Fair comparison of 2 different stents would be very difficult. A commonly used method to compare 2 different stents is a prospective randomized trial, but it is not easy to completely match the background data between the groups, especially when the number of patients included is small. The best way to match the background data completely would be to include the same patients in both groups. It is impossible to include the same patients in different groups at the same time if the treatment is systemic, but it is possible if the treatment is local, such as stenting. With this method, we can evaluate the difference in the vessel reaction such as neointima formation against the different stents under identical circumstances in the same patients. Furthermore, the required number of patients for a similarly powered trial would become smaller with this method. In this issue of the Journal, Miyoshi et al. and that reported by Kim et al. both compared sirolimus-eluting stents with paclitaxel-eluting stents on optical coherence tomography (OCT) with the similar number of stents for each group; the total number of patients enrolled, however, was approximately half in the former study, and patient background was better matched in the former study.

Another question is why we evaluate neointima over DES on intra-coronary imaging. Although DES has dramatically reduced the incidence of re-stenosis and target lesion re-vascularization, it has produced a new problem of very late stent thrombosis (VLST), which has not been regarded as a problem with bare metal stent. But we do not know the mechanisms of VLST or how to evaluate the risk of VLST. Intra-coronary imaging may be able to evaluate the risk of VLST for each patient and for different DES. Then, how can we evaluate the risk? So far, no study has successfully predicted the occurrence of VLST from a specific parameter determined on imaging modalities. There are some possible mechanisms or thrombogenic sources for VLST. First, uncovered stent itself (metal, polymer, or drug) may be a thrombogenic source. Second, a ruptured plaque under the stent that has not been covered by neointima may continue to be a thrombogenic source. Third, a vulnerable plaque under the stent that has not been covered by neointima may rupture in the near future and cause acute coronary syndrome (ACS), which may be regarded as VLST. Finally, newly formed vulnerable plaque in the neointima or in the native vessel wall under the stent may rupture and cause ACS, which may also regarded as VLST. The formation of new vulnerable plaque may be promoted by DES according to the recent angioscopy and pathology results.

The frequency of uncovered stent struts measured on OCT may be a promising parameter to evaluate the risk of VLST according to other studies. Because the development of new atherosclerosis followed by new plaque rupture is also a possible mechanism of VLST, which is promoted by DES, the angioscopically detected yellow color that indicates the presence of atherosclerosis or vulnerable plaque may also be a promising parameter to evaluate the risk of VLST. Presence of thrombus detected on OCT or angioscopy may also reflect the risk. Low-density spots described by Miyoshi et al. on OCT may also be a possible parameter, although their nature is not yet clarified. Prospective studies to clarify which of these parameters can predict VLST should be performed to give clinical meanings to the results coming from the imaging studies.

References

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Cardiovascular Division, Osaka Police Hospital, Osaka, Japan
Mailing address: Yasunori Ueda, MD, PhD, Cardiovascular Division, Osaka Police Hospital, 10-31 Kitayama-cho, Tennoji-ku, Osaka 543-0035, Japan. E-mail: ueda@oph.gr.jp
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