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Topic Overview

Von Willebrand disease (VWD) is the most common hemostatic disorder in domestic dogs. Clinical signs vary greatly: some dogs are asymptomatic, whereas others experience severe clinical signs. VWD is highly prevalent in certain breeds, including the Doberman pinscher. Veterinary nurses play a key role in the management of patients to ensure their safety and wellbeing, in addition to providing thorough and clear client education. This article provides a comprehensive overview of the pathophysiology of VWD, the 3 disease variants, predisposed breeds, diagnosis, clinical signs, and patient management, including client communication and education.

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HEMATOLOGY

A Veterinary Nurse's Guide to von Willebrand Disease

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Von Willebrand disease (VWD) is the most common inherited hemostatic disorder in the domestic dog.¹ It is also known to affect humans and, less commonly, domestic cats and horses.

coagulation pathway. VWF is synthesized in endothelial cells and megakaryocytes and is released in response to endothelial damage and agonists (e.g., thrombin, histamine).^{1,2} It is also a carrier protein for coagulation factor VIII (FVIII), which is a cofactor in the coagulation pathway.

PATHOPHYSIOLOGY

In normal physiology, a large adhesive plasma glycoprotein known as von Willebrand factor (VWF) plays an important role in the intrinsic

The role of VWF is primary hemostasis, and it is metaphorically characterized as “glue,” as it mediates the adhesion of platelets to damaged subendothelium and subsequently promotes

Learning Objectives

- Von Willebrand disease (VWD) is the most common inherited hemostatic disorder in domestic dogs.
- There are 3 variants of VWD that typically present with either a quantitative and/or qualitative abnormality of von Willebrand factor (VWF).
- The type 1 variant is the most common, accounting for more than 95% of cases; therefore, most affected patients seen in clinical practice have a decreased but functional concentration of VWF.
- In each breed affected, a single variant predominates. Type 1 is predominantly observed in Doberman pinschers.
- Each variant arises due to a different genetic mutation of the VWF gene.
- Clinical signs vary greatly. Some dogs are asymptomatic, whereas others experience severe clinical signs.
- Several diagnostic procedures can be conducted, but the concentration and function of VWF should be determined for a definitive diagnosis. No single test can detect all VWD variants.
- Cryoprecipitate or fresh frozen plasma can be administered intravascularly as part of the treatment plan, or presurgical prophylaxis for hemorrhage caused by a deficiency of VWF.
- Veterinary nurses should conduct risk management planning with clients, particularly in newly diagnosed cases.
- VWD is incurable, but with the appropriate care, many dogs can live a normal life.

aggregation to form a platelet plug.³ VWF is made of different-sized molecules known as multimers that vary in molecular weight.⁴ The large multimers with high molecular weight have multiple binding sites for platelet glycoprotein receptors¹ and are the most effective at promoting platelet adhesion and hemostasis.⁵ In VWD, VWF is deficient or defective, leading to inefficient hemostasis due to lack of platelet adhesion and, potentially, excessive hemorrhage. The severity of VWD is based on the deficient or defective nature of VWF. Three known variants exist: types 1, 2, and 3.⁶

DISEASE VARIANTS AND GENE MUTATIONS

The 3 variants of VWD typically present with either a quantitative or qualitative abnormality of VWF (**TABLE 1**). The extent to which an individual is affected often correlates with the level of VWF. For example, a severe deficiency is correlated with clinical signs and an increased risk of hemorrhage.⁵ However, a diagnosis based on clinical signs alone is unreliable due to the variation in clinical signs and the impact of other variables on clinical manifestation; dogs with VWD may be asymptomatic, while some dogs with normal VWF concentrations may experience hemorrhage.¹ Therefore, testing is required for a definitive diagnosis, including disease variant.

Each variant is caused by a different genetic mutation of the VWF gene.^{1,9,10} The type 1 variant is the most common variant in domestic dogs, accounting for more than 95% of cases¹¹; therefore, most dogs with VWD seen in clinical practice have a decreased but functional concentration of VWF. Type 2 is rare; type 3, the most severe variant, is the least common.¹² Type 3 may also be referred to as “severe type 1” due to the severity of VWF deficiency and increased risk of hemorrhage.

AFFECTED BREEDS

Although VWD is most associated with Doberman pinschers, it has been identified in more than 50 other breeds. It was first reported in dogs in 1970 in a group of biologically related German shepherds that displayed laboratory findings and clinical signs similar to those consistent with human VWD, including hemorrhage from an incisional ovariohysterectomy wound, spontaneous gastrointestinal hemorrhage, and chronic intestinal hemorrhage.^{13,14} **TABLE 2** lists some affected breeds according to the variant most commonly identified in each breed.

There is not an extensive body of research on the prevalence of VWD variants in dogs, but it can be concluded that any variant can occur in any breed.²¹

TABLE 1 Overview of von Willebrand Disease Variants^{1,6-8}

TYPE	CHARACTERISTICS	MULTIMERIC STRUCTURE OF VWF	CLINICAL SEVERITY
1	Quantitative deficiency: Proportionate decrease of all multimers but functional concentration of VWF	Normal structure	Variable; subclinical or mild to moderate clinical signs
2	Quantitative deficiency and qualitative defect: Decreased concentration of large multimers and abnormal structure of VWF	Abnormal structure, disproportionate decrease in large multimers	Moderate to severe clinical signs
3	Quantitative deficiency: Trace amount or complete absence of VWF	Absent	Severe clinical signs

VWF = von Willebrand factor

TABLE 2 Types of von Willebrand Disease and Affected Breeds

	TYPE 1	TYPE 2	TYPE 3
Predominantly observed in	Doberman pinscher ^{11,12}	German shorthaired pointer, German wirehaired pointer ^{7,15}	No known predominantly affected breed
Also observed in	Pembroke Welsh corgi, poodle, Bernese mountain dog, Manchester terrier, Akita, dachshund, German shepherd, golden retriever, Shetland sheepdog, mixed breed, other breeds ^{1,10}	German shepherd, Shetland sheepdog, Pembroke Welsh corgi, miniature pinscher, rottweiler, mixed breed ¹⁶	Scottish terrier, ¹⁷ Shetland sheepdog, ¹⁸ Dutch Kooikerhondje, ¹⁹ Chesapeake Bay retriever ²⁰

CLINICAL SIGNS

As noted earlier, clinical signs of VWD vary greatly. Generally, the risk of hemorrhage is greater in dogs with lower VWF concentrations.⁹ Traumatic events, as well as even some routine clinical procedures such as nail clipping, injections, and surgery, can cause excessive and prolonged hemorrhage, which is often the first clinical sign of disease.^{1,22} Affected puppies may also experience excessive gingival hemorrhage during dental eruption. Further clinical signs may include intracranial hemorrhage and spontaneous hemorrhage from the nasal and oral cavities and genitourinary and gastrointestinal tracts.^{1,12,23} Subcutaneous postinjection hematoma, ecchymosis, and hemarthrosis with secondary lameness have also been reported.^{24,25}

Clinical signs may be exacerbated by certain preexisting conditions, including endocrine disease and other clotting conditions, and drugs that interfere with hemostasis such as aspirin, heparin, NSAIDs, and some antibiotics.^{1,24,26} These drugs should therefore be used with caution or avoided in dogs with VWD. Events such as hormonal fluctuations during estrus and pregnancy, systemic conditions such as endocrine disease, and infection may also exacerbate clinical signs.

DIAGNOSTIC TESTS

Diagnosis involves the initial assessment of patient history and clinical signs, if present. No single test can detect all VWD variants; therefore, diagnosis can involve multiple stages. As VWD typically presents with a quantitative and/or qualitative abnormality of VWF, the concentration and function of VWF should be determined. Several laboratory tests can be conducted during the diagnostic workup, many of which go beyond the scope of this article. Some of the most recognized tests are discussed below.

Buccal Mucosal Bleeding Time

Buccal mucosal bleeding time (BMBT) is a straightforward, inexpensive, and often well-tolerated *in vivo* screening test that is used to assess the elapsed time between the creation of a minor incision on the buccal mucosa and cessation of hemorrhage. It is used to determine the presence of a primary hemostatic disorder.⁴ In healthy dogs, BMBT is expected to be less than 4 minutes,²⁷ but times can vary from 1.68 to 4.15 minutes.²⁸ This test is not specific for VWD and thus cannot solely be used for diagnosis. Additionally, elapsed times may vary due to operator dependency

and equipment variation. BMBT is subjective and there is a lack of correlation with clinical outcomes, as it cannot accurately predict the extent of hemorrhage.²⁸ It should be used with caution in dogs suspected or confirmed to have type 2 or type 3 VWD due to the risk of prolonged and/or excessive hemorrhage. Prolonged BMBT requires intervention and further diagnostics to determine an underlying cause.

Plasma VWF Antigen Assay

The plasma VWF antigen (VWF:Ag) assay measures the concentration of VWF in plasma, and results are reported as a percentage compared to a 100% standard from pooled plasma in healthy dogs. The VWF:Ag concentration in a normal dog is greater than 70%.⁸ This assay is the test most commonly used to determine the presence of VWD and predict genetic status. However, as the type 2 variant involves a qualitative defect in VWF, this test cannot be used to truly identify this variant, and assessment of VWF function is additionally required. Due to the effect of systemic stress on plasma VWF concentration, this test should be conducted when the patient is physiologically well and not in estrus, pregnant, or lactating. It can be conducted on puppies from 6 to 8 weeks of age.¹⁰ The key stages of this test and result interpretation are outlined in **FIGURE 1**.

An American- and Canadian-based epidemiological study conducted throughout the mid- to late-1980s reported that 73% of Doberman pinschers ($n = 1075/1466$) were positive for VWD.²⁹ The same study identified 28% of Shetland sheepdogs ($n = 203/730$) and 30% of Scottish terriers ($n = 71/235$) as having VWD. Diagnosis was confirmed by VWF:Ag assay. The researchers did not conduct testing to establish responsible mutations; thus, the variant prevalence is unknown.

Plasma VWF Collagen Binding Assay

The plasma VWF collagen binding assay is a functional test; it assesses the ability of VWF to bind to collagen as it would naturally do in the event of endothelial damage,²¹ with a preference for large multimers.²⁸ Results are correlated to the VWF:Ag assay to determine disease variant (**TABLE 3**).

As the type 2 variant involves a qualitative defect in VWF and a greater decrease in large multimers, this is a valuable screening test for this variant, although it is

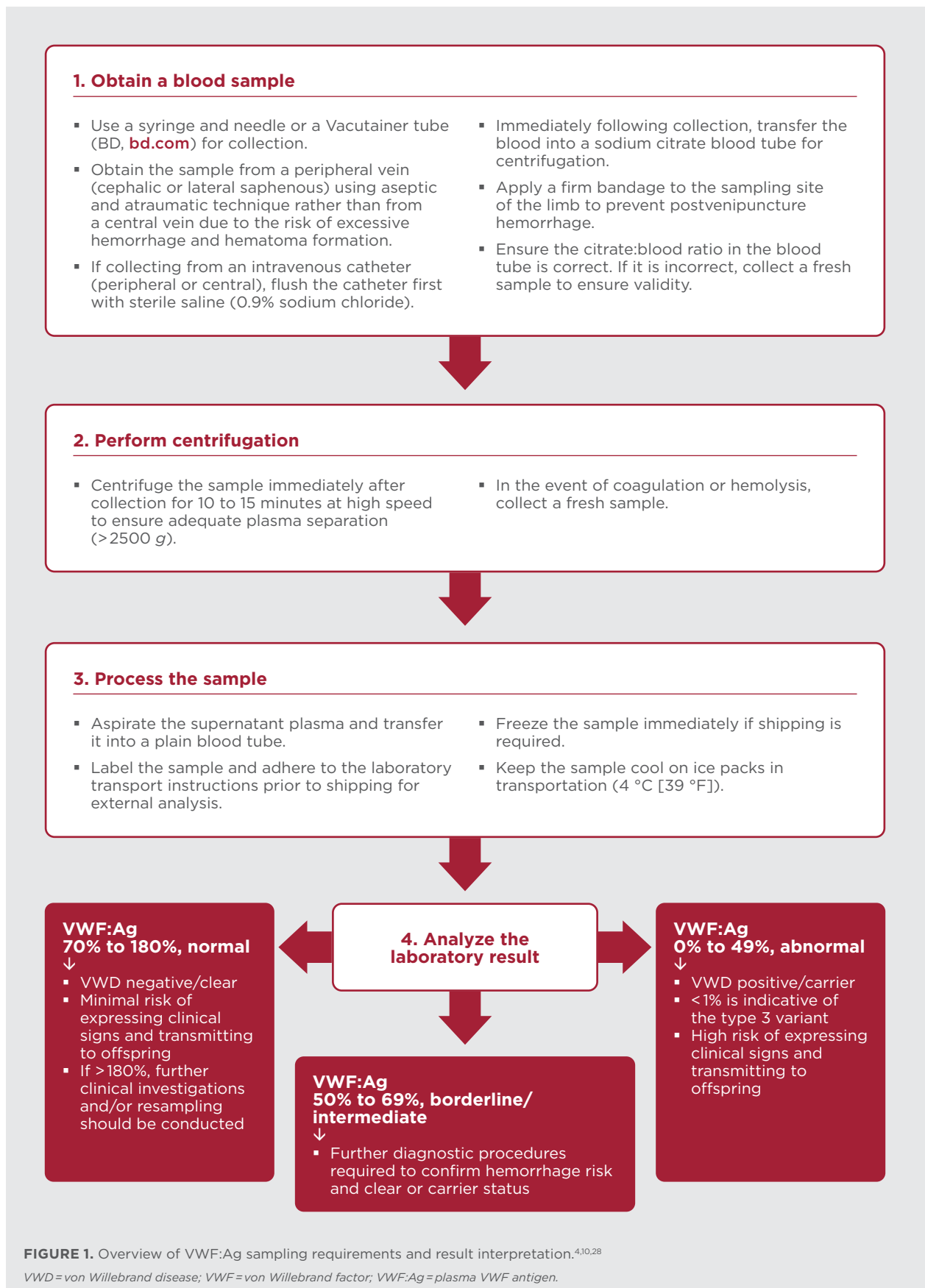


TABLE 3 VWF:Ag to VWF:CBA Ratios^{1,16,28}

VWD STATUS/VARIANT	EXPECTED RATIO
VWD negative	
1	≤1
2	>2
3	Close to 0

VWD = von Willebrand disease; VWF = von Willebrand factor; VWF:Ag = plasma vWF antigen; VWF:CBA = plasma VWF collagen binding assay

not widely available. When used alongside the VWF:Ag assay result, it can also be used to distinguish between type 1 and type 3.²⁸ This is a coagulation test; therefore, the sampling methods described in **FIGURE 1** should be followed.

Genetic Testing

Genetic testing is conducted to confirm a dog's genetic status, which is particularly valuable if their VWF:Ag assay result is borderline/intermediate (50% to 69%). Owners and breeders of high-risk breeds can collect a buccal swab sample from their dog at home, without the need to visit a veterinary practice. The sample is then sent to a laboratory for PCR testing. Additionally, some external laboratories can conduct this test on a whole blood sample in an EDTA blood tube.

Generally, there are 3 possible outcomes: clear, carrier, or affected (**TABLE 4**). Breeding programs can be guided by the result to help reduce the prevalence of VWD.¹⁰ Genetic testing is available for many breeds and should be recommended to clients who own a high-risk breed, especially before breeding and during new puppy consultations.

PATIENT MANAGEMENT AND CLIENT COMMUNICATION

VWD is incurable, but with appropriate care, many dogs can live a normal life. Palliation involves preventing injury and hemorrhage, which is easier than treating the disease. Efforts should be made to keep dogs safe in the clinical setting (**BOXES 1 AND 2**) and educate clients on tips for keeping their dog safe in the home environment (discussed below).

Anesthesia and Surgery

Informed consent for anesthesia and surgery must be obtained after the risks have been discussed with the client and before the patient is admitted. If a patient has not been tested but there is suspicion for VWD (e.g., high-risk breed, history of clinical signs), testing should be encouraged before anesthesia. It is recommended that all Doberman pinschers undergo coagulation testing before anesthesia and surgery due to the high prevalence in this breed, particularly BMBT testing if presented for emergency surgery.³¹

Risk Management

The American Society of Anesthesiologists physical status classification system should be used before anesthesia to identify the level of risk and enable appropriate planning. However, it is subjective (and intended for humans), and the patient may have comorbidities that make grading difficult.

Anesthetic safety checklists should also be used to assist the veterinary team with individual planning to prevent human error and accidents.³²

TABLE 4 DNA Genetic Testing Results^{10,30}

RESULT	INTERPRETATION AND BREEDING GUIDANCE
Clear	● Homozygous: Inherited 2 normal VWF genes (1 from each parent) ● Minimal to no risk of hemorrhage ● Suitable for breeding; will produce 100% clear puppies if bred with a clear mate
Carrier	● Heterozygous: Inherited 1 mutated gene and 1 normal gene (1 from each parent) ● Risk of hemorrhage ● Can be bred with a clear mate to produce 50% clear puppies and 50% carrier puppies ● Carrier-to-carrier breeding is not recommended due to the increased risk of disease inheritance
Affected	● Homozygous: Inherited 2 mutated VWF genes (1 from each parent) ● Increased risk of hemorrhage ● Not suitable for breeding; will pass on the mutated gene to all puppies

VWF = von Willebrand factor

BOX 1

General Patient Management in Clinical Practice

- Before admission, conduct a risk assessment to reduce the risk of injury.
- Implement VWD standard operating procedures to ensure consistent care.
- Display kennel signage to inform others of the suspected or confirmed diagnosis.
- Accommodate restless patients in glass-door kennels to reduce the risk of entrapment.
- When obtaining a blood sample, obtain from a peripheral vein using atraumatic technique (**FIGURE 1**).
- Prevent postvenipuncture hemorrhage with a firm limb bandage.
- Monitor for clinical signs and notify a veterinarian if evident.

VWD = von Willebrand disease

BOX 2

Drug Administration

- Liaise with the veterinarian regarding drugs that interfere with hemostasis (e.g., aspirin, heparin, NSAIDs), as they may need to be avoided due to increased risk of hemorrhage.
- Before anesthesia and surgery, ensure that necessary drugs and products (e.g., desmopressin acetate) and blood products (cryoprecipitate or fresh frozen plasma) are available.
- Administer under the instruction of the veterinarian and monitor for adverse effects.
- Administer via noninvasive routes if possible.

Hemorrhage Control Measures

Intravenous access, preferably via a peripheral vein, must be obtained and secured before anesthesia for routine and emergency use. Equipment, materials, and drugs for hemorrhage control should be available for prophylactic and/or emergency use (**TABLE 5**).

Transfusion Products

Cryoprecipitate is a concentrated transfusion product made from fresh frozen plasma (FFP). It contains multiple labile coagulation factors, including fibrinogen, FVIII, and VWF.³³ This product is administered intravascularly in the treatment or presurgical prophylaxis of hemorrhage caused by a deficiency of VWF. It is often used as an alternative to FFP due to its concentration and ability to replenish FVIII and VWF at lower volumes.³⁴ A clinical study

investigating the administration of FFP and cryoprecipitate in dogs with VWD concluded cryoprecipitate to be the more efficacious product at increasing VWF:Ag concentration and shortening BMBT.³⁵ Furthermore, none of the 9 dogs that received cryoprecipitate experienced a transfusion reaction, in contrast to 6 out of 9 dogs that were treated with FFP. The results of this study suggest that cryoprecipitate is a safer product for use in patients with VWD. For prophylactic use, cryoprecipitate can be administered just before surgery and then as required (every 4 to 12 hours) if indicated.¹⁰

FFP is a transfusion product that contains labile and nonlabile coagulation factors, including VWF.³⁶ It can be used prophylactically prior to surgery and for the treatment of hemorrhage in patients with VWD. It is associated with a higher incidence of nonhemolytic reactions than cryoprecipitate¹⁰ and should therefore be administered via slow infusion. FFP is often more expensive than cryoprecipitate and is sometimes

TABLE 5 Local and Systemic Hemorrhage Control Methods

LOCAL	SYSTEMIC
● Tissue glue ● Sutures ● Diathermy ● Dressings/bandages ● Direct pressure ● Cryotherapy	● Blood products ● Cryoprecipitate ● Fresh frozen plasma ● Desmopressin acetate ● Antifibrinolytic drugs ● Tranexamic acid ● Aminocaproic acid

reserved for when cryoprecipitate is unavailable. Prophylactic use of FFP is similar to cryoprecipitate, and it may be administered every 8 to 12 hours if required.¹⁰

Desmopressin acetate (DA) is a synthetic formula of vasopressin and is frequently used to control hemorrhage in patients with VWD. This drug stimulates the release of endogenous VWF and subsequent elevation of VWF:Ag and FVIII concentrations.³⁷ This effect is particularly useful in suspected or mild type 1 cases when the patient has functional but deficient VWF.¹⁰ DA is less effective in type 2 cases due to the qualitative defect of VWF, and it is ineffective in type 3 VWD due to the lack of synthesis and absence of VWF.¹⁰ In these cases, alternative methods of hemostasis should be implemented, such as the administration of a blood product. DA can be administered via the intravenous, subcutaneous, oral, or intranasal route approximately 30 minutes before surgery to improve hemostasis¹⁰; however, individual response to treatment can vary, and it should not be relied on to control surgical hemorrhage.

Client Communication and Education

An in-depth discussion about clinical signs and risk management planning should be conducted with the client, particularly in newly diagnosed cases. Simple techniques to reduce the risk of injury and subsequent hemorrhage, such as installing nonslip flooring in the home, should be advised, especially for older dogs. Keeping the dog's nails at an appropriate length via safe exercise reduces the need for regular clipping. Physical disputes with other pets in the household should be prevented. Other strategies to prevent trauma include avoiding optional surgeries. Less invasive surgical techniques can be also considered, such as laparoscopic spay surgery rather than via abdominal laparotomy.

A thorough risk–benefit analysis involving the client and veterinary professionals should be conducted before procedures such as surgical neutering, especially if the risk of hemorrhage is significant.

Clients can be advised to purchase a collar tag for their dog to wear to inform others of the diagnosis (**FIGURE 2**). This information could also be included when registering or updating microchip details in case the dog becomes lost and requires care or in the event of rehoming.



FIGURE 2. A collar tag that informs readers about the dog's bleeding disorder.

As VWD is an inherited disease, clients should be educated and encouraged to reduce prevalence by promoting genetic testing and subsequent removal of positive dogs from a breeding program (see **TABLE 4** for breeding guidance). However, this may lead to depletion of breeding stock, particularly in rare breeds. A positive bitch should not be bred due to the risk of hemorrhage during parturition and/or cesarean section if required.

SUMMARY

VWD is the most common inherited hemostatic disorder in domestic dogs. It is incurable, but with appropriate care, many dogs can live a normal life. Palliation aims to prevent injury and hemorrhage, and the veterinary nurse is at the forefront of providing education to clients, as well as providing diagnostic support, aiding in treatment, and preventing disease. **TVN**

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1. Which von Willebrand disease (VWD) variant is characterized by a quantitative deficiency and qualitative defect of von Willebrand factor (VWF)?
 - a. Type 1
 - b. Type 2
 - c. Type 3
 - d. None of the above
2. Which breed of dog is most affected by the type 3 VWD variant?
 - a. Shetland sheepdog
 - b. German shepherd
 - c. Doberman pinscher
 - d. German shorthaired pointer
3. What is the plasma VWF antigen (VWF:Ag) concentration range in dogs with the type 3 VWD variant?
 - a. 0% to 49%
 - b. 50% to 69%
 - c. 70% to 180%
 - d. 180% to 200%
4. What type of blood tube should be used to collect blood for VWF:Ag concentration analysis?
 - a. Plain
 - b. EDTA
 - c. Sodium citrate
 - d. Lithium heparin
5. Which drug may be prescribed by a veterinarian to improve hemostasis during surgery in a patient with VWD?
 - a. Heparin
 - b. Doxapram
 - c. Warfarin
 - d. Desmopressin



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