INVITED COMMENTARY

Role of Carotid Endarterectomy Following Intravenous Thrombolysis

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These Danish surgeons report outstanding 30-day outcomes for carotid endarterectomy (CEA) in the management of symptomatic carotid stenosis. Their review is focused on 22 CEAs performed after thrombolysis for cerebral ischemic attacks.1 The hypothetical question asked is whether post-lytic candidates for CEA differ from those with symptomatic extracranial carotid stenoses who did not qualify for lysis. Is risk of CEA increased following lysis for acute stroke?

The 90-day efficacy of thrombolysis with the recombinant tissue plasminogen activator alteplase (rt-PA) was reviewed in a pooled analysis of US and European randomized placebo controlled trials.2 Infusion of rt-PA is beneficial within 4.5 hours of stroke onset in appropriate individuals. While one may quibble with the definition of stroke requiring 24 hours, these studies excluded minor or resolving neurological symptoms suggesting that transient ischemic attacks were not treated with rt-PA during the trials. It is unknown if indications for lysis have expanded following its widespread introduction into clinical practice.

How often is extracranial carotid stenosis found following rt-PA for acute strokes? Although their initial workup routinely included a duplex ultrasound, the incidence of carotid stenosis in the acute rt-PA population of their two referring institutions is unknown. Likewise, the frequency of carotid stenosis was not reported in the pooled trials validating rt-PA.2 The low incidence of 6% in Bartoli’s report is substantially less than the 20-30% estimated by Rothwell.3 Treatment for acute cerebral attacks has focused on intravenous rt-PA. In an effort to improve the limited success of IV rt-PA, three recent trials randomized concomitant aggressive, intracranial endovascular therapies. No significant benefit beyond rt-PA was found.4

Bleeding is a major surgical concern. The 5 minute half-life of alteplase is unlikely to affect an operative event several days later. Aggressive antiplatelet therapy offers potential for mischief in the OR. However, the unknown risk is hemorrhagic conversion of a cerebral infarct. Eligibility for rt-PA requires a head CT to exclude hemorrhage; of note, intracerebral hemorrhage following rt-PA was reported in 33%, but most were presumably asymptomatic.2 The one reported morbidity resulting from CEA following rt-PA therapy was attributed to hemorrhagic conversion; the dramatic finding of a floating thrombus in conjunction with a tight carotid stenosis following rt-PA therapy was the rationale for CEA at 33 hours.3 Balancing this unknown risk against the benefits of CEA within a 2-week time frame may be the essence of the question raised by this report. Strict, intensive control of blood pressure is recommended to decrease the risk of hyperperfusion syndrome.1–3

As lytic therapy for stroke becomes more common, vascular surgeons throughout the world will be asked to consider CEA in this setting. Since we really do not know whether post-rt-PA patients are different from those with stable strokes, and since recruitment of a sufficiently large study group is daunting, a randomized trial is unlikely. Thus, registry reports or additional reports like this are needed to establish rational guidelines for surgical intervention. While the numbers are small (N = 49), the combined data presented here suggest that CEA may be beneficial.

REFERENCES