In Vivo Illustration of Dendrite Formation on a Mobile Thrombus within a Wallstent in an Iliac Artery: A Fractal Phenomenon

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The mechanism of stent thrombus formation following percutaneous transluminal stent implantation in an artery is unclear. This case report describes a 72-year-old man who had a mobile thrombus in a Wallstent in the right iliac artery despite daily oral administration of 100 mg of aspirin. This Wallstent was implanted 14 years ago. The unique in vivo angioscopic images show a solid mobile thrombus with some projections which rubbed yellow plaque on the stent struts.

Keywords: solid thrombus, stent, platelet

Introduction

The issue of thrombus formation in arterial stent implantation is well-recognized in the field of stent technology. However, the in vivo process of a growing thrombus has not been fully elucidated. This case report provides unique images of a solid mobile thrombus in a stent implanted in the right iliac artery of a patient with arteriosclerosis obliterans.

Case Report

A 72-year-old man was admitted to our hospital with intermittent claudication due to diffuse stenosis in the right superficial femoral artery (SFA). His medical history revealed that he had two Wallstents (Boston Scientific, Marlborough, MA, USA) implanted in his bilateral common iliac arteries 14 years ago. The patient’s risk factors of atherosclerosis included diabetes mellitus with insulin therapy, a smoking habit, and hypercholesterolemia. He took 100 mg of aspirin daily over the 14-year period and had statin therapy for several years. Aortography showed mobile translucency in the Wallstent in the right iliac artery (Fig. 1). We observed this mobile translucency using intravascular ultrasound (IVUS) and angioscopy (Visible, INTER-TEC MEDICALS, Tokyo, Japan). The IVUS revealed heterogeneous regions in the thrombus and angioscopy showed a mobile solid thrombus (Fig. 2) with dendrite morphology (Fig. 2) at the side of the vessel wall and rubbing yellow plaque on the stent struts.

We scheduled femoral–popliteal artery bypass surgery for dealing with the SFA stenosis. However, we did not schedule treatment for the thrombus, because it and its stalk could not be released and distally moved. Moreover, we deemed the thrombus to be too large to remove using...
Discussion

To the best of our knowledge, this is the first report to provide in vivo angioscopic images of dendrite morphology on a mobile solid thrombus after stent implantation. Although the mechanism of solid thrombus formation in the stent is unclear, dendrite formation on the thrombus, similar to activated platelets in morphology, likely contributes to a growing thrombus in the bloodstream in the artery. This may be understood as a fractal phenomenon in that a fractal is a shape made of parts similar to the whole in some way.

IVUS images show a heterogeneous structure in the thrombus, but they do not show the thrombus’ surface. Detailed information regarding the surface was procured using angioscopy. As such, angioscopic images can provide insight into the pathophysiology of thrombus formation in the vessel. Saddle thrombi such as in the present case have occasionally been reported; however, dendrite-formed thrombi similar to activated platelets have not yet been reported.

Because this 14-year-old case had no image available for review, the morphology of the primary plaques prior to stent implantation remains unknown. Therefore, we could not determine the relationship between the primary plaques sealed by the stent and the thrombus. Nevertheless, because the thrombus had its stalk, we believe that it was formed within the stent rather than having migrated out of the stent.

We find the yellow plaque on the stent to be indicative of in-stent neoatherosclerosis. A previous angioscopic study has demonstrated that white neointima often changes into yellow plaque after four years. Angioscopic yellow neointima likely corresponds with foamy macrophages infiltrating into the fibrous cap or underlying lipid accumulation, or both. It is possible that the angioscopic yellow neointima with advanced atherosclerotic degeneration led to the aforementioned thrombotic events. It is noteworthy that the only projection on the thrombus is the yellow plaque on the stent site. Moreover, the formation of yellow neointima is not associated with the use of dual antiplatelet therapy at follow-up.

Statin therapy may control the progression of neoatherosclerosis on the stent leading to thrombus growth.

Conclusion

Angioscopic observation revealed dendrite formation on the mobile thrombus in the Wallstent, providing insight into the mechanism of solid thrombus formation.

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Disclosure Statement

All authors have no conflict of interest.

Author Contributions

Writing: TK
Data collection: SS, TK
Critical review and revision: all authors
Final approval of the article: all authors
Accountability for all aspects of the work: all authors

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Fig. 2 A series of angioscopic images show a solid thrombus in the Wallstent in the right common iliac artery (upper and middle part of Fig. 2). The bottom of the three circles in the angioscopic images in Fig. 2 show dendrite formation (white arrows) on the surface of the thrombus and rubbing yellow plaque (yellow arrows) on the stent struts. Supplementary movie is available at the online article pages on J-STAGE and PMC.