

CASE REPORT

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Surgical intervention with cardiopulmonary bypass in a patient with von Willebrand disease

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Abstract

Background This article presents a clinical case of surgical intervention with cardiopulmonary bypass (CPB) in a patient with von Willebrand disease (VWD). VWD is the most common inherited bleeding disorder, significantly complicating surgical procedures due to impaired hemostasis. Surgical treatment of patients with VWD, particularly in the context of cardiovascular surgery involving CPB, requires an individualized approach. This includes the optimization of von Willebrand factor (vWF) and factor VIII (FVIII) levels, which are critical to preventing perioperative bleeding. The article describes the perioperative anesthetic management of the patient, emphasizing the importance of hemostasis monitoring, an individualized approach, and personalized dosing of replacement therapy to ensure effective treatment and minimize the risk of bleeding and thromboembolic complications.

Case presentation A 36-year-old male (181 cm, 92 kg) with type 2 von Willebrand disease and a history of childhood cardiac surgery (atrial septal defect repair, right ventricular outflow tract reconstruction) was admitted for elective aortic valve replacement with a bioprosthesis due to severe aortic regurgitation. Echocardiography revealed residual ventricular septal defect shunting, aortic root dilation, moderate mitral regurgitation, and cusp prolapse. The patient had a significant bleeding history and laboratory evidence of low vWF antigen/activity and FVIII deficiency. Perioperative management included ultrasound-guided vascular access, balanced general anesthesia, moderate hypothermic cardiopulmonary bypass, antifibrinolytic therapy, and tailored hemostatic replacement with vWF concentrate, FVIII, cryoprecipitate, platelets, and plasma. The patient was extubated 6 h after surgery, required minimal transfusion support, and recovered without hemorrhagic or thromboembolic complications. He was discharged on postoperative day 10 in good condition.

Conclusions Proper management of the perioperative period is identified as a complex challenge for anesthesiologists, requiring a clear understanding of therapeutic mechanisms and their impact on coagulation.

Keywords Von willebrand disease, Anesthetic management, Hemostasis monitoring

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Background

In 1926, Finnish physician Erik Adolf von Willebrand described the symptoms of a previously unknown bleeding disorder in a large family from the Åland Islands off the coast of Finland [1]. The proband was a five-year-old girl who experienced recurrent severe mucosal bleeding. Laboratory tests revealed normal clotting time and clot retraction, but a significantly prolonged bleeding time [1]. Unlike hemophilia, this newly described disorder affected both males and females, who could die from spontaneous bleeding episodes without any apparent trauma.

The condition became known as von Willebrand disease (VWD) and is now recognized as the most common inherited bleeding disorder, affecting approximately 0.6–1.3% of the global population [2, 3], or about 1 in 1,000 individuals [4]. However, clinically significant forms of the disease requiring specialized treatment are found in approximately 125 cases per million people [4].

VWD is inherited in an autosomal pattern, meaning that males and females are affected equally. However, women more frequently exhibit symptomatic bleeding, which is often exacerbated by hemostatic challenges related to menstruation and childbirth [5]. This hemostatic disorder poses a serious challenge both for the patient and the surgical team during operative procedures. The risk associated with surgical interventions depends not only on the type of surgery but also on the type of VWD, baseline plasma levels of von Willebrand factor (vWF), bleeding history, response to previous treatment, and other contributing factors [6].

To date, there is no consensus among clinical guidelines regarding the optimal dosage or target levels of vWF and factor VIII (FVIII) to ensure safe surgery while minimizing the risk of thromboembolic complications in the postoperative period [7].

It is well known that during cardiopulmonary bypass (CPB), blood contact with the extracorporeal surfaces of the oxygenator leads to changes in coagulation integrity and increases the risk of bleeding along with potential perioperative thromboembolic complications – an especially critical concern in cardiovascular surgeries involving patients with preexisting coagulation disorders [8]. Studies by Doyle A.J. and colleagues report that the activation of the coagulation system is initiated by the interaction between blood and synthetic surfaces. The initial deposition of fibrinogen and subsequent activation of clotting factors and complement pathways promote adhesion of platelets and leukocytes to the oxygenator surfaces, enhancing thrombin generation. Evaluation of endogenous thrombin generation potential (ETP) is therefore clinically helpful for predicting both bleeding and thrombotic complications in critically ill patients [9, 10]. Furthermore, preexisting primary hemostatic defects

are worsened by platelet dysfunction and loss of essential adhesive molecules [11].

It is important to note that only a very limited number of surgical cases involving patients with VWD undergoing cardiopulmonary bypass have been described in the literature.

In this article, we discuss the anesthetic management of a patient with VWD who underwent elective aortic valve replacement with a bioprosthesis. The complexity of the surgical procedure and the patient's individual coagulopathy posed several critical challenges in determining the optimal management strategy and therapeutic hemostasis, which are analyzed in this report.

Case presentation

A 36-year-old male patient (181 cm, 92 kg) with confirmed type 2 von Willebrand disease (VWD) and a history of previous cardiac surgery in childhood (perimembranous atrial septal defect repair and right ventricular outflow tract reconstruction) was referred to our institution for elective aortic valve replacement with a bioprosthesis due to severe aortic regurgitation.

Echocardiography revealed residual shunting through a ventricular septal defect with a pressure gradient of 69 mmHg between the left and right ventricles; severe aortic regurgitation; tricuspid aortic valve: prolapse of the right coronary cusp; incomplete separation of the left and non-coronary cusps; aortic root dilation up to 4.2 cm; and moderate mitral regurgitation; left ventricular ejection fraction was 58%.

The patient had a significant bleeding history, including recurrent epistaxis, prolonged bleeding after dental extraction, and a persistent tendency to bruise. Preoperative hemostasis assessment showed:

- Platelet count (PLT): $150 \times 10^3/\mu\text{L}$.
- Activated partial thromboplastin time (aPTT): 45 s.
- International normalized ratio (INR): 1.05.
- Activated clotting time (ACT): 234 s.
- Viscoelastic parameters (ClotPro): ex-test – 48 s; in-test – 166 s; hi-test – 87 s; fib-test – 42 s, indicating fibrinogen and FVIII deficiency.
- Additional analysis:
 - von Willebrand factor antigen (vWF: Ag): 28 IU/dL.
 - vWF ristocetin cofactor activity (vWF: RCoF): 14 IU/dL.
 - FVIII activity (FVIII: C): 34 IU/dL.

The decision to implant a biological prosthesis (Carpentier-Edwards PERIMOUNT, 25 mm) was indeed influenced by the patient's bleeding diathesis (VWD) to avoid

the need for long-term anticoagulation that would be required with a mechanical valve.

To minimize the risk of vascular injury and hematoma formation, peripheral, central venous, and arterial accesses were established under ultrasound guidance.

Anesthesia induction included IV administration of propofol (1.5 mg/kg) and fentanyl (2 µg/kg). Neuromuscular blockade was achieved with rocuronium bromide (0.8 mg/kg), followed by intubation using a thermoplastic endotracheal tube. Anesthesia was maintained with sevoflurane inhalation via a semi-closed system, titrated according to the patient's age-adjusted MAC using the formula: $MAC_{awake} = 0.34 \times MAC_{table} \times 2$, with BIS monitoring maintained between 40% and 50%.

Intraoperative analgesia was maintained with fentanyl; the total dose used during the procedure was 8.73 µg/kg. Mechanical ventilation was performed using a 50% air-oxygen mixture in normoventilation mode (flow rate: 2 L/min), with blood gas monitoring. Cardiopulmonary bypass (CPB) was conducted under moderate hypothermia (central temperature: +34.4 °C). CPB flow was maintained at 2.5 L/min/m². Myocardial protection was provided using Bretschneider's cardioplegic solution.

An anti-fibrinolytic agent – tranexamic acid (TXA) – was administered intravenously throughout the procedure at a total dose of 25 mg/kg. Heparin (300 IU/kg) was administered immediately after sternotomy. Blood from the surgical field was collected via suction into the CPB circuit for reinfusion. The ACT after heparinization reached 856 s. Aortic valve replacement with a 25 mm Carpentier-Edwards PERIMOUNT biological prosthesis was performed via median sternotomy. Arterial cannulation was achieved using a 22 Fr aortic cannula in the ascending aorta, and venous cannulation was performed with a 34/46 Fr dual-stage cannula positioned in the right atrium. Aortic cross-clamp time was 54 min, and total CPB duration was 71 min. At the time of CPB weaning, the patient received inotropic support with dobutamine at 5 µg/kg/min.

Following separation from CPB, protamine (1 mg per 100 IU of administered heparin) was given. Hemostatic therapy included: Fresh frozen plasma: 10 mL/kg; Cryoprecipitate: 2 units; Platelet concentrate: 300 mL; Red blood cell (RBC) concentrate: 250 mL; vWF concentrate: 1500 IU; Human FVIII: 3000 IU. To further reduce active bleeding, two doses of Octaplex (1000 IU total) were administered. Post-hemostatic therapy, ACT was 112 s. A total of 600 mL of blood was collected in the cell saver before and after the use of CPB suction, and 180 mL of processed autologous red blood cells were returned to the patient. Upon ICU admission, hemoglobin was 104 g/L.

The patient was extubated 6 h later. In the first 24 h postoperatively, 340 mL of serosanguinous fluid was

collected via chest drains. On the first postoperative day, one additional unit of RBCs was transfused to achieve a target hematocrit above 35%. At 24 h post-op, aPTT had normalized (33 s) and remained stable throughout the postoperative course. Routine aPTT monitoring was used as a surrogate marker of hemostatic therapy efficacy, reflecting intrinsic pathway activity including FVIII levels. The patient continued receiving vWF concentrate (500 IU) and FVIII (1000 IU) twice daily until the removal of the last intravascular catheter. We administered 1 g tranexamic acid every 8 h intravenously for 72 h postoperatively to minimize the risk of bleeding. No anticoagulant or antithrombotic therapy was administered postoperatively due to the presence of a biological aortic valve prosthesis. Instead, mechanical prophylaxis with compression stockings and early mobilization were implemented to reduce the risk of thromboembolic complications. The total postoperative volume of serosanguineous fluid collected via chest drains was 560 mL.

The patient was discharged in satisfactory condition on postoperative day 10 with no clinical signs of hemorrhagic or thromboembolic complications.

Discussion and conclusions

To date, based on a limited number of case reports, there is no established universal protocol for managing patients with VWD undergoing cardiac surgery [12, 13]. Hemostatic derangements in VWD and acquired von Willebrand syndrome (AvWS) – often exacerbated by high-shear flow, turbulent flow through valves, and contact activation during cardiopulmonary bypass (CPB) – can lead to substantial bleeding risk while complicating conventional coagulation monitoring and management [14]. In this context, structural cardiac lesions that generate high-flow states may further aggravate hemostatic disturbances. In our patient, the residual ventricular septal defect (VSD) was small and hemodynamically insignificant, and therefore left unrepaired in line with standard surgical practice, as surgical closure of minor defects is generally not indicated due to the low risk of hemodynamic compromise and the potential risks associated with additional intervention.

The patient's bleeding risk was confirmed using the ISTH Bleeding Assessment Tool (ISTH-BAT) (8 point) and the shortened MCMDM-1VWD mucocutaneous bleeding score (5 point), indicating a high risk of perioperative hemorrhage. Given these factors, standard laboratory coagulation assays alone were deemed insufficient for real-time guidance. Therefore, we implemented a goal-directed, individualized strategy combining patient blood management following the EACTS/EACTAIC/EBCP blood product management guidelines, autologous blood salvage, and point-of-care monitoring (CLOT-PRO) [15].

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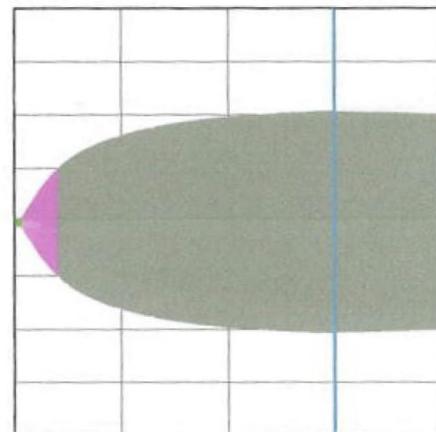
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53

EX-test

21.11.2024 13:06:38

CT	48s	▶ 38-65
A5	25mm	▼ 39-58
A10	33mm	▼ 47-64
A20	40mm	▼ 52-67
MCF	42mm	▼ 53-68
CFT	195s	▲ 42-93
ML	4%	▶ 2-12



Канал: 1

t: c: CP44417CC8708



Fig. 1 Viscoelastic parameters from clot-pro analysis prior to heparin administration

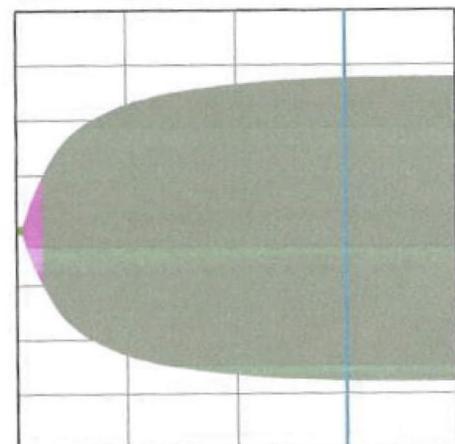
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AP44438AA88C5

AP-test

21.11.2024 13:21:59

CT	35s	▼ 44-76
A5	37mm	▶ 36-59
A10	47mm	▶ 46-65
A20	53mm	▶ 51-67
MCF	56mm	▶ 51-67
CFT	107s	▶ 46-118



Канал: 2

t: c: CP44417CC8708



Fig. 2 AP-test parameters according to Clot-Pro data

Although the use of TEG for monitoring patients with VWD during surgery has been previously reported (Tuman et al., 1987; Pivalizza, 2003; Stolla M., 2018), to our knowledge, this is the first report describing a patient with VWD undergoing redo cardiac surgery with CLOT-PRO-guided coagulation management [16–18].

In our clinical case, analysis of viscoelastometry (CLOT-PRO) parameters revealed decreased Maximum Clot Firmness (MCF) and amplitude at 5 (A5), 10 (A10), and 20 (A20) minutes in the ex-test, indicating impaired fibrin clot formation characteristic of vWF deficiency (Fig. 1). Prolonged Clot Formation Time (CFT) confirmed platelet adhesion defects and fibrin deficiency.

Meanwhile, a normal Clotting Time (CT) suggested relatively normal functioning of the extrinsic coagulation pathway.

Regarding the AP-test (activated intrinsic coagulation pathway) (Fig. 2), relatively normal values of MCF, A5, A10, and A20 indicated that the intrinsic pathway functioned better than the extrinsic pathway, while the shortened CT (35 s) could be a result of compensatory activation of the coagulation system or the influence of other external factors.

Interestingly, the FIB-test (fibrinogen level assessment) showed critically low values of MCF, A5, A10, and A20, indicating a significant fibrinogen deficiency or

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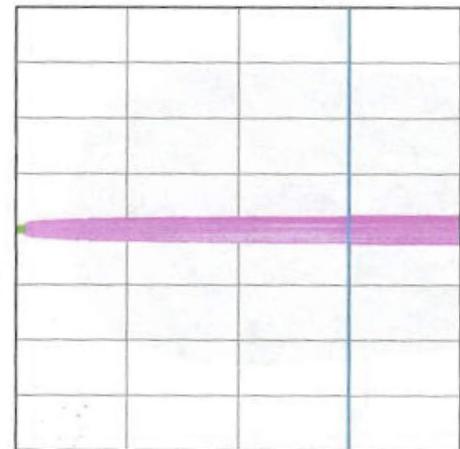
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FIB-test

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CT	42s	▼ 55-87
A5	4mm	▼ 6-21
A10	4mm	▼ 7-23
A20	5mm	▼ 8-25
MCF	5mm	▼ 9-27



Канал: 3

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Fig. 3 FIB-test parameters according to Clot-Pro data

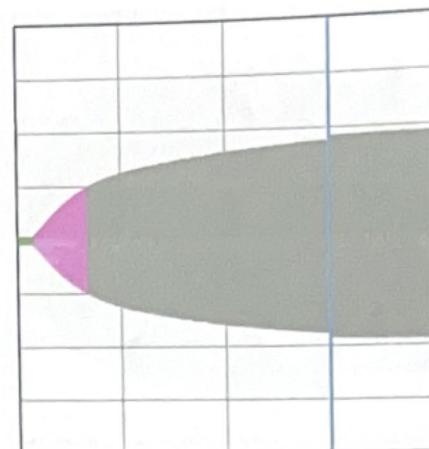
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EC44375AA8AC7

ECA-test

21.11.2024 13:15:42

CT	87s	► 68-100
A5	19mm	▼ 45-58
A10	26mm	▼ 54-66
A20	33mm	▼ 58-70
MCF	38mm	▼ 61-72
CFT	320s	▲ 60-92



Канал: 4

t: c: CP44417CC8708



Fig. 4 ECA-test parameters according to Clot-Pro data

dysfunction, while the low CT could be a consequence of inadequate clot formation (Fig. 3).

ECA-test parameters (assessment of the extrinsic coagulation pathway) according to clot-pro data (Fig. 4). Prolonged CT and CFT values indicated possible von Willebrand factor deficiency and impaired clot formation, while low A5, A10, A20, and MCF values confirmed poor fibrin clot stability. These results support that the patient in this clinical case could have experienced deficiencies in FVIII and vWF.

TPA-test parameters (assessment of fibrinolysis) according to clot-pro data. In our case, the decreased

MCF indicated the formation of a weak and unstable clot, while the increased ML (Maximum Lysis) suggested enhanced fibrinolysis, indicating a tendency toward excessive clot breakdown and a bleeding risk due to possible factor consumption effect (Fig. 5). This finding supported the rationale for the additional administration of tranexamic acid.

Assessment of the intrinsic coagulation pathway using the IN-test revealed a prolonged CT, indicating impairment of the intrinsic pathway (factors VIII, IX), which is typical for von Willebrand disease (Fig. 6). However,

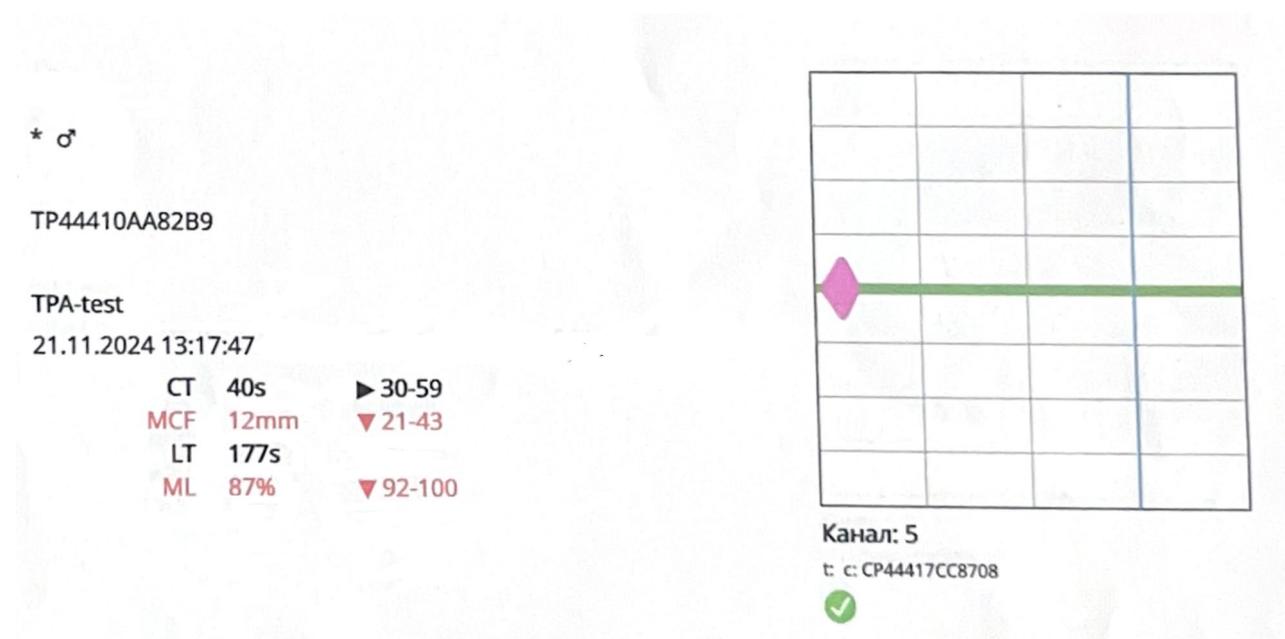


Fig. 5 TPA-test parameters according to Clot-Pro data

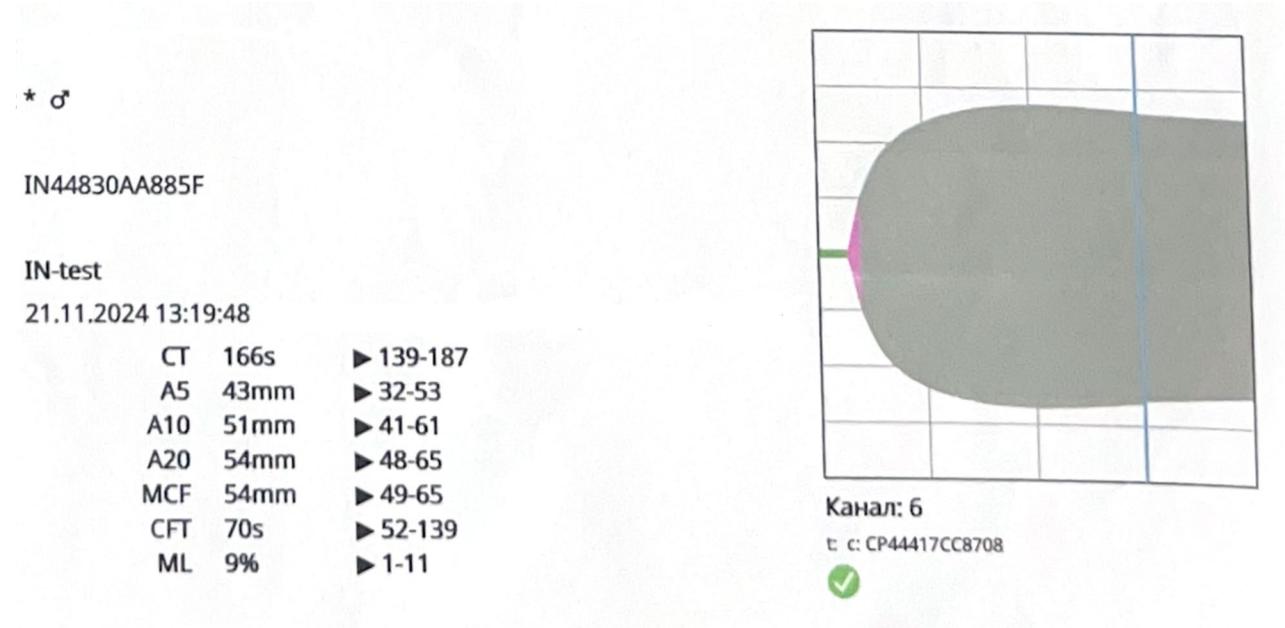


Fig. 6 IN-test parameters according to Clot-Pro data

other parameters remained within normal limits, suggesting relatively preserved fibrin clot formation.

Thus, the preoperative analysis demonstrated that the patient had a pronounced hemostatic disorder characteristic of von Willebrand disease: impaired clot formation (EX-test, FIB-test), fibrinogen deficiency or dysfunction, difficulties with platelet adhesion leading to prolonged CFT (ECA-test), and significant impairment in clot formation and stability (TPA-test).

Due to the high risk of surgical bleeding, a hemostatic therapy regimen was implemented, including antifibrinolytic agents, fresh frozen plasma, cryoprecipitate, platelet concentrates, and anti-hemophilic vWF complex. The patient received 50 IU/kg of recombinant vWF concentrate during surgery, achieving satisfactory levels of vWF: Ag – 135 IU/dL, vWF: RCo – 105 IU/dL, and FVIII: C – 130 IU/dL. An additional dose of 25 IU/kg vWF concentrate was administered 12 h postoperatively. Subsequently, plasma vWF levels were maintained above 50

IU/dL for 10 days. Regarding the use of desmopressin acetate (DDAVP), the patient had previously received desmopressin for the successful treatment of epistaxis. However, the correction of vWF levels with this drug was short-lived. Given the high bleeding risk associated with the planned cardiac surgery, desmopressin was considered unsuitable, and administration of exogenous vWF either plasma-derived or recombinant was chosen to ensure reliable perioperative hemostasis. According to the Universal Definition of Perioperative Bleeding (UDPB) by Dyke et al., the observed blood loss corresponds to mild-to-moderate bleeding, reflecting effective hemostatic management in this high-risk patient [19].

Perioperative management of patients with von Willebrand disease (VWD) presents a complex and unique challenge for anesthesiologists, requiring a thorough understanding of the mechanisms of action of available therapies and their effects on plasma levels of vWF and FVIII. It is essential to adopt a personalized approach for each patient, taking into account baseline vWF levels, bleeding phenotype, previous treatment responses, type of surgery, and comorbidities. Careful monitoring of coagulation and hemostatic changes during surgery, along with precise dosing of replacement therapy in the perioperative and postoperative periods, is crucial to maintain adequate plasma vWF levels and to initiate thromboprophylaxis when the risk of thromboembolism is significantly elevated.

Abbreviations

CPB	Cardiopulmonary bypass
VWD	von Willebrand disease
FVIII	Factor VIII
aPTT	activated partial thromboplastin time
ACT	Activated clotting time
PLT	Platelets
ClotPro	Viscoelastic hemostasis assay
vWF:Ag	von Willebrand factor antigen
vWF	von Willebrand factor
vWF:RCof	Ristocetin cofactor activity of vWF
FVIII:C	One-stage clotting factor VIII activity
TXA	Tranexamic acid
CPB machine	Cardiopulmonary bypass machine
ICU	Intensive care unit
TEG	Thromboelastography
PT	Prothrombin time
FFP	Fresh frozen plasma
MCF	Maximum clot firmness
A5	Amplitude at 5 min
A10	Amplitude at 10 min
A20	Amplitude at 20 min
CFT	Clot formation time
CT	Clotting time
AP	Test-activated pathway test
FIB	Test-fibrinogen level test
ECA	Test-external coagulation activation test
TPA	Test-tissue plasminogen activator test
ML	Maximum lysis
IN	Test-intrinsic pathway test

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Authors' contributions

OAL and JP conceived and designed the study. OAL and DOT provided anesthesiology support during the cardiac surgery. YuIM, DOT, NVK, and SRM collected and analyzed the patient's data. DOL assisted during surgery and contributed to the literature review. BMT performed the cardiac surgery and critically revised the manuscript for important intellectual content. All authors read and approved the final manuscript.

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Data availability

Data sharing is not applicable to this article as no datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

Ethical approval for publication of this case report was obtained from the Ethics Committee of Heart Institute of the Ministry of Health of Ukraine (approval No. 12, dated 10/08/2025).

Consent for publication

Written informed consent for publication of this case report was obtained from the patient.

Competing interests

The authors declare no competing interests.

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