INTERVENTIONAL CARDIOLOGY PERSPECTIVES

Inter Cardio Pers

DOI: 10.4274/intercardiopers.2025.2025-6-4

CASE REPORT

Acute Stent Thrombosis and Myocardial Infarction in a Postsplenectomy Patient with Thrombocytosis

Mustafa Tunahan Öz, Adnan Kaya

Department of Cardiology, Bahçeşehir University Faculty of Medicine, İstanbul, Türkiye

ABSTRACT

We report a case involving acute stent thrombosis in a patient with a history of splenectomy and thrombocytosis. The patient arrived with persistent chest pain and vomiting. The electrocardiogram (ECG) revealed ST-segment elevation in leads D2, D3, AVF, and V4-V6. Primary percutaneous coronary intervention was carried out using a 4.0×23 mm drug-eluting stent following loading doses of 300 mg acetylsalicylic acid and 600 mg clopidogrel. Adequate anticoagulation was ensured with intravenous heparin, monitored by activated clotting time (ACT). An ACT of 258 was achieved within 10 min. The procedure was completed successfully, and the patient was admitted to the intensive care unit. However, a follow-up ECG showed persistent ST-segment elevation, and the patient developed cardiogenic shock with complete atrioventricular block. The patient was returned to the catheterization laboratory, where angiography confirmed stent thrombosis. Balloon angioplasty was performed, and tirofiban was administered along with temporary pacing support. The patient was discharged 3 days after the initial procedure with preserved left ventricular function. This case underscores that acute stent thrombosis can occur despite appropriate intervention and anticoagulation. In patients with splenectomy and thrombocytosis, adequate platelet inhibition and anticoagulation may still be insufficient, increasing the risk of stent thrombosis.

Keywords: Acute stent thrombosis, interventional, myocardial infarction

INTRODUCTION

The causes of thrombocytosis are categorized as either primary or secondary, with secondary (reactive) thrombocytosis being more prevalent than primary.[1]. Secondary thrombocytosis can result from factors such as infections, iron deficiency, splenectomy, medications, malignancies, inflammatory conditions, or hemolysis.[2] Most individuals with secondary thrombocytosis remain asymptomatic and follow a benign course.[3] Nonetheless, in rare instances, it may lead to thrombotic complications including acute myocardial infarction (MI), mesenteric vein thrombosis, or pulmonary embolism. Thrombocytosis following splenectomy accounts for about 1.6% of all cases.[4] MI and other vaso-occlusive events occur in fewer than 5% of patients with secondary thrombocytosis.[5] In our patient, a stroke occurred 1 year after splenectomy, followed by ST-elevation MI 6 years later, and acute stent thrombosis developed just 10 min after a successful percutaneous coronary intervention (PCI). We present this case to illustrate acute stent thrombosis despite an optimized intervention and anticoagulation strategy.

CASE REPORT

Clinical Presentation

A 58-year-old woman presented to the emergency department with persistent chest pain, back pain, and jaw numbness lasting for 2 hours. Her blood pressure was 90/65 mmHg, pulse rate was 52 beats/minute, and oxygen saturation was 98%. The electrocardiogram (ECG) revealed ST-segment elevation in leads V4-V6 and D2-D3, with reciprocal ST-segment depression in V1-V2. Oral loading doses of 300 mg acetylsalicylic acid and 600 mg clopidogrel were given. The patient was then moved to the angiography suite. Coronary angiography identified a 99% subtotal thrombotic occlusion of the right coronary artery (RCA) and 80% stenosis of the circumflex artery (CX), which originates from the right sinus (Figure 1). The left main coronary artery (LMCA) appeared aneurysmal up to the D1 segment, and the left anterior descending (LAD) artery was underdeveloped.

Address for Correspondence: Mustafa Tunahan Öz MD, Department of Cardiology, Bahçeşehir University Faculty of Medicine, İstanbul, Türkiye
E-mail: m.tunahanoz134@gmail.com **ORCID ID:** orcid.org/0009-0003-0674-2523

Cite as: Öz MT, Kaya A. Acute stent thrombosis and myocardial infarction in a postsplenectomy patient with thrombocytosis. *Inter Cardio Pers.* [Epub Ahead of Print]

Received: 11.06.2025
Accepted: 07.07.2025
Epub: 14.07.2025

Management

Predilatation of the RCA osteal region was done with a 4.0×20 mm non-compliant (NC) balloon. A 4.0×23 mm drug-eluting stent was then deployed, followed by postdilatation with a 4.0×20 mm NC balloon. TIMI 3 flow was achieved and the procedure was completed successfully. The patient was transferred to the intensive care unit; however, her chest pain persisted and worsened 10 min later. Her vital signs showed a pulse of 43 beats/minute and a blood pressure of 70/56 mmHg. A repeat ECG showed continued ST-segment elevation. Due to ongoing ST-elevation in V4-V6, she was taken back to the angiography suite. Temporary pacing was applied to the right ventricle, and control coronary angiography was performed with pacing support. This revealed total stent occlusion at the RCA ostium with thrombus and distal bifurcation embolization. The ostial region was redilated with 4.0×20 mm NC balloon (Figure 2). An intracoronary bolus of 32 µg tirofiban was administered. TIMI 3 flow was restored. After the procedure, her chest pain resolved. A tirofiban (glycoprotein IIb/IIIa inhibitor) infusion at 12 µg for 6 hours was initiated.

Medical History

The patient underwent an appendectomy in 2005. In 2019, she had surgery for a right adnexal mass, which included total abdominal hysterectomy, bilateral salpingo-oophorectomy, omentectomy, and splenectomy. That same year, she received four cycles of chemotherapy with carboplatin and paclitaxel. Coronary angiography performed in 2019 showed a 30% plaque at the CX ostium and an aneurysmatic, rudimentary LAD extending to the LMCA D1 level, for which medical management was recommended. She experienced an ischemic stroke in 2020.

The patient was informed that the procedures would be included in a publication and provided written consent. No artificial intelligence assistance was used in preparing this case report.

Follow-up of the Procedure

Twelve hours after the procedure, echocardiography showed an ejection fraction of 45%, inferior wall hypokinesia, mild mitral regurgitation, mild tricuspid regurgitation, and an enlarged ascending aorta measuring 42 mm. Troponin 1 was 35,342.9 pg/mL (reference 0-15.6 pg/mL), decreasing to 16,914 pg/mL on the second day. Hemoglobin was 11.1 g/dL (reference 12.2-16.2 g/dL), hematocrit 34.7% (reference 37.7-47.9%), white blood cell count 17.52 K/uL (reference 4.23-10.2 K/uL), red blood cell count 4.12 M/uL (reference 4.04-5.48 M/uL), and platelet count 546 K/uL (reference 142-424 K/uL). Prothrombin activity was 83% (reference 70-130%) and the international normalized ratio was 1.14 (reference 0.75-1.2). The patient was discharged on dual antiplatelet therapy, which is considered sufficient to prevent further complications.

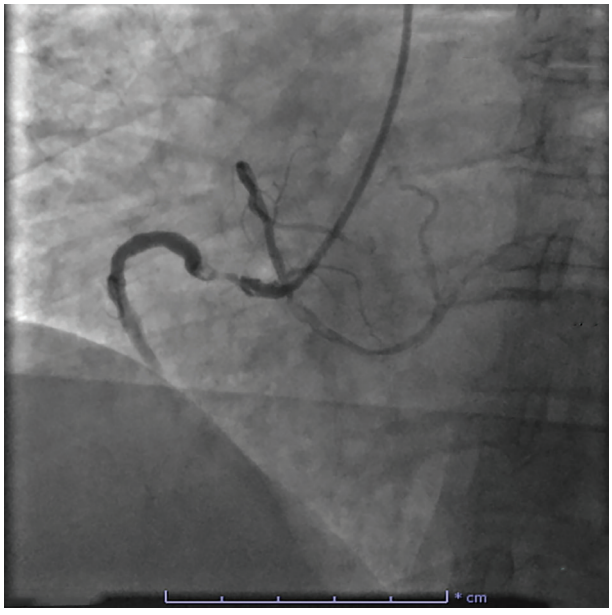

Figure 1. The circumflex artery originates from the right sinus and shows 80% narrowing in its midportion. The right coronary artery is nearly completely occluded with thrombus

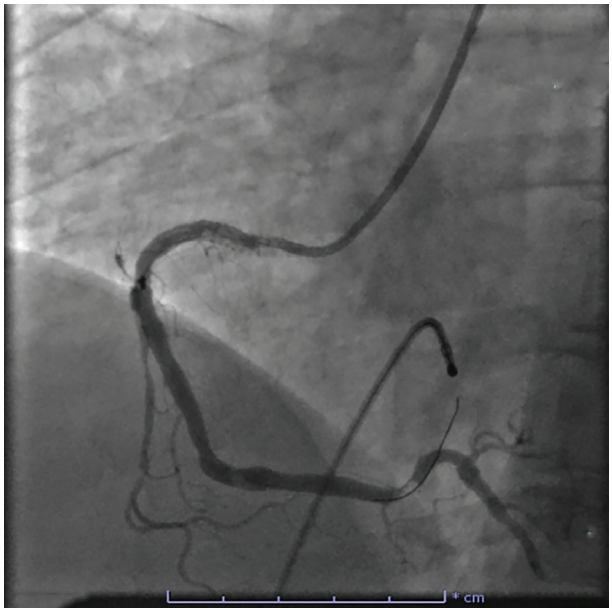

Figure 2. Acute thrombosis following percutaneous coronary intervention. The thrombus resolved after tirofiban administration and balloon inflation within the stent

CONCLUSION

Acute stent thrombosis following PCI is an uncommon occurrence. In this case, it developed in a patient with thrombocytosis secondary to splenectomy. Postsplenectomy thrombocytosis is reported in about 90% of cases due to loss of splenic sequestration.[6] In this patient, a stroke, ST-elevation MI, and acute stent thrombosis occurred after splenectomy. Both predilatation and postdilatation were performed, the stent was appropriately sized, and no residual stenosis was detected. Therefore, the acute stent thrombosis was not procedural in origin. It developed despite activated clotting time (ACT) monitoring. Although periprocedural heparin dosing can vary, the optimal ACT target is 300-350 seconds. The occurrence of acute stent thrombosis after PCI may indicate suboptimal stent patency, and underlying hematologic conditions should be evaluated.

Informed Consent: The patient was informed that the procedures would be included in a publication and provided written consent.

Authorship Contributions: Surgical and Medical Practices: A.K., Concept: A.K., Design: M.T.Ö., Data Collection or Processing: M.T.Ö., Analysis or Interpretation: A.K., Literature Search: M.T.Ö., Writing: M.T.Ö.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

REFERENCES

1. Griesshammer M, Bangerter M, Sauer T, Wennauer R, Bergmann L, Heimpel H. Aetiology and clinical significance of thrombocytosis: analysis of 732 patients with an elevated platelet count. *J Intern Med*. 1999;245:295-300.
2. Rokkam VR, Killeen RB, Kotagiri R. Secondary thrombocytosis. StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025.
3. Williams B, Morton C. Cerebral vascular accident in a patient with reactive thrombocytosis: a rare cause of stroke. *Am J Med Sci*. 2008;336:279-281.
4. Khan PN, Nair RJ, Olivares J, Tingle LE, Li Z. Postsplenectomy reactive thrombocytosis. *Proc (Bayl Univ Med Cent)*. 2009;22:9-12.
5. Tai YT, Yu YL, Lau CP, Fong PC. Myocardial infarction complicating postsplenectomy thrombocytosis, with early left ventricular mural thrombus formation and cerebral embolism--a case report. *Angiology*. 1993;44:73-77.
6. Chia TL, Chesney TR, Isa D, et al. Thrombocytosis in splenic trauma: in-hospital course and association with venous thromboembolism. *Injury*. 2017;48:142-147.