Recurrent stenosis post-carotid endarterectomy (CEA) is not a solitary or unusual phenomenon. Compared to the primary surgery, the reoperation is more challenging for the surgeon and frequently results in local and neurological complications. Carotid artery angioplasty with stenting (CAS) is currently being investigated as an alternative to carotid endarterectomy. In our study, ninety-nine patients underwent CAS in 110 arteries. Procedural success was 99% (109/110). Our results show that CAS treatment in post-CEA restenosis, especially with improved technique and distal protection, is safe with a low neurological complication rate, without any “local” complications and without any cranial nerve palsies. This study suggests that the future primary mode of treatment of post-CEA restenosis might be carotid stenting rather than surgery.

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METHODS

Ninety-nine patients underwent carotid artery angioplasty with stenting in 110 arteries. The average age was 70 ± 8 years; eleven patients (11%) were > 80 years old. Sixty-six patients (66%) were males. Twelve patients (12%) underwent bilateral CAS for bilateral post-CEA restenosis; five of these were in the same sitting. The average duration from CEA to CAS was 25 ± 36 months. All patients underwent a complete brachiocephalic angiography prior to stenting. Ten patients (10%) had an occlusion of the contralateral internal carotid artery. Seventy patients (70%) had hypertension, twenty-two patients (22%) had diabetes mellitus, sixty-one patients (61%) had high cholesterol and fifty-eight patients (58%) had coronary heart disease. Sixty patients (60%) were symptomatic (47 transient ischemic attacks, 13 post-cerebral vascular accidents). The mean diameter stenosis pre-CAS was 79 ± 13%. CAS was performed through the femoral approach using a coaxial system with 6 or 7 French (Fr) 90 cm sheaths, pre-dilatation, placement of the stent and stent dilatation. All patients were pre-treated with antiplatelet medication.
RESULTS

Procedural success was 99% (109/110). The mean diameter stenosis post-CAS was 5 ± 11%. The peri-procedural complication rate (30 days) was as follows: transient ischemic attacks, 2% (temporary minor neurological deficit which resolved within 24 hours); minor stroke, 2% (neurological deficit that either resolved completely within 30 days or increased the NIH stroke scale by 3; major stroke, 1% (neurological deficit that persisted over 30 days and increased NIH stroke scale by 4; mortality, 1% (non-neurological); and myocardial infarction, 1% (development of new pathological Q-waves or elevation of creatinine kinase to more than twice normal). There were no cranial nerve palsies. Since the beginning of 1997, we have had no complications in this subgroup.

Clinical follow-up was conducted on all patients through office visits or phone interviews. Thirty-day follow-up was available on all patients. Patients with periprocedural complications underwent neurological examination at that time. Research coordinators conducted regular phone interviews every 6 months. The mean time to the last follow-up was 20 ± 10 months. There were no minor or major strokes and no neurological deaths in the follow-up interval.

DISCUSSION

Recurrent stenosis after carotid endarterectomy (CEA) is a well-described clinical entity. Recreative carotid surgery for post-CEA restenosis is technically more demanding than the primary operation. Dissection through the scar tissue surrounding the carotid artery and the frequent inability to develop new endarterectomy planes within the artery are major contributing factors to this difficulty. The primary goal of the reoperation is to enlarge the lumen of the carotid artery rather than to remove the “tissue” responsible for the restenosis. This is usually accomplished by performing patch angioplasty. Frequently, a second restenosis can develop in this reoperated carotid artery with the venous patch. These are also very difficult to deal with surgically.

The incidence of recurrent stenosis after CEA is controversial. Initially, the incidence of restenosis was based primarily on patients with recurrent neurological symptoms who underwent angiography. This restenosis rate was considered to be in the range of 4% of the post-CEA population. This method failed to include a much larger group of patients who did not present with symptoms. With routine non-invasive testing (duplex ultrasound, CT angiography, MR angiography), the incidence of post-CEA restenosis appears to have increased markedly from the early series. This incidence in contemporary reports ranges from 1.2–36%. Some authors also reported an even higher incidence of secondary restenosis after repeated CEA.

Increased complication rates following repeat CEA in comparison with primary surgery have been well documented. The complication rate reported varies between 8–20%. An extremely low complication rate (0%) has been reported. In most of the reports, only neurological complications are enumerated and the local and cranial nerve palsy complications are omitted. The post-CEA restenosis subgroup has been excluded from all previously performed randomized trials comparing CEA to medical therapy because of the perceived increased risk.

Our results show that CAS treatment in post-CEA restenosis, especially with improved technique and distal protection, is safe with a low neurological complication rate, without any “local” complications (i.e., wound healing, hematoma, infection) and without any cranial nerve palsies. This study suggests that in the future the primary mode of treatment of post-CEA restenosis might be carotid stenting rather than surgery.

REFERENCES

Carotid Angioplasty with Stenting in Post-CEA Restenosis