Review

Carotid webs: a review of pathophysiology, diagnostic findings, and treatment options

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ABSTRACT

A carotid web (CaW) is an atypical form of fibromuscular dysplasia characterized by a fibrous, shelf-like intimal flap originating from the posterior wall of the internal carotid bulb projecting into the arterial lumen. CaWs disturb normal blood flow and create stasis between the intimal reflection and the carotid wall, thereby promoting thrombogenesis and increasing the risk of downstream embolic strokes. Observational data have suggested that CaWs are associated with strokes with otherwise unknown etiology, particularly in young patients without other stroke factors, and stroke recurrence rates of symptomatic CaWs have been reported to be as high as 20% over 2 years. Despite its clinical importance, there are currently no clear guidelines on the management of CaWs. In this narrative review, we discuss the epidemiology, pathogenesis, pathophysiology, diagnosis, and treatment options for this under-recognized entity.

INTRODUCTION

A carotid web (CaW), also known as carotid diaphragm, is an atypical intimal variant of fibromuscular dysplasia (FMD) characterized by a fibrous, shelf-like intimal flap originating from the posterior wall of the internal carotid bulb projecting into the arterial lumen. The most salient clinical manifestation of CaWs is ischemic stroke; while CaWs are rare, numerous reports, case series, and observational studies have documented strong associations between CaWs and strokes of no known alternative etiology, particularly in younger patients without other vascular comorbidities.[1] Current guidelines do not offer definitive guidance on the optimal management of CaWs.[2]

METHODS

In this narrative review, we explored the Medline Database using the search terms 'carotid web,' 'carotid diaphragm,' and 'stroke' from inception to September 2023 for medical literature on CaWs and sought to summarize the current knowledge regarding their pathophysiology, associations with stroke and stroke recurrence, available diagnostic modalities, as well as data on the outcomes of various treatment strategies.

EPIDEMIOLOGY, PATHOGENESIS, AND PATHOPHYSIOLOGY

The overall population incidence of CaWs is largely unknown. Among non-stroke patients, CaW prevalence may be as low as 1% or less based on control groups of case–control studies of CaW[3 4] (table 1). CaWs are associated with younger age, African American race, and female sex, and a meta-analysis of recent literature suggests that the incidence of CaWs among younger patients (under 60 years old) with otherwise cryptogenic strokes is approximately 13%[1 5] (table 1). Approximately half of patients with CaWs harbor bilateral lesions.[6 7]

For patients with a stroke found to have ipsilateral CaWs, the risk of stroke recurrence is high. In a study of 92 patients with CaW-associated strokes or transient ischemic attacks (TIAs), Olindo *et al*[8] found overall stroke/TIA recurrence risks of 4.4%, 10.8%, and 19.8% at 1 month, 1 year, and 2 years, respectively. In a separate study of 21 patients with symptomatic CaWs, Turpinat *et al* reported an annualized stroke recurrence rate of 11.4%[9] (table 1).

The pathogenesis of CaWs is debated. Histologically, CaWs are characterized by focal intimal hyperplasia,[10] which is distinctly different from classic FMD (which involves medial hyperplasia/fibroplasia) and atherosclerotic lesions. Early reports have suggested that CaWs may be congenital defects due to developmental anomalies of the brachiocephalic system,[11] and CaWs' association with younger age and lack of association with cardiovascular risk factors fit well with this hypothesis. However, in a study of 47 patients with a childhood stroke, Hassani *et al* failed to identify any CaWs, possibly suggesting that CaWs might be acquired lesions instead.[12] Given the association of CaWs with African American race and female sex,[5] it is also possible that the development of CaWs is associated with genetic predisposition or hormonal factors. Future research is needed to further understand the pathogenesis of CaWs.

The pathophysiology of CaWs and their association with strokes are thought to be due to alterations in flow dynamics.[13 14] CaWs can cause altered blood flow and stasis between the intimal reflection and the carotid wall, thereby promoting thrombogenesis and increasing the risk of downstream embolism. In a retrospective analysis of 41 patients, Park *et al* demonstrated, using time–density curve analysis of conventional angiography images, that CaWs are associated with larger hemodynamic disruptions and flow stasis compared to mild to moderate atherosclerotic lesions.[13] The stroke risk of CaWs is probably associated with their size and morphology. In a cross-sectional

Table 1 Prevalence of carotid webs (CaWs) and risk of stroke recurrence by treatment modality

	Statistic	References
CaW prevalence		
Total population	<1%	3, 4
Patients with a stroke with no other known etiology	13%	1
Stroke recurrence risk		
Annualized risk on medical therapy	~10%	8, 19
Risk after carotid stenting	0%	5, 7, 35, 36, 39
Risk after endarterectomy	0%	5, 37, 38, 39

study of 86 CaWs diagnosed by computed tomographic angiography (CTA), Tabibian *et al*[15] suggested that a length of ≥3 mm, acute angle relative to the carotid wall, and greater than 50% CaW occupancy of the carotid bulb were associated with stroke. More recently, Perry da Camara *et al* corroborated these findings, showing that CaW length and volume are significantly associated with TIA or stroke.[16] In a separate study, Haussen *et al*[7] suggested that patients with bilateral CaWs with a stroke have less conspicuous CaWs on the asymptomatic side than on the symptomatic side, although only a minority of symptomatic CaWs were associated with thrombi. Finally, results from the CAROWEB Registry,[17] a multicenter prospective observational study of CaWs conducted in France, suggested that in comparison with asymptomatic CaWs, symptomatic CaWs were associated with a higher percentage of vessel narrowing as measured by the European Carotid Surgery Trial (ESCT) criteria.

Interestingly, CaWs may have a predilection for causing large vessel occlusion strokes. Among all patients with strokes of otherwise unknown etiology, patients with CaWs were more likely to have had a large vessel occlusion stroke.[3 9] In an analysis of the Dutch nationwide MR CLEAN Registry and trial data, Compagne *et al*[18] reported that CaWs were associated with 2.5% of all large vessel occlusion strokes. In a separate analysis, Guglielmi *et al*[19] found that patients with a large vessel stroke who had CaWs had a significantly higher risk of recurrent strokes at 2 years compared to those without CaWs (17% vs 3%, respectively). Thus, for young patients with large vessel strokes of otherwise unknown etiology, a detailed workup to identify CaWs may be warranted.

DIAGNOSIS

CaWs are fibrous, shelf-like intimal flaps projecting from the posterior wall of the internal carotid artery bulb into the arterial lumen. Regardless of imaging modality, oblique views generally visualize the CaWs best, while axial views can sometimes visualize intraluminal filling defects. Below, we discuss specific considerations for diagnosing CaWs with computed tomographic angiography (CTA), magnetic resonance (MR), ultrasound (US), and catheter angiography. We also discuss the emerging role of optical coherence tomography (OCT) in the diagnosis and characterization of CaWs.

Computed tomographic angiography

CTA is the most widely used diagnostic tool to detect and evaluate CaWs. However, despite its high resolution for the visualization of CaWs (figure 1A), CaWs are routinely missed in real-world practice, particularly in facilities with lower levels of cerebrovascular certification.[20] Given that CaWs are best visualized in the oblique view, using multiplanar reconstructions (MPRs) is essential for accurate CaW diagnosis and characterization; doing so can limit missed diagnoses and reduce the risk of mislabeling CaWs as atherosclerotic plaques.[21]

When considering the diagnosis of CaWs on CTA, it is important to distinguish them from other lesions that can mimic

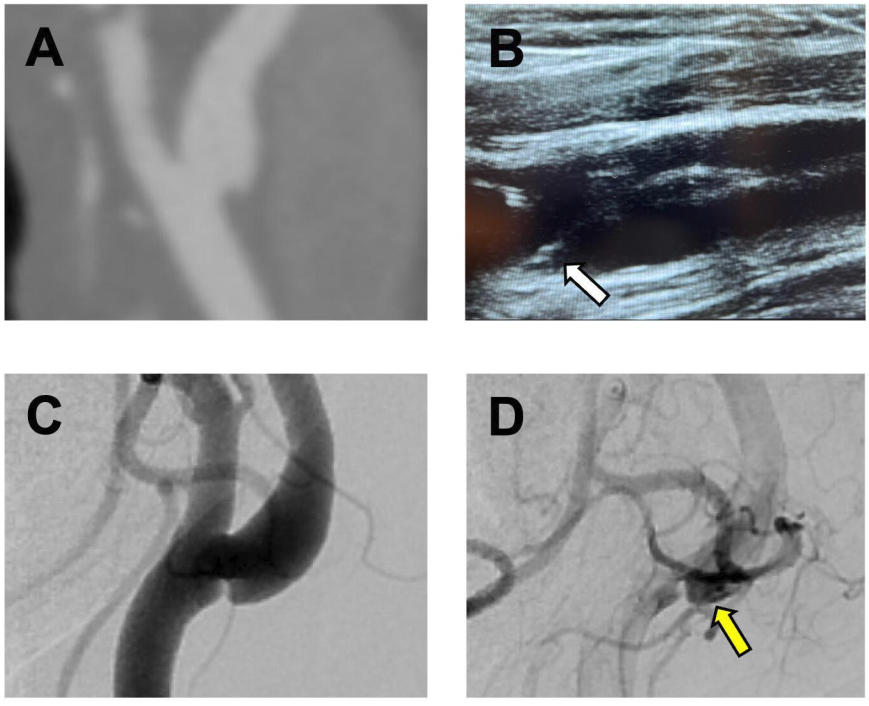

Figure 1 Radiographic appearances of carotid webs (CaWs). (A) The characteristic Intraluminal filling defect arising from the posterior wall of the carotid bulb seen on CT angiography. (B) The thin shelf-like projection seen on ultrasound (white arrow). (C and D) A typical CaW with contrast pooling (yellow arrow) seen on catheter angiography.

CaWs. Atherosclerotic plaques can be mistaken for CaWs, as they share a similar location in the carotid bulb. In addition to using MPRs[21] when looking for CaWs, it is also important to note that atherosclerotic lesions are not usually limited to the posterior carotid wall, are often circumferential, and their surfaces can be irregular and associated with calcifications.[22] Carotid dissections can also be mistaken for CaWs; however, dissections normally start above or extend beyond the carotid bulb, with an overall enlarged vessel caliber. Finally, while CaW is considered an atypical form of FMD, it is distinctly different from classic FMD,[23] which is usually associated with segmental instead of focal defects that affect multiple vessels and centered at the middle cervical segment of the internal carotid artery rather than at the carotid bulb.

It is important to note that while larger CaWs are more likely to be associated with strokes, CaWs are not commonly stenotic lesions, and the use of North American Symptomatic Carotid Endarterectomy Trial (NASCET) or ECST methods for the grading of stenoses is not validated for the measurement of CaWs. Furthermore, vessel narrowing is not a prerequisite for the association of CaWs with strokes; in a case–control study of 102 patients, Kim *et al*[3] demonstrated that none of the CaWs associated with otherwise cryptogenic strokes led to more than 30% vessel narrowing. Thus, providers should take caution with NASCET or ECST measurements of vessel narrowing associated with CaWs, and these measurements should be interpreted differently for CaWs than for classic atherosclerotic lesions.

Finally, while CTA provides excellent spatial resolution for the visualization of CaWs, it is not an ideal modality for the assessment of fluid dynamics. Nevertheless, computational fluid dynamic models based on lumen segmentations of CTA scans can be used to assess hemodynamic parameters; in a case series of nine patients, Compagne *et al*[14] demonstrated the feasibility of CTA for characterizing fluid dynamics and demonstrated that CaWs are associated with larger recirculation zones and metrics suggestive of non-laminar flow. Future investigations are needed to assess the prognostic value of hemodynamic metrics obtained from CT angiographic studies of CaWs.

MR angiography and vessel wall imaging

MR angiography can be performed with or without gadolinium contrast, and the appearances of CaWs are similar to those seen on CTA. However, there are limited data on head-to-head comparisons between MR angiography and CTA. In a case series of five patients with CaWs seen on CTA, Boesen *et al* demonstrated that MR angiography was able to detect CaW in all five patients[24] . Vessel wall MR imaging can also be used to detect CaWs,[25] though its diagnostic accuracy compared with other modalities is unclear. 2D time of flight sequences might be able to detect turbulent flow and stasis,[26] and more advanced hemodynamic parameters such as time-average wall shear stress and oscillatory shear index, might also be helpful in better characterizing CaWs and distinguishing them from atherosclerotic lesions.[27] Future studies are needed to compare the performance of MR imaging modalities with CTA for the detection and characterization of CaWs.

Ultrasound

Carotid webs can also be detected on carotid Doppler US and they are best visualized on a longitudinal view with B-mode US (figure 1B). In a retrospective study of 24 patients, Fontaine *et al*[28] showed that the additional use of US microflow imaging, which can identify slow blood flow, can reveal flow defects surrounding 82% of CaWs and improve the overall sensitivity of US for the detection of CaWs. While US is convenient and often used as a first-line screening tool to explore etiologies of stroke, its diagnostic performance compared with other imaging modalities is unclear. Given its lower resolution, US might not be as reliable as CTA or catheter angiography.[29] For instance, CaWs on US can appear stenotic, which can often lead to misdiagnosis of CaWs as atherosclerotic plaques. While alterations of flow dynamics on US might have prognostic value that can be leveraged into optimizing treatment decisions pending future investigation, US alone is probably insufficient for the diagnostic workup of CaWs.

The use of intravascular ultrasound (IVUS) has been proposed as an adjunctive tool for diagnosing CaWs during catheter angiography. In a small case series of three patients with known CaWs, Hassani *et al*[30] attempted using IVUS to detect CaWs; however, IVUS was only able to reveal focal eccentric areas at the posterior carotid bulb in two patients, and the endoluminal protrusions of the CaWs were indistinguishable from fibrosis, thrombosis, or atherosclerosis. Thus, IVUS may have a limited role in the diagnostic workup of CaWs.

Catheter angiography

While catheter angiography, or digital subtraction angiography (DSA), has been heralded by some as the gold-standard imaging modality for detecting CaWs, its diagnostic performance may not be superior to that of CTA.[29] Thus, given its invasive nature, DSA is not recommended as the first-line diagnostic study for CaWs. The classic morphology of CaWs on DSA is similar to that of CTA—thin, shelf-like endoluminal projections stemming from the posterior wall of the carotid bulb, best visualized on oblique projections (figure 1C). Contrast pooling is often observed in the late venous phase (figure 1D). 3D acquisitions might also be helpful.

Optical coherence tomography

Optical coherence tomography is a powerful imaging modality that can create high-resolution cross-sectional images of biological tissue, and it can be used to visualize neuroendovascular devices[31] and neurovascular lesions.[32] Its use for the diagnosis of CaWs was first explored in a small case series, where Radu *et al*[33] showed that intravascular OCT was able to establish CaW diagnoses in three patients with recurrent strokes of unknown etiology despite extensive previous multimodality workup. Subsequently, in a retrospective analysis of 16 patients with suspected CaWs, Al-Bayati *et al* demonstrated that intravascular OCT can be used to better differentiate CaWs from atherosclerotic lesions, characterize concomitant atherosclerotic changes near CaWs, and identify adherent microthrombi.[34] Future investigations are needed to further validate the usefulness of intravascular OCT in the workup of CaWs and other carotid lesions for patients with a cryptogenic stroke.

Practical recommendations

To maximize the diagnostic accuracy of CaWs, we recommend that providers take the following approach:

1. For embolic stroke patients under 60 years old without traditional vascular risk factors or other overt stroke etiologies, the pre-test probability of CaW is high. Thus, CTA should be considered as the first-line neck vessel imaging modality. MR and US imaging can be considered as adjunctive modalities for patients with atypical lesions.
2. When interpreting CTA scans of the neck, MPRs should be used to ensure optimal CaW detection, particularly with oblique cuts in the plane of the carotid bifurcation.

3. For typical CaW lesions, conventional catheter angiography is not superior to CTA with MPR in terms of diagnostic performance. Thus, catheter angiography should be reserved for patients with lingering diagnostic uncertainty despite multimodality non-invasive imaging or those being considered for carotid intervention.

The clinical usefulness of flow dynamic parameters and the role of intravascular OCT in the diagnosis and characterization of CaW lesions are promising and pending future research.

TREATMENT

Medical management

Currently, there are no evidence-based guidelines for the management of CaWs. Current guidelines recommend antiplatelet therapy for symptomatic CaWs, although the level of evidence is low.[2] In a survey posted on the Society of NeuroInterventional Surgery website, responses from 74 providers showed that medical management with mono- or dual-antiplatelet therapy is considered by most providers to be the first-line treatment for asymptomatic CaWs and symptomatic CaWs with one previous stroke/TIA.[35] Data are limited on the usefulness of anticoagulation for prevention of secondary stroke in patients with symptomatic CaWs. At present, the comparative efficacy of single antiplatelet, dual antiplatelet, or anticoagulation treatments is unknown; however, general observational data of CaWs suggest that despite medical management, patients with symptomatic CaWs might have as high as a 20% risk of stroke recurrence over 2 years.[8 19]

It is important to recognize that patients with a CaW and otherwise cryptogenic strokes tend to be young, and recurrent strokes due to CaW have a higher likelihood of involving large vessels.[3] For patients who had multiple ischemic events related to CaWs, current guidelines recommend considering carotid intervention.[2] Future studies are needed to further assess whether medical management with antiplatelet or anticoagulation regimens alone might offer sufficient protection from strokes in select patients, perhaps those with smaller CaWs.

Carotid stenting

CaWs may be safely and effectively treated with carotid stenting (figure 2A and B). In a prospective study of 24 patients, Haussen *et al* showed that stenting CaWs completely prevented recurrent strokes in all 24 patients at a median follow-up of 12 months, with no periprocedural device-related complications, delayed stenoses, new strokes, or hemorrhages.[7 36] An analysis of the CAROWEB Registry also showed that stenting of 28 CaWs prevented recurrent strokes in all cases (median follow-up 9 months), again with no periprocedural complications or in-stent occlusions or stenoses.[37]

The deployment of stents for the treatment of CaWs is generally straightforward. Unlike stenting atherosclerotic lesions, there is typically no difficulty traversing the lesion or need for pre-stenting angioplasty. Furthermore, given that only a minority of CaWs are associated with adjacent thrombosis, there is a theoretically lower risk of thrombus/plaque disruption that might lead to periprocedural embolic events. Whether angioplasty following stent deployment is necessary is unclear, as most CaWs do not lead to significant luminal stenosis; future studies are needed to investigate whether post-stent angioplasty might achieve better apposition of CaWs to carotid walls and reduce stasis and thrombogenesis.

Carotid endarterectomy

Carotid endarterectomy (CEA) is another potentially effective treatment for CaWs for the prevention of stroke (figure 2C–E). In a case series of six patients with symptomatic CaWs treated with CEA, Haynes *et al*[38] reported no major periprocedural

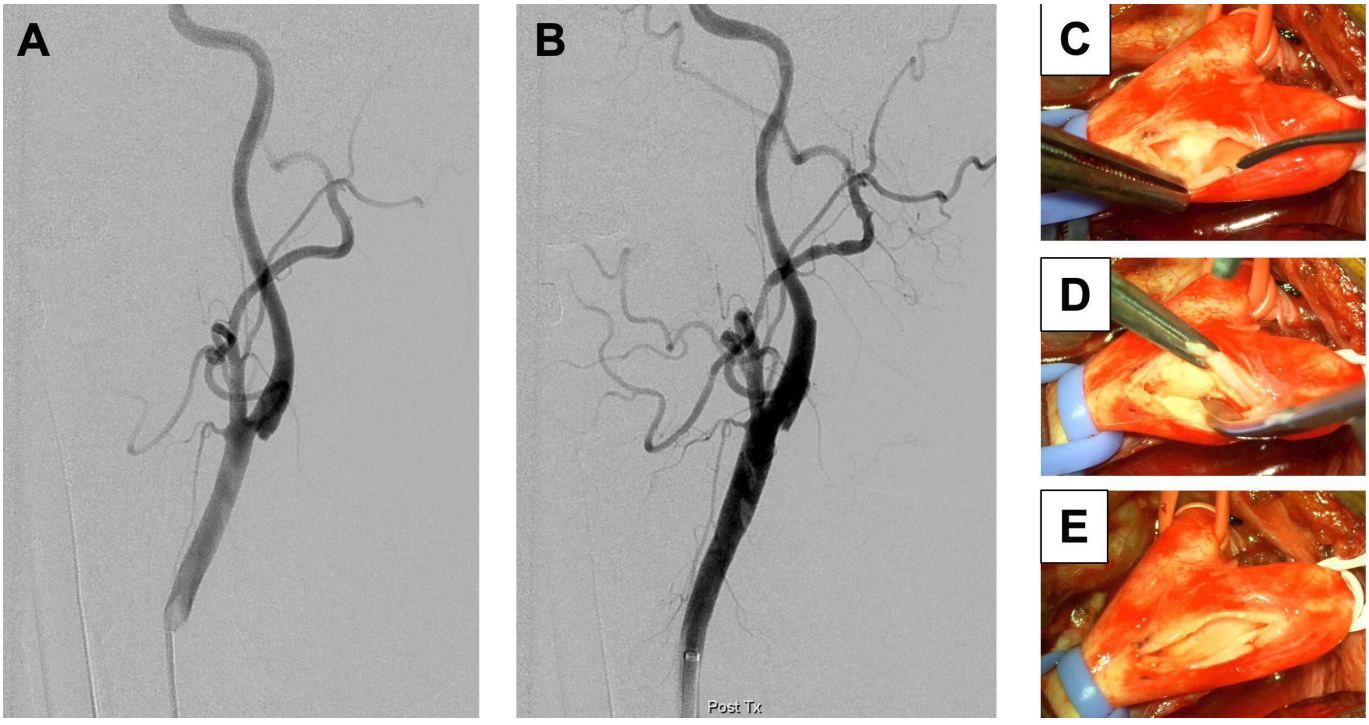

Figure 2 Illustrative cases of carotid web (CaW) interventions. (A and B) A symptomatic CaW before (A) and after (B) endovascular carotid artery stenting. (C, D, and E) A separate case of a CaW in a patient who underwent carotid endarterectomy. (C) The appearance of a CaW after arteriotomy. (D) The CaW retracted. (E) The appearance of the vascular wall after CaW resection.

complications and no stroke recurrence at a mean follow-up of 6 months. In a separate case series of 13 patients, Multon *et al*[39] also reported no perioperative complications following CEA and no recurrent stroke events after treatment. CEA also allows for post-treatment histological assessment of CaWs to confirm CaW diagnosis; while such information is of great value for research endeavors, its clinical usefulness awaits further exploration.

Comparative efficacy

To date, no randomized studies have compared the efficacy of CaW treatments. In 2018, Zhang *et al*[5] performed a systematic review of CaW treatments and identified 158 patients, of whom 57 were treated with stenting, and 57 with CEA. Strikingly, patients who did not receive carotid intervention had a 56% rate of recurrent stroke, while those who did receive intervention had no recurrent strokes or periprocedural complications.[5] A more recent meta-analysis conducted by Patel *et al*[40] in 2022 found similar results—the rate of stroke recurrence was 26.8% for patients treated with medical management, and intervention led to a low rate of periprocedural complications (0.5%) with perfect prevention of recurrent ischemic events. Thus, the current literature suggests that medical management alone may be insufficient for preventing stroke recurrence in patients with symptomatic CaWs and that carotid interventions might be safe and effective treatments in select patients (table 1).

Importantly, the exceedingly low rates of periprocedural complications of both stenting and CEA for CaWs compare favorably with historical rates of complications associated with classic carotid stenting and CEA procedures (~4% of perioperative death, stroke, or myocardial infarction).[41] This discrepancy might be due to younger age and lower overall burden of cardiac and vascular risk factors associated with CaWs.

While both stenting and CEA may be efficacious treatments for CaWs, the choice between the two remains a topic of debate.

Stenting CaWs has a great safety profile due to both easy navigation across the aortic arch (young population with low rates of concomitant vascular risk factors) as well as an easily traversed lesion in the carotid. Furthermore, the minimally invasive nature of the procedure might be attractive to patients and providers alike and expose patients to lower procedural risks associated with open surgeries. On the other hand, CEA offers the advantage of avoiding dual antiplatelet therapy, which might also be preferred in a relatively young population without cardiovascular risk factors. CEA and resection of CaWs also allow for a definitive diagnosis with pathology, which can provide further clarity on stroke etiology. Like stents, CEA procedures for CaWs are generally straightforward, as CaWs are normally associated with healthy individuals who often lack surgical comorbidities and risk factors for complex carotid surgery, such as vessel tortuosity, extensive atherosclerotic plaques, previous neck radiation, and previous surgical procedures in the neck. Moreover, patients typically do not have contralateral carotid disease, making clamping time during CEA less of a concern. Ultimately, the treatment for CaWs should be individualized based on the patient's anatomical features and comorbidities; if intervention is to be pursued, patient preferences and the comfort level of the proceduralist with stenting and CEA should also be taken into consideration.

Of note, while the current literature reports excellent outcomes of carotid interventions for CaWs, these data should be interpreted with caution. Publication biases might account for both the apparently high rates of recurrent strokes with medical management and the excellent efficacy and safety profiles of stenting and CEA. Future prospective studies are needed to further elucidate the comparative outcomes of CaW treatments. Specifically, further investigations on the size and morphologies of CaWs and their associations with stroke recurrence risk[15 16] might be needed to optimize treatment strategies personalized for each patient.

CONCLUSIONS

Carotid webs are common in patients with strokes of otherwise unknown etiology, particularly among young patients with no other cardiovascular comorbidities. CTA with multiplanar reconstructions of oblique cuts in the plane of carotid bifurcations should be considered the first-line diagnostic modality. Stroke recurrence rates for symptomatic carotid webs may be high with medical management alone, and carotid interventions via either carotid stenting or endarterectomy might be safe and effective treatments to prevent future ischemic events. Future prospective and comparative studies are needed to optimize and individualize treatment strategies for this under-recognized but important entity.

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