Options for Management of Carotid Restenosis

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The basis for the treatment of extracranial cerebrovascular occlusive disease is prevention of stroke. Surgical or endovascular intervention for these lesions is warranted if it is determined that they provide a safe and more effective treatment than best medical therapy alone. Patient selection criteria for carotid endarterectomy in the treatment of primary atherosclerotic carotid artery stenosis is well recognized and widely accepted based on the results of rigorously performed prospective randomized trials. However, the indications for intervention on recurrent carotid stenosis remain less well defined.

The incidence of recurrent carotid stenosis is dependent upon the definition of recurrence but estimates range from 1 percent to 17percent of cases. Recurrent stenosis post endarterectomy is typically categorized as early or late. Early recurrence occurs at less than two years and is most commonly secondary to neo-intimal hyperplasia, although some early recurrences may actually represent residual stenosis post carotid endarterectomy due to incomplete endarterectomy or technical error. Intimal hyperplasia leading to some degree of recurrent stenosis may be identified as early as three months post-operatively and is usually apparent at six months. These recurrent hyperplastic stenoses may stabilize or progress but actual regression of the lesions has been observed as well. The intimal hyperplasia is due to smooth muscle cell and fibroblastic proliferation with deposition of a collagen rich extra cellular matrix, resulting in a smooth fibrotic, rubbery lesion. With their smooth homogenous consistency these stenotic lesions carry lower embolic potential and consequently a lower risk of stroke compared to the risk inherent to atherosclerotic lesions of a similar degree of stenosis. Thus, in light of the lower embolic risk and chance for regression of these early fibrotic lesions, the threshold for intervention tends to be higher than for similar degrees of stenosis caused by atherosclerotic disease. Recurrent stenosis following carotid endarterectomy occurs more commonly in women, those patients who had a

primary closure at the time of carotid endarterectomy, active smokers, as well as the use of tacking sutures at the end points of the endarterectomy. Atherosclerotic risk factor management may reduce the likelihood of recurrent disease but prospective randomized trials have failed to confirm any benefit of anti-platelet therapy in reducing the risk of recurrent stenosis. However, prospective randomized trials have demonstrated the value of patch angioplasty closure in reducing the incidence of early and late recurrent carotid artery stenosis.

Intervention is reserved for asymptomatic stenosis of greater than 80 percent diameter in good risks patients or for development of symptoms referable to the recurrent lesion. Options for open surgical repair of recurrent early carotid stenosis would include a patch angioplasty for localized disease, and resection with interposition grafting for more extensive diffuse disease or for second time recurrences. Lesions that develop beyond two years following carotid endarterectomy are typically atherosclerotic in nature and are usually amendable to re-do endarterectomy and patch angioplasty. If a satisfactory endarterectomy plane cannot be developed then resection with interposition grafting may be accomplished. Consideration for intervention for recurrent atherosclerotic disease will include the same clinical factors as in the primary atherosclerotic lesions.

Endovascular stenting of recurrent carotid stenosis may be the preferred treatment of choice due to the higher risk of cranial nerve injury with re-operative surgery, the lower embolic risk of intimal hyperplastic lesions, and for higher relatively inaccessible recurrent lesions. Carotid artery stenting is being performed with increasing frequency in the treatment of primary atherosclerotic carotid artery stenosis in select groups of patients. Early experience with carotid PTA alone suggested an incidence of recurrent stenosis in up to 50 percent of patients. Review of more recent series of carotid angioplasty with self-expanding stent report an incidence of re-stenosis of 2-20 percent and is related to arterial remodeling. Factors predictive of re-stenosis following carotid angioplasty and stenting include female gender, age over 75 years, implantation of multiple stents and 30 percent or greater diameter post-procedural residual stenosis. As in carotid endarterectomy patients, duplex surveillance for recurrent stenosis following carotid artery stenting is important and it has been found to be necessary to revise the

duplex criteria in defining what represents a significant recurrence in stented arteries.

Observation of changes in peak systolic velocity over time appear to be more accurate than strict absolute peak systolic flow velocity criteria in assessing recurrent stenosis and therefore it is paramount to obtain a reliable, immediate post procedure study with which to compare subsequent surveillance studies. Data suggests that recurrent stenosis following carotid artery angioplasty and stenting may be safely and effectively treated with repeat PTA.

Re-stenosis is a substantial clinical problem following open or endovascular intervention in a number of arterial beds. The incidence of recurrent stenosis following carotid endarterectomy and carotid artery stenting appear to be relatively low but should be monitored with serial duplex scan surveillance at regular intervals. The incidence may be positively impacted by meticulous attention to detail during performance of the initial therapeutic procedure, careful patient selection, and aggressive risk factor modification.

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