

IMAGES IN INTERVENTION

Crushed Stent With Acute Occlusion in Superficial Femoral Artery After Enhanced External Counterpulsation



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A 60-year-old woman with hypertension was admitted because of the abrupt onset of resting pain in the left thigh, starting 2 weeks earlier. Three years ago, she received self-expanding nitinol stent implantation (overlapping stents, proximal 6 × 80 mm and distal 6 × 150 mm,

SMART, Cordis Endovascular, Warren, New Jersey) in a totally occluded left superficial femoral artery (SFA) because of claudication (Figures 1A and 1B). At this admission, a computed tomography angiogram showed total occlusion of the left SFA stent with a narrowed stented area in the middle portion of the

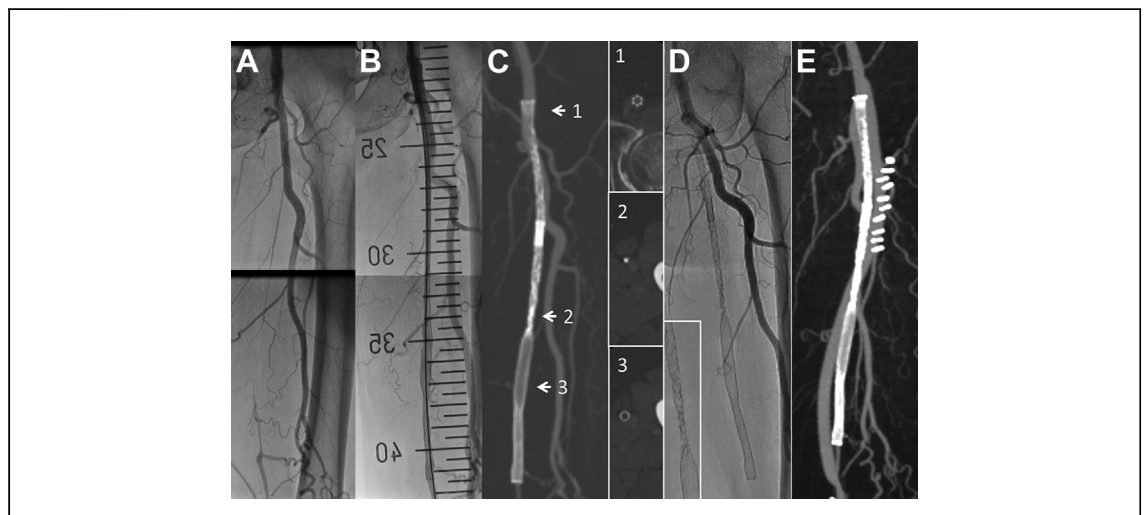


FIGURE 1 A 60-Year-Old Woman With Resting Pain in the Left Thigh After Enhanced External Counterpulsation

(A) Left superficial femoral artery (SFA) was totally occluded. (B) Two SMART stents, 6 × 150 mm and 6 × 80 mm, were implanted from the proximal to middle segments of the left SFA 3 years ago. (C) Computed tomography (CT) angiogram showed left SFA stent occlusion with a narrowed stented area of the middle portion of 2 stents. (D) Left SFA stents were occluded and extensively crushed in the middle portion. (E) CT angiogram after femoropopliteal bypass.

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stents (Figure 1C). Invasive angiography revealed totally occluded SFA stents that were extensively crushed in the middle portion (Figure 1D). After meticulous history taking, we found out that she took enhanced external counterpulsation in a private clinic shortly before symptom onset. For revascularization of the left SFA, we performed femoropopliteal bypass surgery with an artificial graft (Figure 1E).

Compared with balloon-expanding stents, the self-expanding nitinol stent is known to be resistant to mechanical stress, including external compression. Several mechanisms such as joint flexion, external muscular compression, or metal fatigue related with pulsatile blood flow were suggested as causes of stent fracture or compression. In this

report, we demonstrated a case of nitinol stent compression as an iatrogenic cause of restenosis, resulting from medical external compression. Even after nitinol stent implantation, a caution to avoid excessive external compression seems to be necessary to prevent restenosis related to stent deformity.

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