

Risk Factors for Perioperative Stroke after Carotid Endarterectomy: Hypercoagulability Is the Key

NOTES

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Carotid endarterectomy (CEA) has level-1 evidence as an effective treatment for symptomatic or asymptomatic severe carotid stenosis. However, it is only with successful perioperative outcomes that CEA improves the natural history of severe carotid stenosis. Can carotid endarterectomy be effective for treating carotid stenosis? Stroke and death remain the two major adverse outcomes with the incidence of perioperative death decreasing with improved preoperative evaluation, patient selection, and anesthetic management.¹ Perioperative stroke has been traditionally attributed to surgical technique,² presentation with stroke, or certain patient comorbidities.^{3,4} In an effort to better understand the etiology of stroke after CEA, evaluation was undertaken to examine correlates of stroke after CEA in contemporary practice.

During the interval 2000 to 2004, 1,109 isolated (non-coronary artery bypass graft) CEAs were performed. The primary study end point was postoperative stroke and its associated surgical and clinical variables which were analyzed by univariate and multivariate methods. A new stroke after carotid endarterectomy occurred in 16 patients (1.4%) including 3 deaths (0.3%). The etiology of the strokes was classified into three categories: intracranial hemorrhage (1), low-flow watershed stroke (3), and thromboembolic (12, 75%) stroke. The thromboembolic strokes were identified at anesthesia emergence (2), during the early postoperative period (9, 75%; range, 0.3-8 hours), or at a delayed interval of time (1, 14 days). In this subpopulation of 12 patients with thromboembolic strokes, postoperative hypercoagulation profiles were available in 6 patients, all of which were positive. They included heparin-induced thrombocytopenia (3), elevated anticardiolipin antibody (2), and elevated (> 2.5 \times) serum homocysteine level (1).

By univariate analysis, only hypercoagulable state ($p < .001$) and prior stroke ($p < .01$) correlated significantly with development of stroke after CEA, whereas traditional variables as atrial fibrillation, congestive failure, contralateral occlusion, diabetes, hypertension, valvular disease, and preoperative aspirin use were not found to be significant. Multivariate methods verified only hypercoagulable state ($p < .007$, odds ratio 3.21) as having a statistical difference.

The average drop in postoperative platelet count was greater (16% vs 4%) in patients with stroke ($p < .005$) verifying the hypercoagulable nature of this cohort. Three control patients with previously identified heparin-induced thrombocytopenia underwent uneventful CEA with intravenous argatroban.

These data are the first to suggest that there is a significant correlation between a patient's hypercoagulable state and the development of a postoperative stroke after CEA. Although this is not meant to minimize the importance of other etiologies that can contribute to the development of stroke after CEA, these data delineate the importance of screening subpopulations of patients at risk for thromboembolic events before CEA.

References

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