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Interdisciplinary Evaluation and Management of Asymptomatic Carotid Artery Stenosis: Pharmacological Optimization, Nursing Care Coordination, and Radiological Diagnostic Integration

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Abstract

Background: Asymptomatic carotid artery stenosis (ACAS), a narrowing of the carotid artery without recent neurological symptoms, is a common manifestation of systemic atherosclerosis and a marker for increased stroke and cardiovascular risk. Its management balances the prevention of future stroke against the risks of intervention.

Aim: This review provides an interdisciplinary update on the evaluation and management of ACAS, integrating perspectives from radiological diagnostics, pharmacological optimization, and nursing care coordination to guide evidence-based, patient-centered decision-making.

Methods: The synthesis is based on a comprehensive analysis of current guidelines, clinical trials, and cohort studies. It evaluates the roles of various diagnostic imaging modalities (duplex ultrasound, CTA, MRA) for risk stratification and examines the efficacy of best medical therapy (BMT) versus revascularization (CEA, CAS, TCAR).

Results: Contemporary BMT, including statins, antiplatelets, and aggressive risk factor control, has significantly reduced the annual ipsilateral stroke risk to approximately 0.9-1%. This diminished baseline risk narrows the net benefit of routine revascularization. Current management therefore prioritizes BMT for most patients, reserving intervention for those with high-risk features such as stenosis progression, plaque vulnerability (e.g., intraplaque hemorrhage), or impaired cerebrovascular reserve.

Conclusion: The management of ACAS has evolved towards a personalized, medical-first approach. Interdisciplinary collaboration is essential to identify high-risk patients who may benefit from revascularization while ensuring the majority receive optimized BMT. This strategy, supported by rigorous nursing coordination and advanced radiological diagnostics, optimizes outcomes by minimizing stroke risk and procedural harm.

Keywords: Asymptomatic Carotid Stenosis, Best Medical Therapy, Carotid Endarterectomy, Carotid Artery Stenting, Plaque Vulnerability, Stroke Prevention, Interdisciplinary Care.

1. Introduction

Asymptomatic carotid artery stenosis is conventionally characterized by a luminal narrowing of the proximal internal carotid artery of at least 50 percent at its cervical origin, attributable to atherosclerotic plaque formation. This designation applies to patients who have not experienced a recent ischemic stroke or transient ischemic attack affecting

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the ipsilateral carotid distribution within the preceding six months. While many investigations and clinical protocols adopt a 50 percent threshold to denote stenosis, alternative studies and guideline statements commonly employ higher cutoffs, such as 60 percent, with lesions exceeding 70 percent routinely classified as severe stenosis. The term therefore encompasses a range of degrees of luminal compromise, each of which has differing implications for cerebral hemodynamics and embolic potential [1]. Recognition and appropriate management of asymptomatic carotid stenosis carry importance beyond the immediate risk of ipsilateral stroke. The presence of significant carotid atherosclerosis is a marker of systemic atherothrombotic disease and correlates strongly with concurrent coronary artery disease and overall cardiovascular mortality; thus, carotid stenosis functions both as a focal cerebrovascular lesion and as an indicator of broader vascular risk [2].

Atherosclerotic narrowing of the carotid arteries constitutes a principal etiologic substrate for ischemic stroke. The clinical distinction between symptomatic and asymptomatic carotid disease exerts a dominant influence on therapeutic decision making. In patients who present with recent transient ischemic attack or ischemic stroke localized to the ipsilateral carotid territory, the evidence base for carotid revascularization is relatively direct, and management pathways favor more uniform application of interventions [3][4]. By contrast, the management of asymptomatic lesions has engendered substantial debate, generating divergent recommendations across professional societies and a multiplicity of guideline statements [5][6][7][8]. The pathophysiology underpinning ischemic events related to carotid atherosclerosis encompasses both hemodynamic impairment and embolic phenomena. Embolization from unstable plaque at the carotid bifurcation represents the predominant mechanism for cervical carotid territory infarction, whereas flow-limiting stenosis contributes to ischemia through reduced cerebral perfusion. Importantly, contemporary research has underscored that plaque morphology and composition—rather than degree of stenosis alone—are powerful determinants of future plaque behavior and stroke risk. Features such as a lipid-rich necrotic core, intraplaque hemorrhage, a thin or ruptured fibrous cap, and ulceration correlate with higher embolic potential irrespective of stenosis severity [9][10][11][12].

Therapeutic strategies for asymptomatic carotid stenosis have undergone marked evolution during the past three decades. In the era of randomized controlled trials such as the Asymptomatic Carotid Atherosclerosis Study (ACAS) in the 1990s, the standard of "best medical therapy" was limited; antiplatelet therapy consisted primarily of aspirin, and risk-factor control was less aggressive than current practice. Since that time, medical management has broadened substantially and now encompasses comprehensive secondary prevention measures. Contemporary best medical therapy integrates individualized antiplatelet regimens, strict blood pressure control, optimized glycemic management for patients with diabetes, aggressive lipid-lowering therapy using statins and adjunctive agents where indicated, and structured interventions targeting modifiable lifestyle factors including tobacco cessation, weight management, physical activity, and dietary optimization. These therapeutic refinements have materially reduced the baseline risk of ipsilateral stroke among patients managed noninvasively. Historical event rates reported in trials reflect this change: the five-year ipsilateral stroke risk observed in the ACAS cohort approximated 11 percent, whereas later studies, such as the Asymptomatic Carotid Surgery Trial-1 (ACST-1), documented a substantially lower five-year ipsilateral risk of approximately 3.6 percent in more contemporary practice contexts [13]. Aggregate analyses further corroborate this temporal decline; a systematic review and meta-analysis spanning multiple studies reported a reduction in stroke incidence from 2.8 percent to 1.4 percent over the interval from 1985 to 2007, and current estimates suggest that optimized medical therapy can reduce annual ipsilateral stroke risk to near 1 percent [13]. A 2023 comprehensive review encompassing 73 studies and over 28 000 patients treated noninvasively demonstrated a sustained diminution in ipsilateral ischemic events, estimating a 24 percent reduction in risk for each successive five-year recruitment epoch [14].

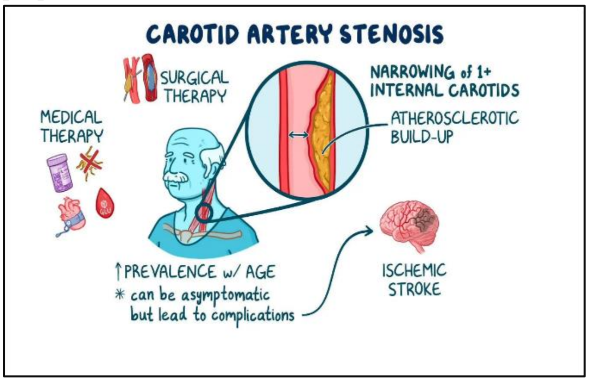

Figure-1: Carotid Artery Stenosis.

These improvements in baseline stroke risk have altered the risk–benefit calculus for carotid revascularization in asymptomatic patients. As the absolute risk attributable to medically managed stenosis has declined, the marginal benefit conferred by carotid endarterectomy or carotid artery stenting has likewise diminished for many individuals. Consequently, indications for revascularization are now more narrowly tailored. Contemporary consensus highlights patient subsets who may still derive meaningful benefit from invasive intervention; these include individuals with very high-grade stenosis (commonly 80 to 99 percent), those exhibiting surrogate markers of plaque instability such as microembolic signals detected on transcranial Doppler, plaques with pronounced echolucency or documented ulceration on duplex ultrasound, patients demonstrating impaired cerebrovascular reserve, subjects with documented progression of stenosis over time, and patients with silent embolic infarcts identified on neuroimaging. Identification of these high-risk phenotypes relies on integration of imaging biomarkers, physiological testing, and longitudinal clinical assessment to stratify risk more precisely than degree of stenosis alone [13][15]. In sum, asymptomatic carotid artery stenosis occupies a nuanced position within cerebrovascular prevention. It signifies a focal atherosclerotic lesion with potential for future ischemic events and concurrently signals systemic atherothrombotic burden. Advances in medical therapy have substantially reduced incident stroke rates in this population, necessitating a more discriminating approach to revascularization that incorporates plaque characteristics, hemodynamic measures, and individualized risk profiling. The contemporary management paradigm therefore emphasizes optimized medical therapy as the foundation of care, reserving invasive strategies for selected patients whose combined clinical and imaging features indicate a sufficiently high residual risk to justify procedural intervention.

Etiology:

Atherosclerotic narrowing of the internal carotid artery develops through a progressive process of lipid deposition, inflammation, and fibrous tissue formation within the arterial intima. Low-density lipoprotein particles infiltrate the intimal layer. Macrophages ingest these lipids and transform into foam cells. Smooth muscle cells migrate from the media and lay down extracellular matrix. Over time these changes form a focal plaque that encroaches on the lumen and alters local flow dynamics. Repeated cycles of injury and repair expand the plaque and may produce a necrotic lipid core, a thin fibrous cap, and neovascularization within the lesion. These structural changes weaken plaque integrity and increase the chance of rupture and thrombosis. The net effect is a lumen that narrows progressively and a plaque that may shed embolic material into the cerebral circulation. Established cardiovascular risk factors accelerate plaque formation and progression. Age exerts a dominant effect; prevalence of significant carotid atherosclerosis increases with advancing decades of life. Male sex associates with higher prevalence in many cohorts, although women may manifest clinically important disease at older ages. Hypertension induces shear stress and endothelial dysfunction, promoting lipid infiltration and intimal fibrosis. Hyperlipidemia, particularly elevated low-density lipoprotein cholesterol, provides the substrate for plaque growth. Cigarette smoking amplifies oxidative stress and inflammation and destabilizes plaques. Genetic predisposition modifies individual vulnerability through pathways that affect lipid metabolism, inflammatory response, and vascular repair mechanisms. Collectively these risk factors create a milieu in which chronic cholesterol accumulation produces focal atheroma at predilection sites such as the carotid bifurcation. [7][16]

Ischemic events associated with carotid stenosis arise from two principal mechanisms. The first is plaque disruption with subsequent distal embolization. When the fibrous cap ruptures or ulcerates, thrombogenic material including platelet aggregates and fibrin can form on the plaque surface or detach as emboli. These emboli travel into the intracranial circulation and occlude downstream arteries, producing focal cerebral ischemia. The second mechanism is hemodynamic compromise. Severe stenosis reduces antegrade flow and lowers cerebral perfusion pressure. When collateral circulation is insufficient, watershed regions and border zones become vulnerable to hypoperfusion, particularly during systemic hypotension. Both embolic and hemodynamic processes can operate within the same patient and can vary over time as plaque morphology and systemic factors change. A minority of carotid stenoses arise from nonatherosclerotic conditions. Fibromuscular dysplasia exemplifies a noninflammatory arteriopathy that causes stenotic, aneurysmal, or dysplastic changes in medium-sized arteries. In contrast to atherosclerosis, fibromuscular dysplasia frequently affects the mid to distal segments of the carotid and may extend into intracranial vessels. Histologically it shows dysplastic smooth muscle and fibrous tissue rather than lipid-rich necrotic cores. Clinically fibromuscular dysplasia often presents in younger patients and shows a marked female predominance. The phenotype includes intermittent cerebral ischemia, arterial dissection, or symptomatic stenosis in a demographic that lacks the conventional atherosclerotic risk profile. Recognition of this etiology matters because management strategies differ and because repair procedures must account for the distinct arterial pathology. [17][18]

Other less common causes of carotid narrowing include radiation-induced arterial injury, vasculitides, and external compression from tumors or neck masses. Radiation to the neck induces endothelial damage, medial fibrosis, and accelerated atherosclerosis in irradiated segments. Systemic inflammatory diseases such as giant cell arteritis or Takayasu arteritis can produce focal stenoses through inflammatory scarring. Extrinsic compression reduces lumen diameter without intrinsic plaque formation and may mimic stenosis on some imaging studies. Accurate etiologic classification requires integration of clinical history, risk factor assessment, and imaging findings that characterize lesion location and tissue properties. Understanding the etiology of carotid stenosis guides risk stratification and therapy. Atherosclerotic plaque burden signals systemic vascular disease and predicts concurrent coronary pathology. Plaque composition and surface morphology inform embolic risk beyond percent stenosis. Nonatherosclerotic causes call for tailored diagnostic and interventional approaches. Effective prevention focuses on modifying the causal factors that drive plaque development including lipid lowering, blood pressure control, smoking cessation, and management of metabolic disorders. Recognition of alternative etiologies ensures appropriate referral and optimizes outcomes. [7][16][17][18]

Epidemiology

Asymptomatic carotid artery stenosis represents a significant subclinical manifestation of systemic atherosclerosis and an important risk marker for future cerebrovascular events. Epidemiological data indicate that the estimated prevalence of severe asymptomatic carotid stenosis, defined as 70% or greater luminal narrowing, ranges between 0.1% and 3.1% in the general population. The prevalence increases with advancing age and varies according to the presence of cardiovascular risk factors such as hypertension, diabetes, and dyslipidemia. Population-level studies estimate that asymptomatic carotid stenosis contributes to approximately 0.7% of the population-attributable risk of ischemic stroke, underscoring its clinical relevance despite the absence of overt neurological symptoms. [13] Stroke continues to rank among the leading causes of death and long-term disability worldwide, and within the United States it remains a major public health burden with significant economic implications. [3][19] Among all ischemic stroke subtypes, approximately 20% are attributable to large artery atherosclerosis, which includes stenosis of the carotid and vertebral arteries. [9][20][21] The pathophysiologic link between carotid stenosis and stroke involves both embolic and hemodynamic mechanisms, and the magnitude of this risk is influenced by plaque morphology, systemic vascular health, and the quality of preventive management. Epidemiologic data estimate that carotid artery stenosis occurs in approximately 13 per 100,000 patients presenting with ischemic stroke. [21] Although this figure represents only a fraction of total stroke cases, the associated morbidity emphasizes the importance of early recognition and optimal management in patients with subclinical disease.

The true incidence of asymptomatic carotid stenosis is difficult to define precisely because the condition is generally silent and often detected incidentally during imaging studies performed for other clinical indications. Many cases are identified through the detection of a carotid bruit on physical examination, though the presence of a bruit has limited sensitivity and specificity for significant luminal narrowing. Population-based screening of asymptomatic individuals is not routinely recommended by major guidelines due to insufficient evidence that such an approach improves clinical outcomes and concerns about overdiagnosis and unnecessary intervention. [22] As a result, epidemiologic estimates rely primarily on cohort studies and cross-sectional data derived from selected populations rather than systematic national surveillance. Sex-related differences have been documented in the epidemiology of carotid stenosis. Asymptomatic carotid stenosis greater than 50% is estimated to occur in approximately 7.5% of men and 5% of women, reflecting the higher prevalence of atherosclerotic disease in men at comparable ages. [23] However, women tend to develop carotid stenosis later in life and may experience a lower absolute risk of ipsilateral stroke when asymptomatic. These sex differences highlight the complex interplay between hormonal influences, vascular biology, and exposure to traditional risk factors. Age remains the strongest determinant of disease prevalence, with a sharp increase observed in individuals over 70 years of age. In this demographic, subclinical carotid atherosclerosis is frequently accompanied by coronary and peripheral arterial disease, illustrating its role as a systemic vascular marker.

Geographic variation also contributes to epidemiologic differences. Studies from Western countries show a higher prevalence of carotid atherosclerosis compared with data from Asian and African populations, likely reflecting differences in diet, lifestyle, genetics, and healthcare access. The adoption of Westernized lifestyles in developing regions has been associated with an increasing burden of atherosclerotic vascular disease, suggesting that the global prevalence of asymptomatic carotid stenosis may rise in parallel with other non-communicable diseases such as diabetes and obesity. Modern epidemiologic trends suggest a gradual decline in stroke incidence and mortality in high-income countries, largely due to improved cardiovascular risk management and widespread implementation of evidence-based therapies such as antihypertensive, lipid-lowering, and antiplatelet medications. However, the prevalence of asymptomatic carotid stenosis has not decreased at the same rate, as improved imaging technologies have increased the detection of subclinical disease. Ultrasound screening in research cohorts has provided valuable insights into population patterns and has shown that mild asymptomatic carotid atherosclerosis carries prognostic significance for future cardiovascular events. Overall, asymptomatic carotid artery stenosis reflects the cumulative burden of systemic atherosclerosis within a population. Although it accounts for a small fraction of total ischemic strokes, its presence identifies individuals at elevated vascular risk who may benefit from aggressive risk factor modification and medical management. The epidemiologic profile underscores the importance of integrating carotid disease assessment into broader strategies of cardiovascular prevention, emphasizing control of modifiable risk factors such as hypertension, hyperlipidemia, smoking, and diabetes. [13][19][21][23]

Pathophysiology

Atherosclerotic plaque formation within the carotid arteries represents a chronic, multifactorial process that develops over many years due to the interaction of hemodynamic, metabolic, and inflammatory mechanisms. The process tends to occur at specific anatomic sites characterized by disturbed blood flow patterns rather than uniform laminar flow. The carotid bifurcation and bulb regions are particularly predisposed to atherosclerotic changes because of their nonlinear vascular geometry and the presence of oscillatory shear stress. These local hemodynamic forces promote endothelial dysfunction, initiating lipid accumulation and inflammatory infiltration in the intimal layer of the artery, ultimately leading to intima-media thickening and plaque formation. [24][25] At the cellular level, endothelial injury plays a central role in the initiation of atherosclerosis. Low shear stress at bifurcation points alters endothelial cell morphology and function, increasing the expression of adhesion molecules and permeability to low-density lipoprotein (LDL) cholesterol. LDL particles infiltrate the intima, where they undergo oxidative modification, stimulating monocyte recruitment and differentiation into macrophages. These macrophages engulf oxidized LDL to form foam cells, which accumulate to form fatty streaks, the earliest visible lesion in atherosclerosis. Smooth muscle cells migrate from the media into the intima and synthesize extracellular matrix components such as collagen and elastin, contributing to plaque growth and fibrous cap formation. Over time, continued lipid accumulation, inflammation, and necrosis within the plaque core can destabilize the fibrous cap, predisposing it to rupture and embolization.

The hemodynamic environment of the carotid bifurcation further amplifies plaque progression. Oscillatory shear stress and turbulent flow contribute to cyclic mechanical stress on the arterial wall, accelerating lipid deposition and calcification. These regions also exhibit increased reactive oxygen species (ROS) production and reduced nitric oxide availability, both of which impair vasodilation and promote vascular inflammation. The cumulative result is a complex plaque composed of lipids, inflammatory cells, necrotic tissue, and calcific deposits that narrow the arterial lumen and impair cerebral perfusion. Asymptomatic carotid stenosis becomes clinically significant when the lesion progresses to a degree that compromises cerebral blood flow or gives rise to embolic phenomena. Two primary mechanisms are implicated in the transition from asymptomatic to symptomatic disease: embolization and hemodynamic failure. Embolic events occur when unstable plaque material, thrombus, or cholesterol crystals dislodge and travel distally into the cerebral circulation, causing transient ischemic attacks or ischemic strokes. Alternatively, severe luminal narrowing can result in reduced perfusion pressure distal to the stenosis, particularly in the presence of inadequate collateral circulation, leading to watershed infarctions or global hypoperfusion.

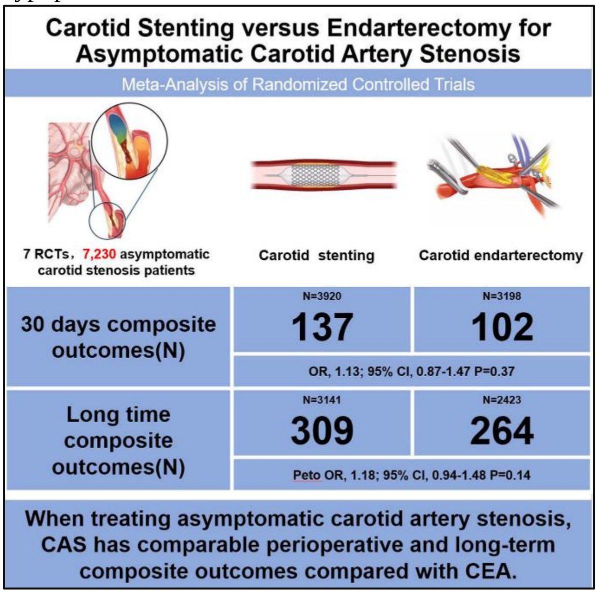

Figure-2: Carotid Stenting Vs. Asymptomatic Carotid Stenosis.

Plaque morphology is a crucial determinant of embolic potential. Certain imaging features, including plaque echolucency, heterogeneity, and ulceration, have been shown to correlate with increased risk of rupture and subsequent embolization. [11][26] Echolucent plaques, which contain large lipid cores and fewer calcified regions, are particularly vulnerable to rupture. Similarly, irregular surface morphology and plaque ulceration disrupt laminar flow, creating localized zones of turbulence that favor thrombus formation and detachment. In contrast, heavily calcified and homogeneous plaques tend to be more stable, although they may still contribute to chronic hemodynamic compromise. Inflammatory activity within the plaque also plays a major role in determining stability. Activated macrophages and T-lymphocytes secrete proteolytic enzymes such as matrix metalloproteinases (MMPs), which degrade the fibrous cap, weakening the structural integrity of the lesion. Systemic inflammatory states, such as those associated with metabolic syndrome, diabetes, and chronic infections, can exacerbate these processes and accelerate plaque instability. Furthermore, oxidative stress and endothelial dysfunction contribute to a prothrombotic state, amplifying the risk of acute ischemic events. While asymptomatic carotid stenosis does not always predict ipsilateral ischemic stroke, it is a well-established marker of systemic atherosclerosis and thus a strong indicator of elevated cardiovascular morbidity and mortality. [2] Patients with carotid stenosis frequently have concomitant coronary and peripheral arterial disease, reflecting widespread endothelial dysfunction and vascular injury. The coexistence of these conditions underscores the systemic nature of atherosclerosis as a generalized vascular disorder rather than a localized pathology confined to a single arterial segment.

In addition, systemic factors such as hypertension, hyperlipidemia, and smoking not only contribute to the development of carotid plaque but also influence its biological behavior. Chronic hypertension increases mechanical stress on the arterial wall, leading to endothelial injury and further intimal proliferation. Elevated serum cholesterol levels promote lipid deposition and oxidation, while smoking introduces pro-inflammatory and pro-coagulant effects that impair endothelial repair mechanisms. These risk factors collectively create a biochemical environment conducive to progressive arterial narrowing and plaque instability. In patients with asymptomatic carotid stenosis, compensatory mechanisms, including collateral flow through the circle of Willis, may maintain adequate cerebral perfusion despite significant luminal narrowing. However, when these compensatory pathways are insufficient or when systemic perfusion decreases, cerebral hypoperfusion may occur. This situation increases susceptibility to ischemic injury, particularly during periods of systemic hypotension or cardiac arrhythmia. Ultimately, the pathophysiology of asymptomatic carotid stenosis reflects a dynamic interplay between mechanical forces, lipid metabolism, inflammatory activity, and systemic vascular health. The degree of stenosis alone does not fully determine the risk of stroke or progression; rather, plaque composition and biological behavior are now recognized as more predictive indicators of adverse outcomes. Understanding these mechanisms provides a foundation for both diagnostic evaluation and targeted therapeutic intervention aimed at stabilizing plaques, restoring endothelial function, and preventing cerebrovascular complications. [24][25][26]

History and Physical

The evaluation of a patient with suspected or known asymptomatic carotid artery stenosis begins with a comprehensive history and physical examination. The goal is to identify vascular risk factors, assess neurologic status, and determine the likelihood of significant carotid obstruction. Because carotid atherosclerosis shares common etiologic pathways with systemic vascular disease, careful attention must be given to the patient's overall cardiovascular risk profile. Major risk factors include advanced age, male sex, hypertension, hyperlipidemia, hyperglycemia, obesity, cigarette smoking, and a positive family history of premature cardiovascular disease. [7][16] Each of these contributes to endothelial dysfunction and the progression of atherosclerotic plaque. Elderly patients, particularly men over 65, represent the demographic most likely to develop hemodynamically significant carotid stenosis. Hypertension accelerates intimal thickening, while elevated serum lipids promote cholesterol deposition within the arterial wall. Diabetes mellitus and metabolic syndrome further exacerbate vascular inflammation and oxidative stress, increasing the likelihood of both carotid and coronary involvement. The history should begin with an assessment of any prior cerebrovascular or cardiovascular events. Patients should be questioned about previous strokes, transient ischemic attacks (TIAs), or unexplained episodes of speech disturbance, facial droop, or limb weakness. Symptoms such as transient monocular blindness (amaurosis fugax) or slurred speech can signal embolic phenomena originating from a diseased carotid artery. However, symptoms like vertigo, diplopia, lightheadedness, or syncope are not characteristic of isolated unilateral carotid stenosis, as these are typically associated with vertebrobasilar insufficiency or other neurologic causes.

The history should also include detailed information on comorbidities that may modify management decisions. Prior neck surgeries, radiation exposure, or cervical trauma can alter vascular anatomy and affect both diagnostic imaging and the feasibility of carotid endarterectomy or stenting. A history of coronary artery disease, peripheral vascular disease, or chronic kidney disease provides additional evidence of systemic atherosclerosis and influences overall risk stratification. The patient's medication profile should be reviewed, with emphasis on antihypertensives, lipid-lowering drugs, and antiplatelet agents, since these directly impact disease control and future management. Physical examination begins with general observation of the patient's cardiovascular status. Blood pressure should be measured in both arms to detect discrepancies that may indicate subclavian stenosis or aortic disease. The clinician should inspect the neck for scars or deformities that might complicate surgical exposure. Auscultation of the carotid and vertebral arteries should be performed with the patient supine and the head slightly extended. The detection of a carotid bruit suggests turbulent flow due to luminal narrowing, but its diagnostic accuracy is limited. A bruit has approximately 53% sensitivity and 83% specificity for detecting stenosis greater than 70%. [27] Absence of a bruit does not rule out significant disease, and the presence of a bruit does not necessarily correlate with severity, as it may disappear when the vessel becomes severely narrowed and flow decreases. A focused neurologic examination is essential. The clinician should evaluate cranial nerves, motor strength, coordination, speech, and visual fields. Attention should be paid to subtle deficits such as transient weakness, facial asymmetry, or sensory changes, which may suggest previous ischemic events. Assessment of gait and balance provides additional information about cerebral function. Fundoscopic examination may reveal cholesterol emboli (Hollenhorst plaques) in the retinal vessels, which are indicative of proximal carotid plaque embolization.

Screening for asymptomatic carotid artery disease remains controversial. Population-wide screening is not recommended due to low overall prevalence and high rates of false positives. However, selected high-risk individuals may benefit from targeted assessment. A grade 2 recommendation supports screening asymptomatic patients with increased risk—those with multiple vascular risk factors or known peripheral or coronary artery disease—provided that the patient is willing to consider intervention if significant stenosis is found. [22][28] The decision to screen should be individualized, balancing the potential benefits of early detection against procedural risks and patient preferences. When physical findings or history raise suspicion, noninvasive imaging such as carotid duplex ultrasonography is typically the first-line diagnostic test. It provides detailed information on flow velocity, plaque morphology, and degree of stenosis. If ultrasound findings are equivocal or if surgical planning is required, additional imaging with CT angiography (CTA) or MR angiography (MRA) can be used to confirm the diagnosis and assess intracranial circulation. Evaluation should not focus solely on carotid pathology but should encompass the patient's overall vascular health. Management of modifiable risk factors is central to preventing progression. Tight control of blood pressure, lipid levels, and blood glucose is critical. Smoking cessation should be emphasized, as tobacco use greatly increases stroke risk and accelerates atherosclerosis. Weight reduction and physical activity improve vascular function and reduce systemic inflammation. In summary, the history and physical evaluation of asymptomatic carotid artery stenosis demand a comprehensive approach that integrates risk assessment, neurologic evaluation, and careful physical examination. Identifying high-risk patients who might benefit from intervention requires clinical judgment grounded in evidence-based practice. The presence of vascular risk factors and a bruit may warrant further investigation, but the final management strategy must always account for the individual's overall cardiovascular risk, life expectancy, and readiness to undergo treatment. Through systematic assessment and targeted risk modification, clinicians can effectively reduce stroke risk and improve long-term vascular outcomes in patients with asymptomatic carotid disease. [7][16][22][27][28]

Evaluation

The evaluation of asymptomatic carotid artery stenosis relies on accurate imaging and hemodynamic assessment to determine the degree of narrowing, plaque morphology, and stroke risk. Diagnostic precision is vital, as management strategies—including medical therapy or revascularization—depend on the severity and characteristics of the lesion. The goal is to identify patients who remain stable under medical management and those at higher risk for cerebrovascular events who might benefit from intervention. The diagnostic approach begins with noninvasive, cost-effective modalities. Carotid duplex ultrasound is the first-line investigation in most cases due to its accessibility, safety, and ability to provide both anatomic and hemodynamic data. This test combines B-mode imaging and Doppler flow assessment to measure the degree of stenosis and characterize plaque features. In the presence of significant narrowing, the waveform exhibits a high-resistance pattern within the carotid bulb and proximal internal carotid artery, with reduced or absent distal flow signals. [30] Duplex ultrasound can also identify the presence of echolucent or heterogeneous plaques, which have been correlated with increased vulnerability to rupture and embolization. [29]

When duplex findings suggest advanced disease or when revascularization is being considered, additional imaging is warranted. Computed tomography angiography (CTA) and magnetic resonance angiography (MRA) are widely employed for this purpose. CTA provides high-resolution, three-dimensional visualization of the vessel lumen and wall, allowing precise measurement of stenosis severity and detection of calcification or ulceration within plaques. CTA also enables assessment of intracranial circulation and collateral blood flow, which is critical in determining the brain's capacity to compensate for reduced carotid perfusion. MRA, on the other hand, offers a radiation-free alternative with excellent soft-tissue contrast, capable of differentiating between lipid-rich necrotic cores and fibrous or calcified components. [30] MRA can also identify intraplaque hemorrhage, a key marker of instability associated with a higher risk of future ischemic events. Digital subtraction angiography (DSA) remains the gold standard for precise vascular imaging, offering dynamic assessment of flow and collateral pathways. However, due to its invasive nature and potential complications such as arterial injury or stroke, DSA is now primarily reserved for cases in which noninvasive modalities are inconclusive or when endovascular intervention is planned. [29] The integration of imaging findings from multiple modalities provides a comprehensive evaluation of both structural and functional aspects of carotid artery disease.

Beyond the degree of stenosis, several imaging-derived markers help stratify risk among patients with asymptomatic carotid stenosis. The identification of microembolic signals using transcranial Doppler ultrasound (TCD) is one such marker. Detection of these signals suggests active plaque instability and ongoing embolization, which may warrant surgical or endovascular intervention. [15][31] Similarly, plaque echolucency identified through duplex ultrasound reflects a lipid-rich core and thin fibrous cap, indicating a higher likelihood of rupture. Progression of stenosis over time, especially when documented on serial imaging, also signifies an elevated stroke risk. Advanced imaging can reveal additional high-risk features, including silent embolic infarcts on brain CT or MRI. These lesions, though clinically asymptomatic, indicate prior embolic activity and a heightened vulnerability to future cerebrovascular events. [31] Carotid plaque ulceration, visible on CTA or ultrasound, represents surface disruption that facilitates thrombus formation and embolization. MRI can further identify intraplaque hemorrhage, an imaging hallmark of vulnerability, which correlates strongly with recurrent ischemic events.

Evaluation of cerebrovascular reserve (CVR) is another crucial element in risk stratification. CVR assesses the capacity of cerebral vessels to dilate in response to decreased perfusion pressure. Techniques such as transcranial Doppler or perfusion MRI during hypercapnia or pharmacologic vasodilation can quantify this reserve. A reduced cerebrovascular reserve suggests impaired autoregulation and limited compensatory ability, increasing susceptibility to ischemia in the event of further flow reduction. [30] The integration of structural and hemodynamic data allows clinicians to determine which patients may benefit from carotid endarterectomy (CEA) or carotid artery stenting (CAS). For instance, patients with severe (≥80%) stenosis and evidence of high-risk imaging features—such as echolucent plaques, intraplaque hemorrhage, or microembolic signals—are more likely to derive benefit from revascularization compared with those managed medically. Conversely, patients with stable plaques, good collateral circulation, and preserved cerebrovascular reserve can often be managed with optimal medical therapy alone. In clinical practice, evaluation should not only focus on the carotid arteries but also consider the patient's systemic atherosclerotic burden. Multivessel disease is common in individuals with carotid stenosis, and imaging findings often prompt further cardiovascular assessment, including coronary or peripheral arterial evaluation. A multidisciplinary approach involving radiologists, neurologists, vascular surgeons, and internists is essential to interpret findings accurately and develop individualized treatment plans. In summary, the evaluation of asymptomatic carotid artery stenosis requires a structured, stepwise approach beginning with carotid duplex ultrasound and advancing to cross-sectional and angiographic modalities when indicated. Imaging provides vital information beyond the degree of luminal narrowing, encompassing plaque composition, embolic activity, and cerebrovascular reserve. These parameters guide clinical decision-making, allowing differentiation between patients who will benefit from intervention and those best managed conservatively. Continuous advances in imaging technology continue to refine this process, improving the precision of risk assessment and optimizing outcomes for patients with asymptomatic carotid disease. [15][29][30][31]

Treatment / Management

Optimal management of asymptomatic carotid artery stenosis begins with rigorous implementation of best medical treatment directed at global vascular risk reduction. Contemporary evidence establishes that multifactorial risk modification reduces the incidence of ipsilateral ischemic events to levels that often obviate the net benefit of routine revascularization. Medical management must therefore be regarded as the foundational therapeutic strategy and the default pathway for most patients. Core components include strict blood pressure control, individualized lipid-lowering therapy with high-intensity statins when indicated, glycemic optimization in diabetic patients, antiplatelet therapy as appropriate, and structured programs to promote smoking cessation, weight control, dietary modification, and regular aerobic exercise. Statins reduce lipid-rich plaque burden and modulate inflammatory activity within atherosclerotic lesions, thereby decreasing the propensity for plaque rupture and embolization. Aspirin remains a commonly used agent for primary prevention in selected patients with asymptomatic carotid disease, although the decision to prescribe antiplatelet therapy must account for bleeding risk and overall cardiovascular profile. Lifestyle interventions that produce durable reductions in cardiovascular risk factors are essential adjuncts to pharmacologic therapy and must be actively reinforced through serial clinical encounters and structured follow-up programs. [32][33][34][7][35][36][37]

The contemporary prognosis for patients managed with optimized medical therapy is substantially improved compared with historical cohorts. Best medical treatment alone can reduce the annual ipsilateral stroke risk to about 1 percent, a magnitude that narrows the absolute advantage obtained from carotid endarterectomy or carotid artery stenting in many patients. This reduction in event rates mandates careful individualization of decisions regarding revascularization. Revascularization should be reserved for patients whose residual risk under optimal medical therapy remains sufficiently high to justify procedural risk and to provide a measurable net clinical benefit. Identifying such patients requires integration of stenosis severity with imaging biomarkers of plaque vulnerability, physiologic measures of cerebrovascular reserve, evidence of silent embolic infarcts on neuroimaging, and documentation of progressive luminal narrowing. Consensus guidance from major vascular societies reflects this individualized paradigm. The European Society for Vascular Surgery recommends consideration of carotid endarterectomy in patients with 60 to 99 percent stenosis, an acceptable perioperative surgical risk of 3 percent or less, and a life expectancy greater than five years, provided one or more high-risk plaque or physiologic characteristics are present. These high-risk features include microembolic signals on transcranial Doppler, plaque echolucency or ulceration on duplex ultrasound, intraplaque hemorrhage on MRI, reduced cerebrovascular reserve, documented progression of stenosis, juxtaluminal hypoechoic expansion, and silent embolic infarcts on brain CT or MRI. Selection criteria emphasize that revascularization should target lesions demonstrating biological aggressiveness or hemodynamic vulnerability rather than stenosis percentage alone. [15][13]

When revascularization is contemplated, the choice between carotid endarterectomy (CEA) and carotid artery stenting (CAS) demands careful consideration of patient-specific anatomical, clinical, and procedural factors. CEA has an extensive evidence base demonstrating efficacy in selected patients but carries operative risks, principally perioperative stroke, myocardial infarction, and cranial nerve injury. CAS offers a less invasive alternative and may be preferable for patients with hostile neck anatomy, prior neck irradiation, prior carotid surgery, or significant comorbidities that increase the risk of open surgical repair. Early randomized data suggested higher periprocedural stroke rates for CAS and higher myocardial infarction rates for CEA, although these findings are procedure and operator dependent and must be interpreted in the context of evolving technique and technology. Those historical comparisons were derived predominantly from transfemoral CAS approaches and from eras with different patient selection and operator experience. Advances in stent design, embolic protection devices, and procedural technique have narrowed outcome differentials, while operator expertise and center volume remain strong determinants of procedural safety. [38][39]

Transcarotid artery revascularization (TCAR) represents an important technical evolution within the spectrum of endovascular options. TCAR employs a direct carotid access via a small incision at the base of the neck and utilizes dynamic flow reversal for cerebral protection during stent deployment. Comparative studies have shown lower periprocedural stroke and mortality rates with TCAR relative to transfemoral CAS in selected cohorts, suggesting that TCAR may combine the benefits of endovascular therapy with improved neuroprotection. Nevertheless, TCAR is not universally applicable and requires anatomical suitability and operator expertise. Device selection, cerebral protection strategy, and antiplatelet regimen must be tailored to the chosen revascularization modality. [40][41] Perioperative and periprocedural medical optimization is critical for minimizing complications and improving outcomes regardless of procedural choice. Preprocedural assessment should document baseline neurologic status, cardiac risk, renal function, and antithrombotic requirements. Perioperative blood pressure control limits fluctuations that could precipitate cerebral hypoperfusion or hyperperfusion injury. Antiplatelet therapy protocols vary by center and procedure; dual antiplatelet therapy is commonly administered periprocedurally for CAS, whereas CEA may proceed on single-agent antiplatelet therapy with consideration of dual therapy in specific circumstances. Periprocedural statin therapy is associated with improved outcomes and should be continued. Meticulous intraoperative technique combined with vigilant postoperative monitoring for neurologic deficits, wound complications, myocardial ischemia, and cranial nerve dysfunction is mandatory. Nursing teams play a central role in early recognition of complications, hemodynamic surveillance, and coordination of multidisciplinary care during recovery.

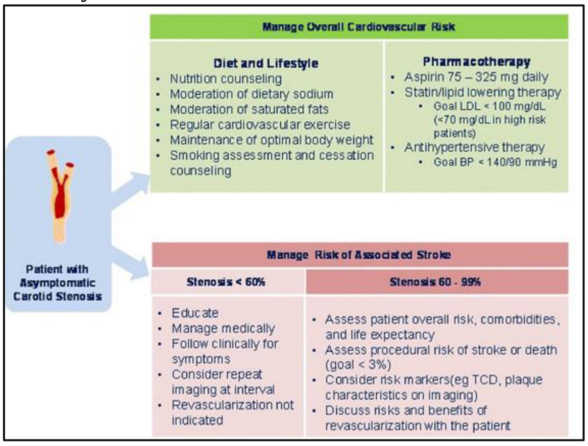

Figure-3: Management of Carotid Artery Stenosis.

Special populations require individualized strategies. Patients with prior neck irradiation, prior cervical surgery, anatomic variants, or connective tissue disorders may be preferentially managed with CAS or TCAR when open surgery is hazardous. Conversely, highly calcified plaques or lesions with extensive common carotid involvement may be better treated with CEA. Age and frailty influence the risk–benefit calculus; older patients, particularly those with limited life expectancy or multiple comorbidities, derive less absolute benefit from invasive revascularization given the competing risks and the low baseline stroke rate under medical therapy. Shared decision making that incorporates patient values, life expectancy, and expected procedural risk is therefore indispensable. The intersection of carotid and coronary disease adds further complexity. A subset of patients with asymptomatic carotid stenosis present concurrently with severe coronary artery disease requiring coronary artery bypass grafting (CABG). Management options include simultaneous CEA and CABG, staged procedures, or hybrid approaches combining CAS with percutaneous coronary intervention. Each strategy entails trade-offs related to cumulative perioperative risk, timing of antiplatelet therapy, and logistical considerations. The ACCF/AHA provides a class IIb recommendation for carotid revascularization in patients undergoing CABG who have bilateral asymptomatic stenosis greater than 70 percent or unilateral stenosis greater than 70 percent with contralateral carotid occlusion. These recommendations reflect the absence of robust outcome data and underscore the necessity of individualized treatment planning in multidisciplinary settings. [42][43][44]

Long-term surveillance after either conservative management or revascularization is essential. For patients managed medically, surveillance focuses on control of modifiable risk factors, adherence to statin and antiplatelet therapy when indicated, smoking cessation maintenance, and periodic imaging to detect progression of stenosis or emergence of high-risk plaque features. Duplex ultrasound intervals should be individualized based on baseline stenosis severity, plaque morphology, and clinical trajectory; typical schedules include an initial follow-up at 6 to 12 months and then annually if stable. After CEA, surveillance aims to detect restenosis, which may occur due to intimal hyperplasia or recurrent atherosclerosis; duplex ultrasound at regular intervals allows early detection and timely consideration of reintervention. After CAS or TCAR, surveillance should similarly document stent patency and identify in-stent restenosis. Pharmacologic stewardship is a longitudinal task. Lifelong statin therapy for appropriate patients, continued antiplatelet therapy as clinically indicated, and aggressive management of hypertension and diabetes remain pillars of secondary prevention. Pharmacy engagement in medication reconciliation, adherence counseling, and management of polypharmacy enhances the durability of medical therapy. Nursing coordination ensures ongoing patient education, symptom recognition, and facilitation of outpatient follow-up.

Health system considerations include ensuring procedure volumes and operator expertise meet quality benchmarks, building multidisciplinary teams that include vascular surgeons, interventional radiologists, neurologists, cardiologists, anesthesiologists, pharmacists, and nursing specialists, and implementing protocols for preprocedural assessment and postoperative pathways to minimize variability in care. Centralized review of outcomes, participation in registries, and adherence to guideline-based indications optimize patient selection and procedural safety. In conclusion, the management of asymptomatic carotid artery stenosis requires a predominantly medical axis complemented by selective revascularization for individuals at sufficiently high residual risk despite contemporary best medical therapy. Treatment planning must integrate patient-specific clinical attributes, detailed imaging biomarkers of plaque instability, procedural risk estimates, life expectancy, and patient preferences. Multidisciplinary collaboration, meticulous perioperative management, and rigorous long-term surveillance maximize the therapeutic ratio and align care with contemporary evidence that prioritizes prevention, safety, and individualized decision making.

Differential Diagnosis:

The differential diagnosis of asymptomatic carotid artery stenosis requires careful clinical and imaging evaluation to identify other vascular or systemic pathologies that may present with similar findings but require different management approaches. Carotid artery dissection is a key differential condition. It occurs when a tear in the intimal layer allows blood to enter the arterial wall, forming an intramural hematoma that narrows the lumen. It can mimic atherosclerotic stenosis on imaging but usually affects younger patients and may be associated with trauma, connective tissue disorders, or spontaneous onset. High-resolution magnetic resonance angiography and computed tomography angiography help distinguish dissection by revealing a tapered stenosis or a double-lumen sign. Vasculitis and arteritis, including Takayasu arteritis and giant cell arteritis, represent inflammatory causes of arterial narrowing. These disorders typically involve systemic symptoms such as fever, malaise, and elevated inflammatory markers. Imaging findings often show long, smooth stenotic segments rather than focal eccentric plaques. Laboratory evaluation for inflammatory markers and biopsy confirmation are essential to differentiate these from atherosclerotic lesions.

Congenital abnormalities, such as fibromuscular dysplasia, should also be considered. This condition produces a characteristic “string of beads” appearance on angiography due to alternating areas of stenosis and dilation. It often affects middle-aged women and can cause carotid narrowing without traditional atherosclerotic risk factors. Connective tissue disorders like Marfan syndrome and Ehlers-Danlos syndrome can cause arterial wall fragility and aneurysm formation, occasionally leading to secondary narrowing or dissection. Similarly, hypercoagulable states—including antiphospholipid syndrome and inherited thrombophilias—can cause thrombus formation within normal carotid arteries, resulting in embolic phenomena rather than true stenosis. Finally, embolization from cardiac sources, such as atrial fibrillation or valvular disease, can cause silent cerebral infarctions and mimic the downstream effects of carotid stenosis. Differentiation depends on cardiac evaluation through echocardiography and rhythm monitoring. Accurate diagnosis ensures appropriate treatment strategies are implemented to prevent stroke and other vascular complications.

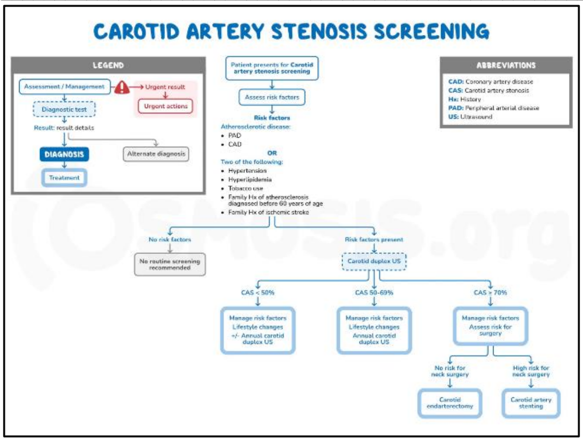

Figure-4: Carotid Artery Stenosis Screening.

Ongoing Trials

As of October 2023, a multicenter cohort comprising 9,830 patients treated across 103 clinical sites reported that 61 percent of enrolled individuals were asymptomatic while 38.9 percent presented with symptoms; the observed 30-day stroke incidence in this aggregate was 1.8 percent and the combined 30-day stroke or death rate equaled 2.6 percent [13]. These outcome metrics derive from large contemporary registries and ongoing trials that seek to define current procedural risk and to compare revascularization strategies against optimized conservative care in an era of substantial advances in medical prevention. The tabulated registry data referenced in the primary report provide context for interpreting procedural safety across diverse centers and operator experience levels [13]. Critical analysis of earlier randomized trials that established the role of carotid endarterectomy in asymptomatic disease underscores important temporal limitations. Landmark studies such as ACAS and ACST were conducted in an era when statin therapy was not routine and global risk factor control was comparatively limited; consequently, their observed absolute benefit from carotid endarterectomy reflected the standard of care of that time rather than present-day best medical therapy. Trial-to-trial heterogeneity in background medical management further complicates direct extrapolation of those trial results to current practice because antiplatelet regimens, lipid lowering strategies, and blood pressure targets varied substantially and did not uniformly incorporate modern high-intensity statin use or aggressive multifactorial risk reduction [48].

Contemporary evidence demonstrates a secular decline in ipsilateral ischemic events among patients with asymptomatic carotid stenosis managed noninvasively. Several observational and prospective cohorts document progressively lower rates of stroke and transient ischemic attack in medically treated patients as recruitment epochs advance and as guideline-driven cardiovascular prevention has become standard. These temporal trends have prompted reappraisal of the net benefit of revascularization for many asymptomatic patients and have driven the design of ongoing randomized studies that explicitly compare modern best medical therapy against revascularization with current techniques and cerebral protection strategies. Critics of routine intervention emphasize that improvements in pharmacologic therapy, multimodal risk-factor control, and systems of longitudinal patient follow up to reduce baseline event rates to levels at which the marginal absolute reduction attributable to surgical or endovascular intervention becomes small and may not exceed perioperative risk for many patients [48][49].

Trialists and guideline panels therefore now focus on more granular risk stratification and on imaging and physiologic biomarkers to identify subgroups in whom the balance of benefit and harm still favors revascularization. Contemporary randomized and registry-based trials incorporate metrics such as plaque morphology, intraplaque hemorrhage on MRI, transcranial Doppler–detected microembolic signals, progression of stenosis on serial imaging, and measures of cerebrovascular reserve. These trials also emphasize standardized perioperative quality metrics and robust adjudication of procedure-related events in order to generate reliable contemporary estimates of procedural morbidity and mortality. Because prior randomized data did not reflect modern secondary prevention, new trials attempt to isolate the incremental value of revascularization over today’s medical standards rather than retesting interventions against outdated comparators. Methodologic challenges remain. Low event rates under contemporary medical therapy require larger sample sizes or enriched high-risk subpopulations to achieve adequate statistical power. Operator and center volume effects introduce heterogeneity that registries may quantify but that randomized designs must control. Long follow up is necessary to observe differences in cumulative stroke incidence and to capture late events related to restenosis or progression of systemic atherosclerosis. Finally pragmatic considerations include patient selection and willingness to accept randomization when equipoise is imperfectly perceived by clinicians and patients. Despite these obstacles, ongoing trials and registries continue to refine the evidence base so that clinical decision making will rest on contemporary risk estimates that integrate procedural safety, individual plaque biology, and the effectiveness of modern comprehensive medical therapy [13][48][49].

Prognosis

Patients with asymptomatic carotid artery stenosis face a low but nonnegligible risk of ipsilateral ischemic stroke under contemporary management. Cohort analyses estimate an annual ipsilateral stroke incidence of near 0.9 percent. [50] This figure represents an aggregate risk that varies substantially according to individual characteristics such as degree of luminal narrowing, plaque morphology, evidence of prior silent embolization, cerebrovascular reserve, and the burden of systemic atherosclerotic disease. High-grade stenosis alone does not uniformly predict outcome; plaques that are echolucent, ulcerated, or contain intraplaque hemorrhage confer a disproportionately greater risk of embolic events than does percent stenosis measured in isolation. Transcranial Doppler–detected microembolic signals and silent infarcts on neuroimaging similarly identify subgroups with a worse natural history despite the absence of clinical events. Temporal trends demonstrate a progressive reduction in stroke rates among patients with asymptomatic carotid disease managed with intensive medical therapy. Multiple observational series and meta-analyses document declining event rates over recent decades as statin therapy, tighter blood pressure control, improved glycemic management, and structured lifestyle interventions have become standard. These improvements have altered the risk–benefit calculus of prophylactic revascularization and shifted management toward individualized selection of candidates for intervention. [48][51] Consequently, overall prognosis for most patients managed conservatively has improved; many patients maintain functional independence and avoid ipsilateral stroke when adherence to secondary prevention is sustained.

Prognostic heterogeneity remains important to recognize. Patients who present with imaging markers of plaque vulnerability, progressive stenosis on serial surveillance, impaired cerebrovascular reserve, or repeated microembolic signals are at substantially higher risk than the population mean. In these individuals the cumulative risk over several years may justify consideration of carotid endarterectomy or carotid stenting when procedural risk is acceptably low and life expectancy is sufficient to realize benefit. Conversely, frail elderly patients and those with multiple competing comorbidities face limited absolute benefit from invasive strategies given their reduced life expectancy and the low baseline stroke rate achievable with optimized medical care. Longitudinal prognosis also depends on systemic cardiovascular risk. The presence of asymptomatic carotid stenosis signals generalized atherosclerosis and predicts heightened risk of coronary and peripheral arterial events. Mortality in this population often relates to cardiac rather than neurologic causes. Therefore, prognostic assessment should integrate cerebrovascular risk with global vascular risk reduction. Rigorous secondary prevention including high-intensity lipid lowering, antihypertensive therapy, antiplatelet strategies where indicated, smoking cessation, and diabetes control improves both stroke-free survival and overall longevity. Surveillance and timely reassessment influence outcomes. Periodic duplex ultrasound and, where indicated, advanced imaging to detect new high-risk plaque features or progression of stenosis permit recalibration of management plans. Patient adherence to medical therapy and access to coordinated multidisciplinary care are modifiable determinants of prognosis. In sum, the contemporary outlook for patients with asymptomatic carotid artery stenosis is more favorable than historical estimates. The average annual ipsilateral stroke risk approximates 0.9 percent; nonetheless individualized prognosis varies widely and hinges on plaque biology, hemodynamic factors, comorbidity burden, and the intensity of preventive care. [50][48][51]

Complications

Asymptomatic carotid artery stenosis carries a principal risk of progression to symptomatic cerebrovascular disease, most notably transient ischemic attack and ischemic stroke. Longitudinal data indicate that the annual transition rate from asymptomatic to symptomatic carotid stenosis approximates 0.9 percent, a figure that encapsulates the heterogeneous natural history of this condition and varies according to plaque characteristics, hemodynamic reserve, and patient comorbidity burden [50]. The clinical consequence of progression is substantial because ischemic events produce enduring neurologic deficits, functional dependence, and increased mortality. In addition to the risk of spontaneous embolic or hemodynamic cerebral ischemia, patients with carotid atherosclerosis face elevated systemic vascular risk; the presence of carotid plaque signifies generalized atherothrombotic disease and correlates with increased incidence of coronary artery disease and cardiovascular mortality [2]. Consequently, complications associated with carotid stenosis extend beyond the cerebral vascular territory and encompass major adverse cardiac events and systemic vascular morbidity. When invasive management is undertaken, complication profiles depend on the chosen modality, the patient’s anatomic and physiologic characteristics, and procedural expertise. Carotid endarterectomy and carotid artery stenting are both effective in selected patients, but each carries specific periprocedural and longer-term risks. Procedural complications that have been documented in contemporary series include perioperative myocardial infarction, perioperative stroke, transient ischemic attack, cranial nerve injury with resultant dysphonia or dysphagia, hemorrhagic complications both at the operative site and intracranially, wound infection, and restenosis due either to intimal hyperplasia or recurrent atherosclerosis [52]. The incidence and severity of these complications are modulated by institutional volume, operator experience, patient frailty, and the presence of comorbid coronary or peripheral vascular disease. Perioperative stroke remains the most feared adverse outcome because it negates the intended prophylactic benefit of revascularization and may produce permanent disability or death.

Beyond immediate procedural risks, longer-term complications warrant consideration. Restenosis after endarterectomy or stent placement may compromise the durability of intervention and necessitate repeat imaging surveillance and, occasionally, reintervention. In-stent restenosis following carotid stenting can present technical challenges and may require alternative endovascular strategies or conversion to open repair. Neurocognitive sequelae and subtle declines in executive function have been observed in some cohorts after carotid revascularization, although the causality and clinical significance remain debated. Additionally, the suppression of platelet function required for stent patency introduces bleeding risk, which must be weighed against thrombotic protection in the individual patient. Given the marked association between carotid plaque and systemic atherosclerosis, complications attributable to extracerebral vascular disease are important determinants of prognosis. Patients with asymptomatic carotid stenosis frequently have concomitant coronary artery disease, and a nontrivial proportion of these individuals subsequently require coronary revascularization. Coexistence of significant coronary disease complicates management decisions because combined surgical strategies—such as simultaneous carotid endarterectomy and coronary artery bypass grafting—carry cumulative perioperative risk, whereas staged or hybrid approaches entail trade-offs regarding timing, antiplatelet therapy, and the sequence of ischemic risk mitigation [42][43][44]. The lack of definitive evidence prescribing an optimal sequence or combination of procedures in patients with concurrent carotid and coronary disease underscores the potential for adverse outcomes arising from misaligned or poorly coordinated management plans.

Consultation:

Consultation with specialists is recommended when high-risk imaging features or severe stenosis are identified. Referral to a vascular surgeon or, in selected instances, a neurosurgeon enables comprehensive appraisal of the individual's anatomic suitability for endarterectomy or stenting, evaluation of perioperative risk, and discussion of alternatives such as transcarotid artery revascularization. Cardiology consultation is appropriate when clinical or investigational data suggest significant coronary pathology because coordinated decision making can mitigate the risk of cardiac complications associated with cerebrovascular interventions. Multidisciplinary evaluation reduces the likelihood of avoidable complications by aligning expertise across vascular, neurologic, and cardiothoracic domains. Prevention of complications centers on aggressive primary and secondary cardiovascular risk management and on patient education. Primary preventive goals recommended by major cardiovascular bodies include maintenance of a healthy body mass index, tight blood pressure control, normalization of serum lipids, optimal glycemic indices in patients with diabetes, and universal smoking cessation [7][35][36][53]. Pharmacologic measures, principally statin therapy, reduce lipid-driven plaque progression and lower the probability of ischemic events. Low-dose aspirin is commonly recommended for stroke prevention in selected patients with asymptomatic carotid disease, although the evidence for aspirin's efficacy in this specific population is more limited than for other vascular indications and requires individualized assessment of bleeding risk [7][35][36]. Regular clinical follow-up to confirm adherence to medical therapy and to reinforce lifestyle modification is a cornerstone of complication prevention.

Patient Education:

Patient education must emphasize recognition of stroke symptoms and the urgency of seeking emergency care when neurologic deficits emerge. Counseling should also address medication adherence, secure storage and disposal of drugs, and avoidance of exposures that could amplify vascular risk, such as tobacco use and poorly controlled metabolic disease. Education strategies that engage family members or caregivers are particularly valuable because these individuals frequently act as first responders in the event of an acute neurologic decline. Several pragmatic considerations influence complication mitigation. Routine population screening for asymptomatic carotid stenosis is not recommended because false-positive findings can prompt unnecessary interventions with attendant procedural risk; screening is reserved for selected high-risk individuals who would accept intervention if significant stenosis were discovered [22][28]. Procedural selection must reflect not only the degree of stenosis but also plaque vulnerability markers, reduced cerebrovascular reserve, patient life expectancy, and surgical fitness. Contemporary evidence arguing for conservative management in many asymptomatic patients is grounded in the declining baseline risk of ipsilateral stroke under optimized medical therapy; therefore, procedural indication should be narrow and targeted to those whose residual risk exceeds the procedural hazard.

Emerging procedural techniques offer potential to reduce complication rates in appropriately selected patients. Transcarotid artery revascularization with active flow reversal provides neuroprotection and has demonstrated lower periprocedural stroke and mortality rates relative to transfemoral carotid stenting in selected series, thereby expanding options for patients with hostile neck anatomy or prior radiation exposure. Nevertheless, any novel approach must be weighed against operator proficiency and institutional experience, as outcomes are sensitive to the learning curve and periprocedural protocols. In summary, complications associated with asymptomatic carotid artery stenosis encompass a spectrum from ischemic cerebrovascular events attributable to plaque embolization or hemodynamic insufficiency to procedure-related morbidity and systemic cardiovascular events. Preventing these complications requires a dual strategy of intensive medical risk factor control and judicious, individualized application of revascularization when the anticipated long-term benefit outweighs immediate procedural risk. Multidisciplinary consultation, robust patient education, and vigilant longitudinal surveillance are essential to minimize harm and to optimize both neurologic and systemic cardiovascular outcomes.

Conclusion:

In conclusion, the management of asymptomatic carotid artery stenosis has undergone a significant paradigm shift. The foundation of contemporary care is rigorous, multifaceted best medical therapy, which has dramatically reduced the baseline risk of stroke to a point where the marginal benefit of routine revascularization for all patients is no longer justified. The modern approach is highly selective, moving beyond the degree of stenosis alone to incorporate advanced imaging biomarkers of plaque vulnerability and physiological assessments of cerebrovascular reserve to identify a high-risk subgroup that may still benefit from intervention. Success in this new paradigm is fundamentally dependent on seamless interdisciplinary collaboration. Radiologists provide critical risk stratification, vascular specialists determine procedural candidacy and technique, and pharmacists and nurses ensure the effective implementation and long-term adherence to optimal medical therapy. This coordinated, patient-centered strategy ensures that invasive procedures are reserved for those most likely to derive a net benefit, thereby optimizing outcomes by effectively preventing stroke while minimizing unnecessary procedural risks.

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