Natural regression of intimal hyperplasia at the site of stent fracture

Tae-Hoon Kim MD, Young-Guk Ko MD, Yangsoo Jang MD PhD

A 46-year-old man with an eight-year medical history of diabetes and hypertension presented with 1 h of chest pain accompanied by ST elevation in leads II, III and AVF on an electrocardiogram. Coronary angiography revealed total occlusion of the proximal right coronary artery (RCA). The culprit lesion was successfully treated with implantation of a 3.5 mm × 33 mm sirolimus-eluting stent after predilation. A routine angiogram after six months revealed a complete fracture of the stent at the distal two-thirds portion accompanied by 70% focal luminal narrowing (Figures 1A and 1B, and Video 1). The fracture was located at the centre of hinge motion of the RCA. Intravascular ultrasound showed intimal hyperplasia with a lumen area of 3.58 mm² (arrowheads), a plaque area of 17.29 mm² and an external elastic membrane area of 20.82 mm² (Figure 1C). It was decided that this restenotic lesion would not be treated with an interventional procedure because the RCA territory showed poor viability on cardiac magnetic resonance imaging. Three years later, the patient was evaluated with angiography because of newly developed chest pain. Surprisingly, angiography showed increased lumen diameter at the fracture site (Figure 1D and Video 2). Intravascular ultrasound also showed regression of intimal hyperplasia, with a lumen area of 4.58 mm² (arrowheads), a plaque area of 11.60 mm² and an external elastic membrane area of 16.18 mm² (Figure 1E).

Fracture of coronary drug-eluting stents is not rare and is occasionally associated with restenosis (1). However, currently, there is no consensus on its treatment methods. The mechanism behind natural regression of intimal hyperplasia at the site of stent fracture in the present case is not clear. The present case suggests that not all restenotic lesions associated with stent fractures require interventional treatment, but can remain free of events with regression of intimal hyperplasia.

Reference