Recurrent Stent Fracture in the Right Coronary Artery

Adnan K. Chhatrwalla, MD; Samuel Unzek, MD; Samir R. Kapadia, MD

Mid America Heart Institute, Kansas City, Missouri (Chhatrwalla); Cleveland Clinic, Cleveland, Ohio (Unzek, Kapadia)

Case Report

An 83-year-old female presented in 2007 with an inferior ST-elevation myocardial infarction (STEMI), accelerated junctional rhythm, and cardiogenic shock. She had undergone percutaneous coronary intervention (PCI) to the ostium of the right coronary artery (RCA) in 2000, with deployment of a 4.0/15 mm Nirox bare-metal stent (BMS; Boston Scientific Scimed, Inc., Natick, MA), without complication (Figure 1). The patient was asymptomatic until 2004, when she began to have angina. At that time, coronary angiography revealed a fracture of the RCA stent and 90% stenosis at the ostium of the RCA. Percutaneous coronary intervention was performed with a 3.5/18 mm Taxus paclitaxel-eluting stent (Boston Scientific Scimed, Inc., Natick, MA) deployed across the prior fractured stent. The procedure was uncomplicated and immediate postprocedure angiography revealed no residual appearance of stent fracture (Figure 2).

On presentation to our institution in 2007 with inferior STEMI, the patient underwent emergent coronary angiography, which revealed occlusion of the proximal RCA, with visible thrombus and stent fracture of the prior RCA stent (Figure 3A). The stenosis in the RCA was crossed with a guide wire with difficulty and the lesion was dilated with serial balloon inflations. Intravascular ultrasound was performed, which confirmed an area of stent separation and revealed significant residual stenosis in the previously stented segment (Figure 3B). Therefore, the lesion was stented with a 3.5/23 mm Multi-link Vision BMS (Abbott Vascular, Abbott Park, IL) spanning the 2 previously fractured stents. The final angiogram demonstrated 0% residual stenosis and thrombolysis in myocardial infarction (TIMI) grade 3 flow (Figure 3C).

The patient was transferred to the coronary intensive care unit for further management and discharged home in favorable condition 16 days later. She has had no recurrent angina over 18 months of follow-up. Lifelong clopidogrel therapy is planned.

Discussion

Coronary stent fracture is likely an under-recognized entity; however, it has been more commonly reported in the

drug-eluting stent (DES) era. In a recent autopsy study, DES fracture was detected in 29% of lesions using high-contrast film-based angiography.¹ The majority of stent fracture reports have involved the Cypher DES (Johnson & Johnson, Cordis Corporation, Warren, NJ)^{1,2}; however, cases of stent fracture have been reported with BMS and Taxus DES as well.^{3–5} Several factors contributing to stent fracture have been suggested. The majority of coronary stent fractures reported in the literature had occurred in the RCA, suggesting that RCA stents may be subject to higher stress due to the exaggerated motion of the RCA in the atrioventricular groove during the cardiac cycle.⁵ Similarly, longer stents may be subject to higher radial forces, and increased stent length has been associated with a higher propensity for fracture.⁶ Furthermore, stent fracture has been reported in tortuous vessels, in calcified lesions, and at regions of stent overlap.⁷ Stent overexpansion may theoretically weaken the stent struts and predispose a stent to fracture. Finally, the higher incidence of fracture with Cypher stents and the tendency of these stents to fracture at certain sites suggests that stent design may play an important role in the propensity of a stent to fracture.¹

In this case, the fact that 2 stent fractures occurred in the same coronary segment is evidence that vascular factors contributed greatly to stent fracture in this patient. This case also suggests a potential variable presentation of patients with fracture of BMS and DES. This patient experienced a stent fracture on 2 separate occasions. In the first instance, the patient presented with angina and in the second instance, she presented with STEMI. This difference in presentation may be related to the type of stent (BMS versus DES) that had fractured. Information from published reports suggests a more benign presentation in patients with BMS fracture. We have hypothesized that when a BMS fractures there may be less exposure of stent material to the vessel lumen, and therefore less stimulus for thrombus formation.² However, DES are associated with less neointimal overgrowth.^{8,9} Therefore, stent material may be more exposed in the setting of DES fracture, potentially stimulating thrombosis and resulting in STEMI. This hypothesis is supported by data from an autopsy study demonstrating pathologic findings of thrombosis associated with 5/9 lesions (55%) with grade V (severe) DES fracture¹ and from the United States Food and Drug Administration (FDA) Manufacturer and User Facility Device Experience (MAUDE) database demonstrating that

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Figure 1. Initial coronary angiogram (2000). (A) Preprocedure angiogram showing 85% stenosis at the ostium of the right coronary artery. (B) Cineradiography still-frame after stent deployment, demonstrating no disruption of the stent struts. (C) Postintervention angiogram demonstrating a good procedural result.

Figure 2. Second coronary angiogram (2004), in the setting of angina. (A) Presenting angiogram with evidence of restenosis at the ostium of the right coronary artery. (B) Cineradiography still-frame demonstrating separation of the stent struts. (C,D) Postintervention angiogram and still-frame demonstrating a good procedural result.

Figure 3. Third coronary angiogram (2007), in the setting of ST-elevation myocardial infarction. (A) Angiographic evidence of complete stent fracture in the proximal right coronary artery. (B) Intravascular ultrasound demonstrating stent fracture. Stent struts are visible in the distal stented segment (B1) and in the right coronary artery ostium (B3), but are absent in the region of stent fracture (B2). (C) Angiographic result after stent deployment spanning the prior fractured stents.

patients with DES fracture presented with acute coronary syndrome in 31% of cases.² Interestingly, at the time of presentation in December 2007, our patient was no longer on clopidogrel therapy and therefore may have been prone to stent thrombosis if stent fracture did in fact result in exposure of stent material to the vessel lumen. Nonetheless, it is important to note that the time course of the association between DES fracture and acute coronary syndrome cannot be determined. In this case, it is possible that stent fracture may have acutely precipitated stent thrombosis and myocardial infarction. However, the stent fracture may have occurred months or years previously and placed this patient at risk for plaque rupture or stent thrombosis at a later date.

Recent data suggest that the incidence of DES fracture is higher than previously thought and the resulting adverse outcomes are clinically important. Increasing awareness of the clinical impact of DES fracture warrants a more careful assessment of future stent design strategies for this infrequent, but potentially catastrophic, complication. Further data are needed regarding the incidence, consequences, and potential treatment of stent fracture with the current generation of DES and in cases of device failure, rigorous reporting is necessary to broaden our understanding of this entity and to improve patient safety following PCI.

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