Academic Journal of Neurology & Neurosurgery

Review

DOI: 10.51271/AJNN-0038

Diagnosis and management of carotid artery stenosis: pathophysiology, advanced imaging, and evolving revascularization strategies

Mehmet Kabalcı

Department of Neurosurgery, Faculty of Medicine, Kırıkkale University, Kırıkkale, Turkiye

Received: 19/11/2025 ◆ **Accepted**: 19/12/2025 ◆ **Published**: 31/12/2025

Cite this article: Kabalcı M. Diagnosis and management of carotid artery stenosis: pathophysiology, advanced imaging, and evolving revascularization strategies. *Acad J Neurol Neurosurg.* 2025;2(4):82-85.

Corresponding Author: Mehmet Kabalcı, kabalci@hotmail.com

ABSTRACT

Carotid artery stenosis is a major and potentially preventable cause of ischemic stroke worldwide. Although the degree of luminal narrowing has traditionally guided risk stratification and treatment decisions, accumulating evidence indicates that plaque composition, biomechanical forces, and hemodynamic factors play a critical role in determining cerebrovascular risk. Atherosclerotic lesions preferentially develop at the carotid bifurcation due to disturbed flow patterns and low wall shear stress, promoting endothelial dysfunction and plaque instability. Recent advances in imaging have shifted diagnostic paradigms beyond luminal assessment toward detailed plaque characterization. Magnetic resonance imaging, particularly with T1-weighted sequences, enables reliable detection of intraplaque hemorrhage and fibrous cap status, both of which are strongly associated with embolic events independent of stenosis severity. These insights have important implications for individualized patient management. Medical therapy remains the cornerstone of treatment for all patients with carotid artery disease. Antiplatelet agents and high-intensity statin therapy play a central role in stroke prevention by reducing thromboembolic risk and promoting plaque stabilization. For patients requiring revascularization, carotid endarterectomy continues to represent the gold standard in appropriately selected symptomatic individuals. Carotid artery stenting has emerged as an alternative for high-risk surgical candidates, although embolic complications associated with transfemoral access remain a concern. Transcarotid artery revascularization, utilizing flow reversal for cerebral protection, has expanded the therapeutic armamentarium and offers a promising hybrid approach with favorable safety profiles. This review provides a comprehensive overview of the epidemiology, pathophysiology, diagnostic advances, and evolving treatment strategies for carotid artery stenosis. Emphasis is placed on multidisciplinary decision-making, patient-specific risk assessment, and the integration of advanced imaging to optimize outcomes in both symptomatic and asymptomatic disease.

Keywords: Carotid artery stenosis, ischemic stroke, plaque vulnerability, carotid endarterectomy, carotid artery stenting, transcarotid artery revascularization

INTRODUCTION AND EPIDEMIOLOGY

Extracranial carotid artery disease accounts for approximately 15–20% of ischemic strokes worldwide. Its prevalence increases markedly with age, exceeding 10% in individuals older than 80 years. Symptomatic carotid artery stenosis represents a particularly high-risk subgroup, as it is associated with a substantially increased likelihood of recurrent ischemic stroke.[1,2]

The development and progression of carotid artery disease are strongly influenced by modifiable risk factors, including cigarette smoking, hypertension, diabetes mellitus, hyperlipidemia, and a sedentary lifestyle.[1,3] Consequently, contemporary management strategies extend beyond the treatment of existing stenosis and emphasize both primary and secondary prevention aimed at halting disease progression and reducing future cerebrovascular events (Figure 1).

ETIOPATHOGENESIS AND BIOMECHANICAL BACKGROUND

Atherosclerosis constitutes the fundamental pathological substrate of carotid artery stenosis. However, the preferential localization of atherosclerotic plaques at the carotid bifurcation cannot be explained solely by systemic risk factors and is best understood through biomechanical principles.[2-4]

This work is licensed under a Creative Commons Attribution 4.0 International License.

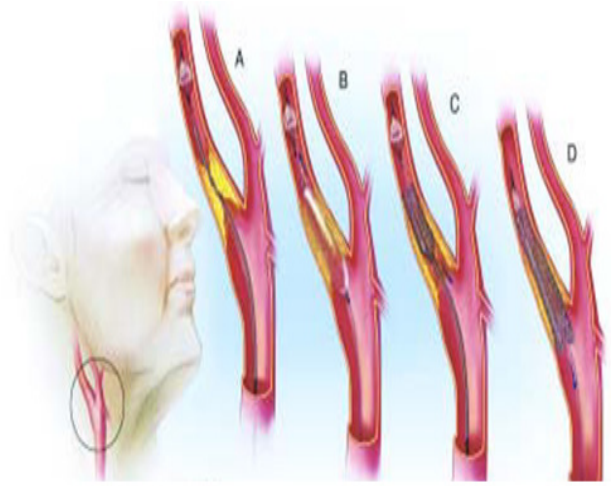

Figure 1. Anatomical localization of extracranial carotid artery stenosis

Hemodynamic Factors and Endothelial Dysfunction

At the carotid bifurcation, abrupt changes in vessel geometry lead to flow separation and disturbed blood flow patterns. These regions are characterized by low and oscillatory wall shear stress. Reduced shear stress suppresses endothelial nitric oxide (NO) production, promotes pro-inflammatory gene expression, and facilitates monocyte adhesion. As a result, the transendothelial migration of low-density lipoprotein (LDL) particles is enhanced, creating a microenvironment conducive to atherogenesis.[3-5] These biomechanical mechanisms influence not only plaque initiation but also the subsequent evolution of plaque phenotype, determining whether lesions remain stable or progress toward an unstable, rupture-prone configuration (Figure 2).

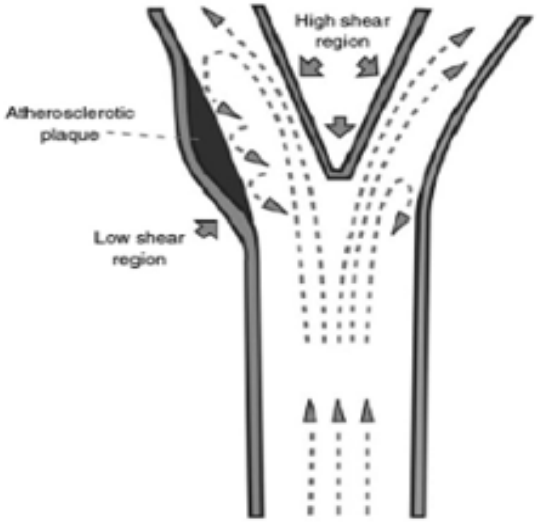

Figure 2. Physiopathogenesis of carotid artery stenosis

DIAGNOSTIC APPROACHES: BEYOND LUMINAL NARROWING

Traditionally, the diagnosis and risk stratification of carotid artery stenosis have relied primarily on the percentage of luminal narrowing. Current evidence, however, indicates that stroke risk is determined not only by the degree of stenosis but also by the structural and biological characteristics of the atherosclerotic plaque.[4-8]

Ultrasonography and Computed Tomography Angiography

Color Doppler ultrasonography remains the first-line modality for screening and follow-up. Peak systolic velocities and spectral waveform analysis allow for noninvasive estimation of stenosis severity.[6-8] Computed tomography angiography (CTA) provides high-resolution visualization of vascular anatomy and calcific burden, offering valuable information for surgical or endovascular planning (Figure 3, 4).

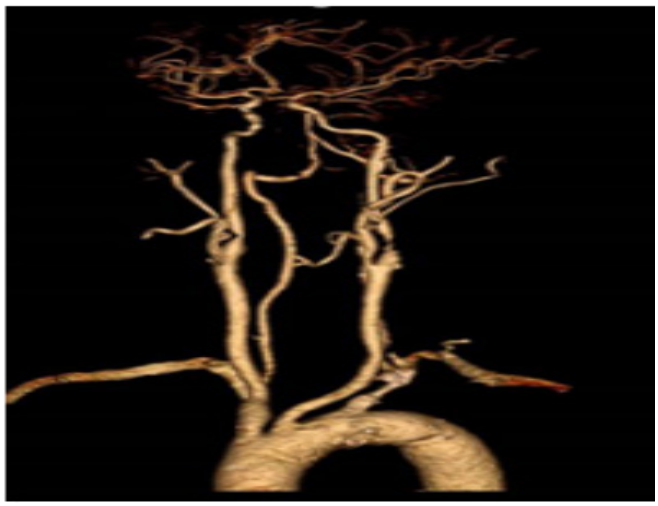

Figure 3. Three-dimensional computed tomography angiographic findings

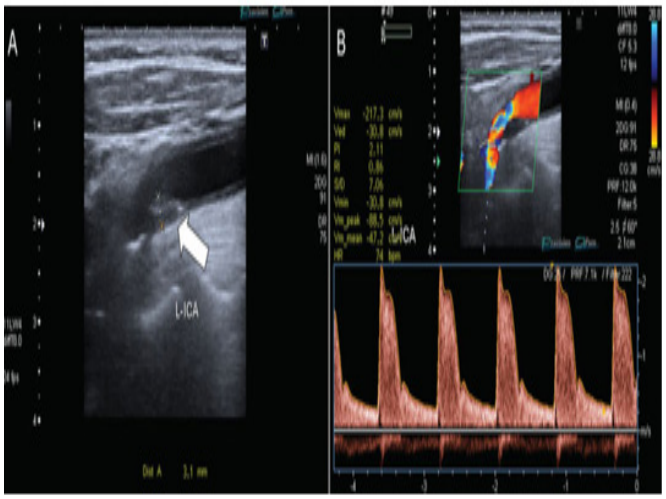

Figure 4. Corresponding Doppler ultrasonography evaluation

Magnetic Resonance Imaging and Plaque Characterization

Magnetic resonance imaging (MRI) has emerged as a powerful tool for assessing plaque biology. In particular, intraplaque hemorrhage (IPH), detected using T1-weighted MP-RAGE sequences, is recognized as one of the strongest predictors of plaque rupture and embolic events. The presence of IPH significantly increases stroke risk, independent of the degree of luminal stenosis.[7,8]

Contrast-enhanced MRI further enables assessment of fibrous cap thickness and lipid-rich necrotic core volume. These parameters have become increasingly influential in guiding treatment decisions in both symptomatic and asymptomatic patients.[7,8]

PHARMACOLOGICAL MANAGEMENT AND PLAQUE STABILIZATION

Medical therapy constitutes the cornerstone of carotid artery disease management and should be implemented in all patients, regardless of whether surgical or endovascular intervention is performed.[9-11]

Antithrombotic Therapy

Antiplatelet therapy represents the standard antithrombotic strategy in carotid artery stenosis. Aspirin or clopidogrel is selected based on clinical context and patient characteristics. Anticoagulation is reserved for specific indications, such as concomitant atrial fibrillation or the presence of a free-floating thrombus.[9,11]

The Role of Statin Therapy

Beyond lipid lowering, statins exert pleiotropic effects that are critical in carotid disease. By attenuating vascular inflammation, reducing lipid core size, and increasing fibrous cap thickness, statins promote plaque stabilization. High-intensity statin therapy is therefore a fundamental component of stroke prevention strategies.[9-12]

SURGICAL AND ENDOVASCULAR TREATMENT OPTIONS

Carotid Endarterectomy (CEA)

Carotid endarterectomy remains the most evidence-based intervention for reducing stroke risk in patients with symptomatic high-grade carotid stenosis. Following the publication of large randomized trials comparing endarterectomy with best medical therapy alone, CEA became the preferred treatment for recently symptomatic patients with severe carotid stenosis. Landmark studies such as NASCET and ACST clearly demonstrated the superiority of CEA over medical therapy when appropriate patient selection is applied. CEA may be performed using conventional longitudinal arteriotomy with patch angioplasty or via the eversion technique. The choice of technique is largely determined by surgeon experience and individual vascular anatomy.[6,10-15]

Carotid Artery Stenting (CAS)

Carotid artery stenting was developed as an alternative to CEA, particularly for patients at high surgical risk. The potential benefit of endovascular treatment, with or without stenting, as an alternative to endarterectomy was first highlighted by the Carotid and Vertebral Artery Transluminal Angioplasty Study (CAVATAS).[10] However, embolic complications associated with the transfemoral approach remain a major limitation.

In the interim analysis of the International Carotid Stenting Study, which compared carotid artery stenting with endarterectomy in symptomatic patients with severe carotid stenosis suitable for surgery, CEA remained the preferred treatment option (International Carotid Stenting Study investigators, 2010).[10,12-17]

Transcarotid Artery Revascularization (TCAR)

Transcarotid artery revascularization represents a significant advancement in endovascular therapy.[11,12] By utilizing direct carotid access and temporary flow reversal during the procedure, TCAR effectively prevents embolic debris from reaching the cerebral circulation. This feature positions TCAR as a hybrid and potentially safer alternative between conventional surgery and transfemoral stenting. Owing to its low rates of perioperative stroke and other complications, TCAR appears to be a promising treatment option for carotid artery stenosis.[11]

SPECIAL CLINICAL SITUATIONS: TOTAL OCCLUSION AND EC–IC BYPASS

In cases of total carotid artery occlusion, revascularization is generally not recommended.[13,15,17,18] Nevertheless, in carefully selected patients who remain symptomatic despite maximal medical therapy and demonstrate severe hemodynamic compromise on perfusion MRI, extracranial–intracranial (EC–IC) bypass surgery, such as superficial temporal artery to middle cerebral artery (STA–MCA) bypass, may be considered as a salvage option. The Carotid Occlusion Surgery Study (COSS) has provided important insights in this context.[15] However, other studies have reported no reduction in the risk of recurrent ipsilateral ischemic stroke within two years[6] (Figure 5).

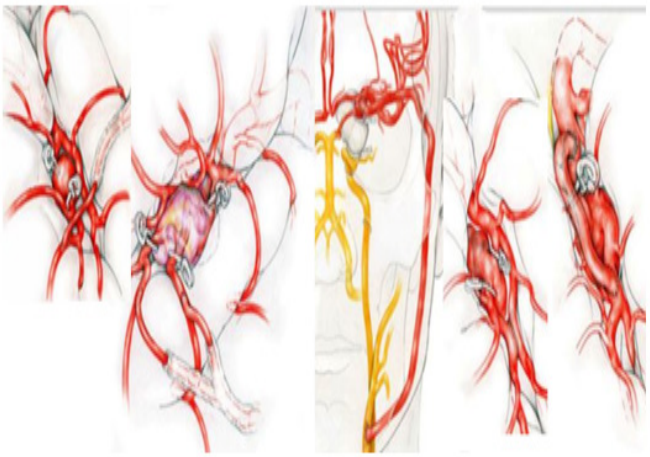

Figure 5. Extracranial–intracranial BYPASS technique

RESTENOSIS AND LONG-TERM FOLLOW-UP

Restenosis following CEA or carotid stenting is closely associated with diabetes mellitus and cigarette smoking. Diabetes accelerates neointimal hyperplasia, whereas smoking exacerbates endothelial dysfunction, significantly increasing restenosis risk. Additional factors such as dyslipidemia, female sex, renal insufficiency, hypertension, and smoking have also been shown to increase restenosis rates, while patch angioplasty during endarterectomy appears to confer a protective effect. Patients with these risk factors require closer imaging surveillance and aggressive risk factor modification.[17,19,20]

CONCLUSION

The management of carotid artery stenosis has evolved beyond the scope of a single specialty. The optimal contemporary approach relies on a multidisciplinary team involving neurology, radiology, cardiovascular surgery, and endovascular specialists. Future research should focus on developing higher-quality evidence to refine patient selection, particularly in asymptomatic carotid stenosis, enabling individualized conservative, surgical, or endovascular treatment strategies. Advanced imaging modalities, including MRI, PET-CT, and duplex ultrasonography, will likely play an increasingly important role in the reliable identification of plaque morphologies predictive of cerebral ischemia. Despite ongoing technological advances, early diagnosis and rigorous control of vascular risk factors remain the cornerstone of successful long-term outcomes.

ETHICAL DECLARATIONS

Peer Review Process

This review was externally peer-reviewed.

Conflict of Interest

The authors declare no conflicts of interest.

Financial Disclosure

No financial support was received for the preparation or publication of this article.

Author Contributions

All authors contributed equally to the planning, writing, and critical revision of the manuscript. All authors approved the final version for submission.

REFERENCES

1. Matsumoto K, Sera Y, Nakamura H, Ueki Y, Miyake S. Correlation between common carotid arterial wall thickness and ischemic stroke in patients with type 2 diabetes mellitus. *Metabolism*. 2002;51(2):244-247. doi:10.1053/meta.2002.28971
2. Barnett HJ, Gunton RW, Eliasziw M, et al. Causes and severity of ischemic stroke in patients with internal carotid artery stenosis. *Jama*. 2000;283(11):1429-1436.
3. Ji X, Leng XY, Dong Y, et al. Modifiable risk factors for carotid atherosclerosis: a meta-analysis and systematic review. *Annals Transl Med*. 2019;7(22):632. doi:10.21037/atm. 2019.10.115
4. Sakakura K, Nakano M, Otsuka F, Ladich E, Kolodgie FD, Virmani R. Pathophysiology of atherosclerosis plaque progression. *Heart, Lung and Circulation*. 2013;22(6):399-411. doi:10.1016/j.hlc.2013.03.001
5. Lee SW, Antiga L, Spence JD, Steinman DA. Geometry of the carotid bifurcation predicts its exposure to disturbed flow. *Stroke*. 2008;39(8): 2341-2347. doi:10.1161/STROKEAHA. 107.510644
6. Randomised trial of endarterectomy for recently symptomatic carotid stenosis: final results of the MRC European Carotid Surgery Trial (ECST). *Lancet*. 1998;351(9113):1379-1387.
7. Rothwell PM, Gibson R, Warlow CP. Current evidence, however, indicates that stroke risk is determined not only by the degree of stenosis but also by the structural and biological characteristics of the atherosclerotic plaque. *Stroke*. 2000;31(3):615-621. doi:10.1161/01.STR. 31.3.615
8. Wilensky RL, Song HK, Ferrari VA. Role of magnetic resonance and intravascular magnetic resonance in the detection of vulnerable plaques. *J Am Coll Cardiol*. 2006;47(8S):C48-C56. doi:10.1016/j.jacc. 2005.11.048
9. Paciaroni M, Bogousslavsky J. Antithrombotic therapy in carotid artery stenosis: an update. *Eur Neurol*. 2015;73(1-2):51-56. doi:10.1159/ 000367988
10. Brown MM, Rogers J, Bland JM. Endovascular versus surgical treatment in patients with carotid stenosis in the Carotid and Vertebral Artery Transluminal Angioplasty Study (CAVATAS): a randomised trial. *Lancet*. 2001;357(9270):1729-1737.
11. Almeida SO, Budoff M. Effect of statins on atherosclerotic plaque. *Trends Cardiovasc Med*. 2019;29(8):451-455. doi:10.1016/j.tcm.2019.01.001
12. Luk Y, Chan YC, Cheng SW. Transcarotid artery revascularization as a new modality of treatment for carotid stenosis. *Ann Vasc Surg*. 2020;64: 397-404. doi:10.1016/j.avsg.2019.11.001
13. Sardar P, Chatterjee S, Aronow HD, Carotid artery stenting versus endarterectomy for stroke prevention: a meta-analysis of clinical trials. *J Am Coll Cardiol*. 2017;69(18):2266-2275 doi:10.1016/j.jacc.2017.02.053
14. Tu J, Wang S, Huo Z, Wu R, Yao C, Wang S. Repeated carotid endarterectomy versus carotid artery stenting for patients with carotid restenosis after carotid endarterectomy: systematic review and meta-analysis. *Surgery*. 2015;157(6):1166-1173. doi:10.1016/j.surg.2015.02.005
15. Esposito G, Amin-Hanjani S, Regli L. Role of and indications for bypass surgery after carotid occlusion surgery study (COSS)? *Stroke*. 2016; 47(1):282-290. doi:10.1161/STROKEAHA.115. 008220
16. Powers WJ, Clarke WR, Grubb RL Jr, et al. Extracranial-intracranial bypass surgery for stroke prevention in hemodynamic cerebral ischemia: the Carotid Occlusion Surgery Study randomized trial. *JAMA*. 2011;306(18):1983-1992. doi:10.1001/jama.2011.1610
17. Megaly M, Alani F, Cheng CI, Ragina N. Risk factors for the development of carotid artery in-stent restenosis: multivariable analysis. *Cardiovasc Revasc Med*. 2021;24:65-69. doi:10.1016/ j.carrev.2020.09.005
18. Eckstein HH, Kühnl A, Dörfler A, Kopp IB, Lawall H, Ringleb PA. The diagnosis, treatment and follow-up of extracranial carotid stenosis: a multidisciplinary German-Austrian guideline based on evidence and consensus. *Deutsches Ärzteblatt International*. 2013;110(27-28):468. doi:10.3238/ arztebl.2013.0468
19. White CJ. Carotid artery stenting. *J Am Coll Cardiol*. 2014;64(7):722-731. doi:10.1016/j.jacc.2014.04.069
20. Patel UK, Malik P, Demasi M, et al. Multidisciplinary approach and outcomes of tele-neurology: a review. *Cureus*. 2019;11(4):e4410. doi:10. 7759/cureus.4410