

Very late bare metal stent thrombosis: The role of restenosis

Çok geç dönem çıplak metal stent trombozu: Restenozun rolü

Cihan Şengül, M.D.,[#] Turan Erdoğan, M.D.,[†] İsmet Dindar, M.D.[§]

Cardiology Departments of, [#]Ordu State Hospital, Ordu; [†]Ordu Medical Park Hospital, Ordu; and

[§]Göztepe Medical Park Hospital, İstanbul

Stent endothelization is complete after one month in the absence of radiation therapy. The incidence of late stent thrombosis associated with bare metal stents is low beyond this one month period. In this paper, we report on a case of very late acute stent thrombosis that occurred after 118 months of first bare metal stent implantation. A 55-year-old male patient was admitted with chest pain and was diagnosed to have acute anterior myocardial infarction. He had a history of bare metal stent implantation for a critical stenosis in the left anterior descending coronary artery. Immediate coronary angiography demonstrated occlusion of the stent in the left coronary artery. Thromboaspiration was not an available option, so a new bare metal stent was implanted and TIMI III flow was established after balloon angioplasty.

Key words: Angioplasty, transluminal, percutaneous coronary; coronary restenosis/complications; myocardial infarction/etiology; stents/adverse effects.

Stent endothelization, in the absence of radiation therapy, is complete after one month. The incidence of late stent thrombosis associated with bare metal stents is low beyond this one month period and its occurrence is unusual. In this paper, we report on a case of very late acute stent thrombosis (ST) that occurred after 118 months following bare metal stent implantation.

CASE REPORT

A 45-year-old, nondiabetic, hypertensive, cigarette smoker, male patient was admitted with chest pain suggestive of cardiac origin and stable angina pectoris in April 1999. After initial evaluation and a positive treadmill test, coronary angiography was performed, which showed a critical stenosis of 80% in the left anterior descending (LAD) coronary artery.

Brakiterapi yapılan olgular dışında, çıplak metal stent takılan olgularda stentin tam endotelizasyonu birinci ayın sonunda tamamlanmaktadır. Birinci aydan sonra çıplak metal stentlerde geç dönem stent trombozu gelişmesi düşük sıklıktadır. Bu yazıda, stent yerleştirilmesinden 118 ay sonra çok geç dönem akut stent trombozu gelişen 55 yaşında bir erkek hasta sunuldu. Göğüs ağrısı ile yatırılan hastada akut anterior miyokart enfarktüsü tanısı kondu. Hastaya daha önce sol ön inen koroner arterde önemli darlık nedeniyle çıplak metal stent takıldığı anlaşıldı. Acil koroner anjiyografide sol ön inen koroner arterde bulunan stentin tıkanıdığı görüldü. Trombüs aspirasyonu imkanı olmadığı için, balon anjiyoplasti sonrasında yeni bir çıplak metal stent takıldı ve TIMI III akım elde edildi.

Anahtar sözcükler: Anjiyoplasti, translüminal, perkütan koroner; koroner restenoz/komplikasyon; miyokart enfarktüsü/etyoloji; stent/yan etki.

A Palmaz-Schatz bare metal stent 3.5 x 24 mm in size was implanted at 14 atm pressure without balloon predilatation. The patient was discharged the following day on indefinite aspirin use and one month of ticlopidine treatment. Statin and ACE inhibitory therapy were also prescribed. After the initial stent implantation, the patient had an uneventful course of 118 months. However, in February 2009, he was admitted to the emergency unit suffering chest pain of two-hour onset. He was still on aspirin therapy and did not quit smoking. The electrocardiogram showed acute anterior myocardial infarction. Immediate coronary angiography revealed occlusion of the implanted stent in the LAD coronary artery (Fig. 1a, b). Insertion of a coronary guide wire did not provide flow restoration. We performed balloon angioplasty with a 3 x 12-mm balloon (Fig. 1c). A bare metal stent, 3 x 28

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Correspondence: Dr. Cihan Şengül, Akyazı Mah., No: 8, D: 4, 52200 Ordu, Turkey.
Tel: +90 452 - 234 32 32 e-mail: drcsengul@yahoo.com

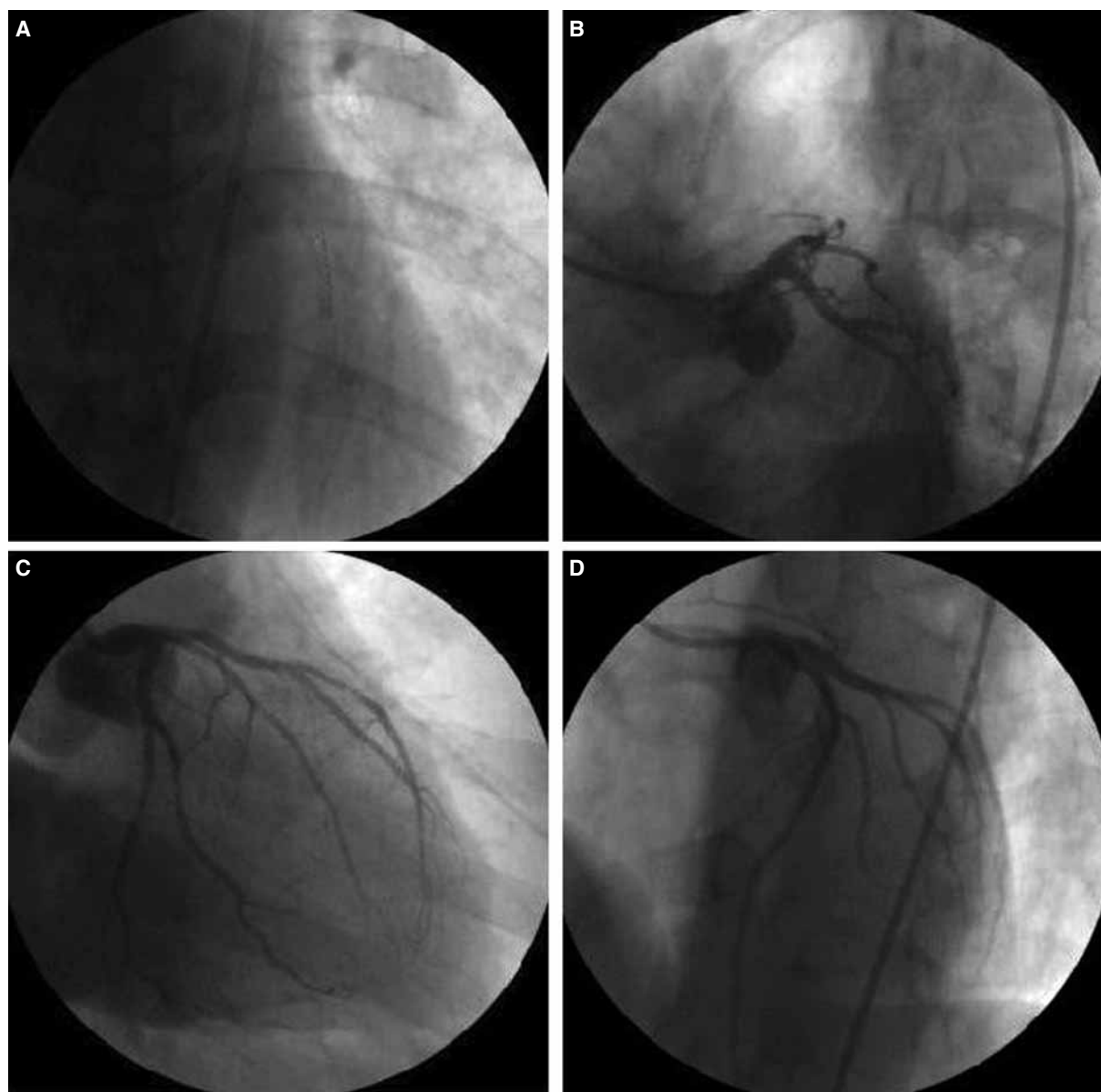


Figure 1. (A) Anteroposterior cranial view without contrast injection shows the Palmaz-Schatz bare metal stent in the left anterior descending (LAD) coronary artery. (B) The spider view shows acute occlusion of the LAD stent. (C) The right anterior oblique caudal view shows distal LAD flow after initial balloon dilatation. (D) The left anterior oblique cranial view shows TIMI III flow after subsequent stent implantation.

mm size, was implanted with restoration of TIMI III flow (Fig. 1d). The patient was sent to coronary care unit. On the fourth day, the peaks of CK and CK-MB enzymes were observed. Transthoracic echocardiography showed modest impairment of left ventricular systolic function with a global ejection fraction of 40%. The patient was discharged on aspirin and long-term clopidogrel treatment.

DISCUSSION

Stent thrombosis is associated with an abrupt onset of cardiac symptoms along with elevations in the levels

of biomarkers or electrocardiographic findings showing myocardial injury after stent deployment. A pooled analysis of multicenter trials involving 6,186 patients undergoing bare metal stent implantation documented the 30-day incidence of ST as 0.9%, most events occurring within one week.^[1] The incidence of ST was associated with a high frequency of major adverse clinical consequences, with 30-day mortality ranging from 20% to 48%, and occurrence of myocardial infarction in 60% to 70% of the patients.^[1]

There are multiple causes for early stent thrombosis. Among them, stent underexpansion, incomplete

apposition, dissection, thrombus, tissue protrusion, and persistent slow flow make up the procedural factors. Acute coronary syndrome, vessel size/lesion length, and left ventricular function are patient- and lesion-related factors. Late bare metal stent thrombosis is almost exclusively associated with intracoronary radiation therapy. A randomized study on late thrombosis in nonirradiated patients reported the incidence as lower than 1%, but did not find any predicting factors.^[2] On the other hand, there are several factors associated with late thrombosis following the use of drug-eluting stents including premature termination of antiplatelet therapy, long stent length, bifurcation lesions, localized hypersensitivity vasculitis, vessel malapposition, incomplete expansion, strut penetration of the necrotic core, diffuse in-stent restenosis, and renal failure.^[3] Very late ST has been described soon after complete termination of dual antiplatelet therapy and as a consequence of left ventricular dysfunction in diabetic patients with drug-eluting stents.^[4] Drug-eluting stent fracture may also result in very late ST.^[5]

Very late ST associated with bare metal stents has appeared in the literature as case reports without any established causes.^[6] Bertrand et al.^[7] reported a case of in-stent restenosis after five years of stent implantation. Trabattoni and Bartorelli^[8] proposed in-stent restenosis as the main mechanism leading to abrupt thrombotic vessel closure and acute myocardial infarction. In our case, there was no distal embolization or no-reflow phenomenon after balloon angioplasty, nor was there much thrombus burden before and after balloon dilatation, suggesting that in-stent critical stenosis was the main cause of very late ST.

Chen et al.^[9] reported that more than one third of episodes of bare metal in-stent restenosis present as myocardial infarction or unstable angina requiring hospitalization. Similarly, Nayak et al.^[10] found that 10.4% of patients having clinical in-stent restenosis presented with myocardial infarction.

In our patient, we did not perform intravascular ultrasound or angioscopic study before coronary angioplasty, so we could not comment on the exact mechanism of ST. Considering minimal thrombus burden, the absence of distal embolization and no-reflow

phenomenon, a high-degree in-stent restenotic lesion seems to be the main pathology. Angioplasty films showed that there was no new atherosclerotic lesion before or after stent implantation as a possible cause of poor distal run-off associated with ST. We also do not think that a 24-mm long stent could fracture easily in the mid-portion of the LAD coronary artery. In addition, the patient was taking 100 mg aspirin, although we did not assess aspirin resistance.

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