Neoatherosclerosis: overview of histopathologic findings and implications for intravascular imaging assessment.

Despite the reduction in late thrombotic events with newer-generation drug-eluting stents (DES), late stent failure remains a concern following stent placement. In-stent neoatherosclerosis has emerged as an important contributing factor to late vascular complications including very late stent thrombosis and late in-stent restenosis. Histologically, neoatherosclerosis is characterized by accumulation of lipid-laden foamy macrophages within the neointima with or without necrotic core formation and/or calcification. The development of neoatherosclerosis may occur in months to years following stent placement, whereas atherosclerosis in native coronary arteries develops over decades. Pathologic and clinical imaging studies have demonstrated that neoatherosclerosis occurs more frequently and at an earlier time point in DES when compared with bare metal stents, and increases with time in both types of implant. Early development of neoatherosclerosis has been identified not only in first-generation DES but also in second-generation DES. The mechanisms underlying the rapid development of neoatherosclerosis remain unknown; however, either absence or abnormal endothelial functional integrity following stent implantation may contribute to this
process. In-stent plaque rupture likely accounts for most thrombotic events associated with neoatherosclerosis, while it may also be a substrate of in-stent restenosis as thrombosis may occur either symptomatically or asymptptomatically. Intravascular optical coherence tomography is capable of detecting neoatherosclerosis; however, the shortcomings of this modality must be recognized. Future studies should assess the impact of iterations in stent technology and risk factor modification on disease progression. Similarly, refinements in imaging techniques are also warranted that will permit more reliable detection of neoatherosclerosis.