Calcified nodule progression-related stent thrombosis after polymer-based paclitaxel-eluting nitinol stent implantation for femoropopliteal artery

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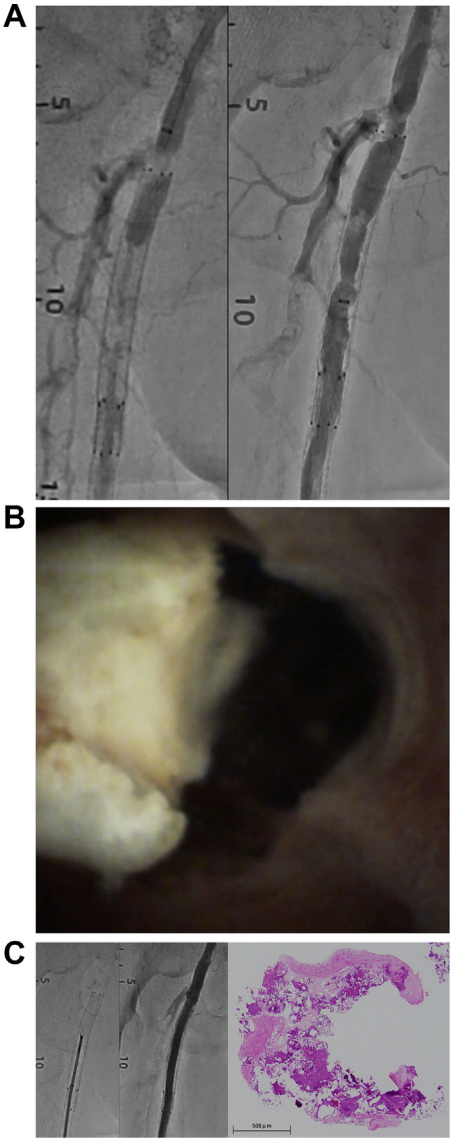
Although a previous study reported favorable durability of Eluvia (Boston Scientific, Marlborough, Mass), a polymer-based paclitaxel-eluting stent (PB-PES), stent thrombosis has been reported in 1.7% of cases.[1] Calcified lesions are considered risk factors for stent thrombosis.[2] Nodular calcification occasionally progresses beyond the stent strut, causing restenosis and stent thrombosis.[3]

CASE REPORT

A 72-year-old man with a history of previous endovascular therapies for moderate claudication (implantation of two balloon-expandable stent grafts [8.0 × 79.0 mm] in the bilateral common iliac arteries using the kissing stent technique 9 months ago; implantation of a 7.0- × 80.0-mm PB-PES and a 6.0- × 100.0- mm stent graft [VIABHAN, W. L. Gore & Associates, Flagstaff, Ariz] in the right superficial artery 6 months ago), complained of sudden right leg pain. Dual antiplatelet therapy (100 mg aspirin and 75 mg clopidogrel) was continued. The pulse at the right groin, but not the popliteal pulse, was palpable. The ankle-brachial pressure index was unmeasurable. Stent thrombosis was suspected. Angiographic findings showed PB-PES occlusion (*A*). Thrombolysis with urokinase (480,000 IU/day) was followed by additional endovascular therapy. A 6F sheath was inserted in a retrograde manner from the popliteal artery. Angiography showed focal stenosis at the mid-PB-PES (*A*). There was no restenosis in the stent graft.

Angioscopy showed the progression of a calcified nodule (CN) beyond the PB-PES strut (*B*/Cover; Video). The CN was successfully extracted with a biopsy forceps (*C*). Additional ballooning and implantation of a 6.0- × 40.0-mm interwoven stent were performed for residual calcified plaque compression. Final angiography showed good flow without residual stenosis (*C*). The patient's symptoms resolved. Pathologic evaluation showed a fibrocalcific plaque and neointima with smooth muscle cells (*C*); these findings were comparable with previously reported pathologic findings for CN progression through the stent strut (not to atheroembolism).[4] No recurrent stent thrombosis was observed at the 6-month follow-up.



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Author conflict of interest: T.N. is a consultant at Boston Scientific, Century Medical, and Medtronic.

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DISCUSSION

Although CN progression beyond the stent strut and related stent thrombosis have been reported in the field of coronary intervention,[4] to our knowledge, this is the first report of CN progression and stent thrombosis in a patient with PB-PES. The findings suggest that the drug technology and open-cell strut cannot prevent CN progression and stent thrombosis, and a more efficient barrier is necessary.

Informed consent. The patient provided written informed consent to undergo the procedure and for publication of this report.

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