REPLY: Coronary In-Stent Restenosis in Patients Treated With Thoracic External Beam Radiation for Cancer

We thank Drs. Montone, Minelli, and Niccoli for their thoughtful letter in response to our paper (1). Our institution is a tertiary referral center, and many patients treated with external beam radiation therapy (EBRT) and percutaneous coronary intervention (PCI) with stents at the Mayo Clinic received their chemotherapy elsewhere. Because precise information regarding chemotherapy duration and specific regimen were unavailable for all patients, we chose not to include data on chemotherapeutic agents in our study. Although we acknowledged in our study limitations that the use of cardiotoxic chemotherapy may have been an unmeasured confounder with respect to adverse impact on long-term outcomes (1), we did not mention the potential of chemotherapeutic drugs to positively influence long-term stent related outcomes.

Montone et al. raise a valid point that certain antiproliferative agents (including paclitaxel and fludarabine) have been used for drug-eluting stent technology to reduce neointimal proliferation. The effects of systemic administration of these medications at high doses (such as those administered as treatment for cancer) on neointimal proliferation and stent restenosis or healing remain entirely unknown. Systemic administration of anti-inflammatory and antiproliferative medications, including prednisone, sirolimus, and colchicine, has been shown to decrease stent restenosis rates in patients with bare-metal stents when given shortly following stent implantation (2–5). It is important to note that in our study, the median intervals between PCI and EBRT (and likely chemotherapy) in both Group A and Group B patients were 3.6 and 2.2 years, respectively, whereas in the aforementioned studies, the systemic agents were administered immediately following stent implantation. Regardless, it remains possible, though unlikely, that antiproliferative chemotherapeutic agents may have affected rates of stent restenosis, particularly in patients treated with bare-metal stents. Thus, the effects of systemically administered chemotherapeutic agents on stent failure certainly merits further study.

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