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A Rare Case of Dysfibrinogenemia in Pregnancy:Management Decisions When Data Are Lacking

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Abstract

Congenital fibrinogen disorders are a rare group of inherited conditions characterized by quantitative and/or qualitative fibrinogen deficiencies. Dysfibrinogenemia is the most common of these disorders and can be diagnosed by standard coagulation blood testing and is often confirmed with pedigree and molecular genetic analyses. Because of variability in genotype-to-phenotype expression and the inadvertent combining of fibrinogen deficiency subtypes in the literature, the optimal management of these individuals in general, and particularly in pregnancy, remains incompletely understood and empirically managed. The authors present a case of a pregnant patient with well-defined congenital dysfibrinogenemia who was managed with laboratory monitoring and expectant care and had a favorable outcome.

The authors review the current literature and discuss alternative approaches to care using new diagnostic screening methodologies. Management was guided by a multidisciplinary team, including maternal-fetal medicine, anesthesiology, pediatric, and adult hematologists.

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Keywords: blood disorder, dysfibrinogenemia, intrapartum management, pregnancy, thromboelastography (teg)

Introduction

Congenital fibrinogen disorders are a heterogeneous group of rare inherited conditions associated with quantitative and/or qualitative fibrinogen deficiencies. Some of these disorders may be more prevalent than reported, as a significant number of affected individuals are asymptomatic [1,2]. Affected individuals may experience a spectrum of symptoms from hemorrhage to thrombosis [1]. Data are limited and confusing regarding management of specific subcategories of these congenital fibrinogen disorders due to the literature combining outcomes for multiple fibrinogen defects, incomplete genotyping of patients, and an expanding recognition of the variability in genotype-phenotype presentation even for the same mutation. Concerning pregnancy, there is an even further paucity of information available to guide the clinician for counseling and management purposes. We present a well-defined case, by family pedigree and genetic diagnosis, of congenital dysfibrinogenemia in pregnancy. Congenital dysfibrinogenemia is an inherited qualitative fibrinogen disorder characterized by reduced fibrinogen functional activity despite normal or elevated fibrinogen antigen levels [2]. The authors use this case example to review the available literature and to present suggestions for the approach to counseling and management.

Case Presentation

A 23-year-old G2P1001 with a known history of congenital dysfibrinogenemia presented for prenatal care. She had a remote history of menorrhagia during adolescence that was adequately controlled with oral contraceptive therapy.The patient denied any other history of abnormal bleeding.The patient's sister was previously evaluated for a bleeding disorder due to menorrhagia. Based on this workup, the patient, her sister, and her mother were identified to have the pathogenic heterozygous FGA gene variant (c.104G>A), resulting in a diagnosis of congenital dysfibrinogenemia. Earlier laboratory testing demonstrated low fibrinogen activity levels with normal fibrinogen antigen values.The patient's mother remains asymptomatic despite these laboratory aberrations.She had three vaginal deliveries without complications and an uncomplicated surgical history, including heart surgery in childhood and a wisdom tooth extraction without significant bleeding complications.

Our patient's previous pregnancy was complicated by chorioamnionitis and a term vaginal delivery without intrapartum bleeding or thrombotic events.She had prophylactically received two units of fresh frozen

plasma (FFP) due to hypofibrinogenemia upon admission. Following delivery, she did not experience any abnormal bleeding or need for additional coagulation agents.Her newborn son tested negative for congenital dysfibrinogenemia.

Throughout the current pregnancy, the patient was managed via a multidisciplinary team consisting of specialists in maternal-fetal medicine, adult, and pediatric hematology. Serial pregnancy-related coagulation laboratory studies are presented in Table *1*.No other pregnancy complications ensued, other than the suspicion for suboptimal interval fetal growth bordering on fetal growth restriction (estimated fetal weight (EFW) 10th percentile) at 36 weeks' gestation.She had the onset of spontaneous labor at 37 weeks with an uncomplicated intrapartum course.

	11 weeks	19 weeks	21 weeks	25 weeks	28 weeks	32 weeks	Day of delivery (37 weeks)	Postpartum day 1
Fibrinogen antigen level (mg/dL)		276		429		368		417
Fibrinogen activity level (mg/dL)	93	195	71	198	57	167	66	50
TEG max amplitude (mm)					63.9		67.1	67.2
Partial thromboplastin time (seconds)		27.1				25.6	26.8	32.3
Prothrombin time (seconds)		16.3				15.6	13.6	16.9
INR		1.6				1.53	1.32	1.66
Reptilase time (seconds)		33		31		32.4		
Thrombin time (seconds)		35.4		29.2		37.3		
Platelet count (K/uL)		276				250	310	323

TABLE 1: Serial pregnancy-related coagulation laboratory studies **

Normal values: Fibrinogen antigen (150-450 mg/dL), Fibrinogen activity level (150-400 mg/dL), Thromboelastography (TEG) max amplitude (52-70 mm), Partial thromboplastin time (23.1-33.1 seconds), Prothrombin time (9.4-11.7 seconds), International Normalized Ratio (INR) (0.86-1.11), Reptilase time (16-22 seconds), Thrombin time (15.8-21.4 seconds)

**Blank cells indicate these laboratory values were not obtained.

A multidisciplinary team conference was held to determine an appropriate course of action for the patient's labor and delivery course. Hematology, obstetrics, and maternal-fetal medicine were involved. It was determined that admission coagulation laboratory testing would be obtained, including fibrinogen antigen, fibrinogen activity, prothrombin time (PT), partial thromboplastin time (PTT), thrombin time (TT), reptilase time (RT), and thromboelastography (TEG). Furthermore, it was decided that the patient would not require fibrinogen replacement if her fibrinogen levels were decreased without evidence of significant bleeding. The patient's history and clinical stability throughout pregnancy were also considered when making this recommendation. However, it was also recommended that 2U FFP and 10U cryoprecipitate be prepared and held when the patient would be admitted. Hematology recommended that FFP was preferred over cryoprecipitate in the event the patient experienced significant bleeding and required replacement therapy. Additionally, it was advised that instrumental delivery be avoided to minimize additional trauma and bleeding risk. Epidural anesthesia could be considered if TEG parameters were within normal limits, as TEG provides additional information about overall clot strength. However, normal findings do not exclude an underlying functional fibrinogen defect. However, the final decision for neuraxial anesthesia would be a multidisciplinary decision between hematology, anesthesiology, and obstetrics in addition to clinical stability and coagulation study results.

Upon admission, the patient progressed rapidly in labor with a spontaneous vaginal delivery of a 2365-gram healthy female infant with good Apgar scores and an estimated blood loss of 200 mL.Cord blood was sent and later showed that the newborn was positive for the family's FGA gene mutation. A baseline fibrinogen antigen level was not obtained prior to delivery due to rapid progression, but the TEG result was reassuring in the setting of the patient's consistent hemodynamic stability. The patient did not receive any FFP, cryoprecipitate, tranexamic acid, or fibrinogen replacement during her hospital course. She did not experience postpartum hemorrhage and was monitored for 24 hours postpartum without a significant

bleeding event. Hematology and Maternal-Fetal Medicine were consulted prior to her discharge. Both teams agreed the patient was stable despite her low fibrinogen levels (Table *1*), given her clinical stability and minimal lochia. The patient was educated to return if she experienced significant bleeding. Her postpartum course remained uncomplicated without further hemorrhage or thrombotic events.

Discussion

Heritable fibrinogen disorders include abnormal clotting conditions due to quality, quantity, or combined aberrations in fibrinogen structure and function, as described in Table *2*. These fibrinogen-deficient conditions are estimated to account for 0.6% of all inherited bleeding disorders. Based on limited data from a 2024 report in the Rare Bleeding Disorders Registry, approximately 45% of congenital fibrinogen disorders are diagnosed as dysfibrinogenemia [3]. Dysfibrinogenemia is a disproportionate qualitative fibrinogen dysfunction in the setting of appropriate antigen levels, as confirmed by genetic testing. During the laboratory evaluation, dysfibrinogenemia is also associated with prolongation of the prothrombin time (PT), activated partial thromboplastin time (aPTT), thrombin time (TT), and reptilase time (RT). Clinically, dysfibrinogenemia can be asymptomatic or associated with excess bleeding and, paradoxically, thrombosis. This latter occurrence has been postulated to be caused by several distinct mechanisms, including elevated thrombin levels due to defective fibrinogen binding, altered stability of the fibrin clot, and impaired fibrinolysis of the abnormal fibrinogen [4].

Afibrinogenemia
Asymptomatic
Bleeding phenotype
Thrombotic phenotype
Hypofibrinogenemia
Severe; activity < 50 mg/dL
Moderate; activity 50 to 90 mg/dL
Mild; activity 100 mg/dL to lower normal limits
Dysfibrinogenemia
Asymptomatic
Bleeding phenotype
Thrombotic phenotype
Mixed bleeding and thrombotic phenotypes
Hypodysfibrinogenemia
Severe; antigenic level < 50 mg/dL
Moderate; antigenic level 50 to 90 mg/dL
Mild; antigenic level 100 mg/dL to lower normal limits

TABLE 2: International Society on Thrombosis and Haemostasis (ISTH) Classification of Congenital Fibrinogen Disorders

Modified from [5]

Obstetric complications of dysfibrinogenemia are frequently reported to include pregnancy loss, placental abruption, hemorrhage, and thrombotic events [4,6]. However, these assumptions are confounded by a mixing of fibrinogen abnormalities in the literature and incomplete reporting. Although bleeding can more accurately correlate with absolute fibrinogen levels, it is not always possible to predict the genotype-phenotype relationship in the congenital dysfibrinogenemias, making it difficult to anticipate whether a specific variant is more likely to cause bleeding, thrombosis, both, or neither. These discrepancies make it exceedingly difficult to adequately counsel such patients or to preemptively plan for pregnancy and surgical procedures. Of historic interest, the first case of congenital dysfibrinogenemia was described at our institution by Mammen and colleagues in an adolescent female who presented with anemia due to heavy menstrual bleeding [7]. Our patient did not have the same genetic variant as was described by Mammen et

al., and there is no apparent familial lineage to that case.

Our patient's dysfibrinogenemia was originally diagnosed based on family pedigree testing because of relatively mild bleeding symptomatology in her sister. She later completed genetic testing with the Congenital Fibrinogen Deficiency Panel at PreventionGenetics. She was found to have the FGA c.104G>A (p.Arg>His) heterozygous pathogenic variant, as listed in Table 3. The autosomal dominant FGA c.104G>A (p.Arg35His) variant is reported to be associated with a bleeding phenotype, although this classification likely overestimates bleeding risk, as individuals are predominantly minimally symptomatic or asymptomatic [1,2]. This specific mutation occurs at a "hotspot" site responsible for the thrombin-mediated cleavage of fibrinopeptide A [4]. Reported symptoms may include heavy menses, excessive postpartum bleeding, and late-onset mild spontaneous bleeding episodes [4,8]. In several cohorts, a majority of patients (up to 58%) with this FGA mutation were asymptomatic and only diagnosed during routine screening or prior to surgery [9]. This variant is typically classified as pathogenic because it significantly disrupts fibrinogen's functional activity [9]. Unlike some other fibrinogen mutations causing dysfibrinogenemia, FGA c.104G>A is not typically associated with a high risk of thrombosis [4].

Gene	Exon	Complementary DNA
FGA	2	c.92C>T
FGA	2	c.103C>T
FGA	2	c.103C>A
FGA	2	c.104G>A
FGA	2	c.107G>A
FGA	2	c.112A>G
FGA	5	c.550T>C
FGA	5	c.1410_1411insT
FGA	5	c.1482-1495del
FGA	5	c.1717G>C
FGB	2	c.130C>T
FGB	4	c.586C>T
FGG	6	c.620A>G
FGG	7	c.837G>C
FGG	8	c.902G>A
FGG	8	c.902G>C
FGG	8	c.917A>G
FGG	8	c.1067A>T
FGG	8	c.1073C>G
FGG	9	c.1173T>A

TABLE 3: Partial list of identified genetic dysfibrinogenemia mutations

Modified from [8]

Available literature exists primarily in the form of case reports, limited case series, and expert opinion pieces. Management discussions are mostly based on empiric considerations. For example, in the case report by Langer and colleagues, they describe a gravida with genetic testing showing a familial dysfibrinogenemia without a more definitive description of the genetic variant [10]. They also raised concern for adverse pregnancy outcomes by combining data related to both hypofibrinogenemia and dysfibrinogenemia, which are two distinct conditions. This led to frequent fibrinogen activity testing and the empiric administration of several rounds of fibrinogen replacement therapy before planning an elective cesarean delivery, additional rounds of fibrinogen replacement therapy, and then prophylactic anticoagulation therapy for the presumed

increased risk of thrombosis. Of note, the patient remained asymptomatic throughout the course of the pregnancy, during, and after the operative procedure. Although the authors surmised the excellent outcome was due to the aggressive monitoring and pharmacologic interventions, it remains a possibility that the same or better outcomes could have been achieved with expectant management. Yan et al. report on the care of a gravida with dysfibrinogenemia who was originally incorrectly thought to have hypofibrinogenemia [11]. Once properly diagnosed to have an FGA Exon 2 1233C>A mutation, the patient was managed expectantly with an uncomplicated term vaginal delivery. These authors advocated for expectant management unless there is clinical bleeding or thrombosis, or a past or family history suggesting an increased risk for these adverse events. They suggest that under these latter circumstances, prophylactic fibrinogen replacement should be administered to raise the fibrinogen above 100 mg/dL for bleeding concerns, or low molecular weight heparin prophylaxis given for thrombosis concerns. He et al. similarly present a case of dysfibrinogenemia in pregnancy that was again expectantly managed in a clinically asymptomatic individual, only empirically administering fibrinogen replacement therapy when cesarean delivery was needed [12].

When our patient presented in active labor, established plans were to draw admission coagulation labs and decide on the need for fibrinogen replacement based on clinical findings, laboratory results, and anticipated interventions. Due to the rapid progression to the second stage of labor, a baseline fibrinogen antigen level was not available at the time of delivery. However, the absence of active bleeding and a normal TEG study were reassuring, and the decision was made to hold any replacement therapy per predetermined guidelines. This decision resulted in a favorable outcome, despite the later identification of very low fibrinogen activity levels.

TEG is a relatively new, rapid point-of-care viscoelastic hemostatic diagnostic tool that measures the entire process of blood clot formation, strength, and breakdown in real-time and can be used in emergency settings to guide transfusion, blood product replacement, and anticoagulation therapies [13]. Although TEG does not always correlate well with other individual blood coagulation studies, it may be a better predictor of both bleeding and thrombosis-related events. This viscoelastic method is widely used and recommended in non-obstetric operative settings, but the data, although encouraging, are limited for its utility in obstetrics and await more definitive pregnancy-specific trials [13]. Of note, viscoelastic assays, such as TEG, have been validated to identify quantitative hypofibrinogenemia [14]. However, their reliability in conjunction with qualitative derangement results has not been formally established. Clinicians should proceed with caution as initial laboratory results may be falsely reassuring. Since our patient had significant variation in fibrinogen activity from lab to lab, we preferred to monitor functional fibrinogen from TEG as one of the parameters to guide replacement therapy.

Because specific guidelines were lacking as to the management of pregnancy in congenital fibrinogen disorder patients, the ISTH Scientific and Standardization Committee developed expert opinion strategies for the management of these patients. Our modified version of these recommendations for dysfibrinogenemia in pregnancy can be seen in Table *4*, and perhaps in the future, the addition of TEG monitoring can be added to these recommendations [5]. The recommendation to measure fibrinogen antigen and activity levels each trimester is intended to establish baseline values and monitor laboratory trends throughout pregnancy. Patients with a history of bleeding or thrombotic events, or those requiring pharmacologic intervention, may warrant closer monitoring with more frequent laboratory assessments to guide decisions regarding fibrinogen replacement. Additional laboratory evaluation in the third trimester is recommended to facilitate planning for delivery, including determining whether fibrinogen replacement is indicated and ensuring appropriate preparations are made. Our modifications to the ISTH recommendations from this case are noted in Table *4*, as our patient was deemed to be a candidate for expectant management of her coagulation disorder. In Table *5*, we present fibrinogen replacement methods, if indicated. In patients requiring fibrinogen replacement, administration of low molecular weight heparin prophylaxis, or an appropriate anticoagulant agent for pregnancy, should be considered based on individualized bleeding versus thrombosis risk.

Prepregnancy
Obtain detailed personal and family histories
Baseline laboratory testing (PT, aPTT, TT, RT, Fibrinogen antigen and activity levels, and TEG if available)
Genetic mutation testing, if not previously obtained
Partner reflex genetic testing, in particular if consanguinity suspected
Provide genetic counseling
Antenatal
Each trimester assessment of fibrinogen antigen and activity (consider more frequent monitoring with bleeding, thrombosis, need for pharmacologic interventions, and in the 3rd trimester)
Monitor for bleeding or thrombotic events
If there is clinical bleeding, fibrinogen replacement to maintain fibrinogen level > 150 mg/dL
*Standard DVT prophylaxis for pregnancy
If high risk for bleeding, withhold low dose ASA and LMWH prophylaxis
If high risk for thrombosis, consider adding LMWH prophylaxis or therapy
Intrapartum
Draw admission coagulation labs (see above)
*With bleeding phenotype, maintain fibrinogen level > 150 mg/dL during labor (if asymptomatic and TEG within normal limits consider expectant management)
If cesarean delivery required, maintain fibrinogen level > 150 mg/dL
If using neuraxial anesthesia, maintain fibrinogen level > 150 mg/dL
When possible, avoid invasive fetal procedures, vacuum, and forceps deliveries
Postpartum
Clinical and laboratory monitoring for 72 hours or until hemostasis obtained
Early use of fibrinogen replacement and tranexamic acid for postpartum hemorrhage
Standard postpartum thromboprophylaxis unless high risk for thrombosis, then add LMWH prophylaxis or therapy for 6-12 weeks postpartum
Neonatal care
Evaluate for clinical evidence of bleeding
Obtain pediatric hematology consultation or similar expertise, especially with clinical bleeding or need for operative intervention
Consider genetic testing for dysfibrinogenemia

TABLE 4: Management guidelines for women with dysfibrinogenemia during pregnancy

Detailed management guidelines for dysfibrinogenemia in mothers and neonates. To our knowledge, this is the first table to describe neonatal care and highlight options for conservative management based on laboratory guidance, clinical presentation, and multidisciplinary evaluation.

*Recommendations modified from The International Society on Thrombosis and Haemostasis (ISTH) [5]

Fibrinogen-containing product	Dosage
Fibryga (human fibrinogen concentrate)	4 gr slow IV injection in adults
Fibrinogen concentrate	Target fibrinogen activity (g/L) – measured fibrinogen activity by the Clauss method (g/L)) x (1/incremental recovery) x body weight (kg) [2]
Cryoprecipitate	Number of bags needed to infuse to increase fibrinogen activity by 100 mg/dl = 0.2 x weight (kg)[2]

TABLE 5: Fibrinogen replacement methods with appropriate dosing information

Source: [15]

Conclusions

This case demonstrates that expectant management may be a safe and effective approach in asymptomatic patients with a history of congenital dysfibrinogenemia. Of note, providers should proceed with caution, as the decision to proceed with expectant management should be based on the patient's individual history and presentation. Close monitoring is essential to quickly identify bleeding or thrombotic complications and guide management through a multidisciplinary team. Consideration of transfusion strategies based on clinical presentation, lab trends throughout pregnancy, and coagulation parameters may help avoid unnecessary interventions.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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