Managing patients with von willebrand disease in dentistry: a case report

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Abstracts

***Aim*: Von Willebrand disease (VWD) is the most common inherited coagulopathy, characterized by a quantitative or qualitative deficiency of Von Willebrand factor (VWF), essential for primary and secondary hemostasis. Patients with VWD have an increased risk of bleeding, which can complicate dental procedures. This case report aims to provide a comprehensive overview of the dental management of patients with VWD, outlining practical strategies and recommendations to address the specific clinical challenges of this condition in the field of oral surgery.**
***Case report*: A 42-year-old male patient with VWD type 1 presented to our Department for extraction of element 3.8 for dysodontiasis.**
***Results*: The surgical extraction was performed successfully. No intraoperative or postoperative issues were encountered.**
Conclusion: Effective communication and collaboration among the dentist, hematologist, and patient are essential to optimize prognosis by reducing the risks of intra- and postoperative complications.

Keywords: Von Willebrand disease, Coagulation factor VIII, FVIII/VWF concentrate, Oral Surgery

Introduction

Von Willebrand disease (VWD) is the most common hereditary haemorrhagic disorder caused by a genetic defect that results in a quantitative or qualitative anomaly in the synthesis of von Willebrand Factor (vWF), located on chromosome 12 (1-3).
The vWF is a glycoprotein with a high molecular weight (260 kDa), synthesized in the blood vessels by endothelial cells and megakaryocytes. Once produced, vWF is accumulated in endothelial cells in specialized storage granules called Weibel-Palade bodies, or it is immediately released into the plasma, where it recognizes and binds to Factor VIII to form the complex Factor VIII/Von Willebrand Factor (2, 3).
The vWF supports the coagulation cascade by binding to and stabilizing factor VIII, thereby increasing its half-life. The vWF protects the action of the active protein C that degrades the circulating factor VIII in the plasma. The vWF transports factor VIII to the injury sites, initiating the coagulation process by acting as a molecular bridge between platelets and the sites of endothelial damage, and protecting factor VIII from the attack of any inhibitors (2-4).
Clinical manifestations of VWD include mucocutaneous bleeding, in particular

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nosebleeds, gingivitis, menometrorrhagia in women of childbearing age, gastrointestinal bleeding, spontaneous hematomas or as a result of minor trauma, prolonged bleeding as a result of minor and major surgeries, and post-dental interventions. More rare are the manifestations of joint bleeding (2).

The International Society of Hemostasis and Thrombosis (SIET) distinguishes three subtypes of VWD: type 1, type 2, and type 3. Type 1, which represents 80% of cases, is characterized by a partial quantitative deficit of vWF, which is qualitatively normal. Type 2, described in 15% of cases, is characterized by qualitative deficits in the synthesis and function of vWF. Sadler categorizes these quality defects into four subcategories: Type 2A, Type 2B, Type 2M, and Type 2N. The reduction of multimer formation characterizes type 2A because Vvwf cannot bind to platelets; type 2B is characterized by a mutation “gain of function” that determines a greater affinity of glycoprotein Ib (gp1b) so there is a greater adhesion-platelet aggregation, often associated with a false thrombocytopenia; type 2M for a reduced platelet binding of vVF and a reduction in the same level of vVW; type 2N is characterized by a decreased binding of vVF to Factor VIII and a significant reduction in levels of Factor VIII. Type 3 represents the rarest and most severe form, characterized by a total quantitative deficiency of vWF. In these patients, vWF antigen concentrations are lower than 5 IU/dL and are often undetectable; similarly, the concentration of factor VIII is reduced (2-4).

Diagnosis of VWD is complex and it requires a history of hemorrhagic diathesis, more or less present, a familiarity with bleeding, as well as laboratory tests confirming the diagnosis.

The Standardization and Scientific Committee of the International Society on Thrombosis and Hemostasis has proposed a standardized questionnaire on the extent of bleeding, accompanied by a well-defined interpretation table, which allows the calculation of a final score, defined as the Bleeding Score (BS).

After the clinical suspicion has been identified, VWD is diagnosed by low levels of vWF antigen (vWF: Ag) and ristocetin cofactor activity (VWF: RCo), as well as sometimes low levels of factor VIII (5-7).

The main therapeutic approaches involve the use of desmopressin (DDAVP) and plasma-derived concentrates of vWF-Factor VIII (pdFVIII/VWF). DDAVP, a vasopressin analogue that promotes the release of vWF into the plasma by endothelial cells, is administered in patients with type 1 VWD who exhibit a positive response to the specific test, and have basic levels of factor VIII and vWF, typically at 10 U/dL. In the cases of type 2 and type 3 VWD, DDAVP is ineffective, and for this reason, the use of plasma-derived concentrates is necessary. More specifically, in type 2B, DDAVP is responsible for the occurrence or transient aggravation of thrombocytopenia. Plasma-derived concentrates of vWF and factor VIII can be used in conjunction with adjuvant drugs, such as tranexamic acid, to reduce the risk of hemorrhage (4-9).

While there are no definitive guidelines in the literature to define a precise therapeutic plan for approaching this type of patient at the level of oral surgery, the hematological protocol allows for the choice of a highly personalized therapeutic plan tailored to the risk of the procedure to be implemented (9, 10).

This case report aims to emphasize the importance of close collaboration between dentists and hematologists in optimally managing outpatient oral surgery in patients with VWD, thereby avoiding the need for hospitalization. Another aim is to identify an adequate surgical protocol that allows for the management of local hemostasis with technical measures to minimize intraoperative trauma and facilitate the postoperative course, reducing potential bleeding risks.

Case report

A 42-year-old male patient with VWD type 1 is reported to the authors. He has been followed at the haematological clinic since November 2011 for an extension of activated partial thromboplastin time (aPTT) (aPTT ratio 1.39), (v.n. < 1.16). The patient had no history of significant hemorrhagic diathesis; the medical history reported only frequent episodes of epistaxis from the right nostril and denied any previous surgery. The patient had an ISTH BAT of 1. vWF: Ag 10% (v.n. 50-126), vWF: RCo 6.25% (v.n. 50-150), FVIII 20.9% (v.n. 58-130), and RIPA 2.5 (v.n. 0.7-1.2) and vWF: RCo/vWF: Ag 0.625. It was then diagnosed with VWD type 1.

Initially, the patient was tested for DDAVP to assess the effectiveness of this drug in inducing the release of endothelial vWF. However, after the administration of the drug the subject developed an episode of lipothymia with the appearance of a knob at the site of inoculum. The DDAVP has been replaced with a pdFVIII/VWF concentrate.

In 2015, following episodes of epistaxis, the patient was treated with the appropriate concentrate, and varicose veins were cauterized.

In March 2025, the patient is referred to the Department of Odontostomatological and Maxillofacial Sciences at Sapienza University of Rome for a visit to the Odontostomatological Clinic.

From the objective intraoral examination and the X-ray examination (Panoramic X-ray) (Figure 1), the extraction of element 3.8 for dysodontiasis.

During the visit, routine haematological examinations were performed (blood count, PT, PTT, INR, glycemia, azotemia, GOT, and GPT), as well as a cardiological examination with an ECG.

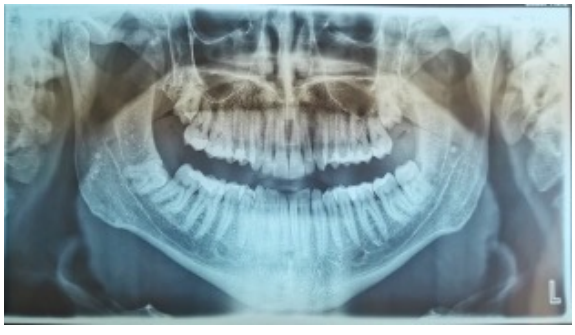

Figure 1. Panoramic-Xray.

Table 1 Hematological protocol

60 IU/Kg 6000 U e.v. total dose hour 0 (about half an hour before surgery)
30 U/Kg 3000 U e.v. total dose hour 12
30 U/Kg 4000 U e.v. total dose hour 24
30 U/Kg 3000 U e.v. total dose hour 48
30 U/Kg 3000 U e.v. total dose hour 72
Continue with 2000 U/day e.v. until the 6th day after surgery.

Given the haematological pathology, the hematologist was consulted. The hematologist has released clearance for extraction and pre- and postoperative therapy to reduce the risk of bleeding. The haematologist then prescribed the infusion of VWF/factor VIII concentrate (Fandhi, Grifols Italia s.p. A.), administered based on the size of the procedure and the patient's weight (95 kg) (Table 1).

Before starting the surgical procedure, the dose of Fandhi was administered.

Surgery was performed under nerve block at the right lower alveolar nerve (3% Mepivacaine) and local anesthesia (2% Mepivacaine with 1:100,000 epinephrine).

During the surgical phase, after an accurate periotomy, the dislocation with an elevator and the avulsion of element 3.8 were performed, followed by revision and washing of the residual alveolar cavity with physiological solution.

In the post-extractive phase, the fibrin sponge was inserted into the remaining alveolar cavity, and an X-suture was performed using 3/0 Vicryl absorbable thread. An antihemorrhagic prevention medication was then applied with a pressure gauze soaked in tranexamic acid (500mg/5ml solution for injection and oral) at the surgical site.

After the haemostasis control, the patient was discharged by providing a prescription for antibiotic and antalgic therapy and post-operative instructions with: Amoxicillin and Clavulanic Acid 1 g (CPR), twice daily for 6 days; Paracetamol 1000 mg (CPR), if necessary not exceeding the limit dose of 3 g per day; Chlorhexidine digluconate mouthwash (0.2% solution) for plaque control twice a day after oral hygiene at home from 24 hours after surgery for 7 days; Compressions with gauze soaked in Tranexamic Acid for 3 days.

The patient continued the replacement therapy prescribed by the hematologist, as outlined in Table 1, at the hematological department.

Back to our observation, after a week for the post-operative check-up, the patient did not report any hemorrhagic symptoms in the days following the extraction, which might have brought forward the date of such appointment.

The post-extractive site showed a good recovery, and to avoid irritation of the mucous membranes, it was decided not to proceed with the removal of the suture, waiting for it to undergo physiological degradation.

Discussion

VWD is the most common inherited coagulation disorder; in this type of patient, any trauma or surgery (including oral surgery) exposes subjects to a high risk of intra- and post-operative bleeding (1-13).

In the literature, the incidence of post-operative bleeding in subjects with proper hemostasis following invasive dental interventions is between 0.2-3.3%, and is estimated to increase between 8.6-32.1% in cases of subjects with hemorrhagic pathologies (8-9). The literature shows that patients with hereditary coagulation pathologies treated with pdFVIII/VWF concentrates in prophylaxis have the same risk of bleeding as patients with physiological haemostasis (5, 8, 10, 15-16). During oral surgery, to minimize the risk of intraoperative bleeding in patients with VWD, it is essential to have the intervention of a hematologist for the correction of the hemostatic defect, which involves increasing the circulating concentration of vWF and, consequently, factor VIII (10).

In dentistry, it is essential to carry out such practices using, as described in the presentation of the case, technical measures to minimize any intraoperative trauma and a correct protocol for controlling local hemostasis, to manage local hemorrhagic emergencies with gauze, local hemostatics, and sutures (10, 15-18). The surgeon will then need to make preoperative considerations regarding the surgical site, the dental pathology, and the ability to control any local bleeding (7, 8). An important preoperative consideration for the surgeon is the choice of surgical instruments to be used. The use of lasers in oral surgery is a valuable tool for patients with von Willebrand disease, offering improved hemostasis control, a rapid recovery, and minimal discomfort. Furthermore, the laser allows the surgeon to work with great precision, preserving surrounding healthy tissues and minimizing collateral damage (19). To prevent medical errors, it's important to remember that oral lesions can present with unusual characteristics requiring a careful differential diagnosis phase to establish a correct treatment plan consistent with the patient's medical history, thereby promoting a favorable prognosis (20).

Local anesthesia by infiltration of the surgical site with local anesthetic and adrenaline reduces the risk of bleeding (8). Among the intraoperative preventive measures that are necessary to avoid, when not strictly required, are surgical procedures involving the

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execution of flaps, which limit further damage to the affected area (15). In the case presented, no surgical flap was carried out to reduce bleeding and soft tissue trauma, preferring an avulsive approach with the sole action of the lever. If a surgical flap is necessary, it must have well-defined margins and a suitable shape for the specific surgery. The flap should then be carefully detached, avoiding any tearing, to ensure an adequate suture in the final phase of the procedure that favors a closure for first intention without excessive tension (8-10). Among local hemostatic agents, chitosan can also be applied to the surface of the alveolus that covers the inside of the suture flaps (5, 10, 25).

The local hemostasis protocol also allows for the use of local hemostatic materials, such as fibrin sponges or fibrin glue, and reabsorbable sutures to ensure a good seal of the wound and a smooth post-surgical recovery, thereby minimizing the risk of new trauma during suture removal (9). At the end of the procedure, it is advisable to use compressive dressings on the affected area, using gauze soaked in Tranexamic Acid to inhibit fibrinolysis by salivary enzymes (1, 7, 26).

Regardless of the techniques used, post-operative bleeding can still occur, especially in the case of early fibrinolysis and hyperfibrinolysis, responsible for late postoperative bleeding; the simplest method to control this complication is to apply a pressure gauze to the wound area, which must then be soaked in Tranexamic Acid (17, 25-27).

In the post-operative course the Tranexamic Acid is administered for 3 days with a simple protocol that allows the patient to regularly monitor the surgical area in the following days, making a compression of 10 minutes with gauze soaked in Tranexamic Acid every hour in the first day, every 2 hours the second and every 3 the third. It was also emphasized that this compression was necessary as a first approach in the event of late bleeding (5, 8, 17-23, 27).

In oral surgery, the patient must carefully follow the appropriate hygiene rules (not washing the teeth for 24 hours after surgery to stabilize the clot and then continue with the use of mouthwash adjuvant brushing to monitor plaque control), recommendations on soft diet and use antibiotic therapy (to reduce the risk of infection that can cause secondary bleeding) and analgesic therapy with paracetamol when needed, avoiding any medication that may interfere with hemostasis (aspirin and FANS are therefore highly discouraged) (2).

In the present clinical case, the practice of outpatient oral surgery is secondary to the close collaboration between the dentist and the haemophilia center. This collaboration enables the preservation of the patient's health by addressing both systemic pathology and the specific oral cavity issues, thereby minimizing the risk of intra- and post-operative bleeding complications through the use of specialized surgical techniques and local hemostasis (8-12, 25-27-29-30).

Furthermore, the potential preparation of the patient to self-administer specific intravenous drugs should not be underestimated, as it promotes the reduction of hospital complication risks and the healthcare costs associated with inpatient care (12).

Conclusions

To date, the pre- and postoperative care adopted in patients with congenital hemorrhagic diseases is a topic still little debated in the literature; there are currently no standard protocols to refer to.

What indeed emerges from this case is the importance of close collaboration between the hematologist and the dentist, which guarantees the adequate framing of the clinical case and the consideration of all factors that can influence the success of the surgery.

In addition, it is also important to note that, with all the precautions mentioned above, VWD patients, if correctly identified and treated, present the same risk of bleeding as an individual with a physiological hemostasis.

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