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Case Report

A 56-Year-Old Male With Acute Stent Thrombosis During Percutaneous Coronary Intervention, How To Resolve This Problem?

Hayla Iqda Milisani[1*], Mohammad Saifur Rohman[2], Sasmojo Widito[2], Indra Prasetya[2]

[1] *Brawijaya Cardiovascular Research Center, Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Brawijaya, Malang, Indonesia.*
[2] *Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Brawijaya, Malang, Indonesia.*

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ABSTRACT

Background: Acute stent thrombosis is the complete occlusion of a coronary artery of the previously implanted stent. This unusual complication occurs in percutaneous coronary intervention (PCI), development of myocardial ischemia, and poor prognosis for the patient. After PCI, acute stent thrombosis can occur within 0-24 hours and cause the symptoms like acute coronary syndrome. Incidents of stent thrombosis are about 0.6% to 3.4% for Drug Eluting Stent (DES) implantation, depending on the lesion and patient factors. The etiology of acute stent thrombosis is multifactorial, and early detection can reduce the mortality rate.
Case Illustration: A 56 yo male visited Rumah Sakit Saiful Anwar Malang with stable angina pectoris (Class III symptoms with medical therapy) planned for elective cardiac catheterization with routine medical treatment. During PCI, he complained the chest pain, and from cine angiography evaluation showed no flow at the diagonal branch because of the acute thrombosis. Then got thrombosuction and got a white thrombus. After the PCI procedure, he got fibrinolytic with streptokinase 1.5 million units for 60 minutes. He was transferred to CVCU for observation and discharged after five days.
Conclusion: Acute stent thrombosis is a severe complication during and after PCI because it is related to high mortality. The mechanisms by which ST arises are complex and multifactorial and must be early detection.

1. Introduction

Percutaneous coronary intervention (PCI) was an invasive therapeutic cardiac procedure in managing ischemic heart disease. In particular, a severe and common complication was associated with morbidity, mortality, and emergency condition o the patient. Stent Thrombosis was the most complication related to PCI, which was associated with a multifactorial process. Most stent thrombosis can occur within 0-24 hours. In general clinical practice, the rate of acute stent thrombosis is 1%. Management of acute stent thrombosis is to restore the coronary blood flow. Several strategies can be applied to decrease the occurrence of acute stent thrombosis.

2. Case Illustration

A male 56 yo came to Rumah Sakit Saiful Anwar Malang with stable angina pectoris (Class III symptoms with medical therapy) and planned for elective cardiac catheterization with routine medical treatment. From past medical history, this patient was hypertension and dyslipidemia since two years ago but did not regularly take medication, history of CVA in 2014 and 2015. The patient with a history of percutaneous coronary intervention with CAD 3 vessel disease post-implantation 1 DES proximal LAD-D1 (in June 2022); 1 DES proximal LAD-D1 (overlapped with old stent) and OM1 (in September 2022); 1 DES at osteal-mid RCA and suggested to staging PCI at LAD if symptomatic (at October 2022). For the treatment of CAD, the patient is routinely controlled by the cardiologist and got Aspilet 1x80mg, Clopidogrel 1x75mg, Simvastatin 1x20mg, Candesartan 1x16mg, Bisoprolol 1x5mg, Isosorbide Dinitrate 3x10mg and Nifedipin 1x30mg. His father had hypertension, and his mother had heart failure.

From the physical examination, the blood pressure was 121/73 mmHg, heart rate 80 bpm regular, respiratory rate 20 tpm, and saturation O2 99% on room air. On examination of the heart was normal with the sound of S1 S2 regular, no murmur, and gallop. On examination of the pulmo, there was no rhonchi or wheezing. At ECG, was normal with sinus rhythm, frontal axis normal, horizontal axis CWR, poor R wave progression, Q pathologist at lead V3-V4, T inverted at lead I, aVL, V3-V6 (Figure 1).

*Corresponding author at: Brawijaya Cardiovascular Research Center, Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Brawijaya, Malang, Indonesia
E-mail address: hayla07@yahoo.com (H. I. Milisani).

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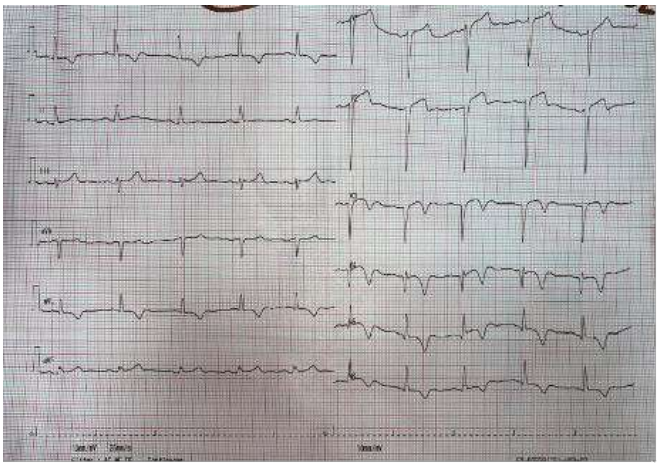


Figure 1. ECG showed sinus rhythm, frontal axis normal, horizontal axis CWR, poor R wave progression, Q pathologist at lead V3-V4,T inverted at lead I, aVL,V3-V6

His CXR examination showed cardiomegaly with aorta sclerotic (Figure 2). The abnormality of the laboratory finding was an increase in creatinine serum level (Cr 1.61). The echocardiography showed systolic LV was 51% with diastolic dysfunction (E/a <1), systolic RV was normal (TAPSE 2.1), and hypokinetic at the anterior segment.

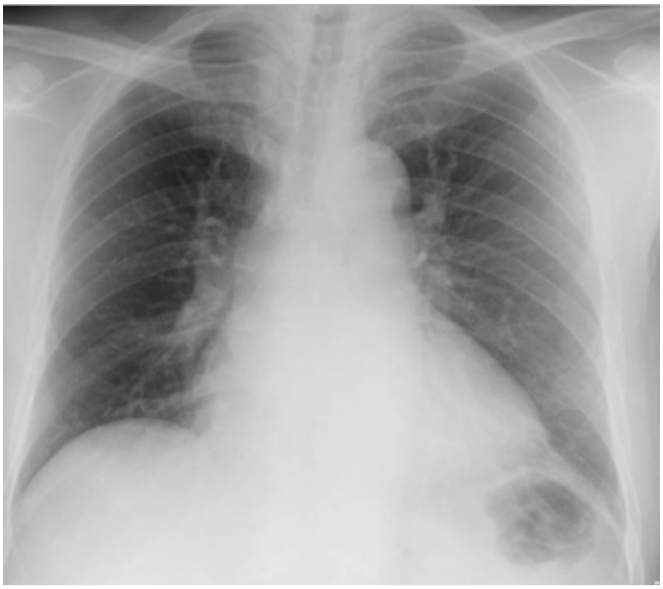
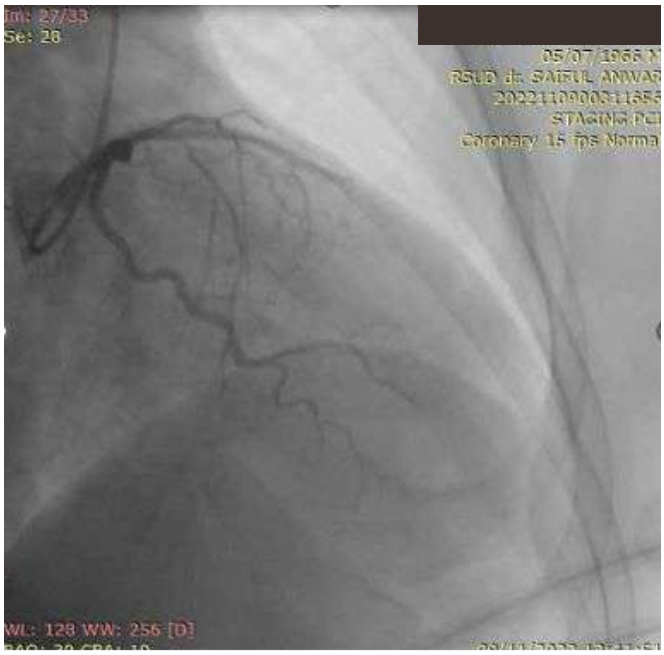


Figure 2. CXR showed cardiomegaly with aorta sclerotic

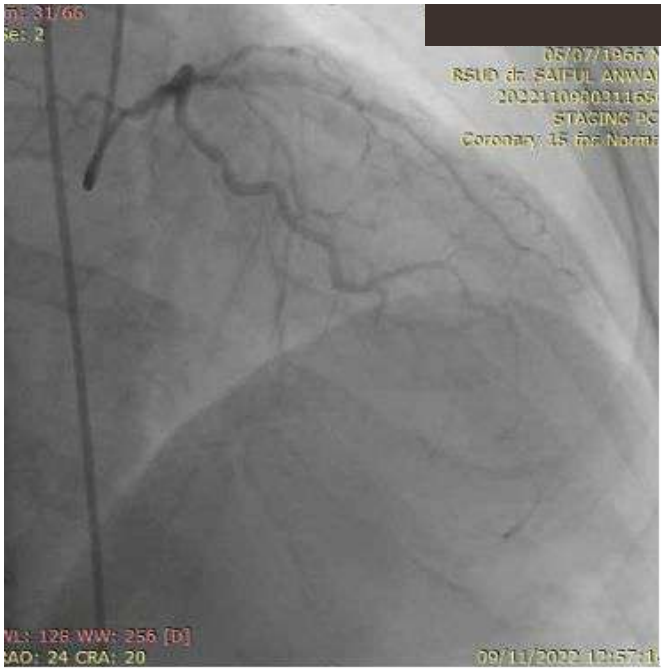
The assessment for this patient was stable angina pectoris, HF stage C Fc II dt CAD HHD, hypertension on treatment, Azotemia pre renal dd renal, and history of CVA. Patient got IVFD Normal saline 0.9% 1cc/kgbw/hour, Aspilet 1x80mg, Clopidogrel 1x75mg, Atorvastatin 1x40mg, Candesartan 1x16mg, Bisoprolol 1x5mg, Isosorbide Dinitrate 3x10mg and Nifedipin 1x30mg. The patient was planning to elective PCI with informed consent to the patient and family at distal LAD if symptomatic.

Before the PCI procedure, the patient was stable with BP 118/71 mmHg, HR 55 bpm, RR 20 tpm, and SpO2 99% on room air. Diagnostic coronary access from right femoral artery) showed the LM was normal. The old stent at proximal LAD-diagonal branch 1 was patent, the diagonal branch 1 was large, CTO at mid-LAD, and distal LAD got collateral from the PLB branch. The old stent at OM 1 was patent, and the diameter was large. The old stent at osteal-mid RCA was patent. We decided on the LAD lesion as the target vessel.

The PCI begins to enter GC BL 3.5 6F with engaged at osteal LMCA. Then enter the Ashahi Sion Blue wire tried to distal LAD. When cine angiography evaluation was found, the GW at the diagonal one branch tried to mid-LAD through the strut stent. There was no flow at the diagonal branch because of the acute thrombosis (Figure 3).



(A)



(B)

Figure 3. (a) Diagnostic Coronary Angiography (b) Coronary angiography showed acute thrombosis at the D1 branch.

Then the patient got thrombosuction with thrombus 6F and appeared a white thrombus. The patient complained of chest pain, then was given a heparin 720 unit/hour, a drip of forbid mg/hour, and a loading of ticagrelor 180mg. Then enter the balloon Sapphire II Pro 1.5x1.0mm into the D1 branch intrastent. Inflate the balloon several times at the D1 branch with maximal pressure of 20 atm for 6 seconds. The cine angiography evaluation found appears no reflow at the D1 branch. The patient was observed in the recovery room at the cath lab and evaluated for hemodynamic, cardiac enzyme, and ECG evaluation. The patient with blood pressure was 171/88mmHg. Heart rate was 78bpm, respiratory rate was 20 tpm, and oxygen saturation was 99% on NC 5lpm. The cardiac enzyme evaluation revealed troponin 23.9 μg/L and CKMB 67 U/L. The ECG evaluation showed sinus Rhythm, HR 78bpm, FA ordinary HA routine, CWR P wave normal, PR interval 160ms, QRS complex 80ms, Q pathologist at lead V4-V5, T inverted at lead I aVL V3-V6. Then the patient got streptokinase IV 1.5 million iu for 60 minutes with hemodynamic and bleeding sign observation. Furthermore, the patient continues to observe at Intensive Care Cardiac Unit and continues therapy DAPT with Aspilet 80mg QD and Ticagrelor 90mg BID.

3. Discussion

The definition of stent thrombosis based on The Academic Research Consortium (ARC) criteria includes based on gradation (definite, probable, and possible stent thrombosis), based on onset (early, late, and very late), it can be distinguished based on pathophysiology mechanism and clinical implantation. Based on the onset, stent thrombosis includes early, late, and very late onset. Early onset of stent thrombosis occurs <24 hours (acute) and 24 hours to 30 days (subacute). Late onset of stent thrombosis occurs 31 days to 1 year. Very late onset if stent thrombosis occurs >1 year. In acute stents, thrombosis may occur by suboptimal procedure results (e.g., inadequate lumen dimension post-procedure, slow blood flow, residual dissection, and tissue prolapse). In our patient, the stent thrombosis occurs during percutaneous coronary intervention and can be acute stent thrombosis.[1–3]

The pathophysiology mechanisms of stent thrombosis consist of 1) turbulence and shear forces, including malposition, dissection, or oversized stent in small vessels; 2) inflammation of endothelial induces congenital or gained hypercoagulability; 3) overactivation of the platelet activation pathway can lead to thrombosis. The risk factors can be divided into several categories when PCI procedure: patient-related, lesion characteristic-related, and procedural-related factors. Patient-related factors include diabetes mellitus, acute presentation, current smoker, reduced left ventricular function, cancer, DAPT non-responsiveness, premature cessation of DAPT, and advanced age. Lesion characteristics related include the long segment of disease, small diameter vessel, chronic total occlusion, and bifurcation lesion. Procedural related include stent under expansion, stent malposition, edge dissection, multiple stent implantation and overlap, residual stenosis, and reduced TIMI flow after the procedure. The type of stent can influence the occurrence of stent thrombosis. From the data, as many as 20% of the patient after BMS implantation had stent thrombosis, which arose within two days after PTCA, and in the era of DES implantation, stent thrombosis occurred more frequently in less than 30 days due to the endothelialization was slow process of drug contained within the stent. Thrombus formation during stent implantation means, as intraprocedural stent thrombosis (IPST), was a rare event in the BMS implantation, which is defined as an angiographically showed intraluminal filling defect within the stent resulting in TIMI grade 0 or 1 antero¬grade flow. One of the risk factors for stent thrombosis is noncompliance with DAPT. However, in this case, patients reported good compliance with clopidogrel and aspirin. The complexity of the procedure itself plays a role in stent thrombosis events and in this patient with a history of stent DES implantation in July 2022 (PTCA at distal LAD), September 2022 (implantation 1 DES at proximal LAD-D1 branch and 1 DES at OM1) and October 2022 (implantation 1 DES at osteal-mid RCA). Furthermore, the characteristic of the lesions at the coronary artery was the predisposition of stent thrombosis, and our patient there was an old stent in proximal LAD-D1 with CTO at mid-LAD, which can increase the risk of stent thrombosis in our patient.[4–6]

The clinical manifestation of stent thrombosis in the patient can occur in chest pain and electrocardiographic changes, but it can be asymptomatic if the patient has a collateral vessel. Intravascular ultrasound can show the filling defect, and it is used to detect the mechanism of stent thrombosis (malposition, under expansion, or edge dissection). Intracoronary thrombus was angiographically identified and scored in 5 grades, including thrombus grade 1 (G1), a possible thrombus is present; thrombus grade 2 (G2), a definite thrombus, with greatest dimensions ≤1/2 the vessel diameter; thrombus grade 3 (G3), a definite thrombus but with greatest linear dimension >1/2 but <2 vessel diameters; thrombus grade 4 (G4), a definite thrombus, with the largest dimension ≥2 vessel diameters; thrombus grade 5 (G5), a total occlusion (unable to assess thrombus burden due to total vessel occlusion). Our patient complained the chest pain with VAS 8/10 after the PCI procedure. The coronary angiography showed no flow (at the diagonal one branch because of the acute thrombosis.[1,7–9]

Anticoagulant therapy during the PCI procedure has been the focus of numerous clinical trials related to complications during or after PCI, for example, stent thrombosis. Anticoagulant therapy, the most commonly used antithrombin agent for PCI, is UFH, supported by practice guidelines (ESC class I recommendation). UFH agent has a binding site for factor Xa, thrombin (factor IIa), or both. Although heparin may be administered intravenously or subcutaneously, the intravenous form is relevant to PCI procedure because subcutaneous delivery delays anticoagulant effects. It has been used since the advent of PCI to inhibit platelet-rich thrombus formation, both at the site of balloon injury and upon angioplasty equipment during PCI. However, the heparin bolus administration for any individual is not predictable because it is influenced by body mass, heparin resistance, concomitant with another drug (e.g., nitrates and other thrombolytic agents), and also influenced by concentrations of platelet factor IV. Heparin doses during PCI based on the recommendation were 70-100 IU/kg body weight at the start of the PCI procedure. In this patient got heparin bolus 70iu/kgbw when starting to PCI procedure.[10–13]

Dual antiplatelet therapy is currently recommended for PCI patients planning for DES implantation. In the basic randomized trial, aspirin and clopidogrel showed decreased stent thrombosis events compared with aspirin alone. Based on guidelines of DAPT duration, the advent of more potent P2Y12 inhibitors, such as prasugrel and ticagrelor, has reduced major adverse cardiovascular events but at the cost of increased bleeding. However, the newer-generation DESs, with their thinner stent struts, a more biocompatible polymer, and favorable drug-eluting characteristics, can decrease the risk of stent thrombosis, particularly late stent thrombosis. Post-DES implantation therapy with aspirin and clopidogrel is currently the standard of care for patients, and the rate of stent thrombosis event also reduced by switching clopidogrel with the more potent antiplatelet such as ticagrelor. However, its benefit must be considered, as the risk of bleeding individual. Intracoronary GP IIb/IIIa inhibitor agents in large trials have demonstrated they can improve epicardial and myocardial perfusion with fewer adverse events. However, clinical trials did not find a significant improvement in outcomes or recurrent myocardial infarction. Furthermore, intracoronary GP IIb/IIIa inhibitor agents are a Class IIb recommendation during PCI procedure, and this agent can administrate if the patient is allergic to P2Y12 inhibitor agents. In this case, the patient

planned to switch from clopidogrel to ticagrelor (after loading Ticagrelor 180mg during PCI) after fibrinolytic therapy. Our patient didn't have an allergy to P2Y12 inhibitor, so he continued DAPT with Aspilet and Ticagrelor.[5,14–17]

Thrombus aspiration can effectively treat stent thrombosis, especially in patients with high thrombus burden, for improving coronary microcirculation. The result of the TAPAS (Thrombus Aspiration During Percutaneous Coronary Intervention in Acute Myocardial Infarction Study) trial showed that thrombus aspiration could improve the outcome of microvascular perfusion and a significant reduction in long-term mortality during the one-year follow-up. Stent thrombosis is a complication during and after the PCI procedure, and the result of the TASTE trial (Thrombus Aspiration in ST-Elevation Myocardial Infarction in Scandinavia) that thrombus aspiration decreased the risk of stent thrombosis by more than half at 30 days but not at one year. The study of thrombus aspiration for stent thrombosis showed the beneficial effect of removing local thrombus, decreasing macrovascular and microvascular occlusion, and preventing distal embolization. The types of thrombus were white thrombus and red thrombus. White thrombi were particularly composed of fibrin and smaller than red thrombi. The patients with white thrombus had less ischemic time than those with red thrombus. In this case, the patient got a thrombus aspiration during the PCI procedure with Thrombuster 6F and a white thrombus.[12,18,19]

The most practiced approach currently to manage stent thrombosis is to re-perform PCI, but in some stent thrombosis cases, the thrombolytic treatment may also be the initial option. The thrombolytic treatment may be considered for the patient, explained by the concise symptoms to treatment time. Based on the previous study showed a similar degree of evidence of thrombolytic treatment compared with percutaneous angioplasty in the management of stent thrombosis from symptom onset to reperfusion was less than 2 hours with Fibrinolytic IV after PCI procedure. In this case, the patient complained of chest pain and stent thrombosis during the PCI procedure. He got thrombosuction during PCI and continued with a fibrinolytic agent with streptokinase 1.5 million units for 60 minutes after the PCI procedure. After fibrinolytic, the patient complained the chest pain (VAS 2/10), and shortness of breath subsided.[20,21]

4. Conclusion

Stent thrombosis is a severe complication associated with a high mortality rate during the PCI procedure. Any potential predictors seem to be involved in the pathophysiology of stent thrombosis. Stent thrombosis is a serious complication resulting from occlusion of the lumen by a thrombus. It has a broad chronological spectrum, occurring anywhere from intraprocedural to years during or after stent implantation. Clinical manifestation of stent thrombosis must be recognized immediately to determine the management of the case from the anamnesis, physical examination, and coronary angiography. Thrombolysis can be the initial treatment of stent thrombosis in the hospital and improve the prognosis for the patient.

5. Declarations

5.1. Ethics Approval and Consent to participate
This study was approved by local Institutional Review Board, and all participants have provided written informed consent prior to involvement in the study.

5.2. Consent for publication
Not applicable.

5.3. Availability of data and materials
Data used in our study were presented in the main text.

5.4. Competing interests
Not applicable.

5.5. Funding source
Not applicable.

5.6. Authors contributions
Idea/concept: HIM, MSR. Design: HIM, MSR.Control/supervision: IP, MSR, SW. Literature search: HIM, MSR, SW, IP. Data extraction: HIM. Statistical analysis: HIM, MSR. Results interpretation: HIM. Critical review/discussion: MSR, IP, SW. Writing the article: HIM, MSR. All authors have critically reviewed and approved the final draft and are responsible for the content and similarity index of the manuscript.

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