Clinical Case Reports Journal

ISSN: 2767-0007

Management of Von Willebrand Disease in Pregnancy, During Delivery and Postpartum Period: A Case Report

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Article Type: Case Report

Compiled date: March 17, 2025

Volume: 6

Issue: 1

Journal Name: Clinical Case Reports Journal

Publisher: Infact Publications LLC

Journal Short Name: Clin Case Rep J

Article ID: INF1000299

Keywords: Von willebrand disease; Hemostasis; Multidisciplinary care; High-Risk pregnancy; Coagulation management; Postpartum hemorrhage

Cite this article: Trupia G, La Via L, Di Grazia S, Conoscenti G, Calvagna C, Scibilia G, Mereu L, et al. Management Of Von Willebrand disease In Pregnancy, During Delivery And Postpartum Period: A Case Report. Clin Case Rep J. 2025;6(1):1–5.

Abstract

Von Willebrand Disease (VWD) is an autosomal hereditary disorder characterized by a deficiency or dysfunction of the Von Willebrand factor (VWF), crucial for hemostasis. The disease manifests in three types, with varying degrees of VWF deficiency and function. Managing pregnancies in women with VWD requires meticulous planning due to the increased risk of bleeding complications.

We report the case of a 40-year-old pregnant woman with VWD type 2A/2M, followed by a high-risk pregnancy service. Her family history included mild bleeding episodes, and she experienced recurrent bilateral nosebleeds and heavy menstrual bleeding since childhood. Despite these challenges, her pregnancy progressed physiologically. Comprehensive prenatal care involved regularly monitoring coagulation parameters and administering iron and folic acid supplements.

At 41+1 weeks, labor was induced due to the absence of spontaneous onset. The patient received coagulation FVIII concentrate rich in VWF and tranexamic acid to mitigate bleeding risks. Labor progressed without hemorrhagic symptoms, resulting in the spontaneous vaginal delivery of a healthy female infant. Postpartum management included tranexamic acid and thromboembolic prophylaxis, with continuous monitoring of coagulation parameters.

The successful outcome in this case underscores the importance of a multidisciplinary approach and meticulous management in pregnant women with VWD. Regular monitoring, appropriate prophylactic measures, and readiness for emergent interventions are crucial. Delivery in a facility with comprehensive obstetric and hematologic expertise is essential to ensure maternal and neonatal safety. This case highlights the feasibility of managing VWD in pregnancy with a well-coordinated clinical strategy.

Abbreviations

VWD: Von Willebrand Disease; VWF: Von Willebrand Factor; FVIII: Coagulation Factor VIII; PPH: Post-Partum Hemorrhage; DDAVP: Desmopressin; VWF Ag: VWF Antigen Concentration; VHF:RCo: Ristocetin Cofactor Activity; aPTT: Activated Partial Thromboplastin Time; Hb: Haemoglobin; C-ADP: Collagen Adenosine Diphosphate; C-EPI: Collagen-Epinephrine; AT III: Antithrombin III; INR: International Normalized Ratio

Background

VWD, first described in 1926 [1], is an autosomal hereditary disorder caused by a deficiency or dysfunction of the VWF, a multimeric protein that plays a crucial role in hemostasis [2]. It contains

binding sites for glycoprotein Ib, promoting platelet adhesion to the sub-endothelium, and FVIII, acting as a carrier and preventing premature clearance and degradation [3,4]. Consequently, low levels of VWF can lead to a reduction in FVIII [4]. There are three types of VWD [2]: Type 1 is characterized by a partial quantitative deficiency of VWF [2]; A qualitative deficiency of VWF characterizes type 2 and is further divided into four subtypes: 2A, 2B, 2M, 2N [2]; Type 3 is caused by an absence of VWF with significantly reduced levels of FVIII [2,5].

The incidence of VWD is equal between males and females, with a peak prevalence between ages 5–14 in males and 15–24 in females [2].

Although Von Willebrand disease is considered the most common bleeding disorder, studies on epidemiology are limited [6]. This affects 0.5%–1.3% of the general population [7], with a prevalence of 1/10000 in symptomatic forms and 1/1000 in clinically irrelevant forms [5].

Pregnancy in women with VWD may be complicated by bleeding, resulting in the risk of anemia, the need for transfusions [5] and thrombocytopenia [7]. However, the greatest challenge for these women is delivery, with increased risk of primary and secondary PPH [2,5]. PPH consist of blood loss greater than 500 ml, defined as severe when it exceeds 1000 ml. It is classified into primary PPH if occurs within 24 hours of delivery and secondary PPH if it appears between 24 hours and 6 week after delivery [2,8]. We will discuss the case of a spontaneously pregnant woman with – VWD type 2A/2M who came to our attention at the 30th week for management of her condition. Pregnant women with VWD type 2 have a high risk of hemorrhage before delivery and in the postpartum, because although VWF levels increase during pregnancy, the deficiency of VWF is qualitative [9]. In particular the type 2A e 2M are characterized respectively by deficiency of high molecular weight multimers of VWF with decreased platelet function and alteration binding with platelet glycoprotein Ib with reduced adhesion of VWF to platelets [10]. In our case, pregnant woman with VWD type 2A/2M has both genetic defects as there may be some overlap between the two types of VWD [11], with greatly impact hemostasis, posing a major hemostatic challenge during pregnancy, delivery, and postpartum.

Inheritance of VWD

In almost all cases, VWD is an autosomal dominant disease, with autosomal recessive characters in VWD type 3 and some subtypes of VWD type 2. It is due to a genetic variant within the VWF gene [12].

VWD type 1 is an autosomal dominant disorder caused by mutations that affect circulating plasma VWF levels through VWF synthesis/secretion, storage, and clearance of VWF, resulting in low plasma VWF levels but preserved function [12]. Most mutations are at exon 8, but many patients have multiple mutations at the gene. In addition, there are cases in which VDW type 1 is not always related to mutations in the VWF gene [13].

VWD type 2 is characterized by mutations in specific regions of the VWF protein [12]. In VWD type 2A, recessive mutations have been found at the D2 domain with impediment of multimer formation and dominant mutations at the D3 and CK domains inhibiting multimerization and dimerization, respectively; dominant mutations at the A2 and A1 domains with increased proteolysis, defective biosynthesis, and intracellular retention were found [13]. Dominant mutations in the A1 domain that can change the binding of VWF multimers to platelets, increasing or decreasing the affinity of VWF for platelet glycoprotein Ib, thus causing an increase (VWD type 2B) or loss (VWD type 2M, 2A/2M) of VWF function [9,13]. In VWD type 2N, recessive mutations were found in the D' domain and part of the D3 domain resulting in FVIII binding defect [13].

In VWD type 3, several recessive mutations have been found scattered throughout the gene, most of which are found in exon 28. In addition, gene conversion between the pseudogene and the VWF gene with multiple substitutions and the addition of a stop codon in the VWF gene has been seen as a common pathogenetic mechanism. This results in the absence of VWF with significantly reduced levels of FVIII [13].

Case Presentation

We present the case of a 40-year-old pregnant woman with VWD type 2A/2M who was followed at the high-risk pregnancy service due to her condition. Women with this genotype typically have low levels of VWF activity compared to VWF antigen values [14].

Her family history includes mild bleeding episodes, such as epistaxis in her father and menorrhagia in her mother. She reported recurrent bilateral nosebleeds since childhood and mild gum bleeding. She experienced menarche at age 12 with very heavy menstrual flows despite no gynecological pathology and had a tendency to bruise easily and develop spontaneous hematomas. She had an episode of microcytic anemia secondary to her bleeding tendency and was treated with oral iron supplements and intravenous iron infusions. She denied any major surgeries or significant bleeding during dental procedures.

Obstetric management: She underwent a DDAVP test, which did not fully correct her VWF defect. DDAVP is a synthetic vasopressin analog that induces the release of VWF from endothelial cells, temporarily increasing FVIII and VWF levels [3]. Due to variability in response, it is recommended to test Desmopressin in all patients with type 1, 2A, 2M, and 2N VWD, barring any contraindications [1], to evaluate its effectiveness, duration of use, and potential utility during delivery [3]. In cases where DDAVP is ineffective, secondary prophylaxis with concentrates and acute treatment with transfusions should be considered [1].

This woman was in her first pregnancy, which proceeded physiologically. She underwent fetal screening tests (echocardiogram, fetal DNA, carrier screening test), all of which

were normal. During pregnancy, she took iron and folic acid supplements. Her pre-pregnancy weight was 59 kg; by the end of pregnancy, it was 71.5 kg, for a total weight gain of 12.5 kg.

The hospital she attended is a second-level university center equipped with an intensive care unit, a transfusion center capable of urgent blood transfusions, and 24/7 obstetric, neonatal, and anesthetic care. She was managed by a multidisciplinary team of gynecologists, hematologists, anesthetists, and neonatologists to handle potential antepartum and PPH, delivery timing and method, and postpartum care. It was decided to await spontaneous labor.

At the end of her pregnancy, her lab results were: VWF Ag 56%, VWF:RCo 34%, FVIII 121%, aPTT 0.92, D-Dimer 964, ferritin 8, transferrin 4.01 g/L, transferrin saturation 12%, hemoglobin (Hb) 10.7 g/dL. Platelet function test results: C-ADP 251 sec, C-EPI > 300 sec.

She had an antepartum hematology consultation recommending the administration of FVIII concentrate rich in VWF (Plitate®) at a dosage of 40 IU/kg at the onset of labor, continuing every 12 hours for at least 4–5 consecutive days, aiming to maintain VWF activity levels between 0.50% and 1.50%. Tranexamic acid three vials (1.5 g) every 8 hours for 10 days–14 days was also advised, given the high risk of late PPH.

At 41 + 1 weeks of gestation, with no spontaneous labor, it was decided to induce labor. Upon admission, with the patient in good general condition, blood samples were taken for complete blood count, coagulation, FVIII, VWF: Ag, VWF: RCo, and platelet function test. No bleeding episodes were noted during the obstetric examination.

Her lab results showed: Hb 13.1 g/dL, Fibrinogen 376, D-Dimer 698, FVIII 147.2%, VWF: Ag 57.5%, VWF: RCo 48.8%.

Given a Bishop score < 6, pre-induction of labor was started with Misoprostol 25 micrograms (Angusta®) orally every 4 hours. After the second dose of Angusta®, a spontaneous rupture of membranes occurred with clear amniotic fluid; after the fourth dose of Angusta®, regular contractions appeared, marking the onset of labor (Bishop 11). A slow intravenous bolus of 3 vials (40 IU/kg) of FVIII concentrate rich in VWF was administered, to be repeated every 12 hours.

Post-therapy blood tests showed: VWF: Ag 206.8% VWF: RCo 11.4%, and FVIII 210%. Continuous cardiotocographic monitoring indicated fetal well-being. Prior to delivery, the anesthesiology team conducted an in-depth review of the patient's medical history, focusing on her bleeding tendencies, previous responses to treatments such as DDAVP, and current coagulation parameters. Laboratory tests, were meticulously reviewed, including complete blood count, coagulation profile, Factor VIII levels, VWF: Ag, and VWF: RCo_1. The patient's hemostatic profile was closely monitored, ensuring that her VWF activity and Factor VIII levels were within safe limits for delivery. The team also assessed her response to Plitate® and planned for its administration.

During labor, continuous monitoring of the patient's vital signs and fetal well-being were paramount. The team of anesthesiologists were prepared to manage pain effectively while minimizing the risk of bleeding. In this context, epidural analgesia was considered, but given the potential for bleeding complications, the decision was made to avoid neuraxial anesthesia unless necessary.

Given the risk of significant hemorrhage [15], the anesthesiological team was prepared for an emergent Cesarean section if required [16,17]. A detailed plan was established for administering general anesthesia [18], ensuring that all necessary blood products, including FVIII concentrates and tranexamic acid, were readily available. The operating room was prepared with equipment for rapid blood transfusion and advanced hemodynamic monitoring [19].

Labor progressed physiologically with no hemorrhagic symptoms and all vital signs within normal ranges throughout its duration. Approximately 3 hours after labor began, the spontaneous vaginal delivery of a female infant in cephalic presentation occurred. The newborn was alive and healthy, weighing 3790 g, with normal biometrics (length 51 cm, head circumference 36 cm) and Apgar scores of 9 and 10 at 1 and 5 minutes, respectively. Prophylactic administration of 10 IU oxytocin intramuscularly was performed upon shoulder delivery. A blood sample was taken from the umbilical cord for fetal pH analysis, which showed a value of 7.39. Spontaneous and complete placental expulsion occurred within the physiological timeframe. The placenta and membranes appeared intact upon inspection. The estimated total blood loss was 50 cc. The uterine contraction was good upon leaving the delivery room. A first-degree vaginal laceration was sutured with absorbable Vycril 1.0. Hemostasis was good and satisfactory. Immediate postpartum therapy with tranexamic acid was administered, with three vials to be repeated every 8 hours. The use of tranexamic acid during the postpartum period for 10 days–14 days is recommended as it reduces the risk of secondary PPH [2]. It also appears safe during breastfeeding [3].

The patient was closely monitored in the delivery room for the next 2 hours, with checks on uterine contractility, lochia, and vital signs, all of which were normal. She was then transferred to the ward due to the physiological progression and absence of complications. Thromboembolic prophylaxis was performed 12 hours after delivery with Inhixa 4000 IU 1 vial/day, considering the corrected VWF: RCo and FVIII values [5]. Total hospital stays: 5 days. Throughout the stay, vital signs were normal, the uterine body contracted and retracted, and lochia was regular. Monitoring of complete blood count, coagulation, FVIII, VWF: Ag, and VWF: RCo was performed as it is crucial to ensure that VWF activity and FVIII levels are maintained above 0.5 IU/ml for at least 3 days post-vaginal delivery [5].

On the third postpartum day, her lab results were: Hb 13.1 g/dL,

fibrinogen 378, D-Dimer 404, INR 1.01, aPTT 24.5 sec, AT III 79%, FVIII 257.8%, VWF: Ag 236.5%, VWF: RCo 195.6%. On the fourth postpartum day, her lab results were: Hb 11.7 g/dL, fibrinogen 357, D-Dimer 400, INR 1, aPTT 28.8 sec, ATIII 74%, FVIII 110.5%, VWF: Ag 220.8%, VWF: RCo 118.6%. On the fifth postpartum day, her lab results showed: Hb 11.7 g/dL, fibrinogen 308, D-Dimer 717, INR 0.99, aPTT 28.2 sec, AT III 83%, FVIII 121.2%, VWF: Ag 215.5%, VWF: RCo 97.8%. These FVIII, VWF: Ag, and VWF: RCo values were communicated to the hematologists, who confirmed their normality and compatibility with Plitate® therapy.

The newborn underwent platelet function tests with the following results: C-ADP 56 sec (normal range 68 sec–121 sec), C-PPI 110 sec (normal range 84 sec–160 sec), FVIII 45.6 s, VWF: Ag 217% (normal range 42.0–176.3), VWF; RCo 119.4% (normal range 48.2–239.8). In healthy neonates, VWF levels are generally elevated at birth [5]. Since VWD is an autosomal hereditary condition, the newborn may be affected, and early diagnosis is necessary [7].

The patient was discharged at the end of Plitate® therapy with a prescription for oral tranexamic acid to be continued for 14 days. According to the ASH 2021 guidelines on diagnosing and managing VWD, it can be administered three times a day for 10 days–14 days or longer if significant blood loss persists [2,20]. At 2 weeks, follow-up blood tests for complete blood count, VWF: Ag, VWF: RCo, and FVIII were normal.

At the 30-day postpartum check-up, the patient was in good general condition. The uterus was in normal puerperal involution, and there were no episodes of vaginal bleeding. Postpartum monitoring of vaginal discharge is crucial to ensure physiological lochia and to check the patient's hemodynamic status [5]. The increase in VWF levels that occurs during pregnancy persists within the first postpartum week and returns to baseline levels by the third postpartum week, predisposing women with VWD to a higher risk of hemorrhagic episodes [21]. Therefore, postpartum check-ups are crucial to prevent secondary PPH and to discuss potential indications regarding the resumption of menstruation [22].

Discussion

There are three types of VWD [2]: Type 1 is present in 75% of cases and is characterized by a partial quantitative deficiency of VWF [2] with functional activity less than 0.3 IU/ml [5].

Type 2 is characterized by a qualitative deficiency observed in 20% of cases and is further divided into four subtypes [2]:

Type 2A involves selective loss of high molecular weight VWF multimers [2];

Type 2B results from a structural change in VWF [2], increasing its affinity for platelet glycoprotein Ib [5];

Type 2M is marked by reduced interaction of VWF with platelet glycoprotein Ib [5];

Type 2N has a reduced capacity of VWF to bind FVIII [2].

Type 3 occurs in less than 1% of cases and is the rarest form, caused by an absence of VWF with significantly reduced levels of FVIII [2,5].

Screening tests include the evaluation of Prothrombin Time (PT), aPTT, fibrinogen, FVIII levels, VWF: RCo, and VWF: Ag [1,23].

Patient history and clinical presentation are crucial for diagnosis, which is later confirmed by laboratory findings showing abnormalities in VWF levels, FVIII, and VWF activity [4,14].

The goal of therapy in VWD is to correct the dual defect in hemostasis. Several therapeutic approaches are available, including tranexamic acid, DDAVP, and concentrates containing either VWF alone or both FVIII and VWF [1]. Clinically, the disease manifests with mucocutaneous bleeding, heavy menstrual bleeding, frequent nosebleeds, and bleeding during minor and major surgeries [2,4].

Pregnancy poses particular interest in managing VWD, as procoagulant factors, including VWF and FVIII, physiologically increase during pregnancy [2], peaking in the third trimester [3]. Each type of VWD responds differently to pregnancy due to its phenotypic and genotypic heterogeneity [24]. Women with type 1 VWD rarely experience excessive bleeding [2] due to the physiological increase in coagulability that compensates for VWF deficiency [5]. This is not the case for women with type 2 VWD, where the deficiency is qualitative, or type 3, where the increase is minimal or absent [5].

Antepartum bleeding risk increases tenfold, PPH occurs in 15%–20% of women, and perineal hematomas, although rare, are more frequent [5,23]. No increased frequency of preterm delivery, pre-eclampsia, fetal growth restriction, spontaneous abortion, or placental abruption has been documented [5].

VWD is not usually an indication for cesarean section and delivery mode is based on obstetric indications [24]. Delivery is critical not only for the mother but also for the newborn, as they are at risk of complications like intracranial and scalp hemorrhages, necessitating a minimally traumatic delivery [7].

During spontaneous labor, the patient was closely observed for any signs of bleeding. The administration of FVIII concentrate began at the onset of labor, continuing every 12 hours to maintain therapeutic levels. Tranexamic acid was also administered intravenously to reduce the risk of PPH. The team maintained a high level of readiness to convert to general anesthesia for a Cesarean section if fetal distress or other complications arose.

Clear communication and coordination among the obstetrics, hematology, and anesthesiology teams were crucial throughout the process. This collaborative approach ensured that the patient received comprehensive care, reducing the risk of complications and promoting a safe delivery outcome.

The management described is in line with studies in the literature. In our case, the management in pregnancy, during delivery, and in the postpartum period of a pregnant woman with VWD type 2A/2M, given the increased risk of antepartum and PPH episodes related to her particular phenotype, highlights how correct management ensures a safe delivery, both for the woman and for the future unborn child.

Conclusions

The rarity of the disease and its difficult management make cases like this a clinical and organizational challenge. In the case in question, the objective during pregnancy was to safeguard the health of both the mother and the newborn, requiring careful and well-organized clinical management. Planning procedures and maintaining close contact with all involved medical professionals were fundamental. This case confirms that careful and multidisciplinary management, targeted and personalized interventions, allow for relatively safe management of VWD in pregnancy. Delivery in a facility with appropriate safety standards is essential.

Conflict of Interest

The authors declare no potential conflicts of interest with respect to the research, authorship, and/or publication of this article. Informed consent was obtained for this publication.

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