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Navigating Rare Coagulation Disorders In Pregnancy: A Case Series Emphasizing Multidisciplinary Care

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Abstract

Background: *Inherited and acquired coagulation factor deficiencies in pregnancy are rare but pose significant risks to both mother and baby. Conditions like Factor XI, Factor VII, Factor XII deficiencies, and acquired hemophilia A present complex clinical and laboratory challenges that necessitate coordinated multidisciplinary care.*

Objective: *To present the clinical features, management, and outcomes of four pregnant women with rare coagulation disorders, emphasizing tailored, team-based strategies for safe delivery and postpartum care.*

Methods and Cases:*Four patients managed at our tertiary center with congenital Factor XI deficiency, Factor VII deficiency, Factor XII deficiency with renal anomaly, and postpartum acquired hemophilia A are described. Multidisciplinary collaboration guided individualized antenatal assessment, hemostatic support, anesthesia choice, and postpartum follow-up.*

Conclusion: *Early identification and personalized multidisciplinary management of coagulation disorders in pregnancy enable favorable maternal and neonatal outcomes. This case series offers practical insights for handling similarly rare, high-risk presentations.*

INTRODUCTION

Inherited and acquired coagulation disorders in pregnancy are uncommon, yet their occurrence can pose substantial risks to both maternal and fetal outcomes if not promptly identified and appropriately managed (1,2). The physiological adaptations of pregnancy produce a hypercoagulable state; however, in the presence of clotting factor deficiencies or other bleeding diatheses, these changes can complicate delivery planning, influence anesthetic decision-making, and impact the safety of the postpartum period (3,4).

Certain congenital deficiencies—such as those involving Factors XI, VII, and XII—are rare autosomal conditions with a broad spectrum of bleeding phenotypes, often demonstrating limited correlation between laboratory measures and clinical severity (5–7). In contrast, acquired hemophilia A, resulting from the development of autoantibodies against factor VIII, is an especially rare but potentially life-threatening postpartum complication, frequently manifesting with severe, unexpected hemorrhage (8–10) . Given their rarity in obstetric practice, most published literature consists of isolated case reports, leaving clinicians with limited practical guidance (11).

In this case series, we present four obstetric patients diagnosed with distinct coagulation disorders: congenital Factor XI deficiency, congenital Factor VII deficiency, congenital Factor XII deficiency associated with a renal anomaly, and postpartum acquired hemophilia A. Each case necessitated an individualized, multidisciplinary management plan developed through close collaboration among obstetric, hematology, anesthesiology, and transfusion medicine teams. Our approach emphasized early antenatal risk stratification, targeted diagnostic evaluation, coordinated peri-delivery hemostatic strategies, and individualized postpartum surveillance. By sharing these clinical experiences, we aim to provide pragmatic insights to inform the management of similarly complex and high-risk pregnancies.

Case Series

Case 1

A 40-year-old woman, G2Ab1, presented at 36 weeks + 3 days gestation for delivery with multiple high-risk conditions: pre-eclampsia, overt diabetes mellitus controlled with oral hypoglycemic agents and insulin, chronic hepatitis B infection (HBsAg positive), hypothyroidism, suspected factor XI deficiency, and positive antinuclear antibody (ANA). She was referred for evaluation due to prolonged activated partial thromboplastin time (APTT) and abnormal coagulation profile detected in the late third trimester. The pregnancy was otherwise uncomplicated with appropriate fetal growth.

Investigations

Laboratory tests revealed a prolonged APTT of 63.5 seconds, normal prothrombin time (PT) and INR, normal fibrinogen, and low factor XI activity at 45.6 IU/dL with normal factor XII. Additional findings showed HbA1c between 5.5 and 8%, TSH at 6.27 μIU/mL, and elevated 24-hour urine protein. ANA was moderately positive, while antiphospholipid antibody (APLA) screening was negative. Hemoglobin dropped from 12.4 g/dL preoperatively to 8.5 g/dL postoperatively, requiring iron supplementation. Cardiology and ophthalmology evaluations noted no hypertensive cardiac changes or diabetic retinopathy.

Management

A multidisciplinary plan included optimization of antihypertensive therapy with labetalol and nifedipine, insulin-based glycemic control, thyroxine supplementation, and monitoring for hepatitis B infection. Hematology recommended no prophylactic fresh frozen plasma (FFP) or tranexamic acid but advised preparedness for emergency administration if needed. A single dose of antenatal dexamethasone was given to enhance fetal lung maturity. She underwent emergency lower-segment cesarean section due to worsening pre-eclampsia and grade 2 meconium-stained amniotic fluid. Intraoperative hemostasis was adequate with no abnormal bleeding. Postoperative care included antibiotics, analgesics, tight blood pressure and glycemic control, iron therapy, close coagulation monitoring, early mobilization, and no pharmacological thromboprophylaxis.

Outcome

The maternal intraoperative and postoperative courses were uneventful, achieving adequate hemostasis without bleeding complications. Hemoglobin levels decreased as expected but improved with iron therapy. Surgical wounds healed well. The mother remained stable, and the neonate was healthy and thriving. She was discharged with detailed counselling on future bleeding risk management, availability of blood products during surgery, and follow-up for endocrine and infectious conditions.

Case 2

A primigravida at 38 weeks + 2 days with an ovulation induction conception and diet-controlled gestational diabetes was found to have congenital Factor VII deficiency during evaluation for delivery. She presented with decreased fetal movements and intermittent abdominal tightening. The deficiency was suspected after markedly prolonged PT/INR with normal APTT and was confirmed on factor VII assay. The pregnancy was otherwise uncomplicated with appropriate fetal growth.

Investigations

Coagulation workup revealed elevated PT up to 61.7 s and INR up to 6.05 with normal APTT ~29 s and fibrinogen 329 mg/dL. Mixing studies showed isolated PT prolongation not corrected with adsorbed plasma, consistent with factor VII deficiency. Factor VII assay was low, confirming congenital deficiency; thrombin time was normal and pre-operative TEG was also normal. Hemoglobin remained >11.6 g/dL, platelet counts were normal, and antenatal scans showed normal growth and amniotic fluid.

Management

A multidisciplinary plan was formulated with obstetrics, hematology, transfusion medicine, and anesthesiology. Vitamin K was administered and 2 mg recombinant factor VIIa (Novoseven) was given immediately before surgery with 2 units FFP on standby. General anesthesia was chosen to avoid neuraxial techniques. She underwent an emergency lower-segment cesarean section on 27 July 2023 for non-progress of labour, delivering a healthy male infant weighing 3.68 kg. Intraoperative hemostasis was adequate and there was no abnormal bleeding. Postoperatively, she received two units of FFP immediately and two more on postoperative day 3 after TEG showed mild hypocoagulability. She was also given antibiotics, analgesics, compression stockings, and encouraged early ambulation, with close monitoring of coagulation status.

Outcome

The maternal postoperative course was smooth, with stable vitals, no hemorrhagic events, and healthy wound healing. She was discharged on postoperative day 8 with detailed counselling on bleeding risks in future surgeries, the need for blood product cover, avoidance of intramuscular injections, use of safe analgesics, and tranexamic acid for mucosal bleeds. The newborn remained stable throughout, feeding well, and was discharged alongside the mother.

Case 3

A 38-year-old woman, G4P2L2Ab1 at 38 weeks + 2 days, with a history of congenital factor XII and XI deficiencies and right hypoplastic kidney, was admitted for safe confinement. She had previously experienced no significant bleeding despite coagulation abnormalities. The current pregnancy was uncomplicated with stable maternal vitals and no family history of bleeding disorders.

Investigations

Pre-delivery laboratory evaluation revealed markedly prolonged activated partial thromboplastin time (APTT >120 seconds), with normal prothrombin time (PT) and INR. Hemoglobin was 10.4 g/dL and platelet counts were normal. Serial antenatal scans showed appropriate fetal growth (estimated fetal weight ~2600 g, 22nd centile), normal amniotic fluid index, and cephalic fetal presentation. Doppler studies were normal. Transfusion medicine assessed coagulation status by thromboelastography (TEG) pre-delivery showed a severe hypocoagulable stage.

Management

The patient received four units of fresh frozen plasma (FFP) prior to labor induction. Labor was induced with oral misoprostol culminating in an induced vaginal delivery. The delivery was uneventful, with a healthy live male infant weighing 2605 g. Post-delivery care included analgesics, antibiotics, regular episiotomy site inspection, and establishment of exclusive breastfeeding.

Outcome

The mother remained stable postpartum without excessive bleeding or complications; the episiotomy site healed well. The neonate was healthy, with no signs of coagulopathy. Both were discharged in good condition with recommendations for postpartum follow-up including coagulation and renal evaluation.

Case 4

Diagnosis

A 32-year-old woman, P1L1Ect1, with prior left salpingectomy for ectopic pregnancy and no personal or family history of bleeding disorders, developed acquired hemophilia A (factor VIII inhibitors) following preterm emergency LSCS at 36 weeks for PPROM and breech presentation. She presented with recurrent rectus sheath hematomas and wound complications. The postoperative course was complicated by multiple laparotomies, arterial embolization, ongoing coagulopathy, and large abdominal wall defects.

Investigations

Initial labs revealed profound anemia (Hb 4.9 g/dL post-op), prolonged APTT (up to >120 s), normal PT/INR, and low platelets intermittently. Factor VIII activity was <1%, with very high inhibitor titers (~230 BU); mixing studies confirmed immediate-acting inhibitors. TEG was repeatedly hypocoagulable. Imaging (CECT and serial ultrasounds) showed large preperitoneal and intramuscular hematomas with intraperitoneal extension, residual bladder-flap hematoma, and large abdominal wall defects. Cultures from wound fluids revealed MDR Klebsiella pneumoniae and E. coli; blood cultures intermittently showed Gram-negative organisms. Autoimmune workup, ANA, and complements were negative except for low C4; APLA and lupus anticoagulant screens were negative. LFTs were frequently deranged, likely secondary to infection and transfusions, while renal function largely remained preserved.

Management

The patient's treatment included multiple surgical interventions including exploratory laparotomies with hematoma evacuation, inferior epigastric artery embolization, and secondary wound resuturing. She received massive transfusion support with 53 units PRBC, 12 units RDP, 68 units cryo and 33 units FFP. Specific hemostatic therapy was provided with activated prothrombin complex concentrate (APCC/FEIBA), recombinant factor VIIa (NovoSeven; ~18 doses), and Emicizumab injections. Four doses of rituximab, high doses of oral and parenteral corticosteroids were administered as inhibitor eradication/immunosuppressive therapy. Tranexamic acid was used for mucosal and wound bleeding. Severe wound infection and abdominopelvic sepsis were managed with broad-spectrum and targeted antibiotics including tigecycline, meropenem, and teicoplanin per sensitivity. Supportive care included wound vacuum dressings, pain control, infection management, nutritional optimization. Multidisciplinary input guided complex wound, hematological, and surgical needs throughout her prolonged hospital stay.

Outcome

Over the course of hospitalization, coagulation parameters normalized (APTT ~22 – 38 s, factor VIII >100%), bleeding was controlled, anemia improved with transfusional and hematinic support, and infection resolved with appropriate antibiotics. The abdominal wall defect was managed with vacuum

dressings, and secondary closure was planned in follow-up. The patient was discharged in stable condition with vacuum dressing in place, advice for weekly wound care, regular hematology and surgical review, and monitoring for any bleeding or infection. The newborn, delivered via LSCS, was stable throughout.

DISCUSSION

Pregnancy complicated by rare coagulation disorders presents complex and often unpredictable challenges. Our case series demonstrates how individualized, multidisciplinary planning allowed safe delivery outcomes across very different disorders — congenital Factor XI, Factor VII, and Factor XII deficiencies, and postpartum acquired hemophilia A. The treatment approaches in this case series illustrate a balance between adherence to established guidelines and individualized modifications based on clinical presentation, laboratory findings, and multidisciplinary input—critical elements when managing pregnancies complicated by rare coagulation disorders.

An individualized management for pregnant women with Factor XI deficiency is emphasized, given the variable bleeding risk and poor correlation between APTT prolongation and clinical outcomes (5,12). Prophylactic use of blood products such as fresh frozen plasma (FFP) or antifibrinolytics is recommended only for women with previous bleeding episodes or anticipated high-risk delivery scenarios (13,14). Neuraxial anesthesia can be considered if factor activity levels are adequate, with close laboratory and clinical monitoring (13,15). In Case 1, the patient had a moderate factor XI deficiency (45.6 IU/dL) and prolonged APTT but no history of significant bleeding. The decision to forgo prophylactic FFP and tranexamic acid, while ensuring readiness for emergency administration, aligns with literature advocating conservative management when bleeding history is absent (5). This tailored approach, coupled with multidisciplinary management of comorbidities such as pre-eclampsia, diabetes, hepatitis B, and hypothyroidism, demonstrates a holistic strategy beyond coagulation correction alone. The positive outcome—uneventful surgery and postpartum course with adequate hemostasis—supports this selective prophylaxis strategy (12).

For congenital Factor VII deficiency, literature highlights the importance of third-trimester coagulation profiling, particularly assessment of Factor VII levels and PT/INR, since factor activity often rises during pregnancy but severe deficiency retains bleeding risk during delivery (6,16). Recombinant Factor VIIa is the preferred hemostatic agent for delivery, supported by FFP as needed, and avoidance of neuraxial anesthesia is generally advised in moderate to severe cases (16,17). In Case 2, the diagnostic approach incorporated mixing studies and coagulation assays confirming isolated Factor VII deficiency with markedly prolonged PT/INR but normal APTT. Multidisciplinary planning ensured administration of recombinant Factor VIIa immediately prior to cesarean section, supported by FFP on standby. General anesthesia was appropriately chosen to mitigate bleeding risks associated with neuraxial techniques. Postoperative proactive use of FFP guided by thromboelastography (TEG) findings of mild hypocoagulability exemplifies dynamic and responsive management recommendations seen in the literature (6,16). The absence of perioperative hemorrhage and smooth maternal and neonatal outcomes validate adherence to recommended protocols.

Unique among coagulation factor deficiencies, Factor XII deficiency rarely predisposes to bleeding despite significant APTT prolongation; indeed, some data suggest a thrombotic proclivity during pregnancy (18). Literature supports routine obstetric management and vigilant thrombosis surveillance without prophylactic factor replacement (18). However, in Case 3, four units of FFP were administered before induction due to severely prolonged APTT (>120 seconds) and hypocoagulable TEG profile. This approach represents a cautious clinical judgment exceeding standard recommendations yet balancing laboratory derangements with the patient's stable clinical status. Subsequent labor, delivery, and postpartum recovery were uneventful, corroborating literature that coagulation abnormalities in Factor XII deficiency do not necessarily translate into clinical bleeding risk (5).

In acquired hemophilia A, sudden onset of severe postpartum hemorrhage with high-titer factor VIII inhibitors necessitates urgent multidisciplinary management (19,20). Guidelines advocate for hemostatic control using bypassing agents such as activated prothrombin complex concentrate (APCC) or recombinant Factor VIIa coupled with immunosuppressive regimens including corticosteroids and rituximab to eradicate inhibitors (20). Supportive care with massive transfusions, surgical interventions, infection control, and advanced wound management is often necessary due to the disorder's complicated course (14,20). Case 4 reflects strict concordance with these protocols: multiple laparotomies, embolization, massive transfusions (over 150 blood product units across types), and specific hemostatic therapies were administered alongside immunosuppression and antibiotics

targeting multidrug-resistant infections. Progressive normalization of coagulation parameters and clinical stabilization mirror reported literature outcomes. This case underscores the resource-intense, multidisciplinary approach critical to survival and functional recovery in acquired hemophilia A postpartum (19,20).

Common Themes and Multidisciplinary Care Importance

Across all four cases, early recognition of the bleeding disorder, careful risk stratification, and sustained multidisciplinary collaboration were essential. Management plans were personalized to clinical history, coagulation profiles, and the anticipated mode of delivery, with blood products and specialist therapies available as needed. Anesthesia decisions were guided by coagulation status to minimize risk. Postpartum monitoring, patient education, and preparation for future procedures were emphasized in every case. Finally, these cases highlight the limitations of relying solely on standard coagulation assays to predict bleeding, and the potential value of global hemostatic testing for more accurate risk assessment.

CONCLUSION

Pregnancy in women with inherited or acquired coagulation disorders warrants prompt recognition and the formulation of an individualized, multidisciplinary management strategy. This series illustrates that, even in the context of uncommon conditions such as Factor XI, Factor VII, and Factor XII deficiencies—and the markedly higher-risk acquired hemophilia A—favourable maternal and neonatal outcomes can be achieved through early antenatal risk stratification, focused diagnostic evaluation, timely specialist involvement, and tailored peripartum planning. The striking disparity between the minimal haemorrhagic potential in some congenital deficiencies and the catastrophic bleeding risk inherent to acquired hemophilia A underscores the necessity of interpreting laboratory data in conjunction with the clinical context. There remains a pressing need for standardized obstetric care pathways, broader interdisciplinary awareness, and vigilant postpartum surveillance to mitigate complications, detect recurrence at the earliest opportunity, and safeguard the management of subsequent pregnancies.

Key Learning Points

- Multidisciplinary Approach is Essential: Optimal outcomes in rare coagulation disorders during pregnancy require early involvement of obstetrics, hematology, transfusion medicine, anesthesiology, and relevant specialties. Coordinated care ensures tailored management for each patient's unique bleeding risk and comorbidities.
- Individualized Risk Assessment: Bleeding severity in factor deficiencies (XI, VII, XII) varies widely and does not always correlate with laboratory factor levels. Personal bleeding history and clinical context should guide prophylactic and therapeutic interventions.
- Peripartum Hemostatic Management: Prophylactic administration of fresh frozen plasma, factor concentrates, or recombinant factor VIIa should be reserved for patients with significant bleeding risk or active hemorrhage. Preparedness for emergency transfusion and factor replacement is critical during delivery.
- Acquired Hemophilia A Requires Aggressive Multimodal Therapy: Unlike congenital deficiencies, acquired inhibitors can cause life-threatening bleeding necessitating bypassing agents, immunosuppressants, massive transfusion support, and multidisciplinary monitoring.
- Careful Anesthesia Planning: General anesthesia is often preferred over neuraxial techniques where coagulopathy poses bleeding risks; however, with adequate prophylaxis and multidisciplinary planning, regional anesthesia can be safely considered in selected cases.
- Close Postpartum Surveillance: Monitoring for bleeding, wound healing, infection, and factor levels postpartum is crucial to identify complications early and adjust therapy.
- Patient Education: Counseling about bleeding risk in future pregnancies or surgeries, avoidance of intramuscular injections, safe analgesics, and recognizing bleeding symptoms empowers patient self-care and timely interventions.
- Need for Better Predictive Assays: Current coagulation tests have limitations in predicting bleeding risk in factor deficiencies; research into clinical assays like thrombin generation or global hemostasis testing may improve individualized management in the future.

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Case	Diagnosis	GA & Parity	Comorbidities	Key Labs	Mode of Delivery	Hemostatic Management	Outcome
1	Factor XI Deficiency	G2Ab1, 36+3w	Pre-eclampsia, DM, hypothyroid, HBsAg+	APTT 63.5 s, XI 45.6	Emergency LSCS	No prophylactic FFP; IV iron	Stable mother & baby
2	Factor VII Deficiency	G1, 38+2w	GDM	PT 61.7 s, INR 6.05, low VII	Emergency LSCS	Pre-op rFVIIa + FFP	Uneventful
3	Factor XI Deficiency	G4P2L2Ab1, 38+2w	Hypoplastic kidney	APTT >120 s	Induced VD + RMLE	4 FFP pre-induction	Smooth recovery

Case	Diagnosis	GA & Parity	Comorbidities	Key Labs	Mode of Delivery	Hemostatic Management	Outcome
4	Acquired Hemophilia A	P1L1Ect1, postpartum	None	APTT >120 s, FVIII <1%, inhibitor 230 BU	Multiple laparotomies post-LSCS	APCC, rFVIIa, Emicizumab, steroids, rituximab	Stable at discharge

Case	Diagnosis	Take-Home Message
1	Factor XI Deficiency	Labs don't always reflect bleeding risk; individualised care with standby products can be safe
2	Factor VII Deficiency	Severe deficiency → peri-delivery replacement is lifesaving
3	Factor XII Deficiency	Despite prolonged APTT, bleeding risk minimal — focus on comorbidities
4	Acquired Hemophilia A	Postpartum severe bleed + prolonged isolated APTT = urgent inhibitor work-up & aggressive therapy