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Non-ST-Elevation Myocardial Infarction in a Case of Von Willebrand Disease: a Case Report

Anees Abdul[1], Arpith Easo Samuel[1*], Fabith Moideen[1], Jayasree Nambiar[2]

1. Department of Emergency Medicine, Baby Memorial Hospital, Kerala, India.
2. Department of Cardiology, Baby Memorial Hospital, Kerala, India.

***Corresponding author:** Arpith Easo Samuel; Email: arpitheasosamuel@gmail.com

Abstract

Introduction: Studies have shown that patients with Von Willebrand disease (VWD) have decreased prevalence of thrombotic events like myocardial infarction (MI). Here we describe a case of VWD with acute non-ST-elevation MI with ongoing bleeding manifestations.

Case presentation: A 37-year-old female patient presented to the emergency department with a complaint of central chest pain since 7 days. She also had a history of hemoptysis since 8 days. Electrocardiogram (ECG) revealed ST-segment depression in leads I, aVL, II, III, aVF, and V_4–V_6 compatible with diagnosis of Non-ST-Elevation Myocardial Infarction (Non STEMI). She was started on nitroglycerine infusion, angiotensin II receptor blockers, and calcium channel blockers along with trimetazidine. Her chest pain and ECG changes settled after 2 days, and she was discharged in a stable condition.

Conclusion: There are limited studies available regarding the management of acute MI in VWD patients with acute bleeding manifestations. Further studies have to be carried out to determine successful ways of managing thrombotic events like MI in this subset of patients.

Key words: Case reports; Disease management; Myocardial infarction; von Willebrand Diseases

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INTRODUCTION

Von Willebrand disease (VWD) is characterized by a reduced level or activity of Von Willebrand factor (VWF), which results in bleeding manifestations. Studies have shown that patients with VWF deficiency have decreased prevalence of thrombotic events like myocardial infarction (MI) and stroke (1). Literature shows case reports of successful treatment of acute MI in VWD with anticoagulant, antiplatelet, and percutaneous coronary intervention (PCI) (2, 3). Previous case reports have also described the successful use of thrombolysis (4). Here we describe a case of VWD with acute non-ST-elevation MI with ongoing bleeding manifestations.

CASE PRESENTATION

A 37-year-old female patient presented to the emergency department with a complaint of central chest pain since 7 days, with the pain radiating to her left shoulder. She also had a history of hemoptysis since 8 days, at a frequency of 10–15 episodes per day. Also, she had one episode of vomiting 2 days prior to presentation with streaks of blood in the vomitus. There was no associated diaphoresis, palpitation, dyspnea, syncope, melena, or fever. She had a history of severe pulmonary valvular stenosis which was treated with balloon valvular dilatation during her childhood. She was detected to have VWF deficiency when she developed a hematoma during the post-operative period. She was not on any regular follow-up or treatment for long periods and did not have any significant bleeding manifestations which warranted medical attention. Her last menstrual period was 16 days back. Examination revealed a heart rate of 71 beats per minute, all peripheral pulses felt, with a blood pressure of 200/130 mmHg, respiratory rate of 18 breaths per minute, and blood oxygen saturation of 99% in room air. She was pale on general examination. Auscultation of the heart revealed an ejection murmur in the left 2nd parasternal area. Her respiratory, abdominal, and neurological examinations were within normal limits. Electrocardiogram (ECG) revealed ST-segment depression in leads I, aVL, II, III, aVF, and V_4–V_6 (figure 1).

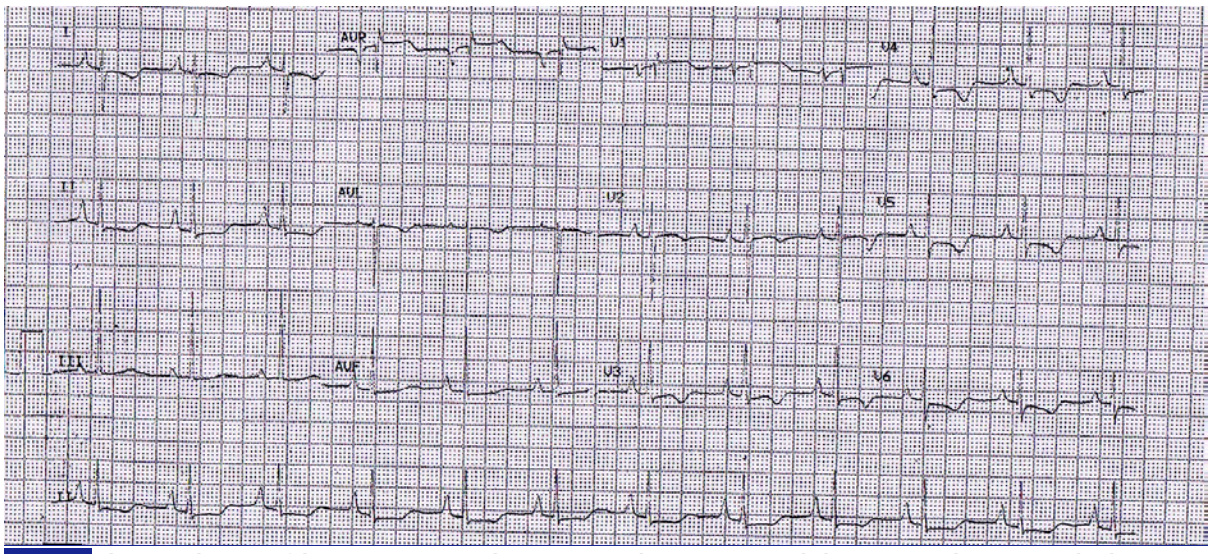

Figure 1: Electrocardiogram of the patients on arrival to emergency department, revealed ST-segment depression in leads I, aVL, II, III, aVF, and V_4–V_6.

Her troponin I level was reported as 0.09ng/ml. Echocardiography revealed severe pulmonary stenosis and right ventricular hypertrophy with no regional wall motion abnormality and good left ventricular systolic function. The patient was transferred to the cardiac intensive care unit. VWF deficiency and presence of bleeding manifestations during the past 8 days was considered as a contraindication for starting the patient on unfractionated heparin and antiplatelet agents. Since echocardiography showed no regional wall motion abnormality and taking into consideration the previous history of complications during balloon valvular dilatation, PCI was withheld. She was started on nitroglycerine infusion, angiotensin II receptor blockers, and calcium channel blockers along with trimetazidine. Her chest pain and ECG changes settled after 2 days (figure 2), and she was discharged in a stable condition.

Discussions

VWF plasma level naturally increases during the course of thrombotic events, including acute MI, and the magnitude of this increase is a predictor of possible adverse outcomes. Some believe that it is an important effector in the pathogenesis of MI, not in the general population but those with

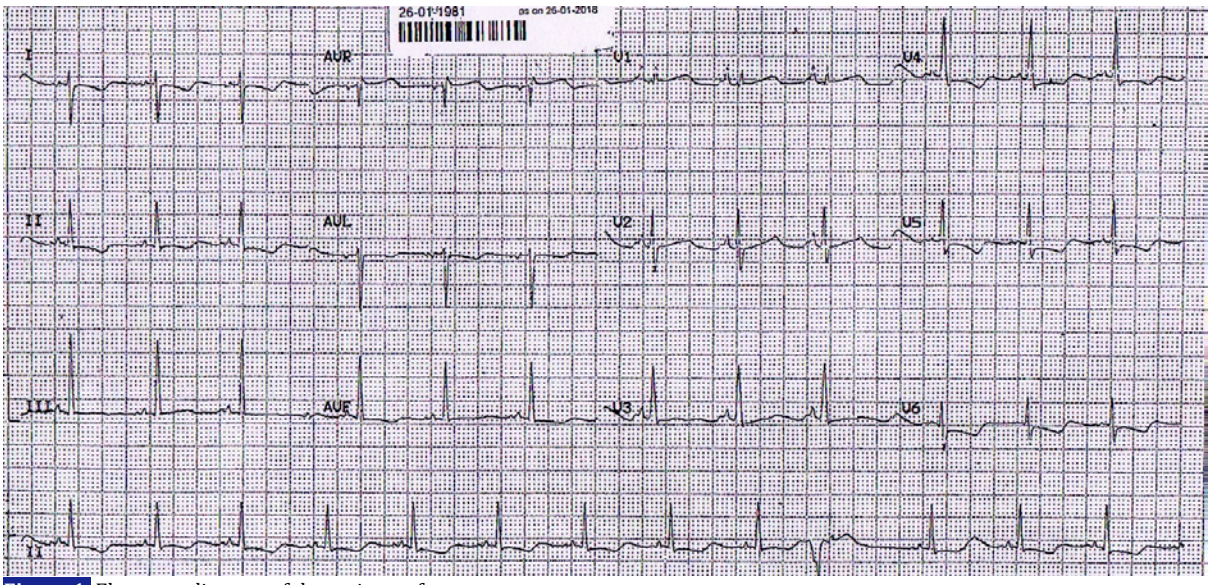

Figure 1: Electrocardiogram of the patients after treatment process

preexisting vascular disease (5-7). Therefore, it was assumed that the occurrence of MI in those with VWD should be rare. Due to its rarity, a well-defined protocol for the management of VWD patients with acute MI is not available. These patients are naturally considered to be vulnerable to bleeding events, so using the common antithrombotic, thrombolytic, and anticoagulant agents is challenging. Literature review shows that acute MI in patients with VWD has been successfully treated with thrombolysis, antiplatelet, and PCI. Hematoma at the site of catheterization is the only reported complication of PCI in such cases, and there is no evidence that these treatments led to acute bleeding manifestations (8). In this case, the presence of ongoing bleeding was a hindrance to the administration of antiplatelet and anticoagulant. The previous history of complication during balloon valvular dilatation prevented considering PCI. The absence of significant echocardiogram findings allowed us to treat this patient symptomatically without antiplatelet and anticoagulant agents.

Conclusions

There are limited studies available regarding the management of acute MI in VWD patients with acute bleeding manifestations. Further studies have to be carried out to determine successful ways of managing thrombotic events like MI in this subset of patients.

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Authors' contribution

A.E.S and A.A conceived and designed the report. A.A collected the data. AES organized and edited the data. A.A wrote the manuscript; A.E.S and A.A approved the final manuscript.

Conflicts of interest

The authors have no conflicts of interest to report. Any conflicts that the editor considers relevant to the content of the manuscript have been disclosed.

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References

1. Sanders Y, Eikenboom J, de Wee E, van der Bom J, Cnossen M, Degenaar-Dujardin M, et al. Reduced prevalence of arterial thrombosis in von Willebrand disease. J Thromb Haemost. 2013;11(5):845-54.
2. Arjomand H, Aquilina P, McCormick D. Acute myocardial infarction in a patient with von Willebrand disease: pathogenetic dilemmas and therapeutic challenges. J Invasive Cardiol. 2002;14(10):615-8.
3. James PR, de Belder AJ, Kenny MW. Successful percutaneous transluminal coronary angioplasty for acute myocardial infarction in von Willebrand's disease. Haemophilia. 2002;8(6):826-7.
4. Fragasso G, Camba L, Pizzetti G, Pagnotta P, Chierchia SL. Successful thrombolysis for acute myocardial infarction in Type I von Willebrand's disease (vWD). Am J Hematol. 1998;57(2):180.
5. Spiel A, Gilbert J, Jilma B. von Willebrand factor in cardiovascular disease: focus on acute coronary syndromes. Circulation. 2008;117(11):1449-59.
6. Rutten B, Maseri A, Cianflone D, Laricchia A, Cristell N, Durante A, et al. Plasma levels of active Von Willebrand factor are increased in patients with first ST-segment elevation myocardial infarction: a multicenter and multiethnic study. Eur Heart J Acute Cardiovasc Care. 2015;4(1):64-74.
7. Sonneveld M, Cheng J, Oemrawsingh R, de Maat M, Kardys I, Garcia-Garcia H, et al. Von Willebrand factor in relation to coronary plaque characteristics and cardiovascular outcome. Results of the ATHEROREMO-IVUS study. Thromb Haemost. 2015;113(3):577-84.
8. Hassan S, Amer S, Qureshi W, Alirhayim Z, Kuriakose P. Treating symptomatic coronary artery disease in patients with Von Willebrand disease. Hematol Oncol Stem Cell Ther. 2013;6(3-4):101-4.