Hindawi
Case Reports in Cardiology
Volume 2021, Article ID 2658094, 3 pages
https://doi.org/10.1155/2021/2658094

*Case Report*

# Simultaneous Multi-Vessel Very Late Stent Thrombosis in Acute ST-Segment Elevation Myocardial Infarction

**Yuefeng Chen, Michael Amponsah, and Cyril Nathaniel**

*Department of Cardiology, Conemaugh Memorial Medical Center, Johnstown, Pennsylvania 15905, USA*

Correspondence should be addressed to Yuefeng Chen; cyf.usa@gmail.com

Received 15 September 2021; Revised 29 November 2021; Accepted 7 December 2021; Published 30 December 2021

Academic Editor: Dirk Bandorski



Simultaneous multi-vessel very later stent thrombosis (VLST) is a very rare complication of percutaneous coronary intervention (PCI). We present a case of simultaneous multi-vessel VLST as the cause of acute ST-segment elevation myocardial infarction (STEMI). PCI of the culprit vessel was performed at acute presentation. Resolution of in-stent thrombosis in non-culprit vessels was noted on coronary angiography 2 days later. Our case suggests that PCI for culprit lesion in acute setting may be a reasonable option for simultaneous multi-vessel VLST.

## 1. Introductions

VLST is defined as stent thrombosis that occurs after first year after the initial implantation [1]. It is a very rare complication of stent implantation with an incidence of less than 1% during a 4-year follow-up [2]. Simultaneous multi-vessel thrombosis in the setting of acute STEMI has been reported [3], but simultaneous multi-vessel VLST in acute STEMI is extremely rare. Here we report a case of acute STEMI with simultaneous multi-vessel VLST with the last stent implantation of 5 years prior.

## 2. Case Presentation

The patient is a 71-year-old male with history of coronary artery disease (CAD) was brought to emergency room for left-sided chest pain for 45 minutes, associated with shortness of breath, diaphoresis and nausea. He has history of PCI of proximal to mid left anterior descending artery (LAD) and proximal left circumflex artery (LCX) with drug-eluting stents (DES) in March, 2012, repeated PCI of mid LAD with DES in March, 2015, and PCI of mid and distal right coronary artery (RCA) in January, 2016. He also has history of hypertension, type II diabetes mellitus and morbid obesity. He was taking Aspirin 325 mg daily, Prasugrel 10 mg daily, Quinapril 40 mg daily, Atorvastatin 80 mg daily. Physical exam revealed body mass index 43.1 $kg/m^2$, blood pressure 157/122 mmHg, heart rate 131 beats/minute, respiration rate 24/minute, oxygen saturation 97%, he was in acute respiratory distress, with diaphoresis, tachycardia, tachypnea and bilateral lungs decreased breath sounds, but no wheezing or crackles. Laboratory studies showed SARS-CoV-2 negative, hemoglobin A1C 6.9%, eGFR 60 ml/min, White blood cell $10.8x10^3$/ul, platelet $316x10^3$/ul, B-type natriuretic peptide 340 pg/ml, Troponin I 0.01 ng/ml, Total cholesterol 199 mg/dl, Low-density lipoproteins 140 mg/dl. Initial electrocardiography showed anterior ST segment elevation. He was intubated due to respiratory distress and unable to lie flat. Emergency cardiac catheterization revealed 100% occlusion of proximal to mid LAD with instent thrombosis, and 90% stenosis in proximal LCX and mid RCA stents (Figure 1(a)–1(c)). PCI of proximal to mid LAD was performed and TIMI-3 flow to distal LAD was restored, a Synergy 3.0 x 24 mm DES was then placed and post dilated with a 3.5 mm non-compliant balloon (Figure 1(d) ). The patient was hemodynamically stable and brought back to catheterization laboratory 2 days later for staged PCI of LCX and RCA lesions. However, angiography showed that previously found severe stenosis in the above vessels was no longer visualized (Figure 1(e) and 1(f)), it turned out that previously noted instent stenosis was actually caused by thrombus at the time of acute STEMI and had dissolved

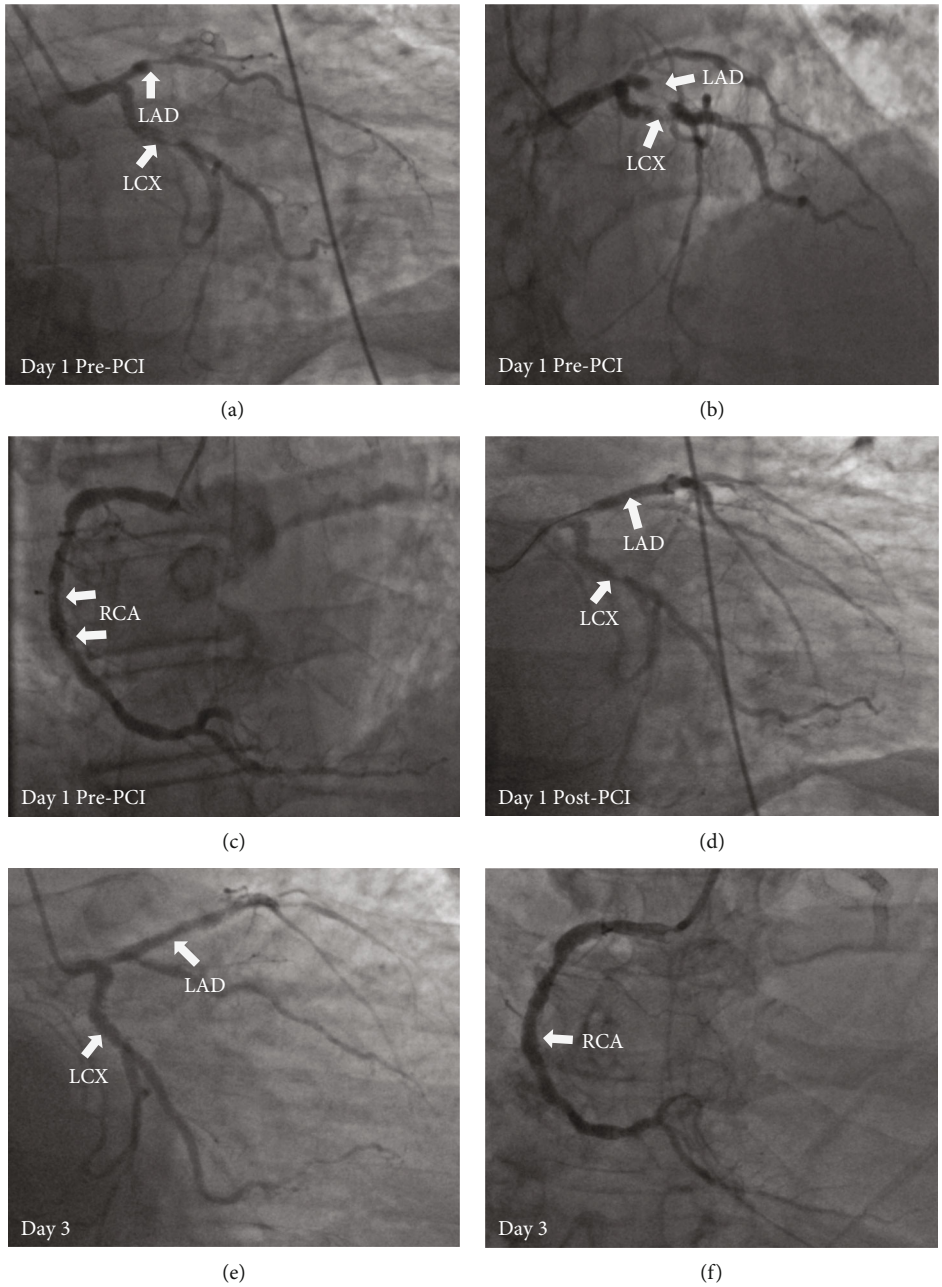


Figure 1: Coronary angiography and LAD primary PCI. Thrombotic occlusion at the site of the proximal LAD stent with TIMI 0 flow (A, B, arrow) and evidence of thrombus at the site of the proximal LCX stent (A, B, arrow) and the mid RCA stent (C, arrows). Following PCI and restoration of TIMI 3 flow in the LAD, there was evidence of remaining thrombus at the site of proximal LCX stent (D, arrow). Resolution of thrombus at the site of the proximal LCX stent (E, arrow) and the mid RCA stent (F, arrow) on day 3 of admission. LAD: left anterior descending artery; LCX: left circumflex artery; RCA: right coronary artery; PCI: percutaneous coronary intervention.

spontaneously over time. No PCI of mid LCX and RCA was performed. Patient recovered well and was discharged later.

## 3. Discussions

VLST is an uncommon complication of stent implantation, the incidence is generally less than 1% in newer generation DES [4], and is especially rare after 5 years of initial stenting. In our case, the last stent in the culprit vessel (LAD) was placed 6 years ago, and the stents in LCX and RCA were placed 9 years and 5 years ago, respectively. VLST has been reported to be more often associated with DES than with BMS [5]. It usually presents as acute STEMI, but compared to patients with early and late ST, patients with VLST had better prognosis with lower incidence of major adverse cardiac events and mortality [6–8]. The mechanism of VLST is not fully understood, but is thought to be related to delayed incomplete stent strut endothelialization [9, 10], stent strut malapposition secondary to positive remodeling [10, 11], stent fracture [12], hypersensitivity reaction to

polymers [13, 14], chronic inflammation [9, 15], rupture of lipid-laden-like neointima within the DES [10], and discontinuation of antiplatelet therapy [16]. In our case, we did not notice anything from his medical history that known to contribute to VLST, and there was no delay in patient transfer or patient care. Simultaneous triple vessel VLST is extremely rare, only a few cases have been reported [17, 18]. Treatment for multi-vessel ST can be challenging, whether PCI should be performed on all lesions at initial presentation is not certain. In our case, VLST caused 100% occlusion of LAD, while LCX and RCA remained TIMI 3 flow during the procedure, only culprit lesion was treated with PCI initially, the thrombus in the other two vessels dissolved spontaneously in 2 days, suggesting that as long as blood flow is not comprised in non-culprit vessels, culprit lesion only management at presentation may be reasonable. VLST is a multifactorial event and prevention can be difficult, some patients may need oral anticoagulation or long term DAPT therapy. More research needs to be done to identify those who are at high risk for VLST, especially in current era of trending toward short DAPT treatment duration.

## 4. Conclusions

Simultaneous triple vessel VLST is a very rare complication following stent implantation. When blood flow in non-culprit vessels is not comprised, PCI for culprit lesion only at initial presentation may be a reasonable option.

## Data Availability

Data available on request.

## Conflicts of Interest

The Authors declare that there is no conflict of interest.

## Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

## References

[1] D. E. Cutlip, S. Windecker, R. Mehran et al., "Clinical end points in coronary stent Trials," *Circulation*, vol. 115, no. 17, pp. 2344–2351, 2007.

[2] L. Mauri, W. H. Hsieh, J. M. Massaro, K. K. Ho, R. Agostino, and D. E. Cutlip, "Stent thrombosis in randomized clinical trials of drug-eluting stents," *The New England Journal of Medicine*, vol. 356, no. 10, pp. 1020–1029, 2007.

[3] A. Mahmoud, M. Saad, and I. Y. Elgendy, "Simultaneous multi-vessel coronary thrombosis in patients with ST-elevation myocardial infarction: a systematic review," *Cardiovascular Revascularization Medicine*, vol. 16, no. 3, pp. 163–166, 2015.

[4] L. Räber, M. Magro, G. G. Stefanini et al., "Very late coronary stent thrombosis of a newer-generation everolimus-eluting stent compared with early-generation drug-eluting stents: a prospective cohort study," *Circulation*, vol. 125, no. 9, pp. 1110–1121, 2012.

[5] B. Brodie, Y. Pokharel, N. Fleishman et al., "Very late stent thrombosis after primary percutaneous coronary intervention with bare-metal and drug-eluting stents for ST-segment elevation myocardial infarction: a 15-year single-center experience," *JACC. Cardiovascular Interventions*, vol. 4, no. 1, pp. 30–38, 2011.

[6] T. Kimura, T. Morimoto, K. Kozuma et al., "Comparisons of baseline demographics, clinical presentation, and long-term outcome among patients with early, late, and very late stent thrombosis of sirolimus-eluting stents: observations from the registry of stent thrombosis for review and reevaluation (RESTART)," *Circulation*, vol. 122, no. 1, pp. 52–61, 2010.

[7] S. Kubo, K. Kadota, T. Ichinohe et al., "Comparison of long-term outcome after percutaneous coronary intervention for stent thrombosis between early, late, and very late stent thrombosis," *Circulation Journal*, vol. 78, no. 1, pp. 101–109, 2014.

[8] E. J. Armstrong, D. N. Feldman, T. Y. Wang et al., "Clinical presentation, management, and outcomes of angiographically documented early, late, and very late stent thrombosis," *JACC. Cardiovascular Interventions*, vol. 5, no. 2, pp. 131–140, 2012.

[9] M. Joner, A. V. Finn, A. Farb et al., "Pathology of drug-eluting stents in humans: delayed healing and late thrombotic risk," *Journal of the American College of Cardiology*, vol. 48, no. 1, pp. 193–202, 2006.

[10] S. Miyazaki, Y. Hiasa, T. Takahashi et al., "In vivo optical coherence tomography of very late drug-eluting stent thrombosis compared with late in-stent restenosis," *Circulation Journal*, vol. 76, no. 2, pp. 390–398, 2012.

[11] S. Cook, P. Wenaweser, M. Togni et al., "Incomplete stent apposition and very late stent thrombosis after drug-eluting stent implantation," *Circulation*, vol. 115, no. 18, pp. 2426–2434, 2007.

[12] Z. Y. Huang, J. Y. Qian, and J. B. Ge, "Very late stent thrombosis due to multiple stent fracture and stent malapposition," *Chinese Medical Journal*, vol. 126, no. 1, pp. 186–189, 2013.

[13] R. Virmani, G. Guagliumi, A. Farb et al., "Localized hypersensitivity and late coronary thrombosis secondary to a sirolimus-eluting stent: should we be cautious?," *Circulation*, vol. 109, no. 6, pp. 701–705, 2004.

[14] R. Rayoo, M. Rayoo, G. Ferrante, and P. Barlis, "Histological confirmation of hypersensitivity as a contributor to very-late coronary stent thrombosis," *International Journal of Cardiology*, vol. 157, no. 2, pp. e29–e30, 2012.

[15] S. Cook, E. Ladich, G. Nakazawa et al., "Correlation of intravascular ultrasound findings with histopathological analysis of thrombus aspirates in patients with very late drug-eluting stent thrombosis," *Circulation*, vol. 120, no. 5, pp. 391–399, 2009.

[16] K. N. Huang, S. M. Grandi, K. B. Filion, and M. J. Eisenberg, "Late and very late stent thrombosis in patients with second-generation drug-eluting stents," *The Canadian Journal of Cardiology*, vol. 29, no. 11, pp. 1488–1494, 2013.

[17] S. Narasimhan, N. R. Krim, G. Silverman, and E. S. Monrad, "Simultaneous very late stent thrombosis in multiple coronary arteries," *Texas Heart Institute Journal*, vol. 39, no. 5, pp. 630–634, 2012.

[18] M. S. Vieira, A. Luz, D. Anjo et al., "Trombose tripla, simultanea, muito tardia de _stents_ coron arios," *Revista Portuguesa de Cardiologia*, vol. 32, no. 3, pp. 247–252, 2013.