepartum:**<br>• **Confirm Diagnosis:** Genetic testing for fibrinogen gene mutations (*FGA, FGB, FGG*) can confirm hereditary hypofibrinogenemia and guide genetic counseling.<br>• **Monitoring:** Continue serial monitoring of fibrinogen levels (Clauss method) and viscoelastic tests (like ROTEM/TEG) every 4-6 weeks and more frequently in the third trimester.<br>• **Target Level:** Aim to maintain fibrinogen > **150 mg/dL** during late pregnancy and especially peripartum to reduce bleeding risk. Her natural levels are below this threshold.<br>**Intrapartum / Peripartum (Delivery Planning):**<br>• **Mode of Delivery:** Vaginal delivery is possible but requires meticulous planning. Cesarean section carries a higher inherent bleeding risk.<br>• **Fibrinogen Replacement:**<br>  ○ **Trigger:** Administer fibrinogen concentrate when the level falls below **150-200 mg/dL** during the peripartum period. |

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| | ○ **Product: Fibrinogen concentrate (e.g., Haemocomplettan®)** is preferred over cryoprecipitate due to viral safety, standardized dosing, and lower volume. Initial dose can be calculated: **Dose (mg) = Target increment (mg/dL) × Plasma volume (dL)**. A typical initial dose is 2-4 grams.<br>○ **Monitoring:** Use **real-time ROTEM (FIBTEM MCF)** and fibrinogen levels to guide repeated dosing. Target a FIBTEM A5 or A10 > 12 mm (her baseline is often at or above this, so aim to keep it there).<br>• **Multidisciplinary Team:** Delivery should occur in a tertiary care center with hematology support, blood bank availability, and an experienced anesthesiology team.<br>• **Avoid:** Regional anesthesia (epidural/spinal) only after consultation with anesthesiology and confirmation of adequate hemostasis, typically with a fibrinogen level > 150 mg/dL.<br>**Postpartum:**<br>• **Vigilant Monitoring:** The risk of postpartum hemorrhage, particularly **secondary PPH**, is elevated and can occur days later. Monitor fibrinogen levels and bleeding closely for at least 3-5 days.<br>• **Continued Replacement:** Maintain fibrinogen levels > 150 mg/dL for the first 24-72 hours postpartum. Her 1st and 4th day PP levels were adequate (105.3, 106.4 mg/dL), but the 2nd day spike (128.9 mg/dL) shows natural variability; reliance on natural levels alone is risky.<br>• **Thromboprophylaxis:** The risk of thrombosis is theoretically low with pure hypofibrinogenemia, but the postpartum state is prothrombotic. Mechanical prophylaxis is essential. Pharmacological prophylaxis (LMWH) should be considered very cautiously, only after ensuring fibrinogen levels are securely in a safe range (>150-200 mg/dL), and in consultation with hematology.<br>**Long-term & Genetic Counseling:**<br>• Counsel the patient on the **autosomal dominant** nature of the condition and the **50% chance of passing it to offspring**.<br>• Discuss implications for future pregnancies and the need for pre-pregnancy counseling and early antenatal management.<br>• Advise about informing family members (siblings, parents) for screening.<br>**In conclusion, this patient requires a carefully planned, proactive approach to peripartum fibrinogen replacement guided by viscoelastic testing and close multidisciplinary coordination to safely navigate delivery and the postpartum period.** |

 

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| **Gemini** | ## Interpretation<br>This clinical case describes a patient with **Severe Hereditary Hypofibrinogenemia (SHH)**, a rare condition where low plasma fibrinogen levels can lead to adverse pregnancy outcomes like recurrent miscarriage and postpartum hemorrhage (PPH).<br>Below is the interpretation of the **FIBTEM** and **NATEM** assays based on the provided data, followed by management recommendations. |
| | **1. Interpretation of ROTEM Assays**<br>**FIBTEM Assay (Fibrin-specific clot strength)**<br>The FIBTEM assay isolates the contribution of fibrinogen to clot formation by inhibiting platelets with cytochalasin D.<br>• **Fibrinogen Levels ($63.2\text{--}128.9$ mg/dL):** These are significantly below the reference range ($180\text{--}450$ mg/dL). This confirms the quantitative deficiency (hypofibrinogenemia).<br>• **Clotting Time (CT: $89\text{--}128$ s):** The CT is prolonged compared to the reference range ($38\text{--}62$ s). This indicates a delay in the initiation of fibrin polymerization due to low substrate availability.<br>• **Maximum Clot Firmness (MCF: $10\text{--}21$ mm):** Interestingly, while these values fall within the standard non-pregnant reference range ($9\text{--}25$ mm), they are **low for a pregnant patient**. In normal pregnancy, fibrinogen levels typically double, and FIBTEM MCF usually exceeds $25$ mm. However, the fact that the MCF is not "zero" (as seen in afibrinogenemia) suggests that the remaining fibrinogen is functionally active.<br>**NATEM Assay (Non-activated global hemostasis)**<br>The NATEM assay provides a holistic view of the coagulation process without using extrinsic activators, relying on the patient's native contact activation.<br>• **Clotting Time (CT):** Mostly remains within the normal range ($300\text{--}999$ s). This suggests that the initiation of the coagulation cascade is largely intact despite the low fibrinogen.<br>• **Maximum Clot Firmness (MCF: $52\text{--}63$ mm):** Despite the low fibrinogen, the NATEM MCF remains **well within the normal reference range** ($40\text{--}65$ mm).<br>• **Clinical Significance:** This is the most critical finding. It indicates that **platelets are successfully compensating** for the low fibrinogen levels, maintaining overall clot stability. This "platelet-driven" |

 

compensation explains why the patient might remain asymptomatic (no significant bleeding history) despite low laboratory fibrinogen levels.

## Suggested Management

The management of SHH in pregnancy is traditionally based on maintaining fibrinogen levels $>100$ mg/dL. However, a more modern, individualized approach—as suggested by this specific case—utilizes **Viscoelastic Testing (VET)** like ROTEM to guide therapy.

**Antenatal Care**

- **Multidisciplinary Approach:** Management should involve hematologists, maternal-fetal medicine specialists, and anesthesiologists.
- **ROTEM-Guided Monitoring:** Instead of solely relying on plasma fibrinogen levels (which may stay low), use **FIBTEM MCF** and **NATEM MCF** to assess functional clot strength.
- **Fibrinogen Replacement:** Fibrinogen concentrate (or cryoprecipitate) should be reserved for:
  - Overt bleeding symptoms.
  - A significant drop in FIBTEM MCF (e.g., $<7\text{--}10$ mm).
  - Preparation for invasive procedures (e.g., amniocentesis).

**Delivery and Postpartum**

- **Mode of Delivery:** Vaginal delivery is generally preferred unless obstetric indications dictate a Cesarean Section (CS). If CS is required, prophylactic fibrinogen supplementation is typically given to target a fibrinogen level $>100\text{--}150$ mg/dL.
- **PPH Prophylaxis:** Use active management of the third stage of labor. Have fibrinogen concentrate and tranexamic acid readily available.
- **Neonatal Screening:** The infant should be tested for fibrinogen levels shortly after birth, as SHH is an inherited condition.

**Summary of Management Strategy**

| Parameter | Threshold for Intervention | Action |
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| **Fibrinogen Level** | $<100$ mg/dL | Consider supplementation |

 

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<tr><td>FIBTEM MCF</td><td>$<10$ mm</td><td>Strong indication for Fibrinogen Concentrate</td></tr>
<tr><td>Clinical Status</td><td>Active Bleeding</td><td>Immediate replacement regardless of numbers</td></tr>
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<tr><td colspan="2">Clinical Vignette 4: Balancing bleeding and thrombosis in chronic liver disease: ROTEM-guided management of life-threatening lower GI bleed</td></tr>
<tr><td>ChatGPT</td><td>

## Interpretation

**1) ROTEM interpretation (from the tracings shown)**

**Key abnormality: severe hypofibrinogenemic / fibrin-based clot weakness**

- **FIBTEM** shows **very low clot firmness** (Panel A: **MCF ~10 mm**; Panel B: **MCF ~19 mm**).
  → This points to **low functional fibrinogen (or impaired fibrin polymerisation)** as the dominant correctable problem.

**Clot initiation is not the primary issue**

- **EXTEM/INTEM CT** are **not markedly prolonged** (INTEM CT ~194–200 s; EXTEM CT ~67–94 s).
  → This argues **against a major coagulation factor deficiency needing immediate FFP/PCC** *based on ROTEM*.

**Platelet contribution appears preserved**

- EXTEM/INTEM clot firmness is **reasonable** (MCF ~57–65 mm) **despite** low FIBTEM early; the "gap" between EXTEM and FIBTEM suggests **platelets are carrying clot strength** (i.e., not primarily a platelet problem *on ROTEM*).

**No strong evidence of hyperfibrinolysis**

- APTEM is not showing a dramatic "rescue" compared with EXTEM, and the traces don't show the classic rapid late lysis pattern.
  → **Do not give tranexamic acid empirically** unless you see clear lysis (e.g., high ML / falling amplitude) or the clinical story strongly suggests it.

**Bottom line:** In a shocked cirrhotic with active GI bleeding, ROTEM is most consistent with **critical functional fibrinogen deficiency** as the main haemostatic driver, with **otherwise acceptable thrombin generation and platelet-driven clot firmness**.

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| | **Suggested Management**<br>**A. Immediate resuscitation (treat this as hemorrhagic shock)**<br>• **Airway:** drowsy + shock → **early airway protection** if mental status deteriorates or ongoing massive bleeding.<br>• **Circulation:** large-bore IV/rapid infuser, **activate massive haemorrhage protocol** if ongoing instability.<br>• **RBC transfusion** to restore oxygen delivery (don't wait for labs if crashing).<br>• **Calcium replacement** during transfusion (ionised Ca often falls and worsens coagulation).<br>• **Warmth + correct acidosis** (both are potent "coagulopathy multipliers").<br>• **Early vasopressor (e.g., norepinephrine)** if hypotension persists after initial blood volume replacement.<br>**B. ROTEM-directed haemostatic therapy (what to give *now*)**<br>**1) Give fibrinogen first (highest-yield correction here)**<br>• With **FIBTEM MCF ~10 mm**, give:<br>  ○ **Fibrinogen concentrate ~3–4 g** (or ~50 mg/kg if weight-based), **OR**<br>  ○ **Cryoprecipitate** (e.g., **10 units / 2 pools**, depending on local unit size).<br>• **Target:** in active bleeding, aim **FIBTEM A5/MCF ≥ 12–15 mm** (many centres push higher for uncontrolled haemorrhage).<br>• **Recheck ROTEM** after replacement (Panel B's FIBTEM MCF ~19 mm would generally be an adequate response).<br>**2) Platelets only if evidence supports it**<br>• ROTEM suggests platelets are contributing well, but in cirrhosis platelet count may still be low.<br>• Give **platelets** if:<br>  ○ Platelet count **<50 × $10^9$/L** in active bleeding / before procedures, **or**<br>  ○ EXTEM A5/A10/MCF remain low **after** fibrinogen correction.<br>**3) FFP / PCC only if CT is clearly prolonged with bleeding**<br>• Here CT isn't screaming factor failure.<br>• Consider **PCC (or FFP)** if **EXTEM CT is prolonged** (or if there's warfarin/DOAC effect) *and* bleeding continues, but avoid reflexive FFP in cirrhosis if ROTEM initiation is acceptable.<br>**4) Antifibrinolytics only if hyperfibrinolysis is present**<br>• Give **TXA** only if you see **significant lysis** (e.g., ML high, falling trace) or APTEM "corrects" EXTEM lysis.<br>**C. Treat the likely GI bleed source (cirrhosis bundle while you resuscitate)** |

 

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| | Even though bleeding is **per rectum**, portal hypertension can still cause significant lower GI bleeding (rectal varices/portal hypertensive colopathy), and empiric therapy is usually low-risk while you localise the source:<br>• **Broad-spectrum antibiotics** (cirrhotics with GI bleed have high infection risk; this also reduces rebleeding).<br>• **Vasoactive therapy for portal HTN** (e.g., **terlipressin or octreotide**) while awaiting definitive localisation, especially if variceal bleeding is plausible.<br>• **Early GI + IR involvement**<br>  ○ **CT angiography** if ongoing brisk bleeding/unclear source.<br>  ○ Endoscopic therapy when feasible (sigmoidoscopy/colonoscopy as appropriate).<br>  ○ **IR embolisation** if arterial source identified.<br>  ○ **TIPS** if portal hypertensive bleeding refractory to endoscopic/medical measures.<br>**D. The tense distended abdomen matters**<br>Progressive distension + shock raises concern for:<br>• **Massive ascites with abdominal compartment physiology**, **or**<br>• **Hemoperitoneum/other intra-abdominal catastrophe**.<br>Do:<br>• **Bedside ultrasound/FAST**, lactate, Hb trend.<br>• If stable enough: **diagnostic paracentesis** (look for blood/SBP), but prioritise haemodynamic stability first. |
| | **Practical "first 15 minutes" order set (what I'd actually do)**<br>1. Airway readiness + $O_2$, 2 large-bore lines, send labs (CBC, CMP, INR/aPTT, fibrinogen, lactate, VBG/ABG) + crossmatch<br>2. Start RBC transfusion, calcium, warming, norepi if needed<br>3. **Give fibrinogen (3–4 g or cryo equivalent) immediately** based on **FIBTEM MCF 10**<br>4. Repeat ROTEM after correction; add platelets/CT-directed factor therapy only if indicated<br>5. Start cirrhosis GI bleed bundle (antibiotics + octreotide/terlipressin) and get GI/IR moving + imaging/localisation |
| **DeepSeek** | Interpretation<br>**1. ROTEM Interpretation** |

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| | The patient has **chronic liver disease**, which typically causes a complex coagulopathy due to:<br>• Reduced synthesis of clotting factors (prolonged CT)<br>• Thrombocytopenia and platelet dysfunction (prolonged CFT)<br>• Possible hyperfibrinolysis (not clearly seen here)<br>• Variable fibrinogen levels (often preserved until late stage)<br>**EXTEM Analysis:**<br>• **CT (Clotting Time):** Prolonged (67–94 sec) → indicates **factor deficiency** (likely II, V, VII, X due to liver disease).<br>• **CFT (Clot Formation Time):** Variable (73–87 sec) → suggests **platelet dysfunction**.<br>• **A10/A20 (Amplitude at 10/20 min): Normal to increased** (57–68 mm) → indicates **adequate clot firmness** once formed, likely due to **adequate fibrinogen**.<br>• **Alpha Angle:** Normal (73–77°) → normal rate of clot formation once initiated.<br>**FIBTEM Analysis:**<br>• **CT:** Slightly prolonged (66–89 sec) → factor-dependent, but FIBTEM mainly assesses fibrinogen.<br>• **A10/A20: Normal to elevated** (18–19.5 mm at A10, 62–64 mm at A20) → confirms **adequate fibrinogen level**.<br>• **CFR (Clot Firmness Rate):** Variable but within acceptable range.<br>**Conclusion from ROTEM:**<br>• **Coagulopathy due to factor deficiency** (prolonged CT in EXTEM and FIBTEM).<br>• **Platelet dysfunction** (prolonged CFT in EXTEM).<br>• **Fibrinogen level is adequate** (normal FIBTEM A10/A20).<br>• **No clear hyperfibrinolysis** (no lysis parameters provided, but EXTEM lysis index not shown).<br><br>## Suggested Management<br>**A. Immediate Resuscitation & Hemodynamic Stabilization**<br>• **IV access × 2 large-bore lines**, begin **crystalloids** (balanced solution) for hypotension.<br>• **Blood transfusion protocol:**<br>  ○ **Packed RBCs** to target Hb > 7–8 g/dL.<br>  ○ **Fresh Frozen Plasma (FFP)** → correct factor deficiency (ROTEM shows prolonged CT). |

 

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| | ○ **Platelets** if count < 50,000/μL or if ongoing bleeding despite FFP (ROTEM suggests platelet dysfunction).<br>○ **NOT fibrinogen concentrate/cryoprecipitate** → fibrinogen is adequate.<br>• **Consider tranexamic acid** if hyperfibrinolysis suspected, but ROTEM does not strongly suggest it.<br>**B. Control of Bleeding**<br>• **Urgent upper GI endoscopy** (likely variceal bleed given liver disease) → band ligation/sclerotherapy.<br>• **IV Octreotide or Terlipressin** (if available) to reduce portal pressure.<br>• **Consider Sengstaken-Blakemore tube** if massive bleeding and endoscopy not immediately available.<br>• **PPI infusion** if peptic ulcer is suspected.<br>**C. Abdominal Distension & Pain**<br>• Tense distended abdomen → consider **abdominal compartment syndrome** or **ascites**.<br>• **Ultrasound abdomen** to assess ascites, portal vein flow, possible hemoperitoneum.<br>• **Monitor intra-abdominal pressure** if indicated.<br>• **Paracentesis** if tense ascites causing respiratory or hemodynamic compromise.<br>**D. Hepatic & Metabolic Management**<br>• **Check ammonia, lactate, electrolytes, renal function**.<br>• **Lactulose/rifaximin** if hepatic encephalopathy (drowsiness may indicate this).<br>• **Vitamin K IV** (10 mg) → may partially correct factor deficiency.<br>• **Avoid NSAIDs, sedatives, nephrotoxic drugs**.<br>**E. Monitoring & Supportive Care**<br>• **Repeat ROTEM** after blood product administration to guide further therapy.<br>• **ICU admission** for monitoring.<br>• **Correct hypothermia** → worsens coagulopathy.<br>• **Antibiotics** (e.g., ceftriaxone) → SBP prophylaxis in cirrhotic with GI bleed. |
| | **3. Summary of ROTEM-Guided Transfusion Strategy** |

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| | ROTEM Finding | Deficit | Transfusion Product |
| | ↑ CT in EXTEM/FIBTEM | Factor deficiency | FFP |
| | ↑ CFT in EXTEM | Platelet dysfunction | Platelets (if low count or bleeding) |
| | Normal FIBTEM A10/A20 | Fibrinogen adequate | Avoid cryoprecipitate/fibrinogen |
| | This patient requires **urgent multidisciplinary care** involving gastroenterology/hepatology, ICU, and hematology to manage bleeding, coagulopathy, and underlying liver disease. | | |
| **Gemini** | ## Interpretation<br>This clinical scenario describes a patient with **Chronic Liver Disease (CLD)** presenting with **acute massive gastrointestinal hemorrhage** and **Class III/IV hemorrhagic shock**.<br>Given the history of CLD and the presentation of bright red blood per rectum with clots, the primary suspicion is variceal bleeding (likely rectal varices or a rapid transit upper GI bleed) or portal hypertensive colopathy. | | |
| | **1. Interpretation of the ROTEM**<br>Rotational Thromboelastometry (ROTEM) provides a real-time assessment of the "cell-based" model of coagulation. Here is the breakdown of the provided traces (referencing Panel A as the primary baseline):<br>**Trace Analysis** | | |

 

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| | • **EXTEM (Extrinsic Pathway):** The **CT (Clot Time)** of 67s and **MCF (Maximum Clot Firmness)** of 57mm are within normal limits. This indicates that the initiation of the clotting cascade via Tissue Factor and the final strength of the clot (platelets + fibrinogen) are preserved.<br>• **INTEM (Intrinsic Pathway):** The **CT** of 200s is within the normal reference range (typically 100–240s). This suggests no significant heparin effect or deficiency in the intrinsic pathway factors.<br>• **FIBTEM (Fibrinogen Function):** The **MCF** of 10mm is at the lower end of the normal range (typically $>7-10$ mm). This indicates that fibrinogen levels and polymerization are currently adequate to support clot formation, though they are not robust.<br>• **APTEM (Fibrinolysis Assessment):** Comparison between EXTEM and APTEM shows no significant difference in MCF or **ML (Maximum Lysis)**. This rules out systemic **hyperfibrinolysis**.<br>**Clinical Synthesis**<br>Despite the patient being in profound shock, the ROTEM shows **rebalanced hemostasis**. In chronic liver disease, patients often have low pro-coagulant factors but also low anti-coagulant factors (like Protein C), leading to "normal" looking ROTEM traces despite an underlying fragility.<br>The "normal" ROTEM here suggests that the bleeding is likely **mechanical/hemodynamic** (due to high portal pressure) rather than a primary failure of the coagulation system (coagulopathy).<br><br>## Suggested Management<br>**2. Management Plan**<br>The immediate priority is **hemodynamic stabilization** and **source control**, rather than "correcting" laboratory numbers that appear relatively stable.<br>**Phase 1: Resuscitation (The "ABCDE" approach)**<br>• **Airway:** The patient is drowsy (GCS 14). While not yet comatose, he is at high risk for aspiration if he develops hematemesis. Consider elective intubation for airway protection before endoscopy.<br>• **Circulation:** * Establish two large-bore IVs (14G or 16G) or a central venous catheter.<br>  ○ **Massive Transfusion Protocol (MTP):** Activate MTP immediately. Use a 1:1:1 ratio of Packed Red Blood Cells (PRBCs), Fresh Frozen Plasma (FFP), and Platelets.<br>  ○ **Goal:** Maintain a restrictive transfusion strategy (target Hemoglobin ~7–8 g/dL) to avoid increasing portal pressure, which can worsen variceal bleeding. |

 

**Phase 2: Targeted Medical Therapy**

- **Vasoactive Drugs:** Start **Terlipressin** (2mg IV initially) or **Octreotide** infusion. These reduce portal venous pressure and are life-saving in variceal bleeds.
- **Antibiotics:** IV **Ceftriaxone** (1g/day). Prophylactic antibiotics significantly reduce mortality in cirrhotic patients with GI bleeds.
- **Corrective Agents:** While ROTEM is normal, if the FIBTEM MCF drops below 7mm during resuscitation, administer **Fibrinogen concentrate** or **Cryoprecipitate**.

**Phase 3: Source Control**

- **Urgent Endoscopy:** Once the blood pressure is somewhat stabilized (MAP > 65 mmHg), the patient needs an urgent EGD/Colonoscopy to identify the bleed and apply endoscopic band ligation or sclerotherapy.
- **Salvage Therapy:** If endoscopic therapy fails, consider a **TIPS (Transjugular Intrahepatic Portosystemic Shunt)** procedure or a Sengstaken-Blakemore tube if the bleed is confirmed to be esophageal.

**Summary Table for ROTEM-Guided Therapy**

| Parameter | Finding | Interpretation | Intervention |
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| **EXTEM CT** | 67s (Normal) | Initiation is good | None needed |
| **FIBTEM MCF** | 10mm (Borderline) | Fibrinogen is borderline | Monitor; give Cryo if $<7$mm |
| **INTEM CT** | 200s (Normal) | No heparin effect | None needed |
| **APTEM ML** | No lysis | No hyperfibrinolysis | No TXA needed currently |

**Clinical Vignette 5: Perioperative ROTEM® evaluation in a patient affected by severe VII factor deficiency undergoing microvascular decompression craniotomy for hemifacial spasm**

**ChatGPT**

## Interpretation

**Overall pattern:** globally *normal viscoelastic clot formation and stability* on **EXTEM** and **INTEM**, with **no hyperfibrinolysis**. **FIBTEM** suggests **adequate fibrin-based clot** (for most adult reference ranges).

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| | **EXTEM (extrinsic pathway + platelets + fibrinogen)**<br>• **CT**: normal (A ~74 s; B ~45 s) → **no clinically relevant factor deficiency** on the extrinsic side.<br>• **CFT / A5 / A10 / MCF**: in normal range (MCF ~60–66 mm) → **good overall clot strength** (platelet + fibrin contribution looks adequate).<br>• **Lysis**: LI30 ~100%, ML ~0–1% → **no significant fibrinolysis**.<br>**INTEM (intrinsic pathway + platelets + fibrinogen)**<br>• **CT**: mildly longer than EXTEM (A ~115 s; B ~141 s) but still not "markedly prolonged" and clot strength is preserved → **no major intrinsic factor problem evident**; would only worry if this were clearly outside your lab's reference range or if bleeding occurs.<br>• **MCF**: ~63–64 mm → **good clot firmness**.<br>• **Lysis**: normal.<br>**FIBTEM (fibrin-based clot)**<br>• **A10/MCF**: ~18–20 mm (A) and ~16–19 mm (B) → typically **adequate fibrinogen contribution** (not suggestive of hypofibrinogenemia).<br>• **Lysis**: normal.<br>**Key caveat (important for this case):** ROTEM **does not reliably "rule out" antiplatelet drug effect** (aspirin/clopidogrel/ticagrelor). EXTEM/INTEM clot firmness can look normal despite clinically meaningful platelet inhibition. If he's on chronic antiplatelets and you're planning **intracranial surgery**, you need **platelet function testing** (e.g., Multiplate/VerifyNow) or manage empirically based on drug history/timing.<br><br>## Suggested Management<br>**A. Pre-op plan (elective neurosurgery = treat as high bleeding risk)**<br>1. **Clarify antiplatelet regimen**<br>  ○ Aspirin alone vs **dual antiplatelet therapy (DAPT)**.<br>  ○ Indication (vascular graft/stent?) and time since repair → discuss with vascular/cardiology about thrombosis risk if stopped.<br>2. **Hold antiplatelets appropriately (typical practice)**<br>  ○ **P2Y12 inhibitors**: stop pre-op (often **5–7 days** depending on agent). |

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| | ○ **Aspirin**: many neurosurgeons prefer stopping **~5–7 days** before intracranial surgery if thrombosis risk permits.<br>3. **Check basics alongside ROTEM**<br>○ Platelet count, PT/INR, aPTT, fibrinogen (Clauss), Hb, renal function, calcium.<br>○ **Add platelet function testing** if available (best way to handle the "ROTEM blind spot").<br>**B. What ROTEM says you *don't* need prophylactically**<br>• **No routine FFP/PCC** (CTs not suggestive of factor failure).<br>• **No fibrinogen concentrate/cryo** (FIBTEM MCF adequate).<br>• **No antifibrinolytic as treatment** (no lysis signal) — though some centers still use TXA prophylactically in selected neurosurgical workflows; that's protocol-dependent.<br>**C. If surgery cannot be delayed / antiplatelet effect likely present**<br>For urgent intracranial surgery where antiplatelet reversal is needed:<br>• **Platelet transfusion** (e.g., 1–2 adult doses/one apheresis unit) timed close to incision.<br>• **Desmopressin (DDAVP) 0.3 mcg/kg IV** (often used to improve platelet adhesion/function in aspirin-like platelet dysfunction).<br>• Consider **repeat assessment** with platelet function testing (or trend bleeding + ROTEM).<br>**D. Intra-op / bleeding algorithm (ROTEM-guided)**<br>If unexpected bleeding occurs, treat based on the pattern:<br>• **Prolonged CT (EXTEM/INTEM)** → consider **PCC or FFP** (depending on context), check heparin effect if suspected.<br>• **Low EXTEM A5/MCF with relatively preserved FIBTEM** → **platelets** (number/function issue).<br>• **Low FIBTEM A5/MCF** → **fibrinogen concentrate/cryo**.<br>• **Increased lysis (ML high / LI30 low)** → **TXA**. |
| | **Bottom line:** This ROTEM looks **hemostatically "fit"** (good clot strength, stable clot, no fibrinolysis). The main perioperative bleeding risk here is **drug-induced platelet dysfunction**, which ROTEM may not detect—so management hinges on **antiplatelet history + timing** and ideally **platelet function testing**, with **platelets ± DDAVP** if urgent or if inhibition is confirmed. |

 

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| **DeepSeek** | ## Interpretation<br>The ROTEM values show a significant coagulopathy consistent with **coagulation factor deficiency**.<br>• **Panel A (likely INTEM or similar):** The prolonged clotting time (CT, represented by the first number/horizontal coordinate: 39, 20, 50, 41, 50 vs. normal typically <100s? Note: values seem low, but scale is 0-160; this suggests very short CT, but let's reconcile - the description implies *long* CT, so the "39" might be a *clotting time* in mm on x-axis? There is ambiguity. However, the clinical picture and "prolonged" description overrides.) The pattern described typically indicates a prolonged initiation of clot formation.<br>• **Panel B (likely EXTEM):** Similarly shows a prolonged clotting time (CT, first number: 30, 20, 40, 60, 80) and likely a reduced maximum clot firmness (MCF, implied by the second number/vertical coordinate amplitude), indicating poor clot strength once formed.<br>• **Key Finding:** The combination of prolonged CT in both INTEM (assesses the intrinsic pathway) and EXTEM (assesses the extrinsic pathway) points to a **global coagulation factor deficiency** (common pathway factors like II, V, X, or I/fibrinogen if also low). The fact that both pathways are affected rules out an isolated heparin effect (which would prolong INTEM much more than EXTEM).<br>• **Context:** In this patient, the most likely cause is **chronic antiplatelet therapy** (which doesn't typically cause this ROTEM pattern) **combined with potential underlying liver dysfunction, vitamin K deficiency, or dilution from recent surgeries**. The history of femoral aneurysm repair might also involve vascular issues affecting coagulation.<br><br>## Suggested Management<br>Given the planned **posterior fossa craniotomy for Microvascular Decompression (MVD)**, meticulous hemostasis is critical due to the confined space and risk of catastrophic brainstem compression from even a small hematoma.<br>**A. Preoperative & Perioperative Management:**<br>1. **Correct the Coagulopathy:**<br>○ **Withhold Antiplatelet Therapy:** The chronic antiplatelet therapy (likely aspirin or a P2Y12 inhibitor like clopidogrel) must be stopped preoperatively. The required washout period (typically 5-7 days for clopidogrel, 5 days for aspirin) must be balanced against the urgency of surgery and the risk of stent |

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| | thrombosis from the femoral aneurysm repair. **A discussion with the neurologist/neurosurgeon and the vascular surgeon/cardiologist who prescribed the antiplatelet is mandatory.**<br>○ **Factor Replacement:** Based on the ROTEM suggesting factor deficiency:<br>▪ Check standard coagulation tests (PT/INR, aPTT, Fibrinogen).<br>▪ Administer **Vitamin K** (e.g., 10 mg IV slowly) to correct any deficiency.<br>▪ If urgent correction is needed, consider **Prothrombin Complex Concentrate (PCC)** or **Fresh Frozen Plasma (FFP)**. PCC is often preferred for rapid, volumetric-sparing correction of multiple factor deficiencies.<br>○ **Myasthenia Gravis Optimization:** Coordinate closely with neurology. Ensure optimal control of myasthenia symptoms preoperatively. Stress-dose steroids may be needed if the patient is on chronic steroids. Anesthesiology must be fully aware to avoid depolarizing muscle relaxants and carefully manage non-depolarizing agents.<br>2. **Intraoperative Strategy:**<br>○ **Anesthesia:** Use a myasthenia-friendly protocol. Prepare for potential need for postoperative ventilation.<br>○ **Hemostasis:** Use meticulous surgical technique. Consider topical hemostatic agents. ROTEM can be repeated intraoperatively to guide further factor or fibrinogen replacement if needed.<br>○ **Monitoring:** Standard neurosurgical monitoring plus likely facial nerve monitoring for MVD.<br>**B. Postoperative Management:**<br>1. **Neurological Monitoring:** Close monitoring in an ICU or step-down unit for any signs of posterior fossa hematoma (altered consciousness, new brainstem signs, respiratory depression) or facial nerve deficit.<br>2. **Coagulation Management:**<br>○ Repeat ROTEM/coagulation tests postoperatively.<br>○ The decision on when to restart antiplatelet therapy is **critical** and requires a multidisciplinary consensus weighing the risk of hemorrhagic complications against the risk of thrombosis in the femoral aneurysm repair site. This is often done 24-72 hours post-op if hemostasis is secure, but must be individualized.<br>3. **Myasthenia Management:** Monitor for myasthenic crisis triggered by surgical stress. Resume pre-operative pyridostigmine and immunosuppressant regimen as soon as possible.<br>**Summary** |

 

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| | The patient presents for high-risk neurosurgery with a significant coagulopathy on ROTEM, primarily due to coagulation factor deficiency, exacerbated by chronic antiplatelet use. **The cornerstone of management is the proactive correction of the coagulopathy pre-operatively and a carefully planned, multidisciplinary perioperative strategy involving neurosurgery, neurology (for myasthenia and HFS), hematology/transfusion medicine, and anesthesiology.** The timing of antiplatelet therapy interruption and resumption is the highest-stakes decision and requires input from all relevant specialists. |
| **Gemini** | ## Interpretation<br>**1. ROTEM Interpretation**<br>The provided image shows two sets of ROTEM traces (A and B), likely taken at different time points (e.g., baseline and intraoperatively). Both sets demonstrate **normal to robust coagulation profiles**.<br>• **EXTEM (Extrinsic Pathway):** In both A and B, the Clotting Time (CT) and Maximum Clot Firmness (MCF) are within normal limits (CT ~45–74s; MCF ~64–66mm). This indicates that the extrinsic coagulation factors and the interaction between platelets and fibrin are intact.<br>• **INTEM (Intrinsic Pathway):** The parameters are within normal ranges (CT ~115–141s; MCF ~63–64mm). There is no evidence of a heparin effect or intrinsic factor deficiency.<br>• **FIBTEM (Fibrinogen Component):** The MCF is notably high (19–20mm; normal is typically 7–12mm). This suggests a **hyperfibrinogenemic state** or a very robust fibrin contribution to the clot, which is common in elderly patients or those with chronic inflammatory states/post-surgical recovery.<br>• **Lysis (ML):** Maximum Lysis is 0% to 2%, ruling out systemic hyperfibrinolysis.<br>**Critical Caveat:** Standard ROTEM (EXTEM/INTEM/FIBTEM) **cannot detect the effect of antiplatelet therapy** (like Aspirin or Clopidogrel). Even with a "normal" ROTEM, the patient's platelets may not be functioning correctly due to his chronic medication.<br><br>## Suggested Management<br>The primary challenge is the patient's **chronic antiplatelet therapy** in the context of **Microvascular Decompression (MVD)**. MVD is a high-risk neurosurgical procedure where even a minor bleed in the cerebellopontine angle can lead to devastating neurological deficits or brainstem compression. |

 

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| | **Pre-operative/Intra-operative Strategy:**<br>• **Antiplatelet Reversal:** Since standard ROTEM doesn't show the defect, consider a **Platelet Function Test** (e.g., Multiplate or VerifyNow) if available. If the patient is still under the effect of antiplatelets, the "normal" ROTEM MCF is misleading because it shows the *quantity* of platelet/fibrin interaction, not the *quality* of platelet aggregation.<br>• **Desmopressin (DDAVP):** In neurosurgery, a dose of DDAVP ($0.3 \mu g/kg$) is often administered to improve platelet adhesion and function in patients on antiplatelet therapy, even if the platelet count is normal.<br>• **Platelet Transfusion:** Because this is an intracranial procedure with a high risk of "critical-site" bleeding, have pooled platelets available. If significant microvascular oozing occurs during craniotomy despite a normal ROTEM, empiric platelet transfusion is justified.<br>• **Blood Pressure Management:** Strict MAP (Mean Arterial Pressure) control is vital. Avoid spikes during emergence from anesthesia to prevent hematoma formation at the surgical site.<br>**Myasthenia Gravis (MG) Considerations:**<br>• **Neuromuscular Blockade:** Avoid or minimize non-depolarizing muscle relaxants. If used, strict quantitative monitoring (TOF) is mandatory, and Sugammadex should be available for complete reversal, as residual weakness can lead to post-operative respiratory failure.<br>• **Post-operative Monitoring:** Monitor in an ICU/HDU setting for "Myasthenic Crisis" which can be triggered by the stress of surgery or infections. |
| **Clinical Vignette 6: Hypofibrinogenemia Following an Anaphylactic Shock Caused by a Neuromuscular Blocking Agent—Case Report** | |
| **ChatGPT** | Interpretation<br>**Big picture**<br>This ROTEM set shows a **transient, profound hypocoagulable state at A**, **partial correction at B**, and **near-normalization by C**.<br>That time course strongly fits an **acute, reversible coagulopathy occurring during the peri-anaphylaxis / shock-resuscitation period** (rather than a chronic inherited bleeding disorder).<br>**A (worst point): severe hypocoagulability driven by weak clot formation + very low fibrin contribution** |

 

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| | **EXTEM (A):**<br>• **CT 272 s (↑)** and **CFT 660 s (↑↑)** → delayed initiation and *very slow* clot build-up<br>• **A5 12 / MCF 30 (↓↓)** → very weak clot overall<br>**INTEM (A):**<br>• CT 290 s (not dramatically abnormal given the wide ref), but **CFT 509 s (↑↑)** and **MCF 37 (↓)** → again, poor propagation/firmness<br>**FIBTEM (A):**<br>• **CT 4823 s (massively ↑)** and **A5/A10/MCF ~4 mm (profoundly ↓)**<br>• This is the key: the **fibrin-based clot is essentially absent** at A.<br>**Interpretation at A:**<br>A **major functional fibrinogen deficit** (or severe inhibition of fibrin polymerization/thrombin generation in the TF-based assay), with **global clot weakness** also implying reduced platelet contribution and/or impaired thrombin generation.<br>In this clinical context (sudden hypotension + bronchospasm after rocuronium), the leading unifying explanation is **anaphylaxis-associated coagulopathy**, often compounded by **dilution from resuscitation**, **consumption**, and sometimes a **heparin-like anticoagulant effect** from mast-cell mediator release. (You'd prove/deny heparin effect with HEPTEM/anti-Xa, and confirm fibrinogen with Clauss fibrinogen.) |
| | **B: improving but still abnormal**<br>• EXTEM: CT 105 (still ↑), CFT 155 (slightly ↑), A5 30 (low), MCF 56 (now within range)<br>• INTEM: propagation still a bit slow, firmness now acceptable<br>• FIBTEM: CT 119 (still ↑), but fibrin clot still very low at early amplitudes in the table<br>**Interpretation at B:** recovering thrombin generation/platelet contribution; fibrin component still catching up. |
| | **C: essentially normal hemostatic profile**<br>• EXTEM CT 63, CFT 72, A5 47, MCF 68 → normal<br>• INTEM CFT 63, A5 49, MCF 65 → normal<br>• FIBTEM CT 65, MCF 18 → normal<br>**Interpretation at C:** the coagulopathy was **reversible** and has resolved (at least by ROTEM). |

 

## Suggested Management

**Immediate priorities (this patient's real emergency is anaphylaxis)**

1. **Treat suspected anaphylaxis aggressively**
    - **Epinephrine** (titrate IV in anesthetized shock; IM if appropriate context), high-flow $O_2$
    - **Large-volume crystalloids**
    - Bronchospasm: inhaled β-agonist ± epinephrine, deepen anesthesia as needed
    - **Adjuncts:** antihistamine + corticosteroid (adjunct only; don't delay epi/fluids)
2. **Abort/postpone surgery** and maintain hemodynamic stability.

**Hemostasis strategy (ROTEM-guided)**

Even if there's no bleeding, this ROTEM A trace is so abnormal that I'd **treat/monitor as "high bleeding risk" until proven corrected**, especially if additional invasive procedures/lines are needed.

**Send labs now (and repeat):**

- CBC/platelets, PT/INR, aPTT
- **Clauss fibrinogen**
- D-dimer, renal function, lactate
- Consider anti-Xa or thrombin time if anticoagulant effect suspected
- Serum tryptase (for anaphylaxis confirmation)

**If there is bleeding, oozing, or urgent need for surgery/line placement while still at "A-like" ROTEM:**

- **Fibrinogen replacement is first-line** (because FIBTEM is essentially flat)
    - **Fibrinogen concentrate** or **cryoprecipitate**
    - Target pragmatically: **FIBTEM A5 ≥ 10–12 mm** (or institution threshold)
- If EXTEM remains weak after fibrinogen correction (low A5/MCF):
    - **Platelets** (platelet contribution is likely impaired/consumed/diluted)
- If **CT remains prolonged** (EXTEM/INTEM) suggesting factor deficiency:
    - **FFP** (or **PCC** in selected settings with specialist input and thrombotic-risk awareness)
- If hyperfibrinolysis is demonstrated (you'd need LI30/ML; not clearly shown here):
    - **Tranexamic acid**
- If a **heparin-like effect** is suspected (especially if HEPTEM corrects INTEM/EXTEM abnormalities):

 

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| | ○ **Protamine** can be considered, but only with evidence/strong suspicion + senior oversight<br>**Re-check ROTEM after each intervention** and trend to the "C" profile. |
| | **After stabilization**<br>• Document rocuronium as the likely trigger; refer for formal allergy work-up.<br>• Consider whether any further surgery should be done in a controlled setting with:<br>○ avoidance of the culprit NMBA,<br>○ anaphylaxis plan,<br>○ and **peri-op coagulation monitoring** if severe shock recurs. |
| | **What I would *not* do**<br>• I would **not** give empiric blood products purely "because ROTEM looks bad" if the patient is stable, not bleeding, and the ROTEM rapidly normalizes to C — but I *would* keep a **low threshold** if bleeding starts or procedures are needed while still abnormal. |
| **DeepSeek** | ## Interpretation<br>The ROTEM results show a **severe, isolated fibrinogen deficiency or dysfunction**, consistent with an **acquired coagulopathy** likely triggered by the acute anaphylactic reaction. This is not a typical presentation of a primary bleeding disorder.<br>**Key Findings:**<br>1. **FIBTEM:**<br>○ **Sample A (baseline/early reaction?):** Shows a **massively prolonged CT (4823 sec)**, indicating a **severe fibrinogen polymerization defect**. The clot firmness parameters (A5-A30, MCF) are **very low (4-18 mm)**, confirming **profound functional fibrinogen deficiency**.<br>○ Samples B & C show progressive normalization with intervention (CT shortening, MCF increasing), indicating response to therapy.<br>2. **EXTEM & INTEM:**<br>○ Both channels in Sample A show **markedly prolonged CT and CFT**, and **severely reduced clot amplitude (A5-A30) and MCF**. |

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| | ○ The fact that both the extrinsic (EXTEM) and intrinsic (INTEM) pathways are affected, **but FIBTEM is the most severely abnormal**, points to the problem being at the level of **fibrinogen/fibrin polymerization**, not a factor deficiency in the coagulation cascade.<br>○ The improvement in Samples B & C parallels the FIBTEM recovery.<br>3. **APTEM:**<br>○ APTEM (which contains aprotinin to inhibit fibrinolysis) shows a similar severe pattern in Sample A, ruling out **hyperfibrinolysis** as the primary cause. The defect persists even when fibrinolysis is blocked.<br>**Conclusion:** The ROTEM pattern is diagnostic for **acute, severe hypofibrinogenemia**. This can occur in anaphylaxis due to mechanisms like:<br>• **Activation of the coagulation system and consumption** of fibrinogen (a form of low-grade Disseminated Intravascular Coagulation - DIC).<br>• **Fibrinogenolysis** (specific breakdown of fibrinogen by proteases like tryptase released during mast cell degranulation).<br>## Suggested Management<br>**1. Immediate Management (Anaphylaxis):**<br>• **Stop the presumed trigger** (rocuronium). Use an alternative muscle relaxant if needed in the future.<br>• **ABCs (Airway, Breathing, Circulation):** Maintain airway, give **100% oxygen**. For bronchospasm, nebulized bronchodilators (salbutamol/albuterol). Secure IV access.<br>• **Epinephrine (Adrenaline):** First-line treatment. Give IM epinephrine (0.3-0.5 mg of 1:1000) immediately. Repeat every 5-15 minutes as needed.<br>• **Fluids:** Rapid IV crystalloid bolus (e.g., 500-1000 mL) for hypotension.<br>• **Adjuncts:**<br>○ **Antihistamines:** H1 (e.g., diphenhydramine) and H2 blockers (e.g., ranitidine).<br>○ **Corticosteroids:** Hydrocortisone IV to prevent biphasic reactions.<br>○ **Monitor closely** in an ICU/high-dependency setting.<br>**2. Management of Coagulopathy (Based on ROTEM):**<br>• **Goal:** Correct functional fibrinogen deficiency. |

 

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| | • **First-line Therapy: Fibrinogen concentrate** (target fibrinogen level >1.5-2.0 g/L) **OR Cryoprecipitate** (if concentrate unavailable). This directly addresses the core problem shown on ROTEM.<br>• **Avoid Fresh Frozen Plasma (FFP) as initial therapy.** FFP is poor in fibrinogen and would expose the patient to unnecessary volume and allergen risk without correcting the primary deficit effectively.<br>• **Transfusion Support:** Monitor for thrombocytopenia or other factor deficiencies, but the ROTEM suggests they are not the primary issue. Platelets and other factors should be given based on lab results/clinical bleeding, not empirically.<br>• **Antifibrinolytics (e.g., Tranexamic Acid):** May be considered given the possible fibrinogenolytic component, especially if there is clinical bleeding. However, APTEM did not show hyperfibrinolysis, so its role is adjunctive.<br>**3. Follow-up and Investigation:**<br>• **Repeat ROTEM / Coagulation Tests:** To monitor response to fibrinogen replacement.<br>• **Laboratory Tests:** Send blood for **fibrinogen level, D-dimer, tryptase** (to confirm anaphylaxis), and a full coagulation panel.<br>• **Allergist Referral:** For definitive testing (skin prick, IgE) to confirm rocuronium (or other agent) allergy and provide future anesthesia guidance.<br>**Summary:** The patient is experiencing **anaphylaxis-induced severe acquired hypofibrinogenemia**. Immediate management focuses on **treating the anaphylaxis** (epinephrine, fluids, bronchodilators) and **correcting the coagulopathy** with **fibrinogen replacement** (concentrate or cryoprecipitate). Avoid empiric FFP. Close monitoring and allergist follow-up are essential. |
| **Gemini** | Interpretation<br>Based on the clinical history and the ROTEM (Rotational Thromboelastometry) traces provided, this is a classic presentation of **anaphylaxis-induced endogenous heparin-like effect (HLE) and transient coagulopathy.**<br>The patient experienced a Grade III/IV anaphylactic reaction (hypotension, tachycardia, bronchospasm) likely triggered by **rocuronium**, which is a frequent culprit in perioperative anaphylaxis. |
| | **1. Interpretation of the ROTEM Traces** |

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| | The traces labeled **A, B, and C** represent a timeline of the event: **A** is during the peak of the reaction, **B** is during early recovery, and **C** is the resolution/baseline.<br>**Phase A (Acute Anaphylactic Phase)**<br>• **FIBTEM:** Shows a severely prolonged Clot Time (CT: 4823s) and a very low Maximum Clot Firmness (MCF: 4mm). This indicates a profound defect in fibrin polymerization.<br>• **EXTEM/INTEM:** Both show significantly prolonged CT and CFT (Clot Formation Time) with reduced MCF.<br>• **Interpretation:** The "spindly" appearance across all channels during the acute event suggests a severe coagulopathy. In the context of anaphylaxis, this is typically due to the **massive release of endogenous heparin and tPA** from mast cell degranulation.<br>**Phase B & C (Recovery Phase)**<br>• **Trend:** You can see a rapid, spontaneous improvement. By Phase B, parameters are moving toward normal.<br>• **Phase C:** The traces have completely normalized (e.g., FIBTEM MCF of 18mm and EXTEM MCF of 68mm).<br>• **Interpretation:** This confirms that the coagulopathy was transient and associated with the acute allergic surge rather than a primary bleeding disorder or consumption (DIC).<br>## Suggested Management<br>The primary goal is the treatment of **anaphylactic shock**, followed by the management of the secondary coagulopathy.<br>**Immediate Resuscitation**<br>• **Epinephrine (Adrenaline):** The cornerstone of treatment. Administer IM (or titrated IV if in a monitored OR setting) to treat hypotension and bronchospasm.<br>• **Fluid Resuscitation:** Aggressive crystalloid boluses to manage the distributive shock caused by massive vasodilation.<br>• **Oxygen & Airway:** Ensure 100% $O_2$ and maintain the airway (bronchospasm management with inhaled salbutamol if needed).<br>**Management of Coagulopathy**<br>• **Conservative Approach:** Since the physical examination revealed **no abnormal bleeding**, the best management for the ROTEM findings is **observation**. The endogenous heparin-like effect usually resolves spontaneously as the heparin is cleared, as evidenced by the progression to Trace C. |

 

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| | • **Avoid Unnecessary Products:** Do not rush to give FFP or Cryoprecipitate based solely on the ROTEM if there is no clinical bleeding, as this may further complicate the allergic picture.<br>• **Protamine:** If the patient were experiencing life-threatening hemorrhage, a low dose of protamine could be considered to neutralize the endogenous heparin effect, though this is rarely necessary.<br>**Follow-up & Prevention**<br>• **Tryptase Levels:** Draw serial serum tryptase (now, at 2 hours, and at 24 hours) to confirm mast cell degranulation and the diagnosis of anaphylaxis.<br>• **Allergy Referral:** The patient must be referred for skin prick testing or IgE testing specifically for rocuronium and other neuromuscular blocking agents.<br>• **Documentation:** Provide the patient with an "Allergy Warning" letter and update their medical records to avoid rocuronium and possibly all NMBA triggers until testing is complete. |
| **Clinical Vignette 7: Paraneoplastic Hyperfibrinolysis in Oesophageal Adenocarcinoma: A Case Report** | |
| **ChatGPT** | Interpretation<br>**1) ROTEM (EXTEM) interpretation**<br>**Overall pattern: hypocoagulability driven by weak clot formation + clinically relevant fibrinolysis.**<br>**Initiation (factors / thrombin generation)**<br>• **CT 71 s (normal 38–79):** *clot initiation is preserved* on EXTEM → no major "start-up" factor deficiency signal on this assay.<br>**Propagation / build-up (fibrinogen + platelets)**<br>• **CFT 155 s (upper limit)** and **α 60° (low)** → **slow clot propagation** (often fibrinogen deficiency/dysfunction and/or platelet contribution issues).<br>• **A5 32 mm** (visually looks low-ish) + **MCF 52 mm (low; ref 54–72)** + **AUC low** → **reduced clot firmness**, most consistent with **low fibrinogen** in the context you gave (and/or thrombocytopenia/platelet dysfunction—can't separate without FIBTEM).<br>**Fibrinolysis**<br>• **LI30 100 and LI45 97 (normal early), but LI60 53 (markedly low)** and the trace **narrows substantially after ~45–60 min** → **late, significant clot breakdown = hyperfibrinolysis signal**. |

 

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| | ○ ML is 11% (within the listed "normal" cutoff), but **the LI60 of 53% is the red flag** here: by 60 minutes, ~half the clot firmness is gone.<br>**Most likely hemostatic explanation (given D-dimer ↑, fibrinogen ↓, PT prolonged, spontaneous bruising)**<br>• **Consumptive coagulopathy/DIC spectrum**, likely **malignancy-associated**, with **hypofibrinogenaemia** and **hyperfibrinolysis contribution** (the ROTEM lysis pattern + high D-dimer strongly supports this).<br>Suggested Management<br>**A. Immediate priorities (if actively bleeding / procedure needed)**<br>1. **Resuscitation + hemorrhage control**<br>○ IV access, hemodynamics, source evaluation (GI bleed? intramuscular? mucosal? post-op site?).<br>2. **Send/confirm core labs**<br>○ CBC/platelets, PT/INR, aPTT, fibrinogen (Clauss), D-dimer, renal/liver panel, blood film (schistocytes), group & screen/crossmatch.<br>3. **Run additional ROTEM assays urgently**<br>○ **FIBTEM** (separates fibrinogen component), ± **APTEM** (confirms fibrinolysis by lysis correction), ± INTEM/HEPTEM if heparin effect is a concern.<br>**B. Targeted component therapy (based on what you already have)**<br>**1) Replace fibrinogen (high priority here)**<br>• With **low fibrinogen clinically + low EXTEM firmness**, treat as **hypofibrinogenaemia until proven otherwise**:<br>○ **Fibrinogen concentrate** *or* **cryoprecipitate**<br>○ Aim for **fibrinogen ≥1.5–2.0 g/L** (higher end if ongoing bleeding/major surgery).<br>○ Recheck **FIBTEM A5/MCF** (or repeat EXTEM + Clauss fibrinogen) after replacement.<br>**2) Correct prolonged PT if bleeding / invasive procedure**<br>• **FFP (or PCC depending on local protocol/context)** if there is clinically significant bleeding *and* coagulation factors are depleted.<br>○ Note: EXTEM CT is normal *right now*, so I'd let **clinical bleeding + INR/PT trend + repeat ROTEM** drive this rather than PT alone.<br>**3) Platelets if thrombocytopenic or platelet dysfunction suspected** |

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| | • If **platelets are low** (common in DIC) or if **EXTEM is weak but FIBTEM is relatively preserved**, give **platelet transfusion** (typical targets: **>50 x10$^9$/L** if bleeding; higher for high-risk sites/procedures).<br>**4) Treat hyperfibrinolysis (carefully)**<br>• Given the **late lysis** signal, if the patient has **ongoing bleeding** *and* hyperfibrinolysis is confirmed/suspected:<br>  ○ **Tranexamic acid (TXA)** can be considered (common approach: 1 g IV, then repeat/infusion per protocol).<br>  ○ Best practice is to **confirm with APTEM** (lysis should improve) and **pair TXA with fibrinogen replacement**, because "locking down" fibrinolysis without substrate correction can be ineffective.<br>  ○ Be cautious if there is strong clinical concern for **macrothrombosis**; DIC can be prothrombotic as well as bleeding—this is a risk–benefit call guided by bleeding severity.<br>**C. Treat the cause (this is non-negotiable in DIC)**<br>• **Search for and treat triggers:** malignancy progression/recurrence, infection/sepsis, tissue necrosis, liver dysfunction, etc.<br>• In malignancy-associated coagulopathy, definitive control often requires **oncology-directed therapy** in addition to supportive transfusion.<br>**D. Monitoring**<br>• **Repeat ROTEM** after each major intervention (fibrinogen/FFP/platelets/TXA) to confirm correction:<br>  ○ Expect **α and MCF to improve** with fibrinogen/platelets.<br>  ○ Expect **lysis to improve** if TXA is effective (and/or APTEM would show reduced lysis). |
| | **What I'd say as a one-liner**<br>"EXTEM shows preserved clot initiation but slow clot build-up with mildly reduced clot strength, plus significant late fibrinolysis—consistent with hypofibrinogenaemia/consumptive coagulopathy with a hyperfibrinolysis component; manage with fibrinogen replacement ± platelets/FFP as clinically indicated, consider TXA if bleeding and lysis confirmed, and treat the underlying driver (likely malignancy-associated DIC)." |
| **DeepSeek** | ## Interpretation<br>The ROTEM trace and parameters indicate a coagulopathy characterized by **impaired clot formation and accelerated fibrinolysis (hyperfibrinolysis)**. This pattern is highly consistent with **Disseminated Intravascular** |

 

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| | **Coagulation (DIC)**, which aligns with the patient's history of adenocarcinoma, clinical presentation (bruising), and lab findings (elevated D-dimer, low fibrinogen, prolonged PT).<br>**Key Abnormal Findings:**<br>1. **Reduced Clot Firmness/Strength:**<br>○ **Maximum Clot Firmness (MCF):** 52 mm (low, below reference range 54-72). This indicates a weak final clot.<br>○ **Amplitude at 5,10,20,30 min (A5, A10, A20, A30):** All values are low for their respective time points, showing slow and suboptimal clot development.<br>○ **Maximum Clot Elasticity (MCE):** 107 dyn/cm$^2$ (low, below reference range 86-230). Another measure of weak clot strength.<br>2. **Delayed/Impaired Clot Formation Kinetics:**<br>○ **Clot Formation Time (CFT):** 155 s (at the upper limit of normal 34-159, indicating slow initial clot formation).<br>○ **Alpha Angle (α):** 60° (low, below reference range 67-83°). This confirms a slowed rate of clot polymerization.<br>○ **Time to Max Velocity (MAXV-t):** 146 s (prolonged, above reference range 46-140 s). The clot reaches its maximum speed of formation later than normal.<br>3. **Evidence of Severe Hyperfibrinolysis:**<br>○ This is the most striking abnormality. The lysis indices show a catastrophic loss of clot stability over time.<br>○ **Lysis Index at 60 min (LI60):** 53% (severely low, well below reference range 85-100%). This means nearly half of the clot has lysed by 60 minutes.<br>○ **Maximum Lysis (ML):** 11% (within normal range 0-15%), but this parameter can be misleading if the clot is weak to begin with. The steep drop in LI30 (100%) to LI45 (97%) to LI60 (53%) on the table, and the visible downward slope after the peak on the trace, are diagnostic of hyperfibrinolysis.<br>○ This explains the clinical bruising and bleeding despite only moderately impaired clot formation parameters.<br>**Summary:** The ROTEM shows a **hypocoagulable profile with severe hyperfibrinolysis**. This is not simply a dilutional or factor-deficient coagulopathy; the accelerated clot breakdown is the dominant feature, characteristic of DIC, often seen in advanced malignancy. |

 

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| | ## Suggested Management<br>Management should be urgent and address both the underlying cause and the specific coagulation defects.<br>**1. Immediate/Therapeutic Interventions:**<br>• **Antifibrinolytic Therapy: Tranexamic acid** is indicated to halt the excessive fibrinolysis. A typical loading dose (e.g., 1g IV over 10 min) followed by infusion should be administered.<br>• **Fibrinogen Replacement:** Given the low plasma fibrinogen (per labs) and low clot firmness (MCF) on ROTEM. Administer **fibrinogen concentrate** (target fibrinogen level >1.5-2.0 g/L) or **cryoprecipitate**.<br>• **Coagulation Factor Support:** Consider administration of **Prothrombin Complex Concentrate (PCC)** or **fresh frozen plasma (FFP)** to address the prolonged PT and support overall coagulation factor levels, especially if active bleeding is present.<br>• **Platelets:** Monitor platelet count. Transfuse if thrombocytopenic and bleeding.<br>**2. Investigate & Treat the Underlying Cause:**<br>• **Malignancy Progression/Recurrence:** The history of oesophagogastric junction adenocarcinoma is the most likely trigger for DIC. Urgent oncology review and investigation for disease recurrence or progression (e.g., CT imaging) is critical.<br>• **Sepsis/Infection:** A common precipitant of DIC. Screen for and treat any source of infection aggressively.<br>• **Other Causes:** Rule out other triggers like acute ischemia or massive transfusion.<br>**3. Monitoring & Support:**<br>• **Repeat ROTEM/VEM:** Monitor the response to tranexamic acid and factor replacement. The goal is to see a normalization of the lysis indices (LI60 >85%) and an improvement in MCF.<br>• **Standard Labs:** Serial CBC, fibrinogen, D-dimer, PT/APTT.<br>• **Thromboprophylaxis:** This is a high-risk decision. While bleeding is the immediate threat, patients with cancer-associated DIC are also at high risk for thrombosis. **Once the hyperfibrinolytic bleeding is controlled with antifibrinolytics, prophylactic-dose anticoagulation (e.g., LMWH) should be strongly considered**, guided by daily clinical and lab assessment.<br>**In summary, this patient has likely developed acute DIC with hyperfibrinolysis secondary to his underlying malignancy. Immediate management requires intravenous tranexamic acid and fibrinogen replacement, followed by urgent investigation and treatment of the cancer recurrence or other precipitating cause.** |

 

**Gemini**

## Interpretation

**1. ROTEM Interpretation**

The ROTEM EXTEM assay results indicate a state of **severe hyperfibrinolysis** and **reduced clot strength**.

**Key Parameter Analysis**

| Parameter | Value | Interpretation |
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| **Clotting Time (CT)** | 71s | **Normal** (38–79s). Clot initiation via the extrinsic pathway is intact. |
| **Alpha ($\alpha$) angle** | 60° | **Low** (67–83°). Indicates a slow rate of clot polymerization, typically due to low fibrinogen levels. |
| **Max Clot Firmness (MCF)** | 52mm | **Low** (54–72mm). Indicates reduced overall clot stability. |
| **Lysis Index at 60m (LI60)** | 53% | **Critically Low** (85–100%). This is the most striking finding. It shows that 47% of the clot has dissolved within 60 minutes of the CT. |

**Visual Analysis of the Trace**

- **Trace (a):** Shows the initial 60 minutes where the clot forms but begins to narrow prematurely.
- **Trace (b):** Shows the extended run time. The characteristic **"cigar-shaped" or "fish-tail" appearance** is pathognomonic for **systemic hyperfibrinolysis**. The clot forms but is rapidly degraded by plasmin.

**Clinical Correlation**

The combination of a known adenocarcinoma (which can release tissue factor and plasminogen activators), low fibrinogen, elevated D-dimer, and spontaneous bruising strongly suggests **Disseminated Intravascular Coagulation (DIC) with a hyperfibrinolytic phenotype**.

 

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| | ## Suggested Management<br>The management must be two-fold: stabilizing the acute bleeding diathesis and addressing the underlying malignancy.<br>**A. Immediate Hemostatic Resuscitation**<br>1. **Antifibrinolytics:** This is the priority given the LI60 of 53%.<br>  ○ Administer **Tranexamic Acid (TXA)**: Typically $1\text{g}$ IV bolus, potentially followed by an infusion, to inhibit plasminogen activation and stop the rapid clot breakdown.<br>2. **Fibrinogen Replacement:** The low MCF and low alpha angle, combined with the laboratory report of low fibrinogen, necessitate replacement.<br>  ○ Administer **Fibrinogen Concentrate** (e.g., $3\text{--}4\text{g}$) or **Cryoprecipitate** (typically 10 units/pools).<br>  ○ **Target:** Aim for an MCF $> 10\text{mm}$ on a FIBTEM assay (though FIBTEM isn't shown here, it is the standard guide for fibrinogen replacement).<br>3. **Prothrombin Complex Concentrate (PCC) or FFP:** If the Prothrombin Time (PT) remains significantly prolonged and the patient continues to bleed after fibrinogen correction, PCC or Fresh Frozen Plasma may be considered to replace clotting factors.<br>**B. Underlying Cause and Monitoring**<br>1. **Treat the Trigger:** The coagulopathy is secondary to the adenocarcinoma. Oncological consultation is required to determine if the cancer is progressing or if there is a secondary complication (like sepsis) triggering the DIC.<br>2. **Serial Monitoring:** Repeat ROTEM (specifically EXTEM and FIBTEM) every 30–60 minutes during active bleeding or after intervention to assess the effectiveness of TXA and fibrinogen replacement.<br>3. **Avoid Platelet Over-transfusion:** Unless platelets are $< 50 \times 10^9/\text{L}$, focus primarily on fibrinogen and antifibrinolytics, as these are the primary deficits shown on the trace. |
| **Clinical Vignette 8: Thromboelastography-Guided Anesthetic Management for Cesarean Section in a Patient With Antiphospholipid Syndrome: A Case Report** | |

 

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| **ChatGPT** | ## Interpretation<br>**1) TEG interpretation (TEG 6s: CK, CKH, CFF)**<br>**Key numbers**<br>• **CK R = 15.9 min (↑)** (ref ~4.6–9.1)<br>• **CKH R = 11.1 min (↑)** (ref ~4.3–8.3)<br>• **K ~ normal (CK 1.9, CKH 1.0)**<br>• **Angle normal–high (CK 68°, CKH 75°)**<br>• **MA normal–high (CK 61, CKH 66)**<br>• **CFF A10 33 / MA 35.8 (↑)** → strong fibrin(ogen) contribution<br>• **LY30 0.1%** → no hyperfibrinolysis<br>**What this means**<br>• The **main abnormality is a prolonged R time** → **delayed clot initiation** (problem at the "start" of clotting).<br>• **R shortens from CK (15.9) to CKH (11.1)**. Because **CKH contains heparinase**, this pattern is **compatible with residual heparin effect** (not completely corrected, but partially), which fits the history of **SC UFH 8 hours prior**.<br>• **Clot propagation and strength are preserved/robust**: normal/high angle, normal/high MA, and **high CFF** (pregnancy is typically hyperfibrinogenemic/hypercoagulable).<br>• **No meaningful fibrinolysis**.<br>**Bottom line: Residual anticoagulant effect (likely heparin) with otherwise strong clot strength.** This is *not* a "bleeding-from-low-fibrinogen/platelets" TEG.<br>## Suggested Management<br>**2) Suggested management (practical, peri-CS anesthesia + hemostasis)**<br>**A. Anesthetic decision (full stomach + neuraxial safety)**<br>Because:<br>• she ate **1 hour** ago → high aspiration risk if GA (needs RSI), **but**<br>• TEG suggests **residual heparin effect** → neuraxial hematoma risk<br>**Safest default in this scenario: General anesthesia with rapid sequence induction (RSI)** *unless you can clearly neutralize heparin and confirm normalization before neuraxial*. |

 

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| | If the team is strongly trying to avoid GA:<br>• **Consider protamine** to reverse UFH effect (even though SC + 8 h makes dosing less exact). A common pragmatic approach is a **small-to-moderate protamine dose (e.g., 25–50 mg IV slowly)**, then **repeat TEG (or aPTT/anti-Xa if available)** to confirm R normalizes.<br>• Only proceed with **neuraxial** if coagulation looks convincingly normalized **and** you are within your institutional guideline thresholds.<br>**B. Hemorrhage / transfusion strategy (TEG-guided)**<br>• Current TEG **does not support empiric fibrinogen, platelets, or factor replacement** (MA and CFF are strong; angle is fine).<br>• Treat the **obstetric cause of bleeding first** (uterotonics, surgical control).<br>• Have blood ready given the clinical bleed:<br>  ○ **Type & cross**, large-bore IVs, warmed fluids.<br>  ○ If bleeding escalates and you need products:<br>  ▪ **R still prolonged without heparin effect** → consider **FFP** (or PCC in select contexts) *guided by labs/clinical picture*.<br>  ▪ If **MA drops** → platelets.<br>  ▪ If **CFF/functional fibrinogen drops** → cryo/fibrinogen concentrate.<br>• **Tranexamic acid (TXA)**: if this is evolving toward postpartum hemorrhage, **TXA is reasonable early** in obstetrics protocols; in **APS** you'd be more thrombosis-conscious, but life-saving hemorrhage control generally takes priority—use with senior oversight and reassess thrombosis prophylaxis plan.<br>**C. Anticoagulation plan (APS, peri-op)**<br>• **Hold further heparin** pre-op.<br>• Post-op: restart thromboprophylaxis once surgical hemostasis is secure (typical obstetric practice: **~6–12 h** after CS for prophylactic dosing, longer if bleeding; coordinate with hematology/OB).<br>• Continue APS plan postpartum (often **LMWH/UFH + aspirin** depending on prior events and institutional protocol). |
| | **One-line summary**<br>**TEG shows delayed clot initiation with partial correction on heparinase tracing → residual heparin effect; clot strength is robust (high fibrin contribution), no fibrinolysis. Manage as residual UFH: avoid neuraxial unless** |

 

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| | **you reverse/confirm normalization; proceed with RSI GA if urgent; no empiric blood products from this TEG, treat obstetric bleeding and reassess with repeat TEG/labs if bleeding worsens.** |
| **DeepSeek** | ## Interpretation<br>**TEG Interpretation:**<br>The TEG assay performed appears to be a **standard kaolin TEG (CK)** and a **heparinase-modified TEG (CKH)**. The comparison between these two tracings is used to detect the presence of residual heparin.<br>1. **Key Finding:** The most critical parameter is the **Maximum Amplitude (MA)**.<br>○ In the **CK** (standard) channel, the MA is **0.1 mm**.<br>○ In the **CKH** (heparinase) channel, the MA is **0.2-2.6 mm**.<br>○ **Interpretation:** There is **no significant difference** between the MA values in the CK and CKH channels. Heparinase neutralizes heparin. If a significant heparin effect were present, the MA in the CKH channel would be substantially larger than in the CK channel. The minimal difference here (0.1 mm vs. 0.2-2.6 mm) indicates that **there is no detectable residual systemic heparin effect** at the time of testing.<br>2. **Other Parameters (CK channel):** The patient's baseline coagulation status (reflected in the CK channel) shows:<br>○ **R time:** 15.9 min (normal range typically ~5-10 min). This is **prolonged**, indicating slower initial clot formation.<br>○ **K time:** 1.9 min & **Angle:** 68.4°. These are within normal ranges, suggesting normal fibrinogen function and clot kinetics once initiated.<br>○ **MA:** 61.3 mm. This is within the normal range, indicating normal platelet function and final clot strength.<br>○ **LY30:** 0.1%, indicating minimal fibrinolysis.<br>**Overall Baseline Coagulation:** The profile suggests a **mild coagulopathy characterized primarily by a prolonged initiation phase (R time)**, which could be due to her underlying condition (APS, mixed connective tissue disease), medications (possibly azathioprine effect), or a dilutional effect from the recent bleed. However, clot strength and stability (MA, LY30) are preserved. |

 

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| | ## Suggested Management<br>**1. Anesthesia Plan:**<br>• The **primary concern was residual heparin contraindicating neuraxial anesthesia**. The TEG results (specifically the CK vs. CKH comparison) provide strong evidence that **there is no significant circulating heparin**.<br>• Given this finding, along with the fact that the last dose of subcutaneous unfractionated heparin was **8 hours ago** (which meets the more conservative ASRA guideline threshold of 4-6 hours for prophylactic dosing), **neuraxial anesthesia (spinal or combined spinal-epidural) can be performed safely** from a coagulation perspective.<br>• **Recommendation: Proceed with neuraxial anesthesia.** This aligns with the initial preference and mitigates the aspiration risk associated with her recent food intake and potential for difficult airway (short stature). A single-shot spinal is often preferred for cesarean delivery for its speed and reliability.<br>**2. Perioperative Coagulation Management:**<br>• **No heparin reversal is required** based on TEG.<br>• The prolonged R time should be noted. While not severe enough to contraindicate neuraxial anesthesia alone, it suggests a potential for increased intraoperative bleeding.<br>• **Ensure ready availability of blood products** (FFP to correct coagulation factors if the prolonged R time correlates with clinical bleeding). However, routine transfusion is not indicated based on TEG alone.<br>• **Monitor blood loss closely** during and after surgery.<br>• Postoperatively, the timing for re-initiation of thromboprophylaxis with heparin should follow standard protocols for postpartum patients with APS, considering the surgical procedure.<br>**Summary:** The TEG effectively rules out a residual heparin effect, clearing the major obstacle for neuraxial anesthesia. The patient's underlying mild coagulation delay (prolonged R time) warrants careful surgical hemostasis and vigilance for bleeding but does not alter the primary anesthesia plan. Proceeding with spinal anesthesia is the recommended and safest course. |

 

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| **Gemini** | ## Interpretation<br>This Thromboelastography (TEG) profile provides critical information regarding this patient's coagulation status, especially considering the timing of her last heparin dose and her current emergency status.<br>**1. TEG Interpretation**<br>The interpretation is based on comparing the three channels: **CK** (Celite-activated), **CKH** (Celite + Heparinase), and **CFF** (Functional Fibrinogen).<br>• **Residual Heparin Effect (CK vs. CKH):**<br>  ○ The **R-time** (time to initial clot formation) in the CK channel is significantly prolonged at **15.9 min** (Normal: 4.6–9.1).<br>  ○ In the CKH channel (where heparin is neutralized), the R-time drops to **11.1 min**.<br>  ○ **Interpretation:** Because the R-time shortened significantly after adding heparinase, there is **confirmed residual heparin activity**. However, because the R-time in the CKH channel is *still* prolonged (11.1 min vs. the reference max of 8.3), there is also an **underlying coagulation factor deficiency** or delay not related to heparin, potentially due to the 500 mL blood loss or her underlying MCTD/APS.<br>• **Clot Kinetics (K and Angle):**<br>  ○ The **K-time** and **Angle** are largely within or near normal limits in the heparinase-corrected sample, suggesting that once the clot begins to form, the speed of clot strengthening is adequate.<br>• **Clot Strength (MA):**<br>  ○ The **MA** (Maximum Amplitude) in the CKH channel is **66.4 mm**, which is at the high end of normal.<br>  ○ The **CFF MA** (Fibrinogen contribution) is high at **35.8 mm**.<br>  ○ **Interpretation:** Platelet function and fibrinogen levels are robust. The patient is actually in a somewhat **hypercoagulable state** once the initial "lag" in clot initiation is overcome, which is common in pregnancy and APS.<br>• **Fibrinolysis (LY30):**<br>  ○ At **0.1%**, there is no evidence of premature clot breakdown (fibrinolysis). |

 

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| | ## Suggested Management<br>This case presents a classic "difficult choice" in obstetric anesthesia: the risk of **aspiration** (Full Stomach) vs. the risk of **spinal/epidural hematoma** (Coagulopathy).<br>**Anesthetic Choice: General Anesthesia (GA)**<br>Despite the risk of aspiration from her recent meal, **General Anesthesia with a Rapid Sequence Induction (RSI)** is the safer path here for the following reasons:<br>1. **Safety of Neuraxial:** Japanese guidelines recommend an 8–10 hour wait after SC heparin. While she is at the 8-hour mark, the TEG explicitly shows that her coagulation (R-time) is **not normalized** even after heparin neutralization. Performing a spinal or epidural in the presence of a prolonged R-time increases the risk of a catastrophic spinal hematoma.<br>2. **Emergency Status:** She has already lost 500 mL of blood and has APS; waiting longer to satisfy the "fasting" or "heparin window" requirements could jeopardize the fetus or the mother if the bleeding accelerates.<br>3. **Aspiration Mitigation:** The risk of aspiration during GA can be managed with professional RSI technique (cricoid pressure, though controversial; rapid intubation with a cuffed tube; and prokinetic/antacid premedication if time allows).<br>**Hemorrhage Management**<br>• **Avoid Protamine (usually):** While there is heparin effect, her MA is high, and her R-time is only moderately prolonged. Given her Antiphospholipid Syndrome (APS), she is at high risk for **thromboembolic events**. Aggressively reversing heparin with protamine could trigger a thrombotic crisis.<br>• **Blood Products:** She does not currently require platelets or cryoprecipitate (as evidenced by her high MA and CFF). If bleeding continues and the R-time remains prolonged, **Fresh Frozen Plasma (FFP)** would be the preferred intervention to address the factor deficiency. |
| **Clinical Vignette 9: Perioperative Management of Single Lung Transplantation for a Chronic Obstructive Lung Disease Patient with Congenital Alpha2-Plasmin Inhibitor Deficiency** | |
| **ChatGPT** | ## Interpretation<br>**1) Interpretation of the intra-op ROTEM (A → B → C)** |

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| | **Overall theme:** *Clot initiation and clot strength look preserved on EXTEM/INTEM, with no obvious hyperfibrinolysis on these tracings; FIBTEM is comparatively low → the fibrin(ogen) component is relatively weak (or at least borderline) and is the main "watch-out."*<br>**A (first set)**<br>• **EXTEM / INTEM:** Rapid build-up to a **large, stable clot** (high A10/MCF visually), with a **flat, sustained plateau** out to ~60 min → **no clear clot breakdown** (i.e., no obvious hyperfibrinolysis on-screen).<br>• **FIBTEM: Low, almost flat** trace compared with the EXTEM/INTEM width → suggests **low fibrin-based clot firmness** (hypofibrinogenaemia / impaired fibrin polymer contribution), possibly masked on EXTEM/INTEM by platelet contribution.<br>**B (second set)**<br>• Very similar to A: **good whole-blood clot firmness** (EXTEM/INTEM) with **persistently low FIBTEM** and **no obvious lysis**.<br>**C (third set)**<br>• **EXTEM / INTEM:** Still **strong, stable clot**.<br>• **FIBTEM: Slightly improved** vs A/B (looks thicker/higher), consistent with **some fibrinogen repletion** and/or improved fibrin contribution.<br>**Key clinical nuance (because she has congenital α2-antiplasmin deficiency):**<br>• This condition predisposes to **severe hyperfibrinolysis**, sometimes dramatic around reperfusion/ECMO transitions. Even if these specific snapshots don't show lysis, you should **actively look for it** with:<br>  ○ **Serial EXTEM LI30/LI60 / ML**, and<br>  ○ **APTEM** (or equivalent antifibrinolysis channel) *if any suggestion of lysis or oozing*.<br><br>## Suggested Management<br>**2) Suggested management for this patient (lung transplant + α2-PI deficiency)**<br>**A. Antifibrinolysis strategy (high priority in α2-PI deficiency)**<br>• **Prophylactic tranexamic acid (TXA)** is very reasonable unless contraindicated:<br>  ○ e.g., **10–15 mg/kg bolus** at induction (or at start of bleeding risk), then **~1 mg/kg/h infusion** intra-op (institutional protocols vary).<br>• If **clinical oozing** or ROTEM suggests fibrinolysis (falling LI30/60, rising ML, narrowing late trace): |

 

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| | ○ **Escalate TXA** (repeat bolus, increase infusion per protocol).<br>○ **Run APTEM** to confirm "lysis corrects with inhibitor" pattern.<br>**B. Treat the weak fibrin(ogen) component (your main ROTEM abnormality here)**<br>• Use **FIBTEM A5/A10/MCF-guided fibrinogen replacement**:<br>○ If FIBTEM is below your trigger (commonly A5 < ~7 mm or MCF < ~9–10 mm, depending on local algorithm) **and bleeding/risk is high**:<br>▪ **Fibrinogen concentrate** (e.g., 3–4 g or ~50 mg/kg) **or cryoprecipitate** (dose per local practice),<br>▪ then **recheck ROTEM**.<br>**C. Platelets / factors only if ROTEM indicates**<br>• If **EXTEM clot strength is low** *and* FIBTEM is adequate → **platelets**.<br>• If **CT is prolonged** (EXTEM/INTEM) with bleeding → **FFP** or **PCC** (per algorithm) + correct hypocalcaemia, acidosis, hypothermia.<br>**D. Replace α2-antiplasmin if bleeding persists despite TXA**<br>• There isn't a widely available targeted α2-antiplasmin concentrate in many settings; **FFP contains α2-antiplasmin**, so in refractory bleeding with a fibrinolytic phenotype, **FFP can be considered** as part of a balanced resuscitation strategy (while continuing TXA and ROTEM guidance).<br>**E. Transplant/ECMO practicals**<br>• Tight control of **temperature, ionized $Ca^{2+}$, pH**, and **heparin/protamine balance** (if on ECMO/CPB) to avoid iatrogenic coagulopathy.<br>**Serial ROTEM** at key phases: pre-anhepatic equivalents aren't relevant here, but **pre-implant, post-reperfusion, after protamine/ECMO wean**, and with any bleeding surge. |
| **DeepSeek** | Interpretation<br>**Interpretation of Intraoperative ROTEM**<br>1. **Isolated Prolongation of EXTEM CT:** The EXTEM clotting time (CT) is significantly prolonged at 606 seconds (normal range typically 38-79 sec). This parameter reflects the initiation of clot formation via the extrinsic (tissue factor) pathway.<br>2. **All Other Parameters Normal:** The FIBTEM and INTEM CT values are normal (20 sec each).<br>○ **Normal FIBTEM CT (20 sec)** indicates that **fibrinogen levels and function are adequate**. |

 

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| | ○ **Normal INTEM CT (20 sec)** indicates that the **intrinsic pathway and heparin-neutralized pathway are functioning normally**. This essentially rules out a significant coagulation factor deficiency (like hemophilia) or the presence of heparin effect in this sample.<br>**Conclusion:** This ROTEM pattern—**isolated, marked prolongation of EXTEM CT with normal INTEM and FIBTEM**—in a patient with **congenital alpha2-antiplasmin (α2-PI) deficiency** is highly characteristic of **severe hyperfibrinolysis**.<br>• **Mechanism:** Alpha2-antiplasmin is the primary, fast-acting inhibitor of plasmin. Its deficiency allows unopposed plasmin activity, leading to rapid breakdown of fibrin clots as soon as they form.<br>• **ROTEM Correlation:** In EXTEM, the reagent contains tissue factor to strongly activate clotting, but also contains an activator that can promote some fibrinolysis. In this patient, the formed clot is immediately and severely lysed by unopposed plasmin, preventing the tracing from reaching the amplitude needed to trigger the CT measurement, resulting in a falsely prolonged or "infinite" CT. The INTEM test, with a different activator, may be less sensitive to this immediate lysis in this specific case, or the lysis occurs just after the CT is recorded.<br><br>Suggested Management<br>The core issue is **uncontrolled fibrinolysis**, not a deficiency of clotting factors or fibrinogen.<br>1. **Antifibrinolytic Therapy (Urgent First-Line Treatment):**<br>○ **Drug of Choice: Tranexamic Acid (TXA).** This synthetic lysine analog inhibits plasminogen activation and plasmin activity, directly countering the pathophysiology.<br>○ **Dosing:** A typical high-dose regimen for major surgery/cardiac surgery can be used, adjusted for the known deficiency. For example:<br>▪ **Loading Dose:** 20-30 mg/kg IV over 20 minutes.<br>▪ **Maintenance Infusion:** 5-10 mg/kg/hr for the duration of the surgery and potentially into the early postoperative period.<br>○ **Rationale:** This will pharmacologically replace the function of the missing alpha2-antiplasmin, stabilizing clots and reducing bleeding.<br>2. **Do NOT empirically transfuse based on EXTEM CT alone.** |

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| | ○ Fresh Frozen Plasma (FFP) or Prothrombin Complex Concentrates (PCCs) are **not indicated** as the coagulation factors are normal (evidenced by normal INTEM).<br>○ Cryoprecipitate or Fibrinogen Concentrate are **not indicated** as fibrinogen is adequate (evidenced by normal FIBTEM).<br>3. **Monitor Response:**<br>○ Repeat ROTEM after initiating TXA infusion. The goal is to see a **correction of the EXTEM CT into the normal range**.<br>○ Continue to monitor surgical bleeding, standard hemodynamics, and hemoglobin.<br>4. **Postoperative Planning:**<br>○ Coordinate with hematology for ongoing management.<br>○ Antifibrinolytic therapy (oral or IV) may be required for a prolonged period or during future hemostatic challenges, as this is a congenital disorder.<br>**In summary, the ROTEM indicates severe hyperfibrinolysis due to the patient's known alpha2-antiplasmin deficiency. The immediate and primary management is the administration of an antifibrinolytic agent, specifically tranexamic acid, to stabilize clot formation.** |
| **Gemini** | ## Interpretation<br>This ROTEM (Rotational Thromboelastometry) profile provides critical information for managing a high-risk surgical patient with a known congenital coagulation disorder ($\alpha_2$-PI deficiency) undergoing a major procedure (lung transplantation).<br>**1. Interpretation of the Intraoperative ROTEM**<br>The three panels (A, B, and C) represent a chronological progression, likely reflecting the administration of blood products or hemostatic agents during the surgery.<br>• **Panel A (Initial State):**<br>○ **EXTEM & INTEM:** Show relatively normal Clotting Times (CT) and a high Maximum Clot Firmness (MCF). This indicates that the extrinsic and intrinsic pathways are functional and that there is a sufficient combination of platelets and fibrinogen to create a "gross" clot. |

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| | ○ **FIBTEM:** The MCF is **severely low** (nearly flat). Since FIBTEM isolates the contribution of fibrinogen to clot strength by inhibiting platelets, this indicates **profound hypofibrinogenemia** or a functional fibrinogen deficit.<br>• **Panels B & C (Progression):**<br>○ There is a visible, stepwise **increase in the FIBTEM amplitude**. By Panel C, the FIBTEM MCF has reached a much more acceptable level (likely $>10$ mm).<br>○ **Absence of Lysis:** Notably, despite the $\alpha_2$-plasmin inhibitor ($\alpha_2$-PI) deficiency, these specific traces do **not** show classic "step-down" or "fish-tail" patterns of hyperfibrinolysis (where the trace narrows significantly after reaching MCF). This suggests that fibrinolysis is either being successfully inhibited pharmacologically or has not yet been triggered by the surgical stress/reperfusion.<br><br>## Suggested Management<br>The management of this patient must address both the **quantitative fibrinogen deficiency** seen on the ROTEM and the **qualitative risk of hyperfibrinolysis** inherent to $\alpha_2$-PI deficiency.<br>**A. Aggressive Antifibrinolytic Therapy**<br>Since $\alpha_2$-PI is the primary physiological inhibitor of plasmin, its absence means that once plasmin is generated, it will degrade clots unchecked.<br>• **Action:** Administer high-dose **Tranexamic Acid (TXA)** or $\epsilon$-aminocaproic acid (EACA).<br>• **Rationale:** These agents prevent plasminogen from binding to fibrin, effectively "blocking" the fibrinolysis that the patient cannot naturally inhibit. This is mandatory in lung transplants where reperfusion typically triggers a surge in fibrinolytic activity.<br>**B. Fibrinogen Replacement**<br>Panel A shows a critical need for fibrinogen to ensure clot stability, especially as lung transplantation involves significant raw surfaces and potential for microvascular bleeding.<br>• **Action:** Administer **Fibrinogen Concentrate** or **Cryoprecipitate**.<br>• **Goal:** Aim for a FIBTEM MCF of $>10-12$ mm (as seen moving toward Panel C).<br>**C. Replacement of $\alpha_2$-Plasmin Inhibitor**<br>• **Action:** If available, administration of **Fresh Frozen Plasma (FFP)**. |

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| | • **Rationale:** While FFP is not as concentrated as specific factor products, it is the primary source of $\alpha_2$-plasmin inhibitor in the absence of a purified concentrate (which is not widely available in most regions).<br>**D. Monitoring for Reperfusion**<br>The "danger zone" for this patient is the **reperfusion of the donor lung**. This often triggers a "fibrinolytic burst."<br>• **Action:** Perform a repeat ROTEM (specifically looking at the **ML/Maximum Lysis** parameter) immediately after reperfusion. If the trace begins to narrow (ML >15%), additional antifibrinolytics and fibrinogen are required immediately. |
| **Clinical Vignette 10: Anesthetic Implications of Factor XI Deficiency: A Clinical Case Study and Review of Literature** | |
| **ChatGPT** | ## Interpretation<br>**1) ROTEM interpretation (pre-op and intra-op panels)**<br>Across **EXTEM, INTEM, and APTEM**, the traces look **"fat and stable" early, with sustained clot width**:<br>• **Clot initiation/propagation:** No obvious delay pattern on the curves that would scream severe factor deficiency (i.e., nothing like a long flat "tail" before the trace widens).<br>• **Clot strength: EXTEM looks robust** (good overall clot firmness → platelet + fibrin contribution preserved).<br>• **Fibrinogen contribution: FIBTEM amplitude/MCF appears preserved** (no "thin/near-flat" fibrin trace), arguing **against clinically important hypofibrinogenemia**.<br>• **Fibrinolysis: No meaningful lysis**, and **EXTEM ≈ APTEM**, so **no hyperfibrinolysis** signal.<br>• **Trend:** Pre-op and intra-op look **similar**, suggesting **no evolving consumption/DIC pattern** on ROTEM.<br>**Bottom line:** ROTEM shows **essentially preserved hemostatic competence** (no hypofibrinogenemia, no platelet/clot-strength problem, no hyperfibrinolysis). A known **mild FXI deficiency (40%) may not produce a dramatic ROTEM abnormality**, especially if overall clot formation is otherwise compensated.<br><br>## Suggested Management<br>This is **active significant bleeding from ectopic pregnancy → source control + structured hemorrhage + targeted factor support**.<br>**Immediate actions (while preparing definitive management)** |

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| | • **Resuscitate:** 2 large-bore IVs, bloods (FBC, PT/INR, aPTT, fibrinogen, group & crossmatch), keep warm, calcium monitoring.<br>• **Early OB/Gyn + anesthesia + hematology** involvement.<br>• **Definitive source control:** urgent operative management (or per local protocol).<br>**Hemostatic therapy (guided by what ROTEM shows + her FXI deficiency)**<br>Because ROTEM doesn't show fibrinogen/platelet/fibrinolysis problems, **don't shotgun cryo/platelets/TXA "because bleeding"**—but you *should* address FXI deficiency because she's actively hemorrhaging and heading to surgery:<br>• **Factor XI replacement (practical first-line):**<br>○ **FFP ~10–20 mL/kg** (often used to raise FXI for urgent surgery/bleeding when FXI concentrate isn't available).<br>○ Reassess clinically and with labs (aPTT/FXI activity if available), but **clinical hemostasis matters most** in the moment.<br>• **Tranexamic acid (TXA):**<br>○ ROTEM does **not** suggest hyperfibrinolysis, so TXA is **not mandatory from the trace alone**.<br>○ However, in **major ongoing hemorrhage**, many teams still give **TXA 1 g IV** early (and repeat if bleeding continues), unless there's a strong contraindication—balance this with her overall thrombotic risk and institutional obstetric hemorrhage protocol.<br>• **RBC transfusion:** based on **hemodynamics, ongoing loss, symptoms, and trajectory** (she's already dropping quickly).<br>• **Fibrinogen/cryoprecipitate: only if** fibrinogen is low or **FIBTEM A5/A10/MCF is reduced** (it doesn't look reduced here).<br>• **Platelets: only if** thrombocytopenic or EXTEM clot strength is poor (it looks preserved here).<br>**If bleeding is refractory despite the above**<br>• Prioritize **surgical hemostasis** (most common reason).<br>• Discuss **rescue agents** with hematology:<br>○ **rFVIIa** is a *last-ditch rescue* option due to thrombosis risk (not first line here). |

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| **DeepSeek** | ## Interpretation<br>**Interpretation of ROTEM**<br>The table shows parameters for four ROTEM assays (APTEM, EXTEM, FIBTEM, INTEM) at two time points.<br>**Key Findings:**<br>1. **FIBTEM (Measures Fibrinogen Contribution):** This is the most critical abnormality.<br>○ **Pre-op:** Amplitude (A5 or A10, implied by the table) is **85**. This is **low** (normal is typically >100 mm or specific to device/clinical context, but a significant drop is key).<br>○ **Intra-op:** Drops further to **80**.<br>○ **Interpretation:** This indicates a **significant fibrinogen deficit or dysfunction**. Given the acute bleeding, this is likely **acquired hypofibrinogenemia** due to consumption and dilution, which is common in major obstetric hemorrhage. This is the primary coagulopathy requiring immediate treatment.<br>2. **Other Assays (APTEM, EXTEM, INTEM):** These assess the overall clotting cascade (EXTEM/INTEM) and the effect of fibrinolysis (APTEM vs. EXTEM).<br>○ The values are stable or show only mild, non-critical reductions (e.g., INTEM drops from 90 to 80). The APTEM (which includes an antifibrinolytic) does not show significant improvement over EXTEM, suggesting **fibrinolysis is not a major driver** of the bleeding at this point.<br>○ The relative normality of INTEM (which is sensitive to factor deficiencies like XI) compared to the stark FIBTEM abnormality suggests that the **acute bleeding-related coagulopathy (low fibrinogen) is more pressing than the underlying factor XI deficiency** in this moment.<br>**Summary ROTEM Diagnosis:**<br>**Acute, severe hypofibrinogenemia** consistent with major obstetric hemorrhage. The known factor XI deficiency (40%) is a background comorbidity that complicates management but is not the primary cause of the current life-threatening coagulopathy.<br><br>## Suggested Management<br>Management must be simultaneous and rapid, following major hemorrhage protocols.<br>**Phase 1: Immediate Resuscitation & Surgical Control (ABCDE)** |

 

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| | 1. **Airway/Breathing/Circulation:** Secure IV access with large-bore cannulas, administer oxygen, and begin rapid transfusion protocol.<br>2. **Surgical Control:** The definitive treatment for a ruptured ectopic pregnancy is **urgent surgery (laparoscopy or laparotomy)** to stop the bleeding source. This is the absolute priority.<br>3. **Blood Product Transfusion (Massive Transfusion Protocol - MTP):**<br>o **RBCs:** Transfuse for hemodynamic instability and low Hgb.<br>o **Fibrinogen Replacement (CRITICAL):** This is the **first-line hemostatic treatment** based on ROTEM.<br>▪ **First Choice: Fibrinogen Concentrate** (e.g., 2-4 grams). It is virus-inactivated, rapid, and allows precise dosing.<br>▪ **Alternative: Cryoprecipitate** (e.g., 10-15 pools). Each pool provides ~2-3g fibrinogen.<br>o **Other Components:** Give **Fresh Frozen Plasma (FFP)** (e.g., 15-20 mL/kg) to replace all clotting factors broadly and help with the factor XI deficiency. Give **Platelets** if count is low (<50-75 x $10^9$/L) or if bleeding persists.<br>**Phase 2: Targeted Coagulation Management**<br>1. **Factor XI Deficiency-Specific Treatment:**<br>o **Factor XI Concentrate** is ideal but may not be immediately available.<br>o **First-Line in Acute Bleeding: Fresh Frozen Plasma (FFP)** (as above) is the standard acute treatment to raise factor XI levels. **Tranexamic Acid** (an antifibrinolytic) is also strongly indicated and should be given (e.g., 1g IV load, then infusion) to provide hemostatic support.<br>2. **Monitor & Guide with ROTEM:** Repeat ROTEM (especially FIBTEM) 15-30 minutes after fibrinogen replacement to confirm correction (target FIBTEM A5 >100-120 mm). Use it to guide further need for FFP, platelets, or antifibrinolytics.<br>**Phase 3: Postoperative & Ongoing Care**<br>• **Hematology Consultation:** Essential for guiding long-term management of factor XI deficiency post-operatively (e.g., further FFP/factor XI dosing, duration of TXA).<br>• **Monitor for Complications:** Watch for signs of thrombosis (balancing pro-thrombotic blood products with a thrombophilic predisposition in some FXI-deficient patients) and recurrent bleeding.<br>• **Emotional & Reproductive Health Support:** Address the psychological impact of ectopic pregnancy and blood loss. |

 

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| | **In summary: The immediate life-threatening issue is hemorrhagic shock from a ruptured ectopic pregnancy, compounded by an acute, severe fibrinogen deficiency. Management is: 1) Immediate surgery, 2) Aggressive fibrinogen replacement (Concentrate or Cryo) guided by ROTEM, 3) Supportive MTP (RBCs, FFP, TXA), and 4) Hematology-guided management of the underlying Factor XI deficiency.** |
| **Gemini** | ## Interpretation<br>**1. ROTEM Interpretation**<br>The traces show a classic pattern consistent with the patient's known **Factor XI (FXI) deficiency** and her **pregnancy status**.<br>• **INTEM (Intrinsic Pathway):** There is a **prolonged Clotting Time (CT)**—visible as the elongated green line at the start of the trace—compared to the EXTEM. This is the hallmark of a Factor XI deficiency, as FXI is a key component of the intrinsic coagulation pathway. However, once the clot begins to form, the Clot Formation Time (CFT) and Maximum Clot Firmness (MCF) appear within normal limits, suggesting that once initiated, the "burst" of thrombin is sufficient to create a stable clot.<br>• **EXTEM (Extrinsic Pathway):** The CT, CFT, and MCF are all **normal**. This indicates that the extrinsic pathway (triggered by Tissue Factor) is intact.<br>• **FIBTEM (Fibrinogen Contribution):** The MCF is **markedly increased** (a very "fat" trace). This is a physiological finding in pregnancy, which is a hypercoagulable state characterized by significantly elevated fibrinogen levels. This high fibrinogen helps compensate for the Factor XI deficiency by increasing the structural strength of the clot.<br>• **APTEM (Fibrinolysis):** This trace is similar to the EXTEM, showing no signs of premature clot breakdown (hyperfibrinolysis).<br>**Summary:** The patient has a delay in clot initiation via the intrinsic pathway, but her overall clot strength is excellent, bolstered by pregnancy-induced high fibrinogen.<br><br>## Suggested Management<br>The management must balance the surgical emergency (ectopic pregnancy) with the risk of hemorrhage due to FXI deficiency. |

 

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| | **Hemostatic Optimization**<br>• **Factor XI Replacement:** Since the patient is actively bleeding and the INTEM CT is prolonged, replacement may be necessary.<br>  ○ **Fresh Frozen Plasma (FFP):** The standard treatment to replace FXI.<br>  ○ **FXI Concentrate:** If available, this is more specific, though it carries a small risk of thrombosis in some patients.<br>• **Antifibrinolytics:** Administer **Tranexamic Acid (TXA)**. FXI-deficient patients are particularly prone to bleeding in tissues with high fibrinolytic activity (like the genital tract). TXA helps stabilize the clots she *is* able to form.<br>• **Avoid Over-correction:** Given her high FIBTEM MCF (hypercoagulability of pregnancy), there is a theoretical risk of thrombosis if coagulation factors are over-replaced.<br>**Surgical & Supportive Care**<br>• **Emergency Surgery:** Immediate laparoscopic or open salpingectomy/salpingostomy is required to stop the primary source of bleeding from the ectopic pregnancy.<br>• **Volume Resuscitation:** Based on the hemoglobin drop and vaginal bleeding, fluid resuscitation and potentially a packed red blood cell (PRBC) transfusion should be prepared to maintain oxygen-carrying capacity.<br>• **Postoperative Monitoring:** Continue to monitor for delayed bleeding, as FXI deficiency often manifests as "oozing" hours after the initial insult. |

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