Mid-and Long-term Clinical Efficacy Analysis of Carotid Artery Stenosis Treated With Carotid Endarterectomy

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Research article

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Abstract

Objective: To explore the mid-and long-term clinical efficacy analysis of carotid artery stenosis treated with carotid endarterectomy.

Methods: Retrospective analysis of 89 cases of patients with carotid artery stenosis

undergoing carotid endarterectomy from our center from Jan,2013 to June,2017. To gather the hospitalization data of patients including the general information, the past medical history, preoperative conditions, the situation during surgery, postoperative situations. All the patients were followed up from 16 months to 63 months, and to make survival analysis on the follow-up status.

Results：Among the 89 cases, 22cases of mild stenosis ,67cases of severe stenosis. 5 cases had postoperation wound hemorrhage or hematoma, 2 cases of cranial nerve injury, 2cases of cerebral stroke. Among the 62 cases of symptomatic carotid artery stenosis,48cases have been improved in term of their clinical symptoms with improvement rate of 77.4%. The Nonparametric test of mRS scores before and after surgery showed that there was significant difference in the preoperative and postoperative scores （P<0.05）.

Conclusion: The mid-and long-term clinical efficacy of carotid artery stenosis treated with carotid endarterectomy is good. The previous history of coronary heart disease and peripheral vascular diseases are the influence factors for the mid- and long-term adverse outcomes after carotid endarterectomy.

Introduction

With the intensification of population aging, stroke has become one of the common cardiovascular and cerebrovascular diseases in the elderly. According to the statistics of the World Health Organization, stroke has become the third disease that endangers human health and life safety, with characteristics of high incidence and high mortality[1]. Among them, the incidence of ischemic stroke accounts for 60–80% [2], and carotid artery stenosis (CAS) is one of the common factors leading to ischemic stroke [3].

90% of the causes of carotid stenosis are atherosclerotic occlusion. Carotid stenosis can cause severe cerebral ischemia, even ischemic stroke, which makes patients' life severely restricted and even can not take care of themselves in their daily life,causes the disability and the high mortality. Therefore, improving the blood supply to thepatient’s brain is important for prolonging patients’ lifespan and improving their quality of life. Carotid atherosclerotic occlusive stenosis usually occurs at the bifurcation of the common carotid artery, especially the bulb of the carotid bifurcation. Carotid atherosclerotic lesions cause cerebral ischemia mainly through the following two mechanisms: plaque or thrombus shedding leads to intracranial artery embolism; stenosis causes distal brain tissue blood flow hypoperfusion. In recent years, studies have shown that carotid stenosis causing ischemia and hypoperfusion leads to a lower incidence of stroke, the vast majority of cerebral ischemic lesions are cerebral infarction caused by plaque shedding. Carotid stenosis is common in middle and old age people, often causes ischemic

symptoms in the brain and plaque or thrombus shedding leading to transient cerebral ischemia and cerebral infarction.

Currently recognized methods for the treatment of carotid stenosis include drug-based medical treatment and surgical treatment based on carotid endarterectomy (CEA) or carotid stenting (CAS). It has been more than 60 years since De Bakey successfully performed the first CEA operation in 1953.With several large-scale multicenter prospective randomized trials: the North American Symptomatic Carotid Endarterectomy (NASCET) [4], asymptomatic carotid atherosclerosis study (ACAS) [5], European carotid surgery test (ECST) [6], Veterans Administration symptomatic carotid endarterectomy test (VAST) [7], all the above trials have proven the efficacy and safety of CEA, and determined the superiority of CEA in the prevention and treatment of stroke from the perspective of evidence-based medicine, CEA is also considered to be the "gold standard" for the treatment of carotid stenosis [8], with the development of CEA in patients with carotid stenosis, more and more patients with carotid stenosis benefit from it. Compared with conventional drug therapy, CEA can effectively reduce the incidence of ischemic stroke caused by carotid stenosis. The American Heart Association (AHA) [9] localizes carotid endarterectomy to grade 1 recommendations. Grade A evidence clearly states that for patients with symptomatic carotid stenosis, carotid endarterectomy is preferred, and stent implantation is considered only when plaque is not available in surgery, the internal carotid artery stenosis closed to the skull base or the proximal stenosis of the common carotid artery, or when the surgical risk is high. Carotid endarterectomy is the only method of removing plaque and reconstructing normal lumen and blood flow.

Although the effectiveness and safety of carotid endarterectomy have been affirmed, there are differences in outcomes after carotid endarterectomy. At the same time, with the development of interventional techniques and the development of vascular instruments, carotid stenting has the advantages of short operation time, small trauma and quick recovery. Some scholars believe that carotid artery stenting is an ideal alternative to treatment. Therefore, in order to understand the efficacy of carotid endarterectomy, reduce the incidence of complications, improve the safety and effectiveness of carotid endarterectomy, this article retrospectively analyzed the detailed information, postoperative complications, and postoperative follow-up of patients with carotid stenosis undergoing carotid endarterectomy in the Department of Vascular Surgery, Second Affiliated Hospital of Nanchang University from January 2013 to June 2017, to provide suggestions for reducing the occurrence of postoperative complications of carotid endarterectomy, thereby further improve the surgical outcome of carotid endarterectomy.

2 Materials And Methods

2.1 Research content

2.1.1Research objects and sources

patients with carotid stenosis who underwent carotid endarterectomy in the Department of Vascular Surgery, Second Affiliated Hospital of Nanchang University, from January 2013 to June 2017, and there

are 89 patients with complete case data.

2.1.2 Inclusion criteria

(1) unilateral or bilateral carotid atherosclerotic stenosis;

(2) symptomatic carotid stenosis >50%;

(3) Asymptomatic carotid stenosis >70%;

(4) Subjects and their families volunteered and signed the informed consent form.

2.1.3Exclusion criteria

- intracranial hemorrhage within 3 months, fresh brain infarction within 2 weeks;

(2) uncontrollable hypertension;

(3)serious tendency to hemorrhage;

(4) complete occlusion of the diseased lateral carotid artery;

(5) intracranial aneurysm which can not be treated in advance or concurrently;

(6) carotid bifurcation position is high or the common arteries are narrow below the clavicle plane.

2.2 Research methods

2.2.1 Preoperative examination

(1)After admission, the patients' blood pressure, pulse and body temperaturewas monitored according to the routinely nursing of vascular surgery.

(2)Improve the examination of blood routine, urine routine, stool routine, blood type, liver and kidney function, blood sugar, coagulation function, etc.,and rule out severe liver and kidney dysfunction and hemorrhagic disease with bleeding disorders.

(3)Preoperative chest X-ray examination and electrocardiogram examination excludes patients with severe cardiopulmonary disease.

(4)Preoperative examinations were performed such as carotid ultrasound, CT angiography or digital subtraction angiography, cranial magnetic resonance imaging, magnetic carotid plaque analysis, craniocerebral perfusion, etc., learn more about the location, extent of carotid stenosis and the charactor of carotid plaques, etc.

2.2.2 Preoperative preparation

(1)Understand the patient's medical history, confirm the diagnosis, and eliminate the surgical contraindications.

(2)Before operation examinate imaging datas of patients like carotid ultrasound, CTA and magnetic resonance , to determine the location and extent of carotid stenosis, stenosis, plaque stability, etc., to guide surgical treatment.

(3)Preoperative oral aspirin or Plavix antiplatelet therapy, oral statin to stabilize plaque, reduce blood lipid treatment, strict control of hypertension, diabetes, hyperlipidemia.

(4)Control risk factors, adjust blood sugar, control blood pressure; quit smoking before surgery, exercise deep breathing, improve breathing function, practice defecating in bed and so on.

(5)Preoperative blood preparation, cross-matching blood to prevent intraoperative bleeding.

(6) Cessation of oral aspirin treatment one day before surgery.

2.2.3 surgical methods

(1)All patients underwent valgus endarterectomy and intravenous anesthesia with endotracheal intubation. The patients were supine, the scapula was high, the head was biased to the opposite side of the surgery field, and a cricoid cushion is placed under the head. The front area of the neck is exposed.

(2)Made oblique vertical incision along the anterior border of the sternocleidomastoid muscle, and then cut the skin, subcutaneous and platysma muscles in proper order, and did longitudinally separation along the anterior border of the sternocleidomastoid muscle, and pull the sternocleidomastoid muscle to the outside. After the carotid sheath was exposed, opened the carotid sheath, and the common carotid artery, internal carotid artery and external carotid artery were exposed freely to avoid injury to the nearby vagus nerve and hypoglossal nerve. In the relatively normal position without hardened plaque, the superior thyroid artery, external carotid artery, internal carotid artery, common carotid artery were blocked respectively. 1% lidocaine closed the carotid sinus and peripheral nerves, after systemic venous heparinization (0.5~1.0mg/ml), made longitudinally incision of the common carotid artery and internal carotid artery wall, stripped carotid intima and plaque, carefully removed the plaque and endometrial tissue from the wall until the vessel wall was smooth and after the distal endometrium was trimmed, it was fixed by suture. The arterial wall was sutured continuously using 5-0 non-invasive vascular suture, and the occlusion forceps of the external carotid artery, common carotid artery, internal carotid artery and superior thyroid artery were sequentially opened.

(3)The blocking time was 20-36 minutes, with an average of 26 minutes. Only one case was treated with a diverter tube and a patch. None of the ramaining cases used drainage tube or patch suture. After the occlusion forceps were removed, 125 ml of mannitol was used to reduce intracranial pressure. Protamine was injected to deheparinized and the ACT index was monitored. The incision was sutured layer by layer

after strict hemostasis, and draining tube was placed in the neck region to drainage . Surgical process is showen in the chart.

2.2.4 postoperative treatment

The patients were sent to the recovery room to wake up. After waking up, they were sent to the general ward or ICU for monitoring.They were given ECG and oxygen saturation monitoring, observed changes in patient consciousness, vital signs and wound drainage, strictly controlled blood pressure (100-130) / (70-90) mmHg, and use mannitol (125ml / time, twice a day for 3 days) to reduce intracranial pressure, postoperative aspirin combined with Plavix double antiplatelet therapy,parents were treated by oral Atto vastatin calcium tablets for hypolipidemic therapy and changed to oral aspirin antiplatelet therapy 6 months later.

2.2.5 Data collection

The clinical data of the subjects were retrospectively collected and collated; the general clinical data of the patients included age, gender, life history (whether smoking, drinking) combined with underlying diseases and risk factors (hypertension, diabetes, coronary atherosclerotic heart history, cerebral infarction, history of peripheral arterial disease). Before operation, CTA, craniocerebral MRI, carotid MRA and other examinations were preformed, improved mRS score was used 1 day before surgery;

Surgical treatment included intraoperative and perioperative complications; follow-up was performed through outpatient, inpatient, or telephone follow-up. Follow-up included complications, stroke, and symptom improvement and improved mRS score.

2.2.6 Determination of risk factors and complications

(1)Hypertension: systolic blood pressure is greater than or equal to 140mmHg and/or diastolic blood pressure is greater than or equal to 90mmHg,with previous history of hypertension and oral antihypertensive drugs.

(2)Diabetes: Symptoms of diabetes plus plasma glucose at any time is greater than or equal to 11.1 mmol/L, or FPG is greater than or equal to 7.0 mmol/L, or OGTT2h PG is greater than or equal to 11.1 mmol/L,with previous history of diabetes and treatment with insulin or oral hypoglycemic agents.

(3)Coronary atherosclerotic heart disease: coronary atherosclerosis causes stenosis or obstruction of the lumen, or (and) heart disease caused by myocardial ischemia or necrosis due to functional changes in the coronary artery (spasm), referred as Coronary heart disease, including acute coronary syndrome and chronic coronary disease.

(4)TIA: transient cerebral ischemic attack, including transient amaurosis, vision loss, visual field defect, or transient vertigo, unstable standing and other symptoms.

(5)Modified mRS score**(Table 1)** [10].

(6) Judgment of carotid stenosis: according to the North American Society of Carotid Surgery (NASCET) criteria [4]: mild (0-29%), moderate (30-69%), severe (70-99%). The calculation formula for the degree of stenosis:(1 - the narrowest diameter of the carotid artery / the diameter of the normal internal carotid artery at the distal end of the stenosis lesion) x 100%.

(7) Symptomatic carotid stenosis: A transient or persistent function defect of ipsilateral cerebral hemisphere or retina associated with stenotic vessel has occurred in the past 6 months. Symptoms include: numbness and weakness in the body and face, dysarthria, aphasia, visual field defect, and transient amaurosis.

(8)Hyperperfusion syndrome [11] is defined as beating pain in the forehead, ankle and eye socket with or without nausea and vomiting, orimaging failure to indicate unilateral focal seizure of cerebral infractionor focal nerve function defect.

(9)Follow-up endpoints: major cardiovascular and cerebrovascular events such as death, stroke or myocardial infarction.

(10)Judgment of cranial nerve injury: Cranial nerve injury was defined as 8, 9, 10, 11, 12 and sympathetic nerve injury. Mandibular branch paralysis often leads to distortion of commissure defined as facial nerve branch injury. Glossopharyngeal disorders are defined as uvula deviation and dysphagia. Assessment of damage of vagus nerve and its branch includes dysphagia, hoarseness, vocal cord paralysis, etc. accessorius injuries include sternocleidomastoid and trapezius muscle defects. Sublingual nerve injury is characterized by a shift of the tongue to one side.

2. 3 statistical analysis

Statistical analysis was performed using SPSS23.0 statistical analysis software. The measurement data were expressed by mean and standard deviation (X±S), and the count data was expressed by the number of cases and percentage; the measurement data of non-normal distribution was measured by Wilcoxon rank sum test. Comparisons of counting data were made using chi-square test; survival analysis was performed with Kaplan-Meier method, and Cox proportional hazard regression model was used for multivariate analysis. Statistical significance was expressed as $p< 0.05$.

Table 1
Modified mRS score

score	Description
0	Completely asymptomatic
1	Despite symptoms, it has no obvious dysfunction and can complete all daily work and life.
2	Mild disability, unable to complete all pre-illness activities, but do not need help to take care of their daily affairs
3	Moderate disability, need some help, but can walk independently
4	Moderately and severely disabled, unable to walk independently, need help in daily life
5	Severe disability, bedridden, incontinence, complete dependence on others in daily life
6	Dead

3 Results

3.1 General information

There were clinical data of 89 patients with carotid stenosis undergoing carotid endarterectomy in Department of Vascular Surgery, Second Affiliated Hospital of Nanchang University, from January 2013 to June 2017, including 68 males and 21 females, age 52–85 years old, with an average of 66.8 ± 6.4 years old. There were 9 cases of unilateral lesions, 80 cases of bilateral lesions, 76 cases of hypertension, 26 cases of diabetes, 13 cases of coronary heart disease, 29 cases of smoking history, and 19 cases of drinking history. There were 62 cases of symptomatic carotid stenosis, including 32 cases of limb weakness, 41 cases of TIA, and 16 cases of blurred vision. The patient data and general information are shown in Table 2.

Table 2
Basic data of patients

	Total number of cases (N)	Percentage (%)
Age (years old)		
50–60	15	16.9
60–70	52	58.4
70–80	19	21.3
80–90	3	3.4
Gender		
male	68	76.4
female	21	23.6
Tobacco and alcohol	29	32.6
Drinking	19	21.3
Hypertension	76	85.4
Diabetes	26	29.2
coronary heart disease	13	14.6
History of stroke	41	46.1
History of peripheral arterial disease	29	32.6
Side number of carotid artery lesions (case)		
Unilateral lesion	9	10.1
Bilateral lesion	80	89.9
Degree of carotid stenosis (case)		
moderate	22	24.7
severe	67	75.3
Symptomatic carotid stenosis (case)	62	69.7
Limb weakness	32	
TIA	14	
Blurred vision	16	

3.2 Perioperative complications of the treatment of carotid stenosis with CEA:

Postoperative wound hemorrhage or hematoma in 5 cases (5.6%) (2 cases with neck hematoma evacuation, 3 cases improved by conservative treatment); 2 cases (2.2%) with cerebral infarction, all improved after conservative treatment, no sequelae occurred; cranial nerve injury in 2 cases (4.5%) (1 case of hypoglossal nerve injury, 1 case of facial nerve mandibular branch damage),improved after half a month of conservative treatment with nutritional nerves and other drugs; there was no death in this group of patients.

3.3 Effect of carotid endarterectomy for the treatment of carotid stenosis

Eighty-nine patients underwent 92 carotid endarterectomy with a success rate of 100%. The preoperative 1dmRS score was 0 in 17 cases, 1 in 35 cases, 2 points in 22 cases, 3 points in 13 cases, and 4 points in 2 cases. MRS score at the time of discharge was 0 in 39 cases, 1 in 20 cases, 2 in 19 cases, 3 in 8 cases, and 4 in 3 cases. In 62 patients with symptomatic carotid stenosis, 48 patients' symptoms had improved at the end of follow-up, and the improvement rate of symptom was 77.4%. The patient's mRS scores on 1 day before operation and at the discharge time were tested by self-matched nonparametric tests. The analysis showed that the m RS scores were different before and after carotid endarterectomy ($P < 0.05$).

3.4 Follow-up

A total of 89 patients were counted and followed up by outpatient or telephone to understand the improvement of symptoms, mRS score, whether having stroke, and perform color Doppler ultrasound. The incidence and timing of stroke, myocardial infarction, and death were recorded. The main adverse events were stroke, postoperative restenosis, myocardial infarction, and death. Postoperative restenosis is defined as surgical side carotid artery ultrasonography suggesting that vascular stenosis is greater than or equal to 50%. The presence and absence of clinical features were studied by KM survival analysis and COX regression analysis. It was considered statistically significant,when $P < 0.05$. There were 81 patients with complete data, the total follow-up rate was 91.0%, the follow-up period was 10 months to 63 months, and the median follow-up time was 29 months.

3.4.1 A total of 3 patients died during the follow-up period. One patient died of sudden acute myocardial infarction 1 month after surgery, 1 patient died of large-area cerebral infarction 10 months after surgery, and 1 patient died of duodenal papillary cancer 30 months after surgery; 1 case of repeated dizziness for 3 months after operation, found acute stenosis by reexamination of MRI , improved after conservative treatment with drugs; 1 case of sudden cerebral infarction after 25 months of surgery improved after conservative treatment with no sequelae left .One patient found a carotid stenosis 41 months after operation, and the stenosis rate was about 60% with no obvious symptoms. The patient data of follow-up are shown in the **Table 3**.

3.4.2Risk factors for patients Kaplan-Meier survival analysis during the single-factor follow-up period showed that the risk factors for cardiovascular events were the presence of coronary heart disease and peripheral vascular disease. Survival analysis was performed for various risk factors such as gender, hypertension, diabetes, coronary heart disease, history of peripheral arterial disease, degree of carotid

stenosis, number of carotid stenosis, preoperative symptoms, stroke history, smoking, and drinking. Survival analysis showed that coronary heart disease (*P*=0.048) and PAD (*P*=0.028) were important factors influencing the prognosis of patients.

3.4.3Establish a Cox proportional hazard regression model for multivariate analysis of follow-up results, see **Table 4**. Coronary heart disease and peripheral arterial disease are important factors influencing the prognosis of patients.

Table 3
Basic information of follow-up patients

	Total number of cases (N)	Number of follow-up endpoints	P
Age (years old)	66.8 ± 6.4	66.5 ± 5.6	0.108
50–60	11	0	
60–70	51	2	
70–80	17	4	
80–90	2	0	
Gender(male)	64	6	0.334
Tobacco	24	1	0.66
Drinking	17	0	0.334
Hypertension	70	4	0.186
Diabetes	22	0	0.182
coronary heart disease	13	3	0.049
History of stroke	38	4	0.432
History of peripheral arterial disease	27	5	0.024
Symptomatic	60	5	1
Bilateral lesion	74	4	0.615
Moderate stenosis	20	1	1

Table 4
Cox proportional risk regression model

Covariant quantity	*P*	HR
coronary heart disease	0.048	6.435
Peripheral vascular diseases	0.028	14.991

4 Discussion

4.1 Carotid endarterectomy for the treatment of carotid stenosis is safe and effective

Stroke is an important disease that jeopardizes the health of the elderly, and carotid stenosis is one of the most common causes of ischemic stroke. The most common cause of carotid stenosis is atherosclerosis, Distal arterial embolization caused by carotid stenosis and carotid plaque detachment caused by carotid atherosclerotic plaque. is an important cause of ischemic stroke. In 1953, Debakey and colleagues successfully performed carotid endarterectomy for the first time for patients with primary carotid artery stenosis.The patient's symptoms were improved, and the method was controversial due to the higher complications and mortality of carotid endarterectomy in early years.With the development of surgical techniques and equipment in recent decades, several large randomized clinical trials in the 1990s confirmed the effectiveness of carotid endarterectomy. Long-term follow-up of the North American Symptomatic Carotid Endarterectomy Trial (NASCET) has shown that CEA surgery significantly reduces the risk of ipsilateral stroke in patients with symptomatic carotid stenosis. The European Carotid Surgery Trial (ECST) and the Veterans Affairs Cooperative Study (VACS) also reached similar conclusions. Compared with conservative treatment of drugs, carotid endarterectomy can significantly reduce the incidence of ipsilateral ischemic stroke, and the curative effect is exact. Therefore, carotid endarterectomy is the gold standard for the treatment of carotid stenosis. The latest AHA/ASA guidelines point out [12],when imaging exams the ipsilateral carotid artery is severely stenotic, and the perioperative disability and mortality are estimated to be less than 6%,carotid endarterectomy is recommended. For patients with moderate stenosis, carotid endarterectomy may be feasible if conditions permit.Carotid endarterectomy is currently a safe and routine procedure for the treatment of carotid stenosis. As a preventive treatment, carotid endarterectomy must be safe to demonstrate its advantages. Therefore,in the guidelines of the American Hearth Association (AHA), there is a requirement for the incidence of perioperative complications of carotid endarterectomy, which requires that the incidence of perioperative stroke and death in symptomatic patients is less than 6%, and that in asymptomatic patients is less than 3%. In the NASCET study published in 2000, the incidence of perioperative stroke and mortality were 5.5% for carotid endarterectomy. In 2013, domestic Jiao Liqun et al [13] reported that the incidence of adverse such as perioperative death and stroke was 3.93% in 494 multicenter cases. There were no deaths in the perioperative period in this group of patients, and only 2 patients had perioperative stroke. The incidence of perioperative stroke and death in this group was 2.2%, which was in line with the requirements of AHA, and was lower than that of NASCT and research results of domestic Jiao Liqun et al. In this study, the preoperative and postoperative mRS scores were significantly improved. For 62 patients with symptomatic carotid stenosis, 48 patients had improved symptoms at the end of follow-up, and the symptom improvement rate was 77.4%.

4.2 Occurrence and preventive measures of carotid stenosis complications

As a preventive procedure, prevention and treatment of carotid endarterectomy postoperative complications is very important. Perioperative complications of CEA mainly include stroke, cerebral

hyperperfusion syndrome, myocardial infarction, cranial nerve injury, and incision hematoma [14]. Postoperative complications need to be discovered timely and treated accordingly.

4.2.1Postoperative stroke

Postoperative stroke is one of the more serious complications after carotid endarterectomy, and is mainly about prevention. There is no uniform evidence for the relevant causes of postoperative stroke. Carotid stenosis, initial symptoms, plaque properties, single and bilateral stenosis, surgical procedures, anesthesia procedures, and application of patches may be associated with postoperative stroke. Data from NASCET and ECST showed no significant association between stroke and stenosis within 30 days after surgery, but the incidence of postoperative stroke was found to be reduced in patients with near-occlusive occlusion. There is currently no clear explanation for this phenomenon, Rothwell puts forward two possible hypotheses,one is that the severe stenosis of the distal carotid artery leads to a slower blood flow velocity and cannot transport the embolus to the brain,another is low perfusion area is less likely to produce embolus. Plaque properties were associated with postoperative stroke, and NASCET studies showed that the incidence of postoperative stroke in the unstable plaque group was approximately 1.5 times that of the stable plaque group, although postoperative stroke in the unstable plaque group was increased due to CEA surgery. The general, CEA still has more harm than good, reducing the incidence of stroke by at least 50%.Patients with bilateral carotid stenosis, especially those with severe stenosis or complete occlusion on the contralateral side, are more likely to have stroke after surgery than patients with contralateral mild to moderate stenosis or unilateral stenosis. NASCET and ACE come to the same in conclusion. Some studies have shown that local anesthesia and the use of patches can reduce the incidence of postoperative stroke, but many studies have shown no correlation between local anesthesia or patch and stroke.

In addition to the patient's own situation, the occurrence of postoperative stroke is also closely related to the operation procedure. Therefore, we fully evaluate the patient's condition and carefully select the treatment method, and it is also important to carefully complete the operation in strict accordance with the technical requirements of the operation for reducing the incidenc of postoperative stroke. According to reports in the literature [15–18], the following measures can reduce the incidence of perioperative stroke: 1 adequately systemic heparinization before blocking blood vessels during operation, gentle operate during operation; 2 in the process of blocking carotid artery and endarterectomy, the blood pressure should be raised appropriately to ensure good cranial perfusion; 3 Detection of craniocerebral perfusion by TCD during operation, patients with poor intracranial compensation can choose a diversion tube to avoid long-term hypoperfusion; 4 repeated irrigating Peel surface during operation, remove the floating inner membrane and debris; 5 fix the distal carotid intima, after the carotid incision suture, check the pulsation of the distal carotid artery; 6 open the external carotid artery ,the common carotid artery and the internal carotid artery in proper order before tightening the suture, flush out residual thrombus and debris; 7 perioperative oral aspirin antiplatelet therapy, adequate antiplatelet therapy after surgery can reduce the incidence of postoperative stroke. In this study, 2 patients had an ischemic stroke within 30 days after surgery and no hemorrhagic stroke occurred.

4.2.2 Craniocerebral hyperperfusion injury

CHS [11] is a clinical syndrome characterized by an increase in acute cerebral blood flow in the early postoperative period after carotid stenosis. It usually occurs 2–7 days after surgery. The main manifestations are [19] cerebral hemorrhage, deliration, seizures and severe headache, etc. The principle of this is because 1 patient with long-term carotid stenosis has intracranial vasodilation, and the autoregulation ability is reduced. After CEA, the carotid artery is dilated, and the blood flow beyond the autoregulation ability of the blood vessel leads to high perfusion. 2During CEA operation the carotid sinus was excessively tracted, and the hypertension was difficult to control after surgery, leading to high perfusion. 3CEA surgical incision stimulation and other factors lead to increased release of vasoactive peptides and other substances, leading to high perfusion [20]. The main risk factors [21, 22] for CHS are highly stenosis with collateral circulation disorders, advanced age, and prolonged hypertension. Therefore, patients with hypertension should control blood pressure in the normal range before surgery,Postoperative-application of hormones and mannitol reduces intracranial pressure to reduce the incidence of complications [23, 24]. The clinical incidence rate is mostly around 3%, and some reports have an incidence rate of 18%. According to the relevant literature [25], intraoperative transcranial Doppler ultrasound (TCD) monitoring can well predict the occurrence of CHS after CEA. In this group, there was no postoperative high brain perfusion in patients, which may have a certain relationship with the preoperative evaluation of intracranial blood vessels, strict control of blood pressure during and after operation, reduction of intracranial pressure caused by mannitol and hormone. Of course, further research is needed on the influencing factors of intracranial hyperperfusion.

4.2.3 postoperative restenosis

Postoperative restenosis is a serious complication of carotid endarterectomy. According to the study, the incidence of restenosis is about 6.2% within 1 year after carotid endarterectomy. In the case of short-term stenosis, it is rarely caused by re-hardening of the carotid artery, mainly by the postoperative proliferation of vascular smooth muscle, often without recurrence of symptoms. Studies have shown that intraoperative use of vascular patches and patients with no smoking history are significantly associated with longer-term survival without longer restenosis [26, 27]. Long-term follow-up show that only 0.47%-1.1. % of patients have restenosis undergoing CEA treatment and have gender differences, while female patients have a higher rate of restenosis than male patients [28, 29]. All cases in this group underwent valgus carotid endarterectomy, surgical lateral carotid stenosis was found in only one patient 41 months after operation, and the stenosis rate was about 60%. The long-term patency rate of other patients also required long-term follow-up.

4.2.4 Cranial nerve injury

One of the more common complications is cranial nerve injury [30], the results of large clinical trials show that the incidence rate is about 3%-23% [31], of which NASCET reported an incidence of 8.9%, NYCAS reported an incidence of 5.5%,common in nerve injury and edema caused by intraoperative nerve involvement, manifested as 1 a facial nerve mandibular branch injury: the incidence of 0.4–12%, this

nerve comes from the lower edge of the parotid gland into the neck, and is parallel with the muscles of the mandibular angle and the lower lip,is one of the frequently injured cranial nerves [31]. 2 vagus or recurrent laryngeal nerve injury: ectopic vagus is often mistaken for sublingual nerve spasm, may be accidentally injured when separating and blocking the carotid artery. Often unilateral vocal cord paralysis, manifested as hoarseness and difficulty swallowing, it is reported that the incidence of recurrent laryngeal nerve damage after CEA is 1.2-7% [32]. 3 hypoglossal nerve injury: often being accidentally injured or getting stretch injury, or being injured mistaken as the second abdominal muscle tendon in the separation and dissection of the distal end of the internal carotid artery. The tongue is often biased toward the injured side, with an incidence of about 1%. Unilateral injury can lead to difficulty in chewing, speaking, and swallowing; bilateral injury can cause upper airway obstruction, which lasts for 4 months, and the incidence is between 2.2% and 10.7% [32]. 4 Glossal nerve injury: extremely rare, most likely happens in the process of separation anatomy of the common carotid bifurcation, this nerve's damage can cause dysphagia and reduction vomiting reflex [33]. The vast majority of cranial nerve injuries are temporary, mostly associated with temporary injury, and most nerve injuries can recover spontaneously after 4–6 weeks [34]. Although CEA postoperative cranial nerve injury will not affect the patient's life safety, cranial nerve injury can seriously affect the patient's quality of life. In order to reduce cranial nerve injury, intraoperative adequate exposure, careful operation, and strict hemostasis should be performed to prevent cranial nerve injury caused by tracting the cranial nerve. In this study, the incidence of cranial nerve injury was 2.25%, including 1 case of hypoglossal nerve injury and 1 case of facial nerve mandibular branch injury. All patients were treated with neurotrophic drugs such as mecobalamin after operation. Basic rehabilitation was achieved in 15 days. No persistent nerves injury were observed.

4.2.5 incision hematoma

Incision hematoma is one of the most dangerous complications after CEA. Severe hematoma compression on the airway often endangers the patient's life. NASCET reported 1415 cases of surgery [4], 101 cases of neck hematoma, accounting for 7%, about half of which required reoperation for hematoma evacuation, and 2 cases died of asphyxia caused by hematoma compression on airway.

Alexander summarized 9308 cases undergoing CEA surgery and showed that CEA postoperative incision hematoma was closely related to stroke and death. The possible cause was that 1 the risk factors for incision hematoma were the same as the risk factors for postoperative stroke or death, such as patients with cervical abnormal anatomical structure or high carotid stenosis, the operation is difficult, and the risk of postoperative incision hematoma or stroke death is relatively high. 2 Incision hematoma or stroke is closely related to the skill level of the surgeon and perioperative management.3 In partial patients,incision hematoma directly leads to death of the patients. If the incision hematoma is obvious, the arterial hemorrhage may result even if the suture is not correct.If not handled properly in time, it may lead to the death of the patient.

There are few literatures on risk factors of postoperative incision hematoma, which may be related to preoperative oral anticoagulant drugs and intraoperative application of heparin [35]. A meta-analysis

report in 2016 showed patients with postoperative application of protamine to heparinize had a significantly lower incidence of postoperative incision hematoma compared with patients who did not use protamine to heparinize [36]. In this group of patients, a total of 5 patients had incisional hematoma after operation, 2 patients had a emergency hematoma evacuation due to a large neck hematoma, and 3 patients were improved with conservative treatment. Among the 2 cases of hematoma evacuation, 1 case of subcutaneous hemorrhage, considered to be related with using too much heparin in the operation, no protamine deheparation was used after surgery and oral anti-platelet therapy for aspirin before surgery; 1 case of small blood vessels bleeding in the patients with a history of diabetes and hypertension, may be related to inproper intraoperative hemostasis, arteriosclerosis and other factors; in 3 cases of conservative treatment, 1 patient with drainage tube blocked were improved by given drainage tube flushing, in the other 2 cases incision hematoma was absorbed by stoppinganticoagulant drugs. For patients underwenting carotid endarterectomy, perform preoperative reasonable antiplatelet therapy and use appropriate protamine and heparin in operation to prevent hematoma caused by local blood leakage, drainage of the drainage tube is also essential, it is especially important to observe the wound condition of the patient postoperatively. Although long-term oral antiplatelet therapy after operation is controversial, it is necessary to control personal high-risk factors [38, 39], such as smoking, blood lipids and blood sugar. Therefore, after CEA, the patient's vital signs should be closely observed to observe whether there is wheezing or poor breathing, and find the neck hematoma in time.

4.3 timing of surgery

At present, there is much controversy at home and abroad for the timing of carotid endarterectomy for the treatment of carotid stenosis ischemic stroke. Some experts believe that patients with mild to moderate stroke in emergency or early surgical treatment can get a better prognosis without increasing the risk and complications of surgery[37]. However, some scholars believe that carotid endarterectomy in early period after stroke will increase the risk of perioperative cerebrovascular events. It is recommended that carotid endarterectomy be performed 4–6 weeks after stroke [38]. The focus of the discussion was on whether early carotid endarterectomy in stroke patients would increase perioperative cerebrovascular events, the risk of stroke recurrence and death. Experts recommending postponing surgery believe that carotid endarterectomy should be performed at least 6 weeks after a cerebrovascular event. Previous literature has shown that early carotid endarterectomy after stroke increases the incidence of hemorrhagic stroke and the recurrence of ischemic stroke. In 2006, Rockman et al. [39] performed a study of 2,537 patients undergoing CEA surgery. For patients with symptomatic surgery within 30 days, perioperative complications accounted for 9.4%. This has led most surgeons to postpone surgery for surgical treatment of patients with acute ischemic stroke with carotid stenosis. The experts recommending early surgery believe that carotid endarterectomy should be performed within 2 weeks after the ischemic stroke event, on the grounds that 1 early surgery can prevent recurrence of ischemic stroke; 2 early surgical treatment can improve cerebral perfusion, especially for ischemic brains; 3 early surgery can prevent carotid occlusion. Rantner et al retrospectively analyzed 468 patients with carotid endarterectomy after acute ischemic stroke. Based on the 30-day postoperative mortality and the incidence of stroke recurrence,it is concluded that the benefits of carotid endarterectomy can offset the risk of perioperative

period. This benefit is maximum in 2 weeks, with the largest gain among patients with mild stroke, and a rapid decline after 2 weeks. However, further research is needed for the postoperative hemorrhagic stroke. For the controversy over the timing of surgery for patients with carotid stenosis in acute ischemic stroke, the current mainstream view is that some patients with non-disabling mild stroke should be actively treated early; for patients with ischemic attacks mild cerebral infarction, imaging evidence of cerebral ischemic and focal neurological impairment, early surgical treatment should be considered; early surgical treatment should be performed with caution in patients with progressive acute ischemic stroke and progressive cerebral infarction.

4.4 Coronary heart disease and history of peripheral arterial disease are important factors affecting the prognosis of patients

Follow-up showed that the risk factors of future cardiovascular events in patients undergoing carotid endarterectomy were history of coronary heart disease and peripheral arterial disease. Because coronary heart disease, peripheral arterial disease, and carotid artery disease have the same predisposing factors, carotid stenosis is often associated with coronary artery stenosis and peripheral arterial stenosis. Foreign Dzierwa et al [40] reported that 1/5 of patients with coronary heart disease had severe carotid stenosis; 80% of patients requiring carotid revascularization had coronary heart disease [41]. Steinvil et al [42] examined 1405 patients with coronary heart disease and found that severe carotid stenosis accounted for 12.8%, and the degree of coronary artery stenosis was positively correlated with the degree of carotid stenosis ($P < 0.001$). Domestic Liu Xiaoying et al [43] reported that cerebral angiography and coronary angiography was performed in 106 patients with coronary heart disease, and found that 36.5% of patients have carotid stenosis,and the degree of coronary artery disease is positively correlated with the degree of carotid stenosis. However, there is no report on the rate of coronary heart disease in patients with carotid stenosis in China, which is 14.6% in this study. In a SMART study involving 2,684 patients with asymptomatic carotid stenosis [44], carotid stenosis was an independent risk factor for vascular events, particularly vascular death. Follow up of this group of patients found that coronary heart disease and peripheral arterial disease were risk factors for prognosis in patients with carotid endarterectomy. This is consistent with the SMART study, indicating that carotid stenosis is an independent predictor of myocardial infarction and vascular death. This result also indicates the importance of screening for the history of coronary heart disease and peripheral arterial disease in patients with carotid stenosis. The importance of preoperative assessment of cardiac function through electrocardiogram, exercise plate test, cardiac ultrasound, or even coronary angiography, and the importance of Doppler blood imaging and examination of the extremity arteries. Some scholars [45] believe that age is also an important factor affecting the prognosis of patients with carotid endarterectomy. However, in this study, the prognosis of patients with carotid endarterectomy was not related to age. Carotid stenosis is only a manifestation of atherosclerotic disease in cervical vessels, not the entire disease. These patients generally have a long history, poor neurological function, poor cardiopulmonary function, and often have multiple complications. Therefore, for the surgical treatment of carotid stenosis, it is necessary to pay attention to the integral prevention, not just the local operation of the carotid artery, in order to achieve the purpose of preventing stroke.

5 Conclusions

In conclusion, carotid endarterectomy is safe and effective for the treatment of carotid stenosis. Carotid endarterectomy can improve the clinical symptoms of patients with symptomatic carotid stenosis effectively. History of coronary heart disease and peripheral vascular disease are factors of the near-term adverse outcome after carotid endarterectomy. Reasonable assessment of the patient's general condition before surgery, knowing the appropriate surgical indications well, choosing the right timing of surgery, paying attention to the perioperative treatment and the skill of the surgeon can reduce the incidence of perioperative complications, improve the patient's prognosis, and make patients get good curative effect.

Abbreviation

CAS Carotid Artery Stenosis CEA Carotid endarterectomy

CAS Carotid Artery Stenting MRI Magnetic Resonance Imaging

AMI Acute Myocardial Infarction TIA Transient Ischemic Attack

NASCET North American Symptomatic Carotid Endarterectomy Trial

ECST European Carotid Surgery Trial

ACAS Asymptomatic CarotidAtherosclerosis Study

VAST Veterans Administration Symptomatic carotid endarterectomy Test

HRmRS Hazard Ratio modified Rankin Scale

Declarations

Ethics approval and consent to participate :

The study was approved by the medical ethics committee of the Second Affiliated Hospital of Nanchang University. All patients involved in the study signed the informed consent voluntarily.

Consent for publication Not applicable.

Competing interests:

There is no conflict of interests in this study: Author Jiasheng Xu declares that he has no conflict of interest. Author KAILI LIAO declares that she has no conflict of interest. Author WEIMIN ZHOU declares that he has no conflict of interest.

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Authors’ contributions

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- searching for references 2：write and revise papers

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Contribution：1：Design research direction 2：Writing papers

Author：Shasha Wan Riwei Wang

Contribution：1：helping to collect clinical data 2：Data summary and Statistical analysis

Author：Qingfu Zeng Contribution：

1：Review and revise the papers 2：Guidance article writing

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Availability of data and supporting materials

Please contact author for data requests.

References

1. Wang Maobin. Rehabilitation of stroke [J]. Chinese Journal of Health Medicine. 2006;3(2):73. Med 75.
2. Rd MCS. Multimodality management of carotid artery stenosis: reviewing the class-I evidence.[J]. J Natl Med Assoc. 2007;99(11):1235–42.
3. Zhu CZ, Norris JW. Role of carotid stenosis in ischemic stroke[J]. Stroke,1990, 21(8):1131–1134.
4. Ferguson GG, Eliasziw M, Barr HW, et al. The North American Symptomatic Carotid Endarterectomy Trial: surgical results in 1415 patients.[J]. Stroke; a journal of cerebral circulation,1999,30(9):1751.
5. Young B, Moore WS, Robertson JT, et al. An analysis of perioperative surgical mortality and morbidity in the asymptomatic carotid atherosclerosis study. ACAS Investigators. Asymptomatic Carotid Atherosclerosis Study[J]. Stroke,1996,27(12):2216–2224.
6. Rothwell PM, Warlow CP. Interpretation of operative risks of individual surgeons. European Carotid Surgery Trialists' Collaborative Group[J] Lancet. 1999;353(9161):1325.

7. Barnett HJ. Symptomatic carotid endarterectomy trials[J]. Stroke; a journal of cerebral circulation,1990,21(11 Suppl):2–5.
8. Moore WS, Barnett HJM, Beebe HG, et al. Guidelines for Carotid Endarterectomy A Multidisciplinary Consensus Statement From the Ad Hoc Committee, American Heart Association[J]. Stroke,1995,91(2):188–201.
9. Zhang Qinyi. American Heart Association guidelines and European guidelines on the application of carotid endarterectomy and differences in primary prevention [J]. Chinese Journal of Cardiovascular Cerebrovascular Diseases. 2009;11(9):648 / 650.
10. De HR, Limburg M, Bossuyt P, et al. The clinical meaning of Rankin 'handicap' grades after stroke[J]. Stroke,1995,26(26):2027–2030.
11. Ogasawara K, Yukawa H, Kobayashi M, et al. Prediction and monitoring of cerebral hyperperfusion after carotid endarterectomy by using single-photon emission computerized tomography scanning. [J]. Journal of Neurosurgery,2003,99(3):504–510.
12. Winstein CJ, Stein J, Arena R, et al. Guidelines for Adult Stroke Rehabilitation and Recovery: A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association[J]. Stroke; a journal of cerebral circulation,2016,47(6):e98.
13. Chen Dong JL, Qun. Wang Jiyue, etc. Multicenter study on efficacy and safety of carotid endarterectomy in the treatment of carotid stenosis [J]. Chinese Journal of Cerebrovascular Diseases. 2013;10(9):452 / 457.
14. Momin TA, Ricotta JJ. Minimizing the complications of carotid endarterectomy[J]. Perspectives in Vascular Surgery Endovascular Therapy. 2010;22(2):106.
15. Rockman CB, Maldonado TS, Jacobowitz GR, et al. Early carotid endarterectomy in symptomatic patients is associated with poorer perioperative outcomes.[J]. Journal of Vascular Surgery,2006,44(3):480–487.
16. Stoner MC, Defreitas DJ. Process of care for carotid endarterectomy: perioperative medical.
17. management[J]. J Vasc Surg. 2010;52(1):223.
18. Qiao Tong L, Changjian H. Fang, et al. Clinical analysis of 179 cases of carotid stenosis treated by endarterectomy [J]. Chinese Journal of practical surgery. 2012;32(8):659 / 661.
19. Diao Yongpeng L, Changwei S, Xiaojun, et al. Analysis of the efficacy of two kinds of carotid endarterectomy in the treatment of carotid stenosis [C].2014.
20. Ziaja D, Biolik G, Koce ak P, et al. Neurological symptoms associated with cerebral hyperperfusion syndrome after CEA and CAS–one centre study.[J]. European Review for Medical & Pharmacological Sciences,2014,18(8):1176–1180.
21. Brott TG, Halperin JL, Abbara S, et al. 2011 ASA/ACCF/AHA/AANN/ AANS/ACR/ASNR/CNS/SAIP/SCAI/SIR/SNIS/SVM/SVS guideline on the management of patients with extracranial carotid and vertebral artery disease: executive summary: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines,

and the American Stroke Association, American Association of Neuroscience Nurses, American Association of Neurological Surgeons, American College of Radiology.

22. American Society of Neuroradiology. Congress of Neurological Surgeons, Society of.
23. Atherosclerosis Imaging and Prevention. Society for Cardiovascular Angiography and.
24. Interventions. Society of Interventional Radiology, Society of NeuroInterventional Surgery, Society for Vascular Medicine, and Society for Vascular Surgery. Developed in collaboration with the American Academy of Neurology and Society of Cardiovascular Computed Tomography[J]. Catheter Cardiovasc Interv,2013,81(1):E76-E123.
25. Chen Yu, Weiwei Wu. Liu Bun, etc. Hyperperfusion syndrome after carotid endarterectomy [J]. Chinese Journal of Stroke. 2010;5(4):338 / 342.
26. Guo Jianming Gu, Yongquan L. Xuefeng, et al. Clinical progress in cerebral hyperperfusion syndrome [J]. Chinese Journal of Medicine. 2012;92(21):1508 / 1510.
27. Hochart A, Lucas C. [Confusion and partial seizures showing cerebral hyperperfusion syndrome after endarterectomy].[J]. Revue Neurologique. 2013;169(4):353–4.
28. Webering N, Weber R, Berlit P. [Focal seizure status caused by a cerebral hyperperfusion syndrome after carotid endarterectomy][J]. Fortschr Neurol Psychiatr,2014,82(5):271–274.
29. Liu Bun L, Zhichao. Ni cold, etc. Transcranial Doppler ultrasound in predicting cerebral hyperperfusion syndrome after carotid endarterectomy [J]. Chinese Journal of surgery. 2013;51(6):504 / 507.
30. Chan RC, Chan YC, Cheung GC, et al. Predictors of restenosis after carotid endarterectomy:17-year experience in a tertiary referral vascular center.[J]. Vasc Endovascular Surg,2014,48(3):201–206.
31. Zhang Liyong Y, Guoyang W, Jiyue, etc. Early complications and influencing factors after carotid endarterectomy [J]. Chinese Journal of surgery, 2015, 53 (7): 533 / 537.
32. Ballotta E, Toniato A, Giau GD, et al. Durability of eversion carotid endarterectomy[J]. Journal of Vascular Surgery,2014,59(5):1274–1281.
33. Baracchini C, Saladini M, Lorenzetti R, et al. Gender-based outcomes after eversion carotid25endarterectomy from 1998 to 2009[J]. Journal of Vascular Surgery,2012,55(2):338.
34. Marcucci G, Antonelli R, Gabrielli R, et al. Short longitudinal versus transverse skin incision for carotid endarterectomy: impact on cranial and cervical nerve injuries and esthetic outcome.[J].Journal of Cardiovascular Surgery,2011,52(2):145–152.
35. Featherstone RL, Brown MM, Coward LJ, et al. International carotid stenting study: protocol for a randomised clinical trial comparing carotid stenting with endarterectomy in symptomatic carotid artery stenosis[J]. Cerebrovasc Dis. 2004;18(1):69–74.
36. Assadian A, Senekowitsch C, Pfaffelmeyer N, et al. Incidence of cranial nerve injuries after carotid eversion endarterectomy with a transverse skin incision under regional anaesthesia[J]. Eur J Vasc Endovasc Surg. 2004;28(4):421–4.

37. Regina G, Angiletta D, Impedovo G, et al. Dexamethasone minimizes the risk of cranial nerve injury during CEA[J]. J Vasc Surg. 2009;49(1):99.
38.] Suyama K, Nagata I. [Carotid endarterectomy for atherosclerotic carotid artery stenosis]. [J]Brain Nerve. 2010;62(12):1301–13.
39. Comerota AJ, Difiore R, Tzilinis A, et al. Cervical hematoma following carotid endarterectomy is morbid and preventable: a 12-year case-controlled review[J]. Vascular Endovascular Surgery. 2012;46(8):610.
40. Rathenborg LK. Commentary on 'Protamine Reduces Bleeding Complications Without Increasing the Risk of Stroke After Carotid Endarterectomy: A Meta-analysis'[J]. European Journal of Vascular Endovascular Surgery. 2016;52(3):308.
41. Rantner B, Schmidauer C, Knoflach M, et al. Very Urgent Carotid Endarterectomy Does Not Increase the Procedural Risk[J]. European Journal of Vascular Endovascular Surgery. 2015;49(2):129–36.
42. Keldahl ML, Eskandari MK. Timing of carotid surgery after acute stroke[J]. Expert Review of Cardiovascular Therapy,2010,8(10):1399–1403.
43. Cheung WK, et al. Attenuation Correction and Normalisation for Quantification of Contrast Enhancement in Ultrasound Images of Carotid Arteries. Ultrasound in Medicine Biology. 2015;41(7):1876–83.
44. Karolina D, Piotr P, Piotr M, et al. Treatment strategies in severe symptomatic carotid and coronary artery disease[J]. Medical Science Monitor International Medical Journal of Experimental & Clinical Research,2011,17(8):A191-A197.
45. de Waard DD, de Borst GJ, Bulbulia R, et al. Diastolic Blood Pressure is a Risk Factor for Peri-procedural Stroke Following Carotid Endarterectomy in Asymptomatic Patients[J]. European Journal of Vascular Endovascular Surgery. 2017;53(5):626–31.
46. Steinvil A, Sadeh B, Arbel Y, et al. Prevalence and predictors of concomitant carotid and coronary artery atherosclerotic disease[J]. Journal of the American College of Cardiology,2011,57(7):779–783.
47. Liu Xiaoying F, Yanguang C, Wei. Incidence and risk factors of extracranial carotid artery stenosis in patients with coronary heart disease [J]. Clinical Meta. 2006;21(14):1008. / 1010..
48. Goessens BM, Visseren FL, Kappelle LJ, et al. Asymptomatic carotid artery stenosis and the risk of new vascular events in patients with manifest arterial disease: the SMART study.[J]. Stroke,2007,38(5):1470–1475.
49. Kragsterman B, Logason K, Ahari A, et al. Risk factors for complications after carotid endarterectomy–a population-based study[J]. European Journal of Vascular Endovascular Surgery the Official Journal of the European Society for Vascular Surgery. 2004;28(1):98.
50. Bonati LH, Dobson J, Featherstone RL, et al. Long-term outcomes after stenting versus endarterectomy for treatment of symptomatic carotid stenosis: the International Carotid Stenting Study (ICSS) randomised trial[J]. Lancet,2015,385(9967):529–538.

Figures

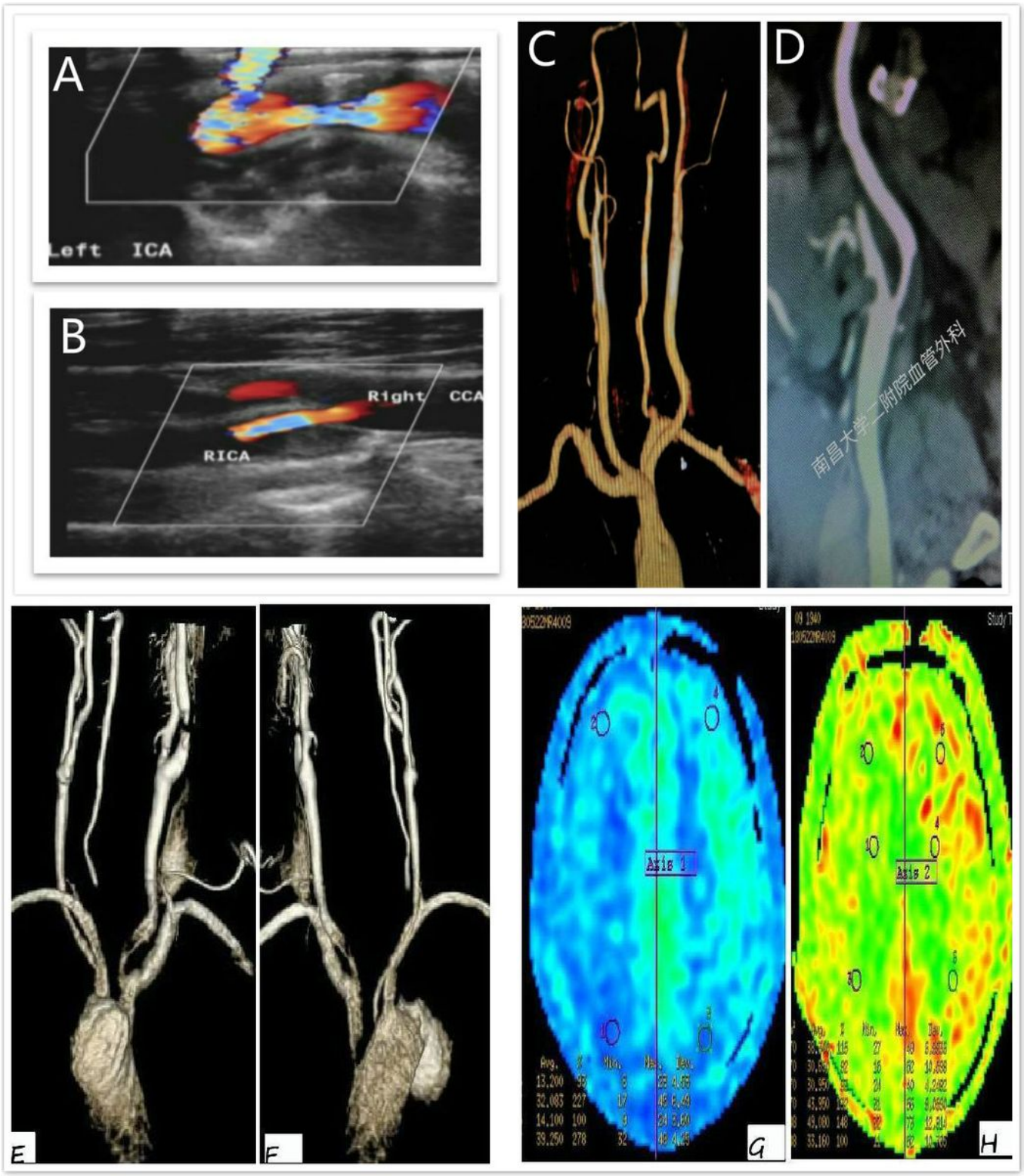

Figure 1

Imaging manifestations of carotid artery stenosis A, B: carotid stenosis color Doppler ultrasound; C, D: carotid artery stenosis CTA; E, F : carotid stenosis MRA manifestations; G, H : carotid stenosis cranial perfusion.

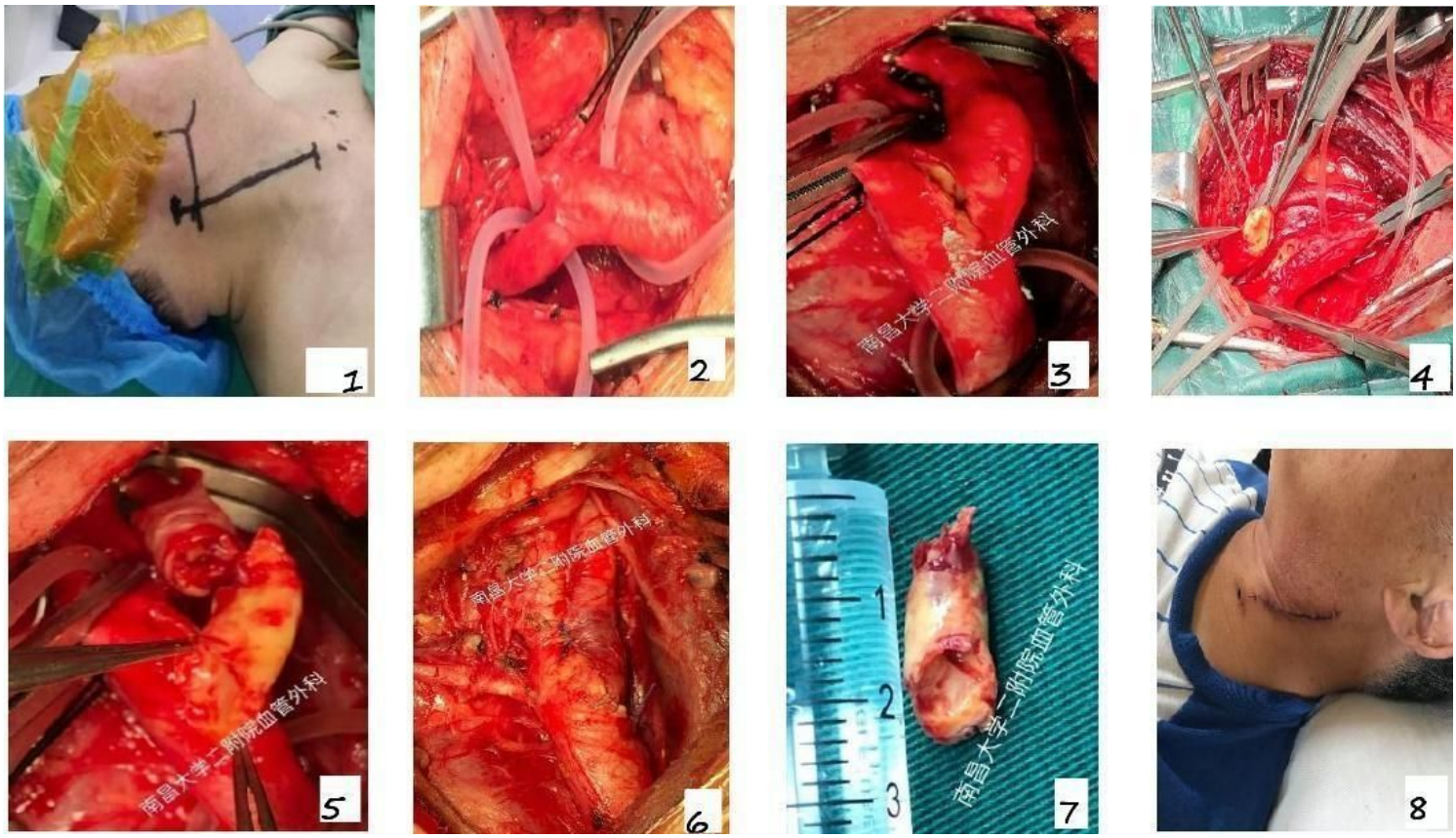

Figure 2

Procedure of carotid endarterectomy (Figure 1 shows the neck incision,figure 2 shows fully dissociated carotid artery,figure 3 shows sequential blocking carotid artery , figure 4 is cuting off the internal carotid artery, figure 5 shows valgus stripping of carotid artery intima, figure 6 carotid artery after anastomosis, Figure 7 shows exfoliated the carotid plaque, and figure 8 shows neck incision 1 weeks after operation)

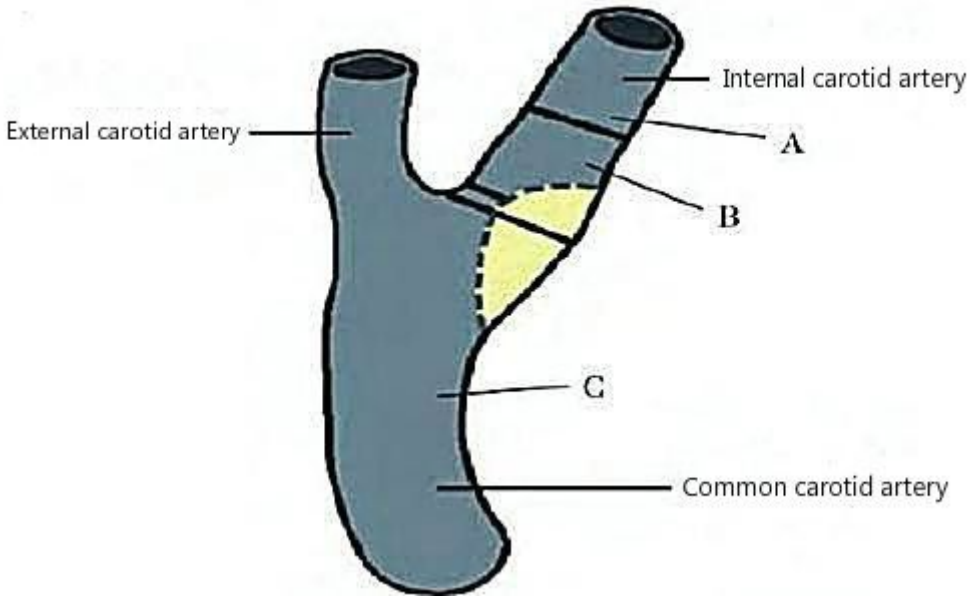

Figure 3

schematic diagram of the measurement of carotid artery stenosis

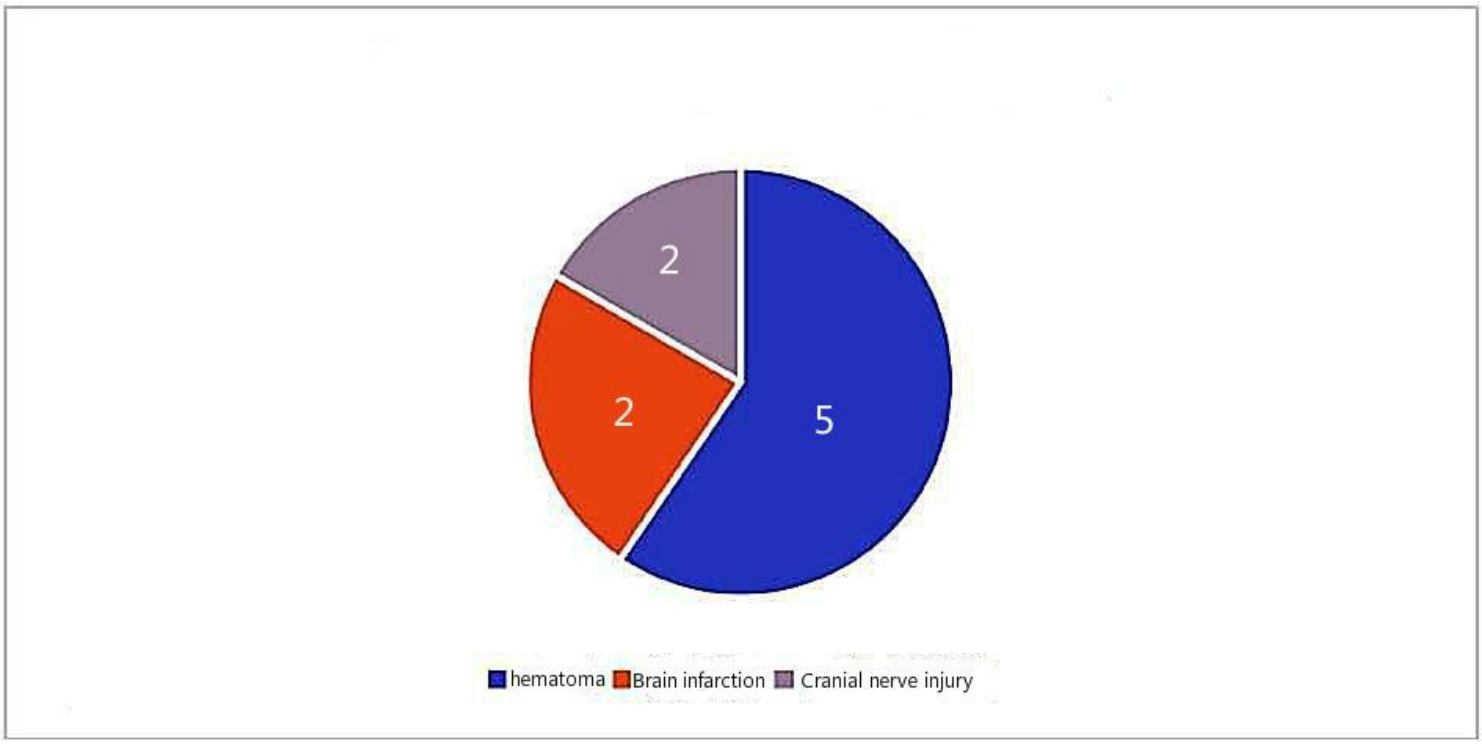

Figure 4

perioperative carotid endarterectomy complications

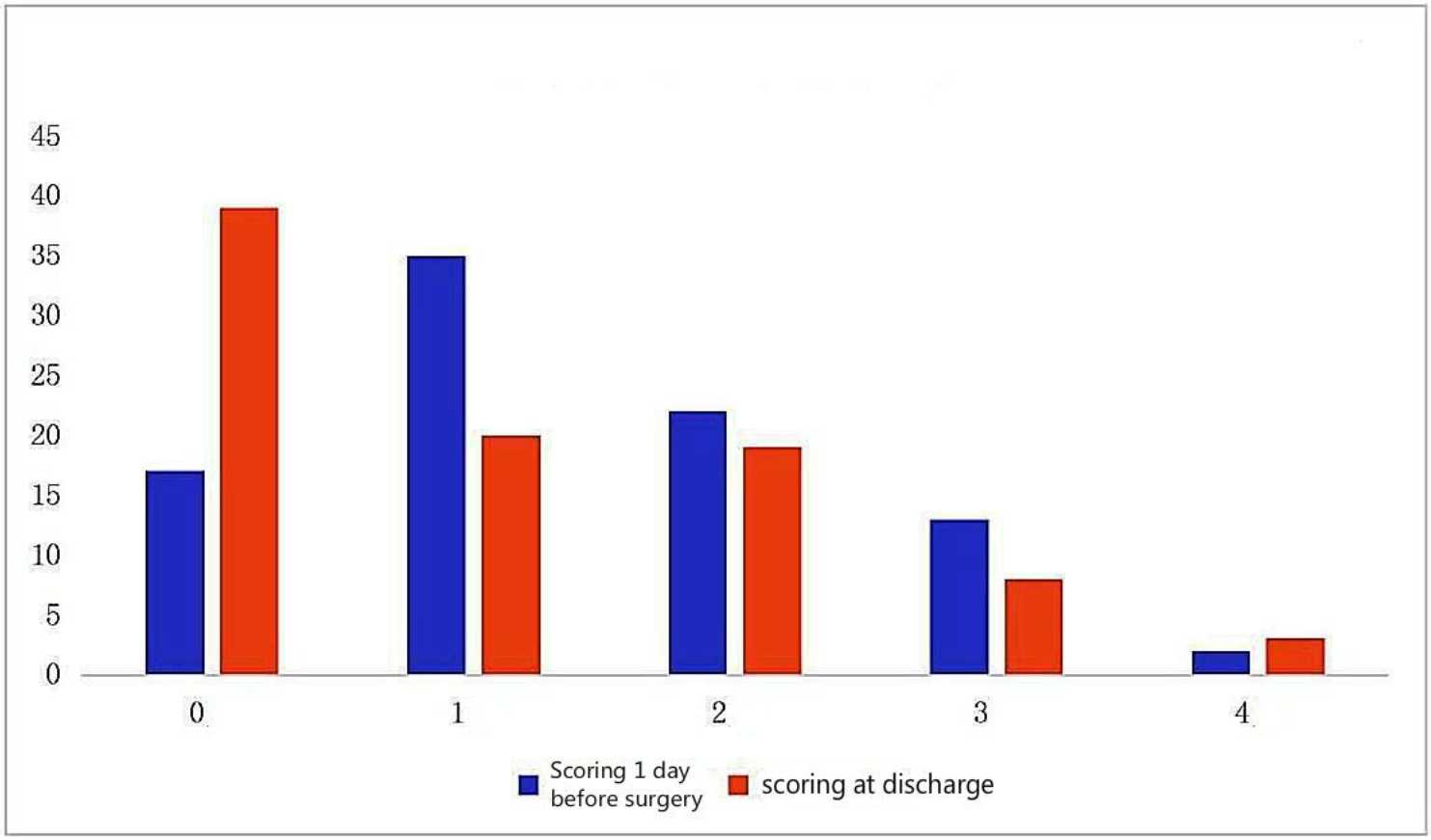

Figure 5

mRS scores before and after surgery

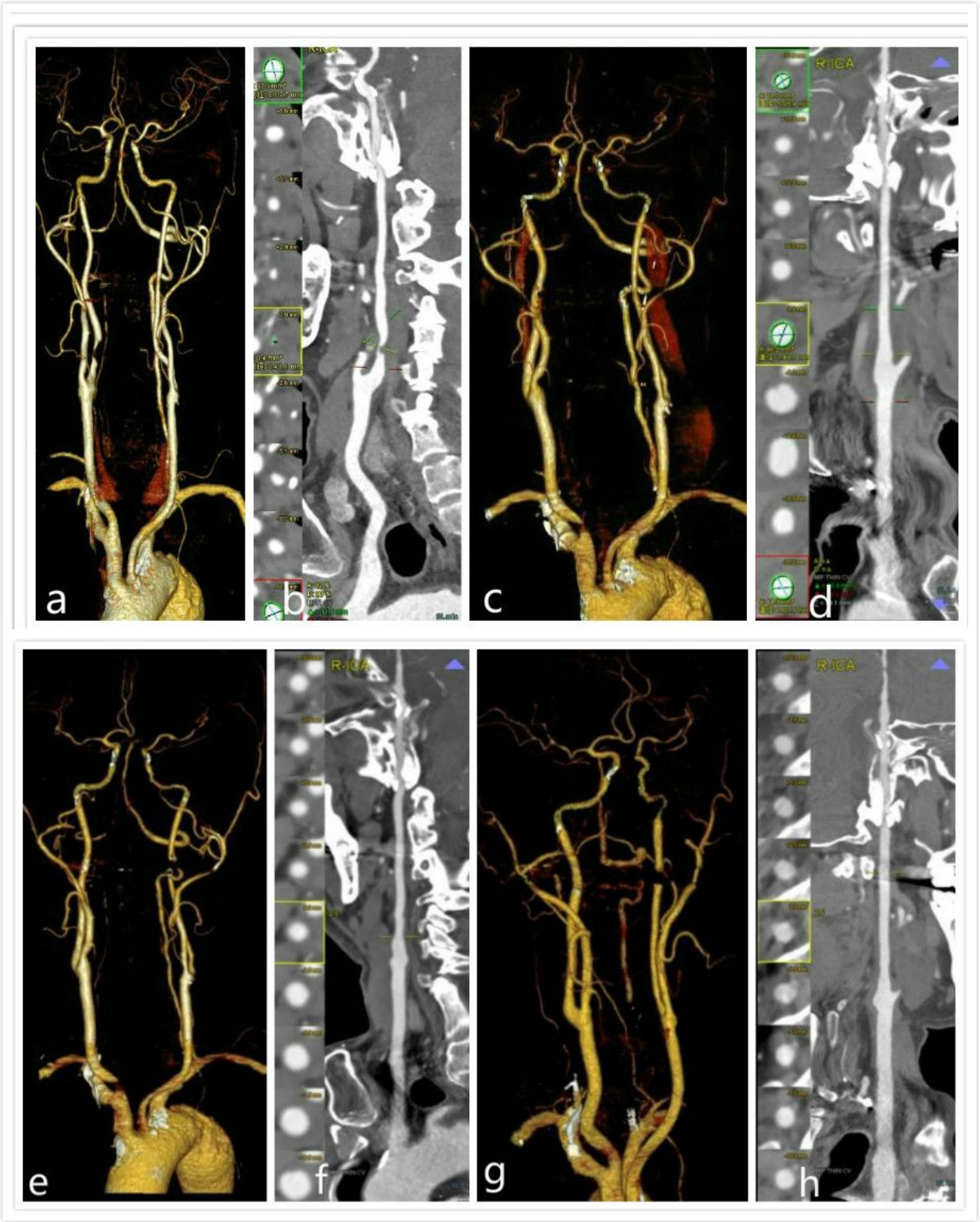

Figure 6

CTA follow-up results after CEA. a, b: preoperative CTA test results in patients with carotid stenosis; c, d: half year after CEA CTA review results; e, f: 1 year CTA review results after CEA; g, h: 3 years after CEA CTA review results

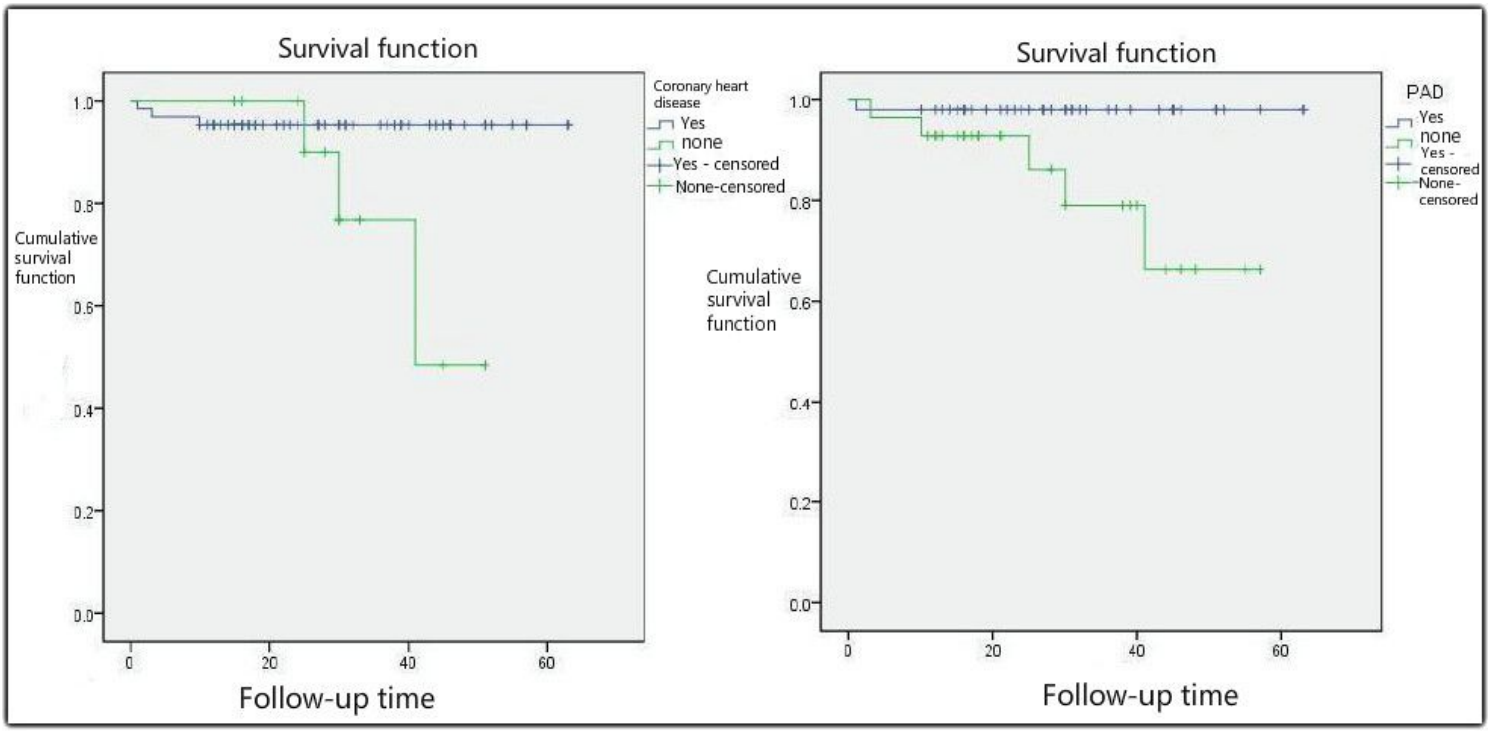

Figure 7

Kaplan - Meier survival analysis during the follow-up period