

**Case Report** 

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# Longest Very Late Stent Thrombosis of Second Generation Drug Eluting Stent

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# Abstract

Stent thrombosis (ST) is a catastrophic complication of coronary stenting, presenting as sudden death or nonfatal myocardial infarction (MI) in almost all cases. ST was considered a time-limited event, with occurrences reported only during the first 30 days after stenting and confirmation of most cases within the first week(1). We are reporting a case in our institution that sustained anterior ST segment elevation myocardial infarction (STEMI) 7 years after initial implantation of Xience Prime second generation DES in the mid segment of LAD. The patient was taken to cardiac catheterization lab and found to have thrombotic occlusion of previously placed Xience Prime DES 7 years ago in mid segment of LAD; TIMI flow was zero distal to the thrombotic occlusion. To our knowledge, this is the longest reported intervening period between second generation Xience Prime DES insertion and the development of an acute coronary event secondary to very late stent thrombosis (VLST).

**Keywords:** Stent Thrombosis; Myocardial Infarction; Very Late Stent Thrombosis.

## **Case History**

Our patient is 72 years old female presented to King Abdulaziz University Hospital with severe substernal chest pain for two hours. Initial ECG shows anterior STEMI. 7 years ago, when she was 65 year old, she had undergone elective placement of Xience Prime DES 2.75\*18 across 90% lesion in mid segment of LAD. The lesion had been post-dilated using noncompliant balloon 3.0\*10. The patient stopped her clopidogrel two weeks prior to her current presentation with acute anterior STEMI under the instruction of her cardiologist.

# **Catheterization findings**

Coronary angiograms revealed thrombotic occlusion of the previously placed Xience Prime DES with TIMI flow zero distal to the thrombotic part of LAD, we gave the patient Tirofiban infusion in addition to bolus of heparin 4000IU intravenously. We crossed the lesion with BMW, red thrombus was aspirated using aspiration catheter, with restoration of TIMI III in LAD. The lesion was predilated with 2.5\* 18 Splinter Balloon and Resolute DES 2.75 \* 20 was deployed. And the stent was post-dilated in the entire length of the stent using 3.0\*15 noncompliant balloon to 20 atm. The patient recovered uneventfully and discharge from the hospital. She was instructed to continue on aspirin and clopidogrel. As of her 15 months evaluation she remains well

### **Discussion**

Our patient sustained an anterior STEMI as a result of VLST 7 years



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after the implantation of a Xience DES. To our knowledge, this is by far the longest reported interval between second generation stent insertion and the development of an acute coronary event consequent to VLST. Accordingly, this case is both unique and cause for alarm. One case report describes VLST 11 years after first generation Cypher DES insertion [1]. Randomized controlled trials initially did not raise any safety issues with first-generation DES [2,3], a subsequent report of 4 cases of angiographically confirmed ST late after elective implantation of SES or PES raised concerns of a possible very late ST risk with DES.

Second-generation DES have been developed with advanced design features, specifically thinner strut stent platforms (most commonly using a cobalt-chromium alloy) and more biocompatible polymers or bioabsorbable polymers. FDA-approved second-generation DES currently in use includes Xience V, Xience Prime, and Xience Expedition (Abbott Vascular, Santa Clara, California), which are cobaltchromium everolimus-eluting stents (CoCr-EES). In the metaanalysis by Baber at al. in which 13 RCTs with 17,101 patients were included, CoCr-EES significantly reduced definite/probable ST and MI compared with pooled PES, SES, and Re-ZES after a median follow-up of 21 months. In the meta-analysis by de Waha et al. [4] in which CoCr-EES were compared with SES in 5 RCTs with 7,370 patients, no significant differences in the risks of death, MI, or definite/ probable ST were apparent between CoCr-EES and SES after a median follow-up of 13.3 months, although a trend toward a reduction in definite/probable ST was apparent in favor of CoCr-EES [4]. Patient, and procedural characteristics have been associated with early and late stent thrombosis; the specific risk factors for VLST are less well defined. Current smoking and longer stent and lesion length have been reported as risk factors [4]. Higher numbers of stents per lesion and stent overlap are also prominent characteristics in patients with DES who sustain VLST [6]. Of importance, the discontinuation of antiplatelets therapy in itself has not been shown to be a risk factor for VLST [7] the crucial aspects of the underlying pathophysiology relate to a combination of delayed arterial healing, ongoing vessel inflammation, neoatherosclerosis, and late stent malapposition.

Mechanisms of Thrombosis includes incomplete endothelialization of stent struts is the primary precipitant of stent thrombosis. Other factors are late stent malapposition secondary to delayed positive remodeling, strut penetration into a necrotic core, and chronic vascular inflammation and hypersensitivity reaction to the metal struts [8,9]. Neoatherosclerosis has an increasingly recognized role in stent restenosis and VLST [10]. As a maladaptive endothelial response to stent implantation, neoatherosclerosis results in the evolution of a fibrous neointima into new instent atherosclerotic plaques. The mechanism underlying neoatherosclerosis might be a pathogenetic link with chronic inflammation [11]. As a group, DES tend to be associated with greater and earlier development of neoatherosclerosis than are bare-metal stents [12].

#### Conclusion

This process appears to be dynamic and to evolve over time, and we are concerned that previously successful temporary cessation of antiplatelet therapy does not necessarily predict immunity from future adverse events. This case highlights the deficiencies in the overall understanding of stent thrombosis and our ability to risk-stratify our patients and treat them accordingly

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