Response to Letter Regarding Article, “Periprocedural Myocardial Infarction After Aortic Endarterectomy and Stenting: Systematic Review and Meta-Analysis”

We thank Dr Galyfos et al1 for their comments on our article. Unfortunately, the diagnostic criteria for myocardial infarction (MI) was unspecified in most of the included studies, leaving the possibility of misclassification bias. However, it is unlikely that differential misclassification bias based on carotid intervention has occurred and all except one of the studies, which described MI criteria met the latest definition of MI.2 When pooling absolute risks, heterogeneity is common and can result from variation in outcome definition, but most commonly from case mix. Random models are used to take this into account.

Despite variations in MI definition by international guidelines and improvement of periprocedural management, no statistically significant variation in absolute risks of MI after carotid endarterectomy (CEA) and carotid angioplasty and stenting (CAS) were observed over time. The increasing number of carotid interventions, especially in the elderly, which are more likely to have cardiac disease, may partly explain these findings.

We found that sex had a differential association with the risk of MI between CAS and CEA. We acknowledge that the reason remains unclear, mainly because of the small number of events observed in studies, precluding any analysis by sex.

We read with great interest the article, in which Galyfos et al1 stated that postprocedural myocardial ischemia occurs after CEA irrespectively from cardiac risk. However, no MI was observed and the interpretation was based on postprocedural asymptomatic troponin elevation. As expected, postprocedural troponin elevation was not uncommonly found and not correlated with MI. Although the maximum rise of troponin occurred within 24 hours after CEA, values spontaneously decreased afterward. The surprising findings of higher troponin elevation in the lower and medium cardiac risk groups compared with the higher risk group raise concern about the validity of the risk stratification score used in this analysis and the meaningful of postprocedural systematic troponin screening in asymptomatic patients after CEA.

We agree that uncertainties remain on the influence of the type of anesthesia after carotid intervention. Most of the literature comes from observational studies of small sample size. The largest randomized controlled trial published, general anaesthesia versus local anaesthesia (GALA) trial, did not show a statistically significant difference in the 30-day absolute risk of MI between local and general anaesthesia after CEA.3 Thus, it seemed difficult to conclude on the potential influence of the type of anesthesia on the risk of MI between CAS and CEA.

Most of the studies reporting a higher risk of long-term mortality in patients with postprocedural cardiac biomarkers elevation included noncarotid procedures, or combined carotid and noncarotid procedures. In Carotid Revascularization Endarterectomy Versus Stenting Trial (CREST), a significant association was identified between postprocedural MI and cardiac biomarkers elevation only and long-term mortality after CEA and CAS, compared with the absence of biomarkers elevation or MI.4 However, the numbers of cardiac events were small (42 MI and 20 biomarkers elevation only), which is more likely to introduce chance findings and several biomarkers were measured and interpreted by various laboratories instead of a central core laboratory. Although patients with carotid stenosis are likely to die from atherosclerosis complications, all-cause mortality was reported. We think that further investigation would be desirable to further understand the association between postprocedural cardiac events and long-term mortality after carotid procedures. However, numbers of cardiac events are small and stratification by factors such as age and previous coronary artery disease, requires a large sample size to show any significant difference.

Disclosures

None.

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