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# A Rare Case of Von Willebrand Disease in Pregnancy: Management Strategies and Challenges

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**Abstract: Von Willebrand disease (VWD), the most common inherited bleeding disorder, is classified into types 1, 2, and 3. Women with VWD are at higher risk of bleeding during menstruation, childbirth, and postpartum. This case of a 24-year-old pregnant woman with VWD underscores the importance of early management to prevent postpartum hemorrhage. Desmopressin and VWF infusion enabled a successful cesarean and uneventful recovery. Diagnosis relies on VWF and FVIII assays, and prophylactic treatment is advised during pregnancy. Vaginal delivery without instrumentation is preferred, with cesarean reserved for obstetric reasons.**

***Keywords:*** *Von Willebrand Disease, Deficiency, Haemorrhage, Pregnancy.*

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## I. INTRODUCTION

Von Willebrand disease (VWD) is the most common hereditary hemostasis disorder caused by qualitative or quantitative deficiency of Von Willebrand factor.(1) VWD affects 0.6%–1.3% of the population, with symptomatic cases requiring treatment affecting both sexes equally. Types 2N and 3 follow autosomal recessive inheritance, while types 1, 2A, 2B, and 2M are autosomal dominant.(2) Women with VWD face elevated bleeding risks during menstruation, childbirth, and postpartum, leading to potential complications like hemorrhage, transfusion reactions, and organ damage.(1, 2) Early treatment planning in pregnant women with VWD is essential to prevent postpartum hemorrhage, and invasive delivery methods like vacuum extraction or rotational forceps should be avoided.(3)

This article presents the case of a 24-year-old pregnant woman with Von Willebrand disease and a favorable perinatal outcome, outlining the institutional management protocol during labor and postpartum.

## II. CASE REPORT

A 24-year-old nulliparous Arab woman with von Willebrand factor deficiency, diagnosed at 22 after unexplained hemoperitoneum, was referred at 24 weeks gestation. She had mild bleeding (epistaxis, bruising) and a family history of fatal hemorrhages. Labs showed low vWF activity (18.7%) and factor VIII (33.9%), confirming diagnosis.

During this pregnancy, clinical examination revealed no significant abnormalities. A pre-anesthetic evaluation at 32 weeks included a risk assessment, with blood tests showing a prothrombin time of 100% (11.4 sec), hemoglobin of 10g/dL, platelet count of 319,000/mm³, activated partial thromboplastin time of 36.9s, FVIII activity of 51%, and vWF activity at 8.1%.

The patient was admitted at 38 weeks for a scheduled cesarean under general anesthesia, receiving von Willebrand factor infusion and oxytocin prophylaxis; she delivered a healthy male infant (3370g) with Apgar scores of 10 at 1 and 5 minutes.

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Due to the high risk of postpartum hemorrhage, the patient was admitted to the ICU for monitoring. She recovered without complications and was discharged after two days. Genetic counseling for the baby was deferred and planned before circumcision.

## III. DISCUSSION

The von Willebrand factor, a multimeric protein encoded by the VWF gene on chromosome 12p, is produced by vascular endothelium and megakaryocytes and stored in Weibel-Palade bodies.(4) VWF is essential for primary and secondary hemostasis, mediating platelet adhesion and aggregation, and stabilizing factor VIII by protecting it from plasma degradation(2).

Von Willebrand disease is classified into three types:

- Type 1 VWD, the most common form (80%), is a partial quantitative VWF deficiency with proportional reduction in antigen and activity. Bleeding is mild to moderate. Diagnosis is made when VWF levels fall below 0.30 IU/mL (with bleeding history) or 0.50 IU/mL (without).(1, 5)
- Type 2 VWD, comprising most remaining cases, features low functional VWF despite normal or near-normal antigen levels. It includes subtypes 2A, 2B, 2M, and 2N, classified by VWF multimer patterns. Bleeding is generally moderate.(1, 5)
- Type 3 VWD is a rare, severe form caused by complete VWF deficiency and markedly low factor VIII levels, leading to serious bleeding. Its global prevalence ranges from 0.1 to 5.3 cases per million (5, 6)

VWD presents with variable symptoms, including spontaneous bleeding and after surgery. Hematomas and hemarthrosis occur in severe FVIII deficiency (Type 3).(4)

The Willebrand Disease Reference Center confirms VWF deficiency using biological and molecular criteria. Initial tests include CBC, PT, aPTT, and fibrinogen assay; hemorrhagic cases undergo additional factor VIII, IX, XI activity, and VWF antigen and activity assays..(4, 7)

The diagnosis of VWD is confirmed through three specific biological tests:

- The ristocetin cofactor activity assay (VWF:RCo), which measures the functional activity of plasma VWF, is the reference and first-line test.
- The VWF antigen assay (VWF:Ag), which quantifies the circulating plasma VWF protein.
- The coagulant FVIII assay (FVIII:C), which measures FVIII levels that decrease in parallel with VWF levels. (7)

During pregnancy, VWF and factor VIII levels rise, but women with VWD have lower levels, increasing their risk of bleeding. However, women with VWF levels ≥50% in the third trimester have no increased risk of postpartum hemorrhage. The rise in VWF and FVIII during pregnancy improves hemostasis, making bleeding rare in women with Type 1 VWD or VWF >50 (IU/dL).(5, 8)

Prophylaxis is advised if factor VIII <25% and VWF <50 IU/dL during pregnancy. Treatments include desmopressin (0.3 µg/kg IV over 15–30 min for Type 1 VWD), transfusions, or antifibrinolytics. For cesarean, desmopressin is given 30 minutes prior and repeated every 12–24 hours due to its short duration..(9, 10) VWF and factor VIII concentrates are recommended if desmopressin fails. Antifibrinolytics, such as aminocaproic acid, serve as second-line treatment for mild bleeding. Vaginal delivery without instrumentation is preferred.(10) The decision to perform an epidural during delivery will depend on the specific case.(11)

Delayed postpartum hemorrhage is common in VWF deficiency due to rapid postpartum decline of VWF and factor VIII, normalizing within 1–3 weeks. Prevention includes oral antifibrinolytics such as tranexamic acid (1 g every 8 hours for up to two weeks).(3)

## IV. CONCLUSION

Von Willebrand disease, a common bleeding disorder, demands careful pregnancy management—including prophylactic desmopressin, antifibrinolytics, timely interventions, and tailored delivery—to prevent complications like postpartum hemorrhage and ensure maternal health.

➢ *Ethics Approval and Consent*

In line with the Declaration of Helsinki; no ethics approval required. Informed consent obtained.

➢ *Consent for Publication*

Written informed consent was obtained for publication of anonymized clinical and biological data.

➢ *Funding*

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➢ *Competing Interests*

The authors declare no competing interests.

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