**ATHEROSCLEROSIS AS A CAUSE OF VERY LATE STENT THROMBOSIS**

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Acute MI sometimes occurs 5-10 years after the implantation of bare metal stent (BMS) at the site where BMS was implanted. Angioscopically, BMS is commonly covered by thick white neointima by 3-6 months after implantation, but the neointima usually has ruptured yellow plaque when acute MI occurs. This phenomenon has been regarded as a natural course of atherosclerosis. On the other hand, very late stent thrombosis (VLST) occurs earlier and continues to occur after drug-eluting stent (DES) implantation. Indeed, 1st generation DES (Cypher and Taxus) has poor neointima coverage and high incidence of in-stent thrombus formation, i.e., delayed healing, when observed by angioscopy at one year after implantation. Furthermore, Cypher stent is known to accelerate the formation of yellow plaques, i.e., neoatherosclerosis. However, 2nd generation DES (Xience and Nobori) are generally well covered by thin neointima and have low incidence of in-stent thrombus formation. Endeavor stent is unique because it is usually well covered by thick white neointima like BMS. VLST and late restenosis are both associated with in-stent thrombus formation, and occur more increasingly after 1st and 2nd generation DES implantation than after BMS or Endeavor stent implantation. Therefore, thrombus formation not only at the site of delayed healing but at newly ruptured plaque (neoatherosclerosis) would be the cause of VLST and late restenosis; and neoatherosclerosis would be the main cause in 2nd generation DES that rarely have delayed healing associated thrombus formation.