Response to Letter Regarding Article, “Validation of Intravascular Ultrasound–Derived Parameters With Fractional Flow Reserve for Assessment of Coronary Stenosis Severity”

We appreciate Dr Layland et al for their interest, and fully agree with the concern that fractional flow reserve (FFR) may underestimate the functional significance of stenotic lesions in patients with ST-segment elevation myocardial infarction (STEMI). Increased microvascular resistance may have an influence on FFR by altering distal pressure, which is especially remarkable in the culprit lesions with slow flow. Mostly, previous observations were based on “culprits” in the setting of early after STEMI. To the contrary, our inclusion was totally different from the previous studies. Although our data included a small number of patients with non-STEMI (NSTEMI) (16 [8%] of a total 201 patients, with 19 [8%] of a total 236 lesions),1,2 all lesions were “nonculprits.” Moreover, FFR and intravascular ultrasound were performed 72 hours after onset. Because culprit vessels of infarction or thrombi-containing lesions were completely excluded as described, all showed initial thrombolysis in myocardial infarction grade 3. Furthermore, we excluded patients with STEMI as well as patients with scarring myocardium or regional wall motion abnormality of the studied vessel territories. The mean ejection fraction in the NSTEMI subgroup was 59±7%, representing a relatively small myocardial damage. Thus, we believe that the inclusion of a small number of nonculprits in patients with stabilized NSTEMI rarely affected the overall results.

When an additional analysis excludes the 16 patients with NSTEMI, the best cutoff value of minimal lumen area to predict FFR <0.80 was <2.41 mm² (89.1% sensitivity, 61.4% specificity, P<0.001), which was consistent with the data shown in the overall population. The FFR at maximal hyperemia in 19 nonculprit lesions of NSTEMI was not significantly different compared with the remaining 217 lesions without infarction (0.86±0.08 versus 0.84±0.09, P=0.43).

With regard to the impact of microvascular dysfunction in myocardial infarction on the FFR, there still remains debate. Tamita et al1 demonstrated no correlation between FFR and anatomic parameters in patients with STEMI. Conversely, McClish et al4 reported that FFR in culprit vessels was not different from noninfarct controls, indicating that recent myocardial infarction with microvascular injury may not alter FFR. As Layland et al suggest in their letter, the influence of microvascular resistance on the FFR measurement should be more validated, particularly in the settings of early versus late after myocardial infarction and in culprit versus nonculprit lesions.

Disclosures

None.

References

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