Review Article

Surgical Treatments of Stenosis of the Internal Carotid Artery

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There are three categories among ischemic cerebrovascular diseases. One is lacunar infarction, another is atherothrombotic infarction, and the last is cardioembolic infarction. Strokes from atherothrombotic origin are increasing in Japan. Stenosis of the cervical carotid artery is a typical atherothrombotic disease. Carotid endarterectomy have been a choice of treatment for carotid stenosis; however, carotid stenting is developing as an alternative treatment for carotid stenosis. In this review, indication and operative techniques of carotid endarterectomy are presented.

Keywords: Carotid endarterectomy; Carotid stenting; Carotid stenosis

Introduction

Although the death rate of cerebrovascular diseases in Japan has decreased in recent decades, number of medical care is increasing remarkably. Approximately 300 patients per 100,000 persons per year have medical cares. This rate is three fold higher compared with medical care bill for ischemic coronary diseases. Among ischemic cerebrovascular diseases, incidence of atherothrombotic type is increasing. In contrast with this, incidence of arteriosclerotic lacunar stroke has decreased especially in male.

Elevated serum cholesterol level in recent Japanese population may be the cause of change. Stenosis of the internal carotid artery is one of the main lesions of the atherothrombotic cerebrovascular diseases. Approximately 4,000 patients with carotid stenosis may be treated surgically per year in Japan. Carotid endarterectomy is major surgical procedure for carotid stenosis; however, carotid stenting has become an alternative procedure in recent years.

Indication of carotid endarterectomy

Symptomatic carotid stenosis

In Japanese Guidelines for the Management of Stroke 2004,1 carotid endarterectomy (CEA) is strongly recommended (grade A) for symptomatic severe carotid stenosis (stenosis over 70%), and is recommended (grade B) for those with moderate carotid stenosis (stenosis of 50-69%). There are no controlled randomized trial (CRT) in Japan concerning CEA. Thus, these recommendations were based on CRTs of western countries. The major CRT of CEA is North American Symptomatic Carotid Endarterectomy Trial (NASCET).2 NASCET showed effectiveness of CEA for symptomatic carotid stenosis over 70%. The absolute risk reduction (ARR) of any ipsilateral stroke and death was 19.4% at 3 years after CEA. The relative risk reduction (RRR) was 68.5%. For patients with stenosis of 50-69%, ARR was 6.5%, and RRR was 29.2%. Surgical risk should not exceed 6%. The number needed to treat (NNT) was 5 and 15 for stenosis over 70% and that of 50-69%, respectively. In Guideline for CEA of American Heart Association (AHA), proven indication of CEA is for patients with carotid stenosis over 70%, and acceptable indication is for those with stenosis of 50-69%.3 NNT for stenosis of 50-69% tells us that surgical indication for mild carotid stenosis may be uncertain if surgical morbidity increased. Thus, from the viewpoint of health economics, CEA for moderate carotid stenosis might not pay.

Asymptomatic carotid stenosis

Japanese Guidelines for the Management of Stroke 2004 recommends CEA for asymptomatic patients with severe carotid stenosis over 60% (grade B). This recommendation is mainly based on the results of Asymptomatic Carotid Atherosclerosis Study (ACAS).4 In this study, ARR of ipsilateral stroke and death was 5.9% at 5 year, and RRR at 5 year was 53.6% (p=0.004). However, NNT was 17, which is larger than that of CEA for symptomatic carotid stenosis.
of 50-69%. ARR of disabling or fatal stroke with CEA was 2.7%, which is not statistically significant (p=0.26). NNT to prevent ipsilateral major stroke plus perioperative stroke, and death was 38. Perioperative stroke and death was 2.3% in ACAS. AHA proved the indication of CEA for asymptomatic carotid stenosis over 60% if the surgical morbidity-mortality does not exceed 3%.^7^ Recently, results of another CRT, ACST (Medical Research Council Asymptomatic Carotid Surgery Trial), have been published.^8^ This study proved the effectiveness of CEA for the patients with asymptomatic severe carotid stenosis over 60%. In this study, ARR of any disabling stroke and death was 2.5%, which is statistically significant (p=0.004). However, NNT for any stroke and death was 19, and that for disabling stroke and death was 40. An additional result of ACST is that CEA was proved to be effective for asymptomatic female patients. In the United States, approximately 150,000 CEA's are performed every year and at least half of them are for asymptomatic carotid stenosis.

Although large CRTs proved the effectiveness of CEA for asymptomatic patients with severe carotid stenosis, the cost-effectiveness should carefully be studied in near future. If high risk asymptomatic carotid plaques could be diagnosed precisely, the cost-effectiveness of CEA for asymptomatic carotid stenosis will increase.

**Techniques of carotid endarterectomy**

Basic concepts of CEA are: (1) to prevent ischemic events during carotid clamping; (2) to prevent embolic complication during procedure; (3) to remove completely the plaque especially one at distal end; (4) to preserve nerves in the operative field; and (5) to suture arteriotomy securely. Cerebral ischemia may be caused by carotid clamping. Some surgeons always use internal shunt, while others never use it. We use the shunt selectively according to the intraoperative carotid occlusion test. Tolerance against carotid clamping is checked by intraoperative monitoring. Measuring of the stump pressure, transcranial Doppler spectroscopy (TCD) and near-infrared spectroscopy (NIRS) are physiological monitors. On the other hand, electroencephalography (EEG) and sensory evoked potential (SEP) are electrophysiological monitors which are functional. We use SEP, stump pressure and NIRS for intraoperative monitoring. NIRS is also very useful for detecting post-operative hyperperfusion syndrome.

Systemic heparinization, careful dissection of vessels and enough washout of debris or air before declamping may prevent distal embolism.

Carotid bifurcation is high in Japanese patients compared with western patients. It is essentially important to dissect the internal carotid artery high enough to remove atherosclerotic plaque completely. Otherwise, residual plaque may cause acute occlusion of the vessel or may result in restenosis. Transnasal intubation, dissection or cut of the digastic muscle, and cut of the descending rami of hypoglossal nerve or the occipital artery are the techniques to approach distal segment of the internal carotid artery.

Secure suturing of vessels including adventitia is important step to reduce postoperative bleeding.

**Complications of carotid endarterectomy**

Surgical morbidity plus mortality should be lower than 3% in asymptomatic patients and 6% in symptomatic patients with carotid stenosis. However, there are several types of surgical complications with CEA: ischemic, hemorrhagic, wound and systemic complications. Ischemic and wound complications have been mentioned above. Post-operative intracerebral hemorrhage may be caused by hyperperfusion syndrome and by recanalization after embolic complications.

Hyperperfusion syndrome consists of headache, seizure, and intracerebral hemorrhage. Incidence of intracerebral hemorrhage after CEA is approximately 0.5%.^3^

Risk factors of hyperperfusion syndrome are decreased vascular reserve, uncontrolled hypertension, history of recent stroke and use of anticoagulants.^9^ The most important systemic complication is myocardial infarction which is a major causative factor of peri- and post-operative mortality. Approximately 30% of the patients with significant carotid stenosis have coronary disease in Japan. Thus, preoperative assessment of coronary artery steno-occlusion is essential. Perioperative morbidity plus mortality of CEA is quite different among clinical categories.^9^ The absolute risk (morbidity plus mortality) of CEA is 5.1% for symptomatic patients, 2.8% for asymptomatic patients and 19.2% for patients with ongoing symptoms. CEA for restenosis is 1.95 times higher than that for primary lesion. But there is no difference in the risk between patients with stroke and those with transient ischemic attacks.

**Restenosis after carotid endarterectomy**

Restenosis after CEA usually means stenosis over 50% in diameter. Mild recurrent carotid stenosis with symptoms is also regarded as restenosis. Restenosis after CEA usually occurs within 2 years with the rate of approximately 10%.^10^ This rate is quite small compared with that of coronary restenosis after percutaneous transluminal angioplasty or stenting. Restenosures after CEA are known rarely symptomatic. Restenosis is a result of myointimal hyperplasia after CEA and may occur within 2 years postoperatively. Re-CEA for carotid restenosis is risky as mentioned above. However, these restenoses are treated with acceptable morbidity by carotid stenting. Female patients, younger patients, CEA with primary closure, small caliber of the carotid artery and residual hemodynamic abnormality are thought to be causative factors of carotid restenosis. There are several methods for preventing carotid restenosis: patch graft and eversion endarterectomy are being tried to lower the risk of restenosis.
Carotid stenting

In recent years, carotid stenting (CAS) has been accepted as an alternative method for treating carotid stenosis. Distal embolism was a major disadvantage to CEA in the initial experiment. However, many protection devices have been developed to prevent distal migration of plaque debris or thrombi and CAS is spreading as an alternative procedure for the treatment of carotid stenosis. Although CAS is not accepted in medical treatment insurance in Japan, 1/3 of patients with carotid stenosis are now treated with CAS. CAS is thought to be superior to CEA in comorbid patients because CAS can be performed without general anesthesia. In the recent CRT, 11 CAS with emboli-protection device is proved not inferior to CEA for the treatment of patients with medical risks. Criteria for high risk were: (1) clinically significant cardiac disease; (2) severe pulmonary disease; (3) contralateral carotid occlusion; (4) contralateral laryngeal palsy; (5) previous treatment of neck by surgery or radiation; (6) recurrent carotid stenosis; and (7) aged over 80 years. In this study, perioperative stroke or death plus ipsilateral stroke or death was fewer in CAS group (5.5%) than in CEA group (8.4%) (p=0.36). However, CAS is inadequate to tortuous or highly calcified lesions. Both of aortic atheromatous plaque and presence of peripheral artery disease are also contraindications for CAS.

References