Figure 1 Vulnerable lipid rich plaque with point of rupture at the level of thin fibrous cap (TFC) and intraluminal thrombus formation. White lines indicate the interface between the lipid pool and the fibrous cap.

Fig. 2 A Red thrombus with typical high signal attenuation. B White thrombus appear like signal-rich protruding structure.
Fig. 3 Example of various types of atherosclerotic plaque. A and B Calcified plaques. C Fibrous plaque. D Lipid plaque and detail of the fibrous cap with corresponding size.

Fig. 4 Spontaneous intimal dissection.
Fig. 5  Six month follow up after primary PCI with BMS. Complete stent struts coverage (A); Incomplete stent struts coverage with co-existing struts malapposition (B).

Fig. 6  Example of late stent thrombosis. A Thrombus at proximal stent edge with coexisting malapposed and uncovered struts (arrow). B Cross section close to subocclusion with uncovered stent struts (arrows). C Cross section at the level of the thrombotic
Figure 1Fig. 7 Different patterns of in stent restenosis (ISR). A Eccentric critical in-DES restenosis on left main coronary artery with layered pattern. B Microvessel (arrow) in the contest of homogeneous ISR with high backscatter. C Concentric critical resten
Fig. 8 Example of OCT assessment of an angiographically intermediate stenosis of ostial left descending artery. The OCT-derived MLA of 3.88 mm$^2$ at the level of the stenosis cross-section indicates a significant stenosis.

Fig. 9 A Complete stent apposition. B and C Examples of stent malapposition (white arrows) of a self-expandable coronary BMS (B) and of a balloon-expandable BMS. D Stent malapposition after stent implantation on a thrombotic lesion in a patient with acute myocardial infarction.
Fig. 10  Thrombus protrusion into the stent lumen after primary PCI.

Fig. 11  Two examples of plaque shift. A Left descending artery (**) - Diagonal branch (*) bifurcation before PCI. B Plaque shift (arrow) in the same diagonal branch after PCI.
Fig.12 Intimal dissection at distal stent edge after PCI.