Chapter

Carotid Disease

Minerva Irene Hernández Rejón
and Manuel Alexis Vargas Robles

Abstract

Atherosclerotic carotid disease causes about 30% of cerebrovascular ischemia transitory or permanent in the world; the severity of symptoms is variable. The clinical manifestations are varied from focal neurological alterations to transient or permanent vascular events. The treatment of the disease will depend on the location, degree, and risk, which can be surgical, endovascular, or medical. Open surgical treatment, endarterectomy, has been preferred as the first option and, however, has been reported to have associated complications like infection, hematoma, stroke, heart attack, restenosis, etc. With the advent of new technologies, endovascular treatment has been described as an option in patients with high risk or restenosis.

Keywords: atherosclerotic carotid disease, carotid endarterectomy, endovascular treatment, medical management of carotid disease, complications

1. Atherosclerotic disease and carotid stenosis

Atherosclerosis is a chronic disease of the arteries characterized by inflammation and plaque building in the arterial wall, eventually leading to stenosis of the vessel. Carotid atherosclerosis is associated with the increased risk of cardiovascular diseases [1].

Ischemic cardiovascular disease is a combination of progressive atherosclerosis and acute thrombotic complications [2].

The risk factors in atherosclerosis can be modifiable or not: non-modifiable are genetic predisposition, gender, and age, and modifiable factors are blood pressure, smoking, diabetes, cholesterol levels, obesity, and physical activity [2].

The earliest pathologic studies described the predilection of atherosclerosis near branch ostia, bifurcations, and bends, suggesting the important component of flow dynamics plays an important role in its initiation and development. Atherosclerotic plaque tends to form at regions where flow velocity and shear stress are reduced, in particular at the carotid bifurcation where disturbances in blood flow deviate from a laminar unidirectional pattern. Thus, the unique geometry and flow properties presented by the carotid bifurcation contribute [3].

Metabolic syndrome is a clinical entity characterized by multiple risk factors for cardiovascular disease and diabetes mellitus such as high-normal or elevated blood pressure, hyperglycemia, elevated triglycerides, low high-density cholesterol level, and abdominal obesity [4].

The increase of cardiovascular risk related to metabolic syndrome is reported; over the past decades, the impact of metabolic syndrome on large arterial vessels has been analyzed [4].

Carotid intima-media thickness has been proven to be a valuable predictor of myocardial infarction and ischemic stroke independent of traditional risk factor. The intima-media thickness was higher in patients with metabolic syndrome [4].

In the study IMPROVE, a multicenter observational European study, it showed that the intima-media thickness was a strong predictor of cardiovascular disease [4].

The maximum intima-media thickness of the common carotid artery, as measured by carotid artery ultrasound, has been used as a marker of atherosclerosis and cardiovascular disease. In contrast, there are reports suggesting that the predictive ability of intima-media thickness for cardiovascular disease is inferior than that of the carotid plaque score assessed by ultrasonography [5].

Recent studies have focused in adding pharmacological strategies like antithrombotic therapy, lowering lipid levels, improving glycemic control, and addressing inflammation present in the metabolic syndrome. On the other hand, patients with coronary and peripheral diseases have a high risk of cardiovascular death and disabling vascular events [2].

Carotid artery stenosis and lower limb peripheral arterial occlusive disease usually share the same pathological changes and can coexist. It has been reported that the prevalence rate of significant carotid stenosis increased with the stage of lower limb peripheral occlusive arterial disease. So the screening for significant carotid disease in these patients [6].

Screening for carotid stenosis in patients who are neurologically asymptomatic may therefore be acceptable when there are two or more risk factors or when ankle/brachial index is less than 0.4 with reevaluation of 6 months. Investigations have identified combinations of risks that identify populations in whom the risk of stenosis between 50% and 99% approaches 60%. Screening patients in these categories who are suitable operative candidates and who would undergo operation were the found to have clinically important disease [7].

2. Definition and epidemiology

Stroke diagnosis has been based on the World Health Organization's definition of a focal, occasionally global loss of neurological function lasting >24 h and which has a vascular etiology. A transient ischemic attack is defined in a similar way, but the duration is <24 h [8].

The principal causes of ischemic carotid territory stroke are thromboembolism from the internal carotid artery or middle cerebral artery (25%), small vessel intracranial disease (25%), cardiac embolism (20%), and other causes (5%). About 10–15% of all strokes follow thromboembolism from previously asymptomatic internal carotid stenosis >50% [9].

Moderate and severe carotid artery stenosis is an important public health issue; this condition affects 10% of the general population by their eighth decade and accounts for 10% of all strokes [10].

3. Clinical manifestations

The symptoms of the carotid disease were fist described by Fisher (1951). Stroke produced by carotid stenosis is caused by a combination of affection of the blood vessels, clotting system, and hemodynamics that in conjunction cause embolism or/and low cerebral flow [11].

Carotid atherosclerosis is usually most severe within 2 cm of the bifurcation of the common carotid artery and involving the posterior wall of the vessel. The

definition of asymptomatic or symptomatic carotid artery stenosis is based on the history and physical examination [11].

Symptomatic symptoms include transient ischemic attacks produced by embolization that causes low flow with inadequate collateral blood supply. The symptoms depend on the cerebral artery territory involved, and total carotid artery occlusion can cause low flow or embolic ischemic events.

Various features of plaque morphology can be used to identify risk and include plaque ulceration, plaque structure and composition, and plaque volume [12].

A history of more than one episode of neurological alterations occurring in the same carotid territory is very suggestive of underlying carotid disease. The carotid bruit is an important sign over the site of the stenosis; however, is a poor predictor for development of stroke; and is not sufficiently predictive of high grade of carotid disease. However, 75% of patients with bruit had a moderate to severe stenosis (more than 60). Ischemic symptoms reflect ipsilateral ocular and cerebral hemisphere ischemia like partial or complete blindness, hemianopsia, hemiparesis, and hemisensory loss [12].

4. Diagnosis

There are four diagnostic modalities to directly image the internal carotid artery: cerebral angiography, carotid duplex ultrasound, magnetic resonance angiography, and computed tomography angiography.

Cerebral angiography is the gold standard for imaging the carotid arteries; it permits an evaluation of the entire carotid artery system, providing information about tandem atherosclerotic disease, plaque morphology, and collateral circulation; however, it is invasive, costs high, and has a high risk of morbidity and mortality [13].

There are three methods to measure carotid stenosis that predominate worldwide (NASCET, ECST, and CC):

- The North American Symptomatic Carotid Endarterectomy Trial (NASCET) measures the residual lumen diameter at the most stenotic portion and compares it with the normal internal carotid diameter [14].

- The European Carotid Surgery Trial (ECST) measures the lumen diameter at the most stenotic portion of the vessel and compares this with the estimated probable original diameter at the site of maximum stenosis [15].

- Common carotid (CC) measures the residual lumen diameter at the most stenotic portion of the vessel and compares this with the lumen diameter in the proximal common carotid artery [15].

Equivalent measurements for the three methods have been determined.

Carotid duplex ultrasound uses B-Mode to help detect focal increases in the blood flow velocity indicative of a high-grade carotid stenosis. The peak systolic velocity is the most frequently used measurement to gauge the severity of the stenosis (end-diastolic velocity, spectral configuration, and carotid index provide additional information) and correlate with the residual lumen diameter [16].

The sensitivity reported is 89% and the specificity 84%. It is a noninvasive, safe, and relative inexpensive technique, although it has limited utility in obtaining information about plaque composition and intraplaque hemorrhage and is less precise in determining stenosis of <50% compared with stenoses of higher degrees.

Transcranial Doppler is often used in conjunction with ultrasound carotid duplex to evaluate the hemodynamic significance of internal carotid artery stenosis [17].

Magnetic resonance angiography technique is used for evaluating the extracranial carotid arteries and produced a reproducible three-dimensional image of the carotid bifurcation with a good sensitivity for detecting high-grade carotid stenosis; however, it had been reported overestimated degree [18].

Computed tomography angiography provides an anatomic depiction of the carotid artery lumen, allows imaging of adjacent soft tissue structures, and is particularly useful when carotid duplex ultrasound is not reliable or when an overall view of the vascular field is required. It had a sensitivity and specificity of 97 and 99%, respectively [19].

In earlier reports, magnetic resonance, angiography, and carotid duplex ultrasound had difficulties distinguishing very severe stenosis from occlusion, so false-positive and false-negative results occurred [20].

The location most frequently affected by atherosclerosis is the carotid bifurcation. Progression of atheromatous plaque results in luminal narrowing, often accompanied by ulceration, process that can lead to ischemic stroke and transient ischemic attack from embolization, thrombosis, and hemodynamic compromise [20].

5. Treatment

5.1 Endarterectomy

The endarterectomy is the choice treatment in those patients with transient ischemic attacks or who suffered a cerebral infarction with a minimal sequel [21]. It is beneficial for symptomatic or asymptomatic patients.

The indications for carotid endarterectomy (CEA) are:

1. Bilateral carotid stenosis: in which the combined death and stroke rates in patients were almost twice than that of matched patients with unilateral disease (5.6% versus 2.4%) [22].

2. Prophylactic carotid endarterectomy: in patients with severe carotid stenosis prior to another surgery, it is rarely needed, and a decision to proceed should be individualized depending upon the clinician's best estimate of the risk of perioperative stroke.

3. Coronary artery bypass surgery: a new stroke or transient ischemic attack occurs in approximately 3% of patients following coronary artery bypass grafting (CABG); also in general surgery, the incidence of stroke appears to be lower in nonvascular surgical procedures than following cardiac surgery with a reported incidence in patients undergoing general anesthesia of less than 0.5%.

4. Vascular procedures: there are not enough trials for prophylactic CEA prior to abdominal aortic aneurysm repair or other major peripheral vascular procedures, because of the involvement of significant hemodynamic fluctuations.

5. Patients with intracranial aneurysm: ipsilateral intracranial aneurysms that are distal to a cervical internal carotid artery stenosis may be susceptible to sudden hemodynamic changes associated with CEA, leading to aneurysm rupture [23].

6. Contraindications for carotid endarterectomy

Absolute: asymptomatic complete carotid occlusion.

Relative contraindications: history of neck radiation, concurrent tracheostomy, prior radical neck dissection with or without radiation, contralateral vocal cord paralysis from prior endarterectomy, atypical lesion location, either high or low, that is surgically inaccessible, severe recurrent carotid stenosis, unacceptably high risk [24].

Carotid endarterectomy is established as safe and effective by randomized controlled trials for reducing the risk of ischemic stroke in both symptomatic and asymptomatic patients. However, carotid angioplasty and stenting are proposed as alternative for carotid endarterectomy.

Symptomatic carotid disease is defined as focal neurologic symptoms that are referable to the appropriate carotid artery distribution including transient ischemic attacks or ischemic strokes. The definition is contingent on the occurrence of carotid symptoms within the previous 6 months.

Carotid endarterectomy and stenting are recommended in patients with recently symptomatic carotid stenosis of 70–99% and a life expectancy of at least 5 years who meet all of these conditions: accessible carotid lesion, absence of clinically significant cardiac, pulmonary or other diseases that increase greatly the risk of anesthesia and surgery, and no prior ipsilateral endarterectomy.

It is suggested that carotid endarterectomy have to be done 2 weeks after a nondisabling stroke or transitory stroke, because between the first 48 hours, the risk of recurrent stroke elevates compared with later surgery. In patients who have a large infarction with brain swelling, hemorrhagic brain infarction and progressing stroke have long been thought to have high perioperative risk and expose the patient to an increased risk of recurrent stroke, emergent CEA for progressing/fluctuating stroke, or crescendo transitory attack that may have a high operative risk.

The risk of stroke can be calculated based on patient age, patient sex, degree of carotid stenosis, type of presenting symptomatic event, time since last symptomatic event, and carotid plaque morphology.

6.1 Surgical complications in carotid endarterectomy

Carotid endarterectomy complications can usually be related to comorbid conditions or cardiovascular preexisting diseases and also to the surgical technique. Most common complications of CEA include myocardial infarction, perioperative stroke, postoperative bleeding, and the potential consequences of cervical hematoma, nerve injury, surgical site infection, and carotid restenosis. However, the rates of complications after carotid endarterectomy is low [25–33].

There are two important trials in which the benefit is higher than the possible complications: the European Trial (ICSS) reports a mortality at 120 days of post-op of 0.8% and complications of 4.2%. The North American Trial (CREST) [33, 34] reported combined results for symptomatic and asymptomatic patients. In 1240 patients assigned to endarterectomy (47.3% asymptomatic), the 30-day death rate was 0.3%, and the rate of any periprocedural (30-day) stroke or death or postprocedural ipsilateral stroke was 2.3%.

Appropriate perioperative medication management is important to reduce the risk of cardiovascular and procedure-specific complications.

There are three mains risk factors:

1. Myocardial infarction

2. Stroke

3. Death

Others:

1. Hyperperfusion syndrome

2. Cervical hematoma

3. Nerve injury

4. Infection

5. Restenosis

There are a lot of risk factors, some of them are:

1. Age (>80 years)

2. Smoking

3. Previous stroke including transient ischemic attack

4. Previous stenosis of carotid artery

5. Special or chronic conditions such as cancer, heart diseases, hypertension, and diabetes

6.2 Myocardial infarction

In randomized trials, myocardial infarction has occurred at a slightly higher rate for carotid endarterectomy than carotid artery stenting [25, 26, 28–31]; with a reported incidence between 0 and 2%, pooled absolute risk of perioperative (30-day) myocardial infarction was 0.87%. The risk factors for myocardial infarction included older age, coronary heart disease, peripheral artery disease, and carotid restenosis.

6.3 Perioperative stroke

Perioperative stroke is the second most common cause of death in carotid endarterectomy [25, 26, 28, 32, 33, 35, 36] with a rate of less than 3% for symptomatic patients and less than 5% for the symptomatic patients depending on the indication of the CEA and the experience of the surgeon. But also there are some factors that can contribute to postoperative stroke [28, 37–42] like plaque emboli, platelet aggregates, improper flushing, poor cerebral protection, and relative hypotension.

If perioperative stroke is suspected, ultrasound is a good option; but if it shows good flow throughout the carotid artery with no thrombosis or intimal flaps, a head computed tomography (CT) scan should be obtained to rule out intracranial bleeding [38, 43–45]. If there is access to a hybrid operating room, another approach to obtain may be head CT first, and, if no bleeding is identified, intraoperative arteriography to identify any correctable lesions is performed [28].

6.4 Hyperperfusion syndrome

It is an uncommon complication of endarterectomy, occurring after carotid revascularization in less than 1%; it causes the most postoperative intracerebral

hemorrhages and seizures in the first 2 weeks after CEA. It is caused after the surgery; because of the small vessels that chronically compensated the patient, and after endarterectomy blood flow is restored to a normal or elevated perfusion pressure and those vessels are unable to vasoconstrict sufficiently to protect the capillary bed, causing edema and hemorrhage and clinical manifestations. Hypertension is a frequent predecessor of the syndrome, underscoring the importance of good perioperative blood pressure control [46].

Hyperperfusion syndrome appears to be more likely with revascularization of a high-grade (80% or greater) stenosis.

Hyperperfusion syndrome is characterized by the following clinical features:

1. Headache ipsilateral to the revascularized internal carotid, typically improved in upright posture, may herald the syndrome in the first week after endarterectomy.

2. Focal motor seizures are common, sometimes with postictal Todd's paralysis mimicking post-endarterectomy stroke from carotid thrombosis.

3. Intracerebral hemorrhage is the most feared complication, occurring in approximatelys 0.6% of patients after CEA, usually within 2 weeks of surgery.

Treatment consists of prevention, strict control of postoperative hypertension maintaining a systolic blood pressure below 150 mmHg, and using labetalol, nitroprusside, and nitroglycerin aggressively. If complication is suspected in any patient with severe headache following CAE, a head CT is required to confirm or discard this syndrome [46, 47].

6.5 Cervical hematoma

This is a catastrophic and real urgent complication, because it can result in abrupt loss of the airway, with an incidence of 4% after CAE and requiring a re-exploration of the neck. Uncontrolled hypertension during emergence from anesthesia or in the postoperative period can also lead to hematoma formation [26, 35, 48].

6.6 Nerve injury

Cranial nerve or other nerve injuries occur in approximately 5% of patients following carotid endarterectomy [27, 33].

According to the European Carotid Surgery Trial, cranial nerve injury rate declined to 3.7%. In the Vascular Study Group of New England (VSGNE) database, the overall rate of nerve injury at discharge was 5.6%; 0.7% of patients had more than one nerve affected.

Hypoglossal nerve is the most frequently injurie manifested by a deviation of the tongue to the side of the injury, the facial nerve is the second one resulting in paresis of the lateral aspect of the orbicularis oris muscle with asymmetric smile; and the vagus nerve which result in unilateral vocal cord paralysis, glossopharyngeal nerve can be damaged with excessive dissection in the carotid bifurcation [26, 32, 36].

Injury to the sympathetic nerves can result in Horner's syndrome or, rarely, an entity called "first bite syndrome." Horner's syndrome can be complete (miosis, ptosis, anhidrosis) or partial (no anhidrosis) [29].

First bite syndrome is characterized by unilateral pain in the parotid region after the first bite of each meal felt to be due to sympathetic denervation of the parotid gland.

6.7 Infection

Surgical site/patch infection (SSI) rarely occurs following carotid endarterectomy, and, when they occur, most are superficial and self-limiting with antibiotic treatment. The prophylaxis is the best practice to prevent SSI.

Clinically the patient will have neck swelling and drainage from incision in acute events; in delayed infections, a draining sinus tract or pulsatile neck mass could be indicative of a carotid pseudoaneurysm. If infection persists, a patch excision is indicated with early carotid ligation, reconstruction with autogenous vein, or bypass; nowadays the use of negative pressure systems is a good option to perform a better treatment of these infections, resulting in better outcomes and reducing complications of a new exploratory surgery in these patients [3, 13, 19, 25–41].

6.8 Carotid restenosis

The pathology of the restenotic lesion is related to the time of presentation after initial surgery. Most patients with restenosis are asymptomatic and are identified with routine follow-up carotid imaging [29].

It is called early restenosis in those that occur within 2–3 years after CEA and late in those that occur more than 2–3 years after surgery. Patients at increased risk for restenosis include those below age 65, smokers, and women. Patch angioplasty appears to be associated with a decreased risk of long-term recurrent stenosis compared with primary closure [29, 31].

Reintervention is indicated in patients who develop neurologic symptoms referable to the carotid artery and those with restenosis >80% [29, 45].

6.9 Endovascular treatment

Carotid artery angioplasty and stenting are the standard for endovascular carotid intervention that is preferred for most patients with symptomatic internal carotid atherosclerosis. Patients with symptomatic carotid disease treated by endarterectomy that are considered to be less invasive have long-term benefits; nevertheless, it is reported that patients have an increased risk for poor outcomes with endarterectomy such as stroke periprocedural (30 days). So it is recommended in patients with 70–99% stenosis with the following conditions: not suitable surgical access, stenosis radiation induced, restenosis after endarterectomy, and clinically significant cardiac, pulmonary, or other disease that increment risk.

Low dose of aspirin treatment is recommended for all patients who are having an endarterectomy and have to be continued for at least 3 months after surgery; posterior only is indicated with cardiovascular disease.

The endovascular treatment is nowadays the first-line treatment for many vascular diseases. The percutaneous intraluminal angioplasty (PTA) consists in dilatation with a balloon for stenotic lesions, making a dehiscence effect, leading to fracture and separation of the arterial media from the intima; it was the first performed endovascular treatment. The introduction of stents was a major step in the evolution of the endovascular management of carotid stenosis [49].

There are a lot of diapositives that can be used in the endovascular treatment [22, 41, 50]; some of them are the following:

1. Cryoplasty is a type of balloon angioplasty that has liquid nitrous oxide to get inflated and uses cooling a-10°C (14°F) and pressure to dilate the plaque and vessel wall. It is well studied in iliac, infrainguinal, femoropopliteal, and popliteal lesions.

2. Focal pressure balloon was designed to reduce dissection and restenosis and to exhibit focal pressure to the lesions; it is used in infrapopliteal lesions.

3. Drug-coated balloon: drug-eluting stents (DES) achieve local administration of an agent capable of inhibiting intimal hyperplasia without systemic side effects. Cypher stents release sirolimus which has a potent immunosuppressive drug that controls intimal hyperplasia. TAXUS is a second-generation DES, with greater durability and which reduces restenosis. These devices are used in femoropopliteal lesions.

4. Stents are a method to reduce the incidence of restenosis or address balloon PTA failure due to elastic recoil or dissection. They are classified as balloon-expanding (BES) and self-expanding stents and also either as bare metal or covered stents. Self-expanding stents are composed typically of nitinol.

5. Stent grafts

    a. Balloon-expanding stent grafts can be expanded beyond the stated stent diameter; they are used for tracheobronchial strictures but not for PAD, for iliac or renal artery vascular beds, for occlusive lesions, as well as for perforation after PTA or stenting.

    b. Self-expanding stent grafts are used for various applications and in femoropopliteal disease and biliary applications.

    c. Drug-eluting stents have been associated with improved patency for the treatment of PAD compared to conventional balloon angioplasty. These are loaded with paclitaxel and sirolimus usually using polymers; these are used for femoropopliteal disease.

    d. Multilayer stents:

        i. Multilayer flow modulator (MFM) is designed to exclude peripheral or visceral aneurysm while maintaining branch vessel flow. It is a three-dimensional braided tube composed of multilayer wire without any covering prostheses.

        ii. Bioabsorbable stents are initially developed for coronary intervention, now is used also for peripheral arterial beds.

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Author details

Minerva Irene Hernández Rejón* and Manuel Alexis Vargas Robles
Department of General Surgery, Hospital Central Norte PEMEX, Mexico City, Mexico

*Address all correspondence to: minehdezr@gmail.com

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