Article type: Editorial

Home Page URL: https://www.alliedacademies.org/hematology-blood-disorders/

Von willebrand disease: The most common clotting disorder you haven't heard of.

Isak Dinesen*

Department of Hematology, Anna University, India

Correspondence to: Isak Dinesen, Department of Hematology, Anna University, India, E-mail: isak@chester.ac.uk

Received: 02-Sep-2025, Manuscript No. aahbd-25-171225; **Editor assigned:** aahbd-25-171225, PreQC No. aahbd-25-171225 (PQ); **Reviewed:** 17-Sep-2025, QC No. aahbd-25-171225; **Revised**: 24-Sep-2025, Manuscript No. aahbd-25-171225 (R); **Published**: 30-Sep-2025, DOI: 10.35841/aahbd-8.3.228.

Introduction

When people think of bleeding disorders, hemophilia often comes to mind. Yet, the most common inherited bleeding disorder is not hemophilia—it's Von Willebrand Disease (VWD). Affecting up to 1% of the population, VWD is a condition that impairs the blood's ability to clot properly, often going undiagnosed due to its subtle symptoms and lack of public awareness. Von Willebrand Disease is a genetic bleeding disorder caused by a deficiency or dysfunction of von Willebrand factor (VWF), a protein essential for platelet adhesion and clot formation. VWF acts like biological glue, helping platelets stick to damaged blood vessels and form a clot. Without it, bleeding can persist longer than normal [1].

Living with a chronic bleeding disorder can take a toll on mental health. Anxiety about bleeding episodes, social stigma, and limitations in physical activities can affect quality of life. Support groups and counseling can help patients cope and connect with others facing similar challenges. Advancements in genetic testing, recombinant therapies, and personalized medicine are improving outcomes for VWD patients. The most common and mildest form, where individuals have lower-than-normal levels of VWF. It accounts for about 85% of diagnosed cases. Characterized by dysfunctional VWF. It has four subtypes—2A, 2B, 2M, and 2N—each with distinct molecular defects. The rarest and most severe form, where VWF is virtually absent, often leading to serious bleeding episodes. VWD is typically inherited in an autosomal dominant pattern, meaning a child can inherit the condition from just one affected parent. However, Type 3 VWD is inherited in an autosomal recessive manner, requiring both parents to pass on the defective gene [2].

VWD affects up to 1% of the U.S. population, or roughly 3.2 million people. Globally, prevalence estimates vary due to underdiagnosis and lack of awareness. Many individuals with mild symptoms never seek medical attention, contributing to the disease's hidden burden. Despite its prevalence, public awareness remains low. Advocacy groups like the National Hemophilia Foundation and World Federation of Hemophilia are working to educate communities and improve access to care. Unlike hemophilia, which primarily affects males, VWD affects men and women equally. However, women are more likely to experience noticeable symptoms due to menstruation and childbirth. Symptoms of VWD can range from mild to severe and often mimic everyday issues, making diagnosis difficult. Common signs include: Frequent or prolonged nosebleeds, Easy bruising, Heavy menstrual bleeding (menorrhagia), Excessive bleeding after surgery, dental work, or childbirth [3].

With proper management, most people with VWD can lead normal, active lives. Education is key—patients must understand their condition, recognize symptoms, and communicate with healthcare providers before surgeries or procedures. Women with VWD face unique challenges during menstruation, pregnancy, and childbirth. Obstetricians and hematologists often collaborate to ensure safe delivery and postpartum care. In severe cases, joint and muscle bleeding, similar to haemophilia. Because these symptoms can be subtle or mistaken for other conditions, many people live with VWD for years before receiving a diagnosis. Diagnosing VWD involves a combination of blood tests and clinical evaluation. Key diagnostic tools include: Measures the amount of von Willebrand factor in the blood. Assesses how well VWF functions. Since VWF stabilizes factor VIII, its levels may also be low [4].

Citation: Dinesen I. Von willebrand disease: The most common clotting disorder you haven't heard of. Hematol Blood Disord. 2025;8(3):228.

Evaluate how well platelets interact with VWF. Because VWD symptoms overlap with other bleeding disorders, accurate diagnosis requires specialized testing and often referral to a hematologist. Treatment depends on the type and severity of VWD. Common therapies include: A synthetic hormone that stimulates the release of stored VWF and factor VIII. Effective for Type 1 and some Type 2 cases. Infusions of plasma-derived or recombinant VWF for patients with Type 3 or severe bleeding episodes. Medications like tranexamic acid help prevent clot breakdown, especially useful during dental procedures or menstruation. Birth control pills may help manage heavy menstrual bleeding in women. Patients are advised to avoid medications that impair clotting, such as aspirin and NSAIDs, unless prescribed by a doctor [5].

References

1. AJahoda CA, Whitehouse CJ, Reynolds AJ, Hair follicle dermal cells differentiate into adipogenic and osteogenic lineages. Exp Dermatol ; 12(6):849-859.
2. Driskell RR, Lichtenberger BM, Hoste E, Distinct fibroblast lineages determine dermal architecture in skin development and repair. Nature; 504(7479):277-281.
3. Horne KA, Jahoda CA, Oliver RF Whisker growth induced by implantation of cultured vibrissa dermal papilla cells in the adult rat. J Embryol Exp Morphol; 97(1): 111-124.
4. Jahoda CA, Oliver RF, Reynolds AJ, Human hair follicle regeneration following amputation and grafting into the nude mouse. J Invest Dermatol; 107(6): 804-807.
5. Ferlay J, Soerjomataram I, Dikshit R, Cancer incidence and mortality worldwide: sources, methods and major patterns in GLOBOCAN 2012. Int J Cancer; 136(5): E359–E386.

Citation: Dinesen I. Von willebrand disease: The most common clotting disorder you haven't heard of. Hematol Blood Disord. 2025;8(3):228.