Response to Letter Regarding Article, “Coated-Platelets Improve Prediction of Stroke and Transient Ischemic Attack in Asymptomatic Internal Carotid Artery Stenosis”

We thank Dr Sabour1 for the interest expressed in our work. Our results demonstrate the ability to stratify asymptomatic patients with ≥50% stenosis into a low-risk category if they had <45% coated platelet levels and into a high-risk category if they had coated platelet levels ≥45%, based on prospective follow-up for ≤39 months.2 The associated risk of transient ischemic attack and stroke among the high-risk stratum was 21.5% versus 1.27% in the low-risk stratum.2 This is relevant clinically in that the risk of events in this group without revascularization treatment far outweighs the traditionally cited risk of carotid intervention.2 These findings were derived from a cohort of subjects with higher than expected rates of transient ischemic attack and stroke, and extrapolation of these results to a population of patients with a lower pretest probability of carotid-related transient ischemic attack and stroke would lead to overestimation of risk. Our results exemplify the need for study of event rates among local practice networks as compared with the use of published rates from randomized control trials.3

The ability to stratify patients into groups with such widely differing risk would suggest that a similar approach would be applicable to lower risk populations, and further study among patients with a broad range of risk factors are warranted to validate and improve the generalizability of our results.2 Dr Sabour wishes to stress what we already clarified in our discussion: “…these findings suggest that coated-platelet levels may be useful in this setting to identify a subgroup of patients at high risk for stroke for whom revascularization would be warranted or who may qualify for investigation of novel primary prevention strategies.”2 We agree with the need for a validation cohort; however, a careful reader of our discussion section is unlikely to find a misleading message. Moreover, Dr Sabour seems to minimize the clinical value of our findings. Most clinicians will understand that a test with a high negative predictive value will be useful to define the necessity of intervention, which in this case would be carotid artery revascularization. Validation of our results will be instrumental in avoiding potential complications of unnecessary procedures.

Altogether, our results provide a strong foundation for further necessary research of this subset of procoagulant platelets as one determinant of carotid-related stroke or transient ischemic attack in asymptomatic carotid stenosis.

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