Review Article

The pitfalls and challenges in the diagnosis and treatment of patients with carotid near-occlusion: a narrative review

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Background and Objective: Carotid near-occlusion (CNO) is defined as a severe stenosis of the internal carotid artery (ICA) resulting in a reduction in the distal diameter. It is a specific type of carotid stenosis accounting for 34% of the patients with symptomatic carotid stenosis ≥50%. The current guidelines recommend the best medical treatment (BMT) as the treatment of choice. Therefore, it is of the utmost importance to accurately diagnose CNO to avoid unnecessary surgical treatment. Although imaging can detect the majority of cases, there are still a few cases that remain unexplained. The purpose of this review is to discuss the mechanism of stroke, diagnostic imaging, and therapeutic approaches in patients with CNO and to examine the importance of diagnostic imaging in determining the treatment and prognosis of patients with CNO.

Methods: The PubMed database was searched for clinical studies on CNO published in English from 1950 to 2024. The selected literature was screened for articles related to the formation mechanism of CNO, clinical diagnosis, and the latest developments in treatment methods.

Key Content and Findings: The primary mechanisms of stroke in CNO include embolism and hypoperfusion. Imaging techniques such as computed tomography angiography (CTA) and digital subtraction angiography (DSA) can identify most instances of CNO; however, anatomical variations may pose challenges in accurately diagnosing this condition. Currently, revascularization offers limited benefits for patients with CNO. According to the 2023 European Society for Vascular Surgery (ESVS) guideline update, BMT is the preferred treatment for CNO, and patients with CNO and distal vessel collapse with recurrent carotid territory symptoms may be considered for revascularization after multidisciplinary team discussion.

Conclusions: The diagnosis of CNO based on feature interpretation necessitates a cautious approach. It is imperative to conduct large prospective clinical trials to identify the optimal treatment for patients with various types of CNO.

Keywords: Carotid stenosis; near occlusion; medical imaging; carotid artery stenting (CAS); carotid artery endarterectomy

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Introduction

Carotid near-occlusion (CNO) is a severe stenosis of the internal carotid artery (ICA) resulting in a reduction in the distal diameter. CNO can be classified into two categories: collapse of the distal lumen with a "thread-like" appearance (CNO with full collapse) and less pronounced collapse of the distal lumen with a normal appearance (CNO without full collapse) (1). Initially discovered as CNO with full collapse, CNO is typically characterized by the gradual filling of the contrast medium, visually manifested as a "string sign" (2). In 1960, Newton and Couch (3) referred to the condition of "slow and incomplete filling of the contrast medium due to increased intracranial pressure" first described by Riishede and Ethelberg (4) as "pseudo-occlusion". Clark *et al.* (5) subsequently suggested severe stenosis at the carotid artery to be another cause of pseudo-occlusion. In 1970, Lippman *et al.* (6) first noted the phenomenon of distal lumen collapse resulting from atherosclerosis of the ICA, which they termed "poststenotic carotid slim sign" and "spurious hypoplasia". On this basis, Mehigan *et al.* (7) summarized the five pathological types of string sign (dissection of the ICA, radiation-associated carotid artery disease, preocclusive atherosclerosis at the carotid bifurcation, subacute partial thrombosis of the ICA, and chronic subtotal thrombosis of the ICA). Since then, "string sign" has been frequently used to describe CNO with full collapse. In addition, "pseudo-occlusion", "incomplete occlusion", "subtotal occlusion", "hairline residual lumen", "string sign", "slim sign", "poststenotic narrowing", "poststenotic carotid slim sign", "hypoplasia", "near total occlusion", and "99% stenosis" (2,5-15) have also been used to describe the phenomenon of a marked reduction in the diameter of the lumen of the ICA. However, these terms do not accurately describe the subtle partial reduction in distal lumen diameter due to severe stenosis of the ICA, and some terms have been used primarily to describe other entities (e.g., pseudo-occlusion and string sign) (1). The first comprehensive description of CNO was provided in 1997 by the North American Symptomatic Carotid Endarterectomy Trial (NASCET) (16), and later Fox *et al.* provided a detailed description of CNO in 2005 (2). In 2016, Johansson *et al.* (1) further clarified the term "near occlusion" to distinguish it from other terms. With the growing understanding of CNO, it has been reported that CNO accounts for 34% of patients with symptomatic carotid stenosis ≥50% (17). However, there is currently no consensus regarding the pathological mechanisms, diagnostic imaging, or treatment related to CNO. Correctly identifying patients with CNO through diagnostic imaging can guide clinicians in selecting the appropriate treatment options. Moreover, identifying different CNO stroke pathomechanisms and stages of CNO formation can allow clinicians to determine whether to treat patients conservatively or not, and each treatment modality results in different prognoses for patients with CNO, which is related to their quality of life. This review outlines the potential mechanisms of CNO formation and stroke occurrence and provides an overview of the current status and advances in imaging-based diagnosis and treatment of CNO. We present this article in accordance with the Narrative Review reporting checklist (available at https://qims.amegroups.com/article/view/10.21037/qims-24-1037/rc).

Methods

A search was conducted in the PubMed database to explore the formation mechanism, diagnosis, and management of CNO. The search terms included the following: "near-occlusion", "carotid arteries", "critical stenosis", "small or narrow distal internal carotid artery", "preocclusive stenosis", "subtotal stenosis", "subtotal occlusion", "functional occlusion", "subocclusion", "hypoplasia", "99% stenosis", "hairline residual lumen", "incomplete occlusion", "near total occlusion", "pseudo-occlusion", "string sign", and "slim sign". These terms were primarily derived from a critical review (1). English-language articles published from 1950 to May 2024 were included. The search resulted in 986 studies. Non-English language literature and conference papers were excluded, resulting in 109 included reports. The detailed search strategy is presented in *Table 1*.

Pathomechanism

The natural course of CNO remains poorly understood. CNO is characterized by a thinning of the distal lumen due to severe ICA stenosis. When stenosis occurs, the pressure differential across the stenotic region drives an increase in blood flow velocity to preserve blood volume and maintain normal vessel diameter. However, it should be noted that velocity is not equivalent to flow volume, when stenosis exceeds 80%, the increase in flow becomes insufficient, leading to a decrease in flow. This decrease promotes the development of collateral circulation to compensate for the reduced blood volume (*Figure 1*). The transition from ordinary stenosis to CNO without full collapse proceeds according to the shape of Spencer's curve (18). Several

Table 1 Narrative review search strategy

Items	Specification
Date of search	February 1, 2024 to June 1, 2024
Databases searched	PubMed
Search terms used	Near-occlusion, carotid arteries, critical stenosis, small or narrow distal internal carotid artery, preocclusive stenosis, subtotal stenosis, subtotal occlusion, functional occlusion, subocclusion, hypoplasia, 99% stenosis, hairline residual lumen, incomplete occlusion, near total occlusion, pseudo-occlusion, string sign, slim sign
Timeframe	1950–2024
Inclusion and exclusion criteria	Included: full-text articles published in English; excluded: non-English language literature and conference papers
Selection process	Two doctors (F.X. and J.Z.) conducted the review process. Differences were resolved by discussion

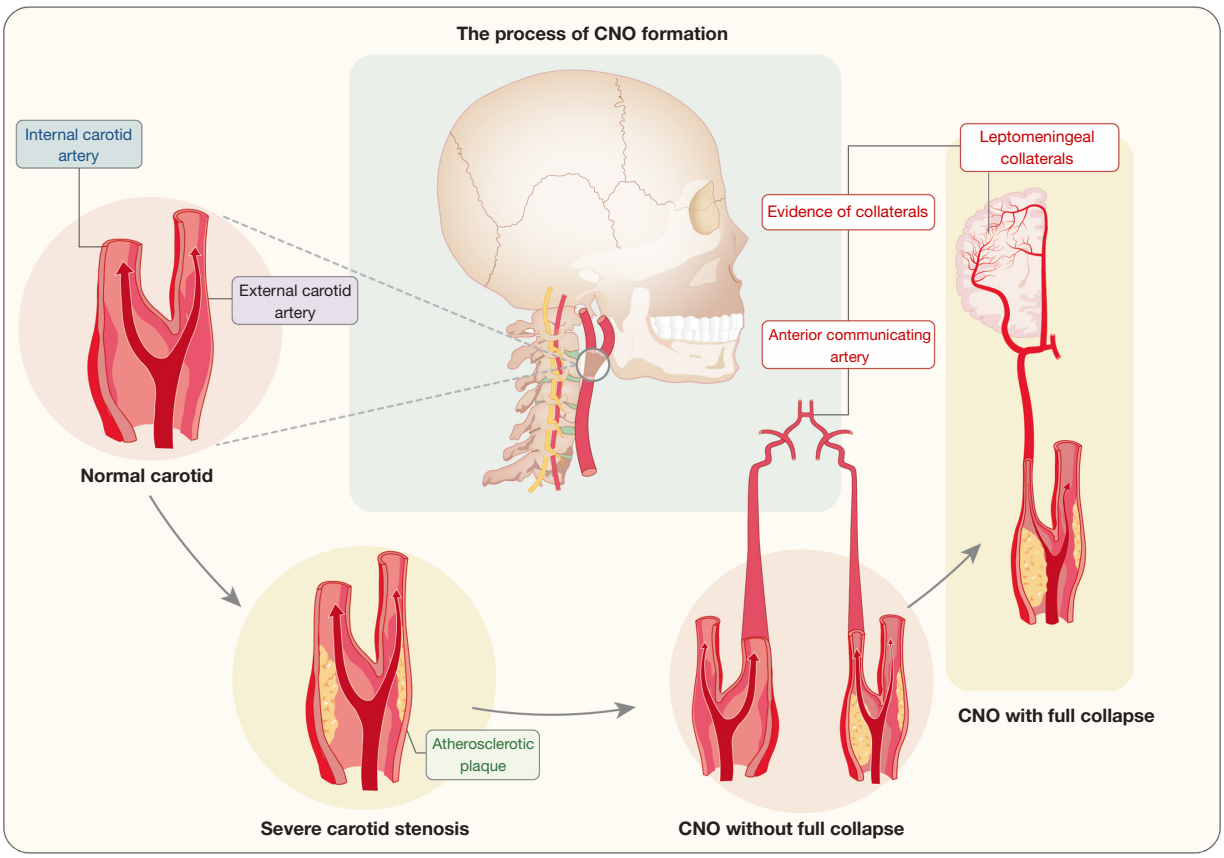

Figure 1 The transformation of a normal ICA into a CNO with full collapse. Initially, the appearance of atherosclerotic plaques in normal carotid arteries leads to lumen stenosis, while the distal lumen diameter of ICA remains normal. However, as stenosis progresses, the lumen diameter of the distal ICA diminishes, becoming smaller than that of the contralateral ICA and the ipsilateral ECA. At this stage, ordinary stenosis evolves into a CNO without full collapse and is accompanied by the opening of the intracranial branch circulation. Once the distal diameter completely collapses and assumes a "linear" configuration, a type of CNO with full collapse is formed. CNO, carotid near occlusion; ICA, internal carotid artery; ECA, external carotid artery.

studies have indicated that patients with CNO have high flow velocities, particularly those with CNO without full collapse (19-21). Moreover, 74% of CNO cases have been observed to exhibit high peak systolic velocity (PSV) (≥125 cm/s), as indicated by Khangure *et al.* (20). However, when a stenosis threshold is exceeded, the ICA's blood volume is not maintained at optimal levels, leading to an increase in back pressure generated by collateral circulation blood flow. The pressure differential is such that the distal ICA diameter begins to decrease significantly and becomes "linear". It is important to note that Spencer's curve is derived under ideal conditions and does not account for compensatory dilation of blood vessels; thus, the "other side" of Spencer's curve is not well described. Nevertheless, multiple studies have indicated that patients with CNO can ultimately progress to complete carotid occlusion (CO) (22,23).

The mechanisms underlying stroke in CNO primarily involve hypoperfusion and embolism. In ICA of the CNO, the gradual narrowing of the lumen leads to a progressive decrease in blood flow, ultimately resulting in CO. This impairment in cerebral hemodynamics prompts the activation of collateral circulation in compensating for reduced cerebral blood flow (CBF). The establishment of collateral circulation can mitigate the impairment of cerebral hemodynamics in patients with CNO to some extent (24), thereby lowering the risk of stroke. Studies indicate that 63.6–96% of patients with CNO exhibit collateral circulation (16,24-26). In the study by García-Pastor *et al.* (22), almost one-third of the 122 patients diagnosed with CNO ultimately developed CO. However, only 17.5% of these patients exhibited symptoms of stroke ipsilateral to the affected side, which may be attributed to the fact that patients with CNO tend to have well-developed collateral circulation. Palacios-Mendoza *et al.* (26) found that decreased or depleted cerebrovascular reserve (CVR) is more prevalent in patients with poor or absent collateral circulation. However, numerous studies have reported that some patients with CNO inevitably experience a reduction or failure of CVR (26-31). Oka *et al.* (29) found that CBF and CVR prior to carotid artery stenting (CAS) in patients with CNO are significantly lower than those observed in patients with traditional carotid artery stenosis and healthy individuals. Reduced CVR in patients is closely linked to the incidence of stroke (32,33). Rothwell and Warlow (8) suggested that patients with CNO may experience poor cerebral vascular perfusion but are unlikely to develop cerebral embolism. They proposed the low-flow cerebral protection hypothesis, positing that insufficient blood flow in the ICA fails to remove emboli from the plaque surface due to reduced CBF. This aligns with the concept of impaired clearance of emboli (34), in which reduced perfusion hinders the ability to flush and carry emboli. Furthermore, the number of intracranial microemboli is significantly reduced when the carotid artery is severely stenotic (35). Additionally, plaques in patients with CNO tend to have thicker fibrous layers and smaller lipid cores, potentially decreasing the likelihood of embolism (12). However, Fox *et al.* (2) argued that the low stroke risk associated with CNO indicates that the cause of strokes in these patients is not due to insufficient cerebral blood perfusion but rather arises due to embolism. This was supported by the findings of Johansson *et al.* (36,37) who observed a phenomenon of frequent early recurrent strokes followed by stabilization in patients with CNO. Slow blood flow in patients with CNO leads to blood coagulation and thrombus formation, which can result in embolism and subsequent stroke. Furthermore, some plaques in patients with CNO appear to be thrombi accompanied by neovascularization, which may partially support the presence of an embolic mechanism (12). Another study proposed that if patients with CNO experience strokes due to hypoperfusion, there should be relatively few asymptomatic patients with CNO with full collapse; however, this was not substantiated (38). A recent study demonstrated that symptomatic and asymptomatic CNO have similar total cerebral and hemispheric blood flow rates in (25). This finding does not support the hypothesis of CBF hypoperfusion. However, it is important to note that blood flow rate alone cannot directly indicate the extent of damage to cerebral hemodynamics. Both pathological mechanisms may account for the occurrence of stroke, yet neither has been confirmed with sufficient evidence, and a definitive link remains elusive.

Diagnostic imaging

CNO is a transitional stage between common stenosis and CO, and any inappropriate selection and interpretation of imaging methods can lead to a misdiagnosis of CNO. The earliest complete identification of CNO was performed by digital subtraction angiography (DSA) with feature interpretation. The greatest advantage of feature interpretation is that it allows the clinician to fully and flexibly utilize all the detailed information presented in the images to arrive at the most consistent diagnosis of carotid stenosis. However, due to the complexity

Table 2 Summary of diagnostic imaging

Diagnostic imaging	Advantage	Type of misdiagnosis	Reason
DSA	Provides accurate information regarding ICA stenosis, as well as comprehensive information on the collateral circulation, blood flow filling, and flow direction	(I) ICA bulb with no stenosis but a thinner distal lumen; (II) ICA bulb with no severe stenosis but a thinner distal lumen; (III) CO; (IV) stenosis	(I) Dissection, high intracranial pressure, radiation-associated disease, hypoplasia, and intracranial thromboembolism or stenosis; (II) ECA anatomical variations, asymmetric development of the circle of Willis; (III) slow filling of contrast medium; (IV) distal lumen collapse of CNO not significant
CTA	In comparison to DSA, this method is noninvasive and relatively straightforward to perform. It is also highly accurate in identifying the majority of CNO cases and their type		
Ultrasonography	Identifying CNO with full collapse	CO; stenosis	Narrow flow not visible; CNO without full collapse has high flow velocities
MRI			
TOF-MRA	No contrast media required	CO; stenosis	Signal absence; uncertain
PC-MRI	Provides blood flow information to distinguish CNO from stenosis		
CE-MRA	Distinguishing CNO with full collapse from CO		

DSA, digital subtraction angiography; CTA, computed tomography angiography; MRI, magnetic resonance imaging; TOF, time of flight; MRA, magnetic resonance angiography; PC-MRI, phase contrast magnetic resonance imaging; CE-MRA, contrast-enhanced magnetic resonance angiography; ICA, internal carotid artery; CNO, carotid near occlusion; CO, complete occlusion; ECA, external carotid artery.

of the procedure and the procedural risks involved, in clinical practice, it is not as commonly used as much as computed tomography angiography (CTA). CTA shares similar imaging principles to those of DSA and can better identify patients with CNO, but with less imaging detail. Ultrasound can detect most cases of completely collapsed CNO via severely altered carotid artery profiles and typical manifestations of low blood flow velocity. However, since many cases of CNO without full collapse do not exhibit low flow velocity, ultrasound presently has limited efficacy in identifying this condition. Time-of-flight magnetic resonance angiography (TOF-MRA) may misdiagnose CNO as CO due to signal loss, while contrast-enhanced MRA (CE-MRA) is beneficial for accurate identification. Phase-contrast magnetic resonance imaging (PC-MRI) shows potential in differentiating ordinary stenosis from CNO through flow metrics. There currently exist certain challenges and difficulties in generating high-quality and accurate diagnoses of CNO, as the awareness of CNO is not widespread. Moreover, there is a lack of knowledge and incomplete understanding of CNO in the diagnosis of carotid stenosis, and anatomical differences [circle of Willis variants and external carotid artery (ECA) collateral circulation formation] may lead to the misdiagnosis of CNO (*Table 2*).

DSA

Conventional angiography assesses the severity of carotid artery stenosis by calculating the ratio of the diameter of the most severe stenosis of the ICA to the distal normal diameter (39). However, when the NASCET method is applied to assess the CNO, the degree of stenosis of the CNO may be underestimated when the distal normal diameter decreases or collapses completely as a result of severe stenosis of the ICA. Accordingly, Fox *et al.* (2) initially proposed DSA-based feature interpretation to characterize the CNO, which includes the four following criteria: (I) delayed time for contrast arrival; (II) evidence of collaterals; (III) obvious diameter reduction of the ipsilateral distal cervical ICA in comparison to the opposite ICA; and (IV) obvious reduction of the ipsilateral distal ICA in comparison to the ipsilateral distal ECA. DSA offers unique advantages in demonstrating the filling speed of carotid artery contrast medium and the status of collateral circulation, and some ICA vessels appear to be initially occluded with no contrast filling but gradually fill with time. This allows for the differentiation of CO from CNO with full collapse, thus enhancing the precision of CNO diagnosis. When criteria (III) and (IV) described above are applied, it is essential to consider the possibility of ECA anatomical variations, as the ECA may have

a wider lumen than that of the ICA in the presence of vascular malformations, such as collateral circulation or fistula formation. Asymmetric development of the circle of Willis, such as a hypoplastic/aplastic ipsilateral A1 or large contralateral posterior communicating artery, can lead to a reduction in the normal lumen of the distal ICA on the same side (2,40,41). In the presence of these conditions, the application of criteria (III) and (IV) alone is insufficient for the diagnosis of CNO. Instead, the use of additional evidence, such as the delayed arrival of the contrast medium and the presence of collateral circulation, is necessary to make a conservative diagnosis of CNO. Furthermore, subtle differences should not be used as evidence for diagnosing severe stenosis as CNO. Additionally, congenital dysplasia of the ICA may also lead to a slender lumen, but it is important to note that CNO is characterized by severe stenosis of the ICA. Moreover, it is critical to differentiate between the ascending pharyngeal artery, a slender vessel originating from the ECA and conforming to the course of the ICA, and CNO with full collapse. Despite the absence of quantitative standards for feature interpretation, a high degree of consistency can be achieved in diagnosing CNO after training (2).

CTA

Thin-layer CTA has become a well-established technique for evaluating both intracranial and extracranial vasculature. It is gradually replacing DSA as the gold standard for carotid stenosis assessment due to its noninvasive nature and reduced procedural risks compared to those associated with DSA (42-44). The value of CTA in diagnosing CNO has been demonstrated in previous studies (45,46), but early identification was predominantly limited to cases of CNO with full collapse. Bartlett *et al.* (47) further developed the complete diagnostic criteria for CNO based on DSA as proposed by Fox *et al.* (2) by quantifying the criteria and applying them to CTA. This includes ratio of the distal ICA diameter to that of the contralateral distal ICA ≤0.87, diameter of the distal ICA ≤3.5 mm; narrowest diameter of the ICA bulb stenosis ≤1.3 mm. and ratio of the distal ICA diameter to the distal ECA diameter ≤1.27 (*Table 3*). The combination of the ratio of the distal ICA diameter to that of the contralateral distal ICA and diameter of the distal ICA has a sensitivity of 91.9% and a specificity of 96%. Bartlett *et al.* underscored the necessity of applying these four criteria in conjunction with feature interpretation. However, their study did not provide further detail regarding the application of the four criteria. Additionally, in the context of contemporary clinical practice, where images are presented in millimeters, the position of the patients, the marginal effect of the vessels, and the proficiency of the operator can potentially introduce errors of a few millimeters, which may ultimately result in misdiagnosis. In one study, a diagnosis of CNO based on meeting at least three of the Bartlett *et al.* criteria achieved a sensitivity of 100% and a specificity of 87% (48). The study by Manrique-Zegarra *et al.* (49) is the only one of its kind to validate the diagnosis of CNO using CTA based on the Bartlett *et al.* criteria against the diagnosis of CNO using feature interpretation based on DSA. Their findings indicate that compared to CNO diagnosed by ≥2 feature interpretations, using ≥3 Bartlett *et al.* criteria for diagnosing CNO yielded a sensitivity of 75–82% and a specificity of 87–90%. Notably, the observers measured and diagnosed CNO on CTA without knowledge of the DSA diagnosis, suggesting that the results may more accurately reflect daily clinical practice. In the early days of research, CNO was less well understood. As a result, the incidence of documented CNO was reported to be approximately 0.5% to 2% (50). However, in a recent study, 34% of 186 patients examined with ≥50% carotid stenosis had CNO (17). Furthermore, CNO is underestimated in clinical practice assessment. The sensitivity of CTA imaging reports for CNO is only 20%. Additionally, only 16% of radiologists can detect any CNO, and most cases are misdiagnosed as ordinary stenosis (48). Johansson *et al.* posited that those new to the field can gain a foothold by learning the criteria proposed by Bartlett *et al.* However, a lack of attention to detail can result in the confusion of common stenoses with CNO (48). Therefore, the popularization of diagnosis consisting of CNO-based feature interpretation in clinical practice should be emphasized.

In addition to using intricate diagnostic methods, researchers have conducted studies to quickly identify patients with CNO using ≥1 convenient criterion (49,51). However, it is important to note that variations in the circle of Willis and congenital hypoplasia of the ICA may result in misdiagnosis. Additionally, in one study, CNO with full collapse and CNO without full collapse were classified on images according to clinical symptoms. A distal ICA diameter ≤2.0 mm and/or an ICA ratio ≤0.42 was indicative of a higher risk for recurrent stroke. In such cases, the patient can be diagnosed with CNO with full collapse, which is better reflected in imaging for clinical prognosis (52,53). Other research has focused on developing

Table 3 Summary of the main results of the CTA Bartlett *et al*. criteria for CNO diagnosis

Authors	Total No. of patients with CNO	Ref. diagnostic criteria (diagnostic technique)	Bartlett *et al.* criteria	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
Bartlett *et al.*, 2006, (47)	42	Feature interpretation (CTA)	Crit. 1	90.2	84.1	62.7	96.7
			Crit. 2	95.2	89.9	66.7	98.9
			Crit. 3	97.3	89.4	65.5	99.4
			Crit. 4	90.5	87.3	61.3	97.6
			Crit. 2 and Crit. 3	91.9	96.0	81.0	98.4
			Crit. 3 and Crit. 4	89.2	95.4	78.6	97.9
			Crit. 2 and Crit. 4	88.1	95.4	80.4	97.4
			Crit. 1 and Crit. 2	87.8	96.8	85.7	97.4
			Crit. 1 and Crit. 3	86.8	96.3	82.5	97.3
			Crit. 1 and Crit. 4	82.9	96.8	85.0	96.3
Johansson *et al.*, 2020, (48)	104	Feature interpretation (CTA)	At least 3 criteria	100.0	87.0	NR	NR
			Fulfilling 4 criteria	93.0	97.0	NR	NR
Manrique-Zegarra *et al.*, 2022, (49)	28	≥2 of 4 feature interpretation (DSA)	Crit. 1	57–64	77–97	72–94	70
			Crit. 2	75–82	83–86	82–84	78–83
			Crit. 3	82–93	55–63	67–68	79–89
			Crit. 4	75–82	52–80	60–79	68–83
			At least 2 criteria	82–89	62–70	68–74	78–88
			At least 3 criteria	75–82	87–90	85–88	79–84

Crit. 1: narrowest diameter of the ICA bulb stenosis ≤1.3 mm; Crit. 2: distal ICA diameter ≤3.5 mm; Crit. 3: ratio of the ICA diameter to that of the contralateral distal ICA ≤0.87; Crit. 4: ratio of the distal ICA diameter to the distal ECA diameter ≤1.27. CTA, computed tomography angiography; CNO, carotid near occlusion; PPV, positive predictive value; NPV, negative predictive value; DSA, digital subtraction angiography; Crit., criteria; ICA, internal carotid artery; NR, not recorded; ECA, external carotid artery.

new imaging techniques, such as four-dimensional CTA, to aid in the diagnosis of CNO (54).

MRA

MRA is widely recognized as a safe, convenient, and noninvasive method for diagnosing carotid artery stenosis. Two-dimensional (2D) TOF is commonly used to evaluate the carotid vasculature, while only a few studies have used MRA to diagnose CNO. One study has suggested that 2D TOF may outperform three-dimensional (3D) TOF. However, it is important to note that the degree of stenosis is often exaggerated due to signal loss (19), which can potentially lead to the misdiagnosis of CNO as complete occlusion, especially in CNO with full collapse. Furthermore, CNO without full collapse may be mistakenly identified as a common type of stenosis. CE-MRA is superior to TOF-MRA due to the contrast agents' superiority in depicting the structure of the lumen (19,55). However, both CE-MRA and 2D TOF-MRA have low sensitivity and specificity for the diagnosis of distal stenosis and signal attenuation (56). In one study, PC-MRI demonstrated (57) 100% sensitivity and specificity in diagnosing CNO as reflected by an ICA flow ≤110 mL/min and a relative flow <35%, indicating great potential. Two subsequent studies provided further validation of the diagnostic potential of PC-MRI for CNO. Holmgren *et al.* (58) stated that an ICA-to-CBF ratio of ≤0.225 on PC-MRI yielded a sensitivity of 90% and a specificity of 99% in differentiating CNO from ≥50% stenosis. The same study further indicated that the mean ICA flow in CNO (70 mL/min) was lower than that observed in conventional ≥50% stenosis

(203 mL/min; P<0.001) (25). However, PC-MRI is unable to ably differentiate between CNO with full collapse and CNO without full collapse or to differentiate between CNO and CO. Consequently, it may be considered as a supplemental imaging tool for diagnosing CNO in daily practice in the future.

Ultrasonography

Ultrasonography is commonly used as the primary diagnostic tool for detecting carotid stenosis. Several studies have shown that both color and power Doppler can differentiate CO and CNO with full collapse (14,19,59,60), which are characterized by severe stenosis of the carotid arteries, with thin, narrow flow bundles that move slowly and systolic triangular spikes with or without diastolic flow at peak systole (14,59,60). Hetzel *et al.* (59) reported that color-coded duplex sonography detection of preocclusive carotid stenoses exhibited a sensitivity of 88% and a specificity of 99%. However, the early recognition of CNO was constrained to instances of CNO with full collapse, and as the definition of CNO broadened to encompass instances of CNO without full collapse, the sensitivity of conventional ultrasound features (severe stenotic vascular profiles and thin, slow flow) for identifying CNO diminished. Spencer's curve does not specify the specific blood flow rate for CNO (61). The relevant guidelines state that velocity parameters may not apply in cases of CNO because the velocities can be high, low, or undetectable (62). In studies that have included CNO without full collapse, it has been found that most patients with CNO exhibit high flow velocities (19-21,36). In one study, 12 of 14 patients with ultrasonography-confirmed CNO had a high PSV at the ICA stenosis on ultrasound, which ranged from 250 to 706 cm/sec (19). A subsequent study found that only 13% of the patients with CNO had a low PSV and 74% of CNO exhibited a PSV of ≥125 cm/sec (20). Many patients with CNO would have been classified as conventional stenoses based on high PSV value and thus potentially overlooked. A subsequent study further corroborated these findings, reporting the sensitivity of carotid ultrasound in diagnosing CNO to be only 22% (21). In the study by Palacios-Mendoza *et al.*, 45% of patients with CNO with full collapse were diagnosed by ultrasound as CO and 40% those with CNO without full collapse were categorized as severe stenoses (26). However, some studies have also demonstrated the favorable benefits of ultrasound in the diagnosis of CNO. Bowman *et al.* suggested that a low end-diastolic velocity can help to diagnose CNO (11). A small study demonstrated the feasibility of using contrast-enhanced ultrasound to differentiate CNO from CO (63), and in another study, the sensitivity and specificity of distal PSV ≤50 cm/s in differentiating CNO from common stenosis were 63% and 94%, respectively (64). Other distal parameters demonstrated similar sensitivity and specificity. This approach is anticipated to address the limitations of ultrasound in differentiating ordinary stenosis from CNO with high PSV. Nevertheless, at present, the sensitivity and specificity of any combination of ultrasound flow parameters do not exceed 75% due to significant overlap (21). Ultrasound is susceptible to mistaking CNO with full collapse for CO and mistaking CNO without full collapse for common stenosis; it is therefore currently not a viable diagnostic method. However, it can be considered a supplementary examination technique.

Comparative analysis

Imaging methods for diagnosing CNO

Due to the procedural risks associated with DSA, ultrasound and MRA are not yet fully reliable for identifying CNO. Consequently, CTA is commonly employed for diagnosing CNO given its low risk and relatively high accuracy (*Figures 2*,*3*). The feature interpretation of Fox *et al.* and the four criteria proposed by Bartlett *et al.* are commonly used to differentiate CNO from common stenoses and occlusions (2,47), with the Bartlett *et al.* criteria being more suitable for beginners. However, it is important to note that DSA provides more comprehensive information, including the direction of blood flow and collateral circulation. The use of ultrasonography to measure distal blood flow velocity and the use of PC-MRI to identify low carotid blood flow may be beneficial for supporting the diagnosis of CNO. Nevertheless, there remain a few unexplained occurrences, and the accurate diagnosis of CNO remains challenging across all imaging modalities due to the potential pitfalls related to anatomical factors and equipment limitations (2,21). This indicates the need for further research to aid in devising specific or new diagnostic criteria to distinguish CNO from other causes of a reduced distal lumen. Johansson *et al.* (41) were the first to propose using postoperative lumen expansion in patients with CNO as a reference in diagnostic research, aiding in the identification of cases with reduced distal ICA diameter that could not be accurately classified preoperatively. Holmgren *et al.* (58) were the first to use postoperative diagnosis as an auxiliary

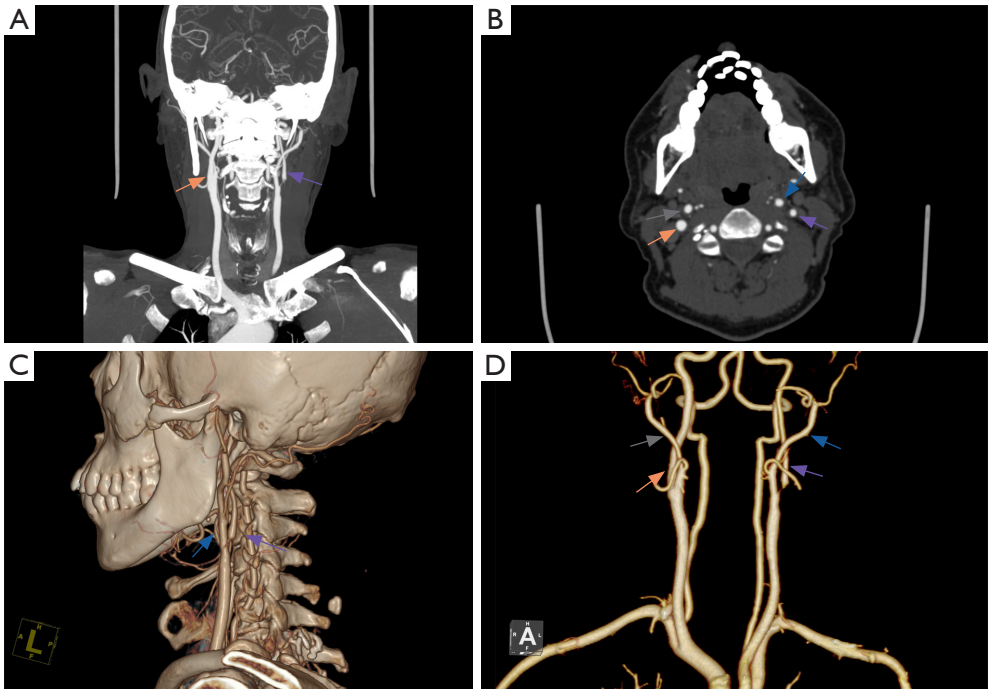

Figure 2 MPR images (A,B) and VR reconstruction images (C,D) of a case with left-sided carotid near-occlusion without full collapse. The left ICA is severely stenotic at its origin, with a partially reduced distal lumen (purple arrow) that is smaller than that of the contralateral ICA (orange arrow) and the ipsilateral ECA (dark blue arrow), and the right-sided ICA (orange arrow) is thicker than is the ipsilateral ECA (gray arrow). MPR, multiplanar reconstruction; VR, volume rendering; ICA, internal carotid artery; ECA, external carotid artery.

reference method for PC-MRI in the diagnosis of CNO and reported that the optimal threshold for PC-MRI could achieve 75% accuracy in diagnosing ICAs of unknown etiology. However, it is crucial to recognize that postoperative imaging cannot guide preoperative clinical practice, and thus this method can only serve as a complement to clinical information.

Imaging methods for diagnosing pathologic mechanisms of CNO

Determining a threshold for differentiating between therapeutic measures or prognoses for patients with CNO through imaging is an area of heightened research interest. If a stroke is mainly caused by embolization, then using imaging methods, such as transcranial Doppler imaging, to determine the degree of carotid stenosis when there is a significant increase in the value of emboli and a decrease in the number of microemboli, can guide clinicians in determining the patient's timing for revascularization treatment. If the stroke is due to inadequate perfusion, ultrasonography, cerebral perfusion, and metabolic imaging techniques can provide insights into the hemodynamic condition of the CNO. Furthermore, analysing the sequential relationship between changes in the ICA lumen and the onset of hemodynamic compromise leading to stroke symptoms can assist in deciding whether conservative or revascularization treatment is more suitable for patients with CNO.

Treatment

Best medical treatment (BMT)

BMT can help prevent the progression of atherosclerotic plaques in the blood vessels through the management of blood pressure and regulation blood lipid levels. This reduces the effects of high-risk factors on patient prognosis. Nonsurgical treatments currently include smoking cessation, glucose-lowering therapy, lipid-lowering therapy via statins, antiplatelet agents, anticoagulation therapy, and lifestyle improvements such as a balanced diet and regular exercise. According to the European Society for Vascular Surgery (ESVS) guidelines, BMT should be first considered

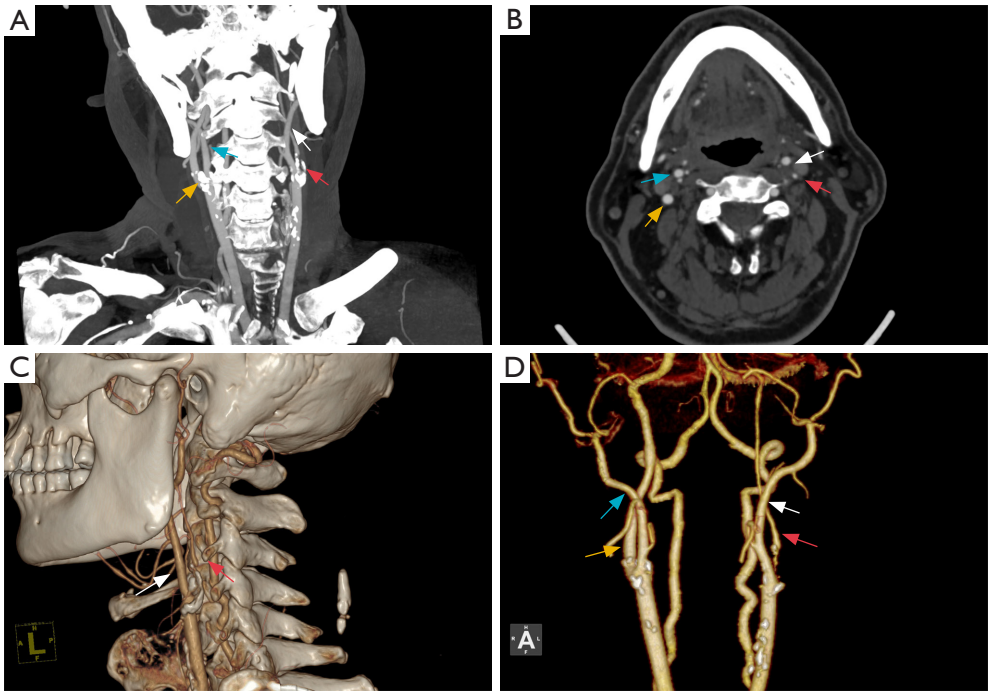

Figure 3 MPR images (A,B) and VR reconstruction images (C,D) of a case with left-sided carotid near-occlusion with full collapse. The left ICA is severely stenotic at its origin, and the distal lumen is completely collapsed, "thread-like" (red arrow), and smaller than the contralateral ICA (yellow arrow) and the ipsilateral ECA (white arrow), while the right ICA (yellow arrow) is thicker than is the ipsilateral ECA (light blue arrow). MPR, multiplanar reconstruction; VR, volume rendering; ICA, internal carotid artery; ECA, external carotid artery.

for patients with CNO (65,66). Initially, the BMT regimen for the NASCET and European Carotid Surgery Trial (ECST) involved aspirin therapy (67,68). According to the NASCET, the risk of ipsilateral stroke within 1 year for patients with CNO without full collapse is 18.3% with medical therapy and 9.1% with surgical therapy. In comparison, patients with CNO with full collapse have stroke incidences of 11.1% and 6.7% after medical and surgical therapy, respectively (16). However, newer additions to the therapy now include clopidogrel, newer statin drugs, and a more effective combination of cardiovascular medications. One meta-analysis found a negative correlation between pooled stroke rate after BMT and publication year (50), suggesting that advances in medication regimens provide long-term benefits for patients. In the ECST, poststenotic stenosis was a significant predictor of reduced stroke risk in the medication group (8). Neves *et al.* (69) reported that BMT has long-term benefits in patients with asymptomatic CNO. However, treating CNO with BMT alone may carry the potential risk for progression to CO (22,23).

Carotid endarterectomy (CEA)

CEA plays a crucial role in preventing stroke in symptomatic patients with severe stenosis (16,67,70). Ringelstein *et al.* (71) reported that in the early stages, patients presenting with string sign must be identified and undergo emergency surgery. Similarly, Sekhar *et al.* (72) noted that performing CEA is beneficial once a diagnosis of carotid artery pseudo-occlusion (i.e., CNO with full collapse) is made in a patient. Conversely, O'Leary *et al.* (23) posited that patients with ICA pseudo-occlusion is associated with a high risk of angiography and surgery. However, Fox *et al.* (2) rescreened 262 patients with CNO from patients with moderate or severe stenosis in the NASCET and ECST based on feature interpretation, of whom 6.1% had CNO with full collapse. A pooled analysis indicated that a 3-year ipsilateral stroke risk in patients with CNO was 15.1% and 10.9% for medically and surgically treated patients, respectively (P=0.33). The 3-year ipsilateral stroke risk in patients with severe stenosis but

no CNO was 26.0% and 8.2% for medically and surgically treated patients, respectively (P<0.001). The 2018 ESVS guidelines recommend BMT rather than revascularization for patients with CNO (65). However, this conclusion was based on outdated data and has been questioned. First, the benefits of CEA may be underestimated because most patients in the NASCET underwent surgery more than 4 weeks after the last clinical symptom, while the recurrence rate of stroke symptoms substantially reduced over time (36). Additionally, the crossover rate between the medical and surgical groups in patients in the NASCET was as high as 50% (68), and patients in the ECST had a lower prevalence of stroke risk factors such as advanced age, diabetes, ischemic heart disease, and hyperlipidemia (2). This suggests that the trial results may not be applicable to real-world scenarios due to the lack of adequate capture of the impact of patient risk factors on prognosis in randomized trials. Finally, the NASCET and ECST tended to exclude CNO with full collapse. The 2023 ESVS guidelines also recognize some of these aforementioned issues (66). Subsequently, several small studies sought to demonstrate the safety and efficacy of CEA in patients with CNO (73-77); however, the majority of these studies were retrospective, and these results primarily addressed the short-term prognosis within 1 year. One prospective study involving patients with CNO appeared to include individuals with severe carotid artery stenosis (77). One study indicated that the stroke rate in a mean period of 28.3 (range, 3–60) months in patients with CNO treated with CEA was only 2.6% (78). In contrast, findings from a nonrandomized, prospective, observational, multicenter study revealed that the complete revascularization success rate with CEA was 87%. At 24 months, the cumulative ipsilateral stroke rates for patients in the CEA group and the BMT group were 17.4% and 13.1%, with corresponding mortality rates of 4.5% and 5.6%, respectively (79). These results suggest that revascularization may not significantly reduce the risk of stroke in patients with CNO.

CAS

CAS was introduced in 1994 as an alternative to CEA for stroke prevention in patients with carotid artery stenosis (80). Most CAS procedures use embolic protection devices (EPDs) to reduce distal embolization, decrease the incidence of postoperative stroke, and improve patient survival (81-84). The survival and stroke recurrence rates after CAS and appeared to be similar to those after CEA (78). Furthermore, Stěchovský *et al.* (85) demonstrated no statistically significant difference in the 5- and 10-year survival rates between patients with CNO and those with severe stenosis. This finding suggests that performing CAS in patients with CNO may perhaps not influence their long-term survival. Although the reduction of distal blood flow in patients with CNO can, to some extent, reduce the risk for distal embolism, it can also lead to impaired hemodynamics and reduced vascular reactivity (26,30). CAS can improve cerebral perfusion in patients with CNO (29,86-88) and improve cognitive function in patients with CNO (89). Thus far, few large-scale prospective studies on CAS for the treatment of CNO have been conducted. Some research indicates that the success rate of CAS for CNO ranges from 79.1% to 100% (13,29,30,69,81,90-94). In short-term follow-up within 30 days after surgery, the incidence of stroke or fatal events ranges from 0% to 8.3% (13,28-30,81,85,90,95), while the 5-year survival rate ranges from 69.8% to 85.8% (78,85). These results appear to indicate a potential clinical benefit of CAS for the treatment of CNO. However, the majority of the related studies have been retrospective and of a limited sample size and may thus represent an insufficient evidence base.

At present, there is a scarcity of studies specifically addressing the treatment of both symptomatic and asymptomatic CNO with CAS, and whether patients with asymptomatic CNO should receive CAS treatment remains contentious. Neves *et al.* (69) demonstrated that the incidence of neurological events in symptomatic patients with CNO with full collapse successfully treated with CAS was comparable to that of asymptomatic patients with CNO without full collapse treated with BMT. However, the short-term and midterm prognosis for patients who experienced failed revascularization was poor. Additionally, in the study by Cay *et al.* (92), the permanent morbidity and mortality rates for CNO in the asymptomatic group were 0%, compared to 4.9% in the symptomatic group, with a similar incidence of hyperperfusion syndrome observed in both groups. Meanwhile, Stěchovský *et al.* (85) conducted a matched cohort and found that the 30-day incidence rates of the primary outcome (any stroke or death) were 10.5% for the symptomatic CNO group and 9.0% for the severe stenosis group (P=0.748). In the asymptomatic CNO group and the severe stenosis group, the 30-day incidence rate of the primary outcome was 6.5% and 4.4%, respectively (P=0.688). Regardless of whether CNO was symptomatic, there was no significant difference in the 30-day incidence rates in the primary outcome between the severe stenosis

group and the CNO group. However, this study did not further compare the symptomatic CNO group with the asymptomatic CNO group.

A meta-analysis (50) revealed a negative correlation between the pooled stroke rate after CAS and the publication year, indicating promising prospects for CAS in preventing postoperative embolization and restenosis in CNO due to advances in EPDs and the introduction of new stents. Transcarotid artery revascularization (TCAR) is an emerging technology that plays a critical role in complementing traditional CAS (96,97), yet there is currently a lack of research on TCAR for the treatment of CNO.

Comparative analysis

The latest ESVS guidelines recommend BMT as the first-line treatment. For cases of recurrent symptomatic CNO with distal vessel collapse, CEA or CAS may be considered following discussions by a multidisciplinary team (66).

Prognosis and treatment of CNO

Acute ischemic stroke is associated with highly mortality and disability, and stroke survivor can expect a poor long-term prognosis (98,99). To ascertain the optimal treatment for patients with CNO, it is first necessary to establish the risk of stroke in patients with CNO throughout the natural course of CNO and to determine the risk of complications associated with different treatment modalities. However, the evidence regarding the risk of stroke in patients with CNO is limited and largely inconsistent. The early pooled evidence from the NASCET and ECST indicates a 3-year ipsilateral stroke risk of 15.1% in patients with CNO treated with BMT, while in patients with severe stenosis without near occlusion, the 3-year ipsilateral stroke risk is 26.6% (2). The incidence of stroke in patients with CNO is lower than that in patients with severe stenosis. However, the risk of stroke might have been underestimated because 51% of patients in these trials had a 4-week gap between the time of final symptom onset and inclusion in the study, and there were only 16 CNO patients with full collapse (2,68,100). Subsequent studies have indicated that patients with CNO may be at an elevated risk of stroke. Johansson *et al.* (36) observed that symptomatic patients with CNO with full collapse exhibited a heightened risk of stroke at 90 days compared to patients with 50–99% stenosis (43% *vs.* 18%; P=0.041). Furthermore, the 90-day risk of stroke in patients with CNO without full collapse was 0%. However, as the diagnosis of most patients with CNO is primarily based on ultrasound, there is a possibility of misdiagnosis, with CNO being incorrectly identified as 50–99% stenosis or occlusion. Additionally, the sample size of patients in the related studies has been insufficient, impairing the assessment of the risk of stroke recurrence. Gu *et al.* (101) employed the CTA method for diagnosing CNO and concluded that symptomatic CNO patients exhibit a markedly elevated short-term risk of ipsilateral ischemic recurrence at 90 days. This risk was particularly pronounced in patients with CNO with full collapse. However, a different study indicated that the 90-day cumulative incidence of stroke, transient ischemic attack (TIA) or retinal ischemia in patients with symptomatic CNO patients was only 10.6% and that patients with CNO with full collapse were not associated with a high risk (102). Henze *et al.* (53) first assess the risk of recurrent stroke in symptomatic near-occlusion with full collapse defined by distal ICA diameter ≤2.0 mm and/or ICA ratio ≤0.42. Their findings indicated that the risk of stroke in patients with CNO without full collapse and those with CNO with full collapse within 2 days of the event was 3% and 16%, respectively (P=0.01); moreover, a 28-day stroke risk of 16% and 22% was observed, respectively (P=0.22), indicating that patients CNO with full collapse may have a high risk of stroke recurrence at an early stage.

Several meta-analyses have indicated that BMT in patients with symptomatic CNO does not yield better long-term outcomes than do CEA or CAS (50,103-105). However, two of these meta-analyses did not incorporate the results of all studies (103,104), one meta-analysis did not provide study comparisons (105), and another meta-analysis omitted the time of occurrence of the consequences of different studies when discussing the consequences of the treatment modality (50). The most recent meta-analysis revealed that the combined incidence of acute ischemic stroke occurring at >30 days for BMT, CAS, and CEA was 9.90%, 0.79%, and 0.80%, respectively. Additionally, a subgroup analysis of mortality beyond 30 days was statistically significant difference (P<0.016). However, meta-regression did not indicate a clear preference for a specific treatment modality over another, and the remaining outcomes were not statistically significant. Additionally, the meta-analysis of studies of consequential events after >30 days similarly failed to consider the time to event (106). Following the NASCET and ESCT, there has been a notable absence of large, multicenter, prospective trials. Despite the evidence indicating a high success rate of revascularization and a low risk of stroke recurrence

Table 4 Summary of the main results from revascularization and BMT

Author	Study type	Total No. of patients with CNO [CNO with full collapse, without full collapse]	Diagnostic technique	Mean age (years)	Treatment			Follow-up		
					Revascularization		BMT No.	Mean follow-up period (months)	Revascularization, BMT	
					No. (method)	No. of any strokes and death within 30 days			No. of carotid occlusion, n/N	No. of ipsilateral strokes and cases neurogenic or cardiac death
O'Leary, *et al.*, 1989, (23)	Retrospective study	34 [NR]	DSA	66	25 (CEA)	2	9	34	4/23, 9/9	0, 5
Ascher, *et al.*, 2002†, (10)	Retrospective study	17 [NR]	Ultrasonography	73	8 (CEA)	0	5	8	NR, NR	0, 1
Radak *et al.*, 2010, (77)	Prospective cohort study	309 [NR]	Ultrasonography, DSA and/or CTA	64	259 (CEA)	0	50	12	NA, 15/40	8, 11
Fox, *et al.*, 2005, (2)	Re-analysis of NASCET and ECST trial	262 [246, 16]	DSA	NA	148 (CEA)	8	114	36	NR, 5/19	16, 17
Neves, *et al.*, 2018‡, (69)	Prospective cohort study	46 [46, 0]	Ultrasonography and CTA	65.7	19 (CAS)	1	22	63.9	2/19, 3/22	2, 2
Garcia-Pastor, *et al.*, 2022, (79)	Prospective cohort study	141 [41, 100]	DSA	68.71	67 (CEA, CAS)	4	74	24	2/60, NR	12, 13

†, four patients with internal carotid artery ligation were excluded; ‡, five patients with unsuccessful CAS placement were excluded. BMT, best medical treatment; CNO, carotid near occlusion; NASCET, North American Symptomatic Carotid Endarterectomy Trial; ECST, European Carotid Surgery Trial; NR, not recorded; DSA, digital subtraction angiography; CTA, computed tomography angiography; CEA, carotid endarterectomy; CAS, carotid artery stenting; NA, not available.

(23,75,78,81,95), there is currently a paucity of comparative studies of revascularization versus BMT treatments, with some findings being contradictory (*Table 4*). In the study by Radak *et al.* (77), the incidence of TIA, ipsilateral stroke, and neurologic mortality within 12 months in patients CNO treated with CEA and those treated with BMT was 5% and 24% (P<0.001), 1.5% and 14% (P<0.001), and 1.5% and 8% (P=0.034), respectively. Neves *et al.* (69) reported that asymptomatic patients with CNO with full collapse had no neurologic events during the follow-up period, with a cumulative survival rate of 81.8%. In contrast, the cumulative survival rate of the successful versus the unsuccessful group of CAS placement was 89.4% versus 40%, respectively. Song *et al.* (89) demonstrated that CAS equipped with EPD can reduce the incidence of ipsilateral TIA and stroke while improving cognitive function in patients with CNO. In contrast, cognitive function in patients treated with BMT decline compared to baseline after 12 months. However, a recent prospective, multicenter, and nonrandomized study found that neither CAS nor CEA reduced the risk of stroke during follow-up when compared to BMT (79). García-Pastor *et al.* found that CO was more prevalent in patients with CNO receiving BMT compared to those treated with revascularization (55.7% *vs.* 9.8%; P<0.001), but the frequency of symptomatic CO was higher, and symptoms were more severe in those treated with revascularization (11.8% *vs.* 50%) (22). This may be attributed to the inherent collateral circulation that patients with CNO possess for maintaining CBF. This finding is consistent with the results reported by Neves *et al.* (69).

Therefore, it is crucial to consider the optimal indications and timing for CAS, CEA, and BMT. In patients with chronic CNO, the benefits of surgery decrease as the risk for stroke recurrence decreases (103). Therefore, BMT may be a preferable treatment option for patients with chronic CNO, asymptomatic CNO, and high-risk factors such as congestive heart failure (69,103). For patients with CNO who require revascularization according to multidisciplinary team discussion, CAS may be a more suitable option in those with highly situated plaques, ipsilateral CEA, a history of neck surgery, or cranial nerve injury. However, CEA may be a better choice for patients with a curved aortic arch or carotid arterial course and severely calcified plaques. In patients with CNO, CEA and CAS should be viewed as complementary—rather than competing—treatment options (76).

CNO with full collapse and CNO without full collapse

Compared to CNO without full collapse, CNO with full collapse is associated with a narrower lumen, reduced flow rate, and diminished blood flow (25,64). However, there is limited pathophysiological information available for comparing CNO without full collapse and CNO with full collapse, particularly regarding collateral circulation and cerebral blood perfusion. Palacios-Mendoza *et al.* (26) reported there being no statistically significant difference in collateral circulation, CVR, mean velocities, or pulsatility indexes of the middle cerebral artery between CNO without and with full collapse. The stroke risk associated with CNO with full collapse remains a subject of debate (16,36,53,101). Different treatment modalities also carry varying risks. Patients receiving BMT alone appear to have a higher risk of recurrent stroke (36,53,101) and the potential for progression to CO (22,23). Furthermore, compared to CNO without full collapse, CNO with full collapse may have a poorer prognosis following revascularization (73,79,92,107). This increased risk of poor outcomes may be attributed to a higher incidence of postoperative recanalization failure, restenosis, and intracerebral hemorrhage, which may arise from less ICA blood flow in CNO with full collapse. When the ICA is severely stenotic, cerebral perfusion pressure decreases, prompting local arterial blood vessels to dilate to maintain CBF. However, in the regions where arterial dilation occurs, cerebral vascular reactivity diminishes. Following revascularization, cerebral perfusion pressure rises sharply, which passively leads to an increase in CBF, ultimately resulting in cerebral hyperperfusion syndrome and cerebral hemorrhage. Additionally, balloon dilatation and stent placement may interfere with carotid pressure receptor reflexes (108), contributing to overperfusion. The risk of postoperative intracerebral hemorrhage may counterbalance the clinical benefits of revascularization. Furthermore, the significantly slender lumen distal to the CNO with full collapse presents challenges for stent expansion and can complicate the placement of a drainage tube during CEA, thereby increasing the incidence of postoperative restenosis (69,79). Moreover, thrombotic recanalized plaque may heighten the likelihood of guidewire puncture failure during CAS (12).

In summary, the optimal treatment of CNO remains a topic of debate, with current guidelines recommending BMT based on two large studies conducted in the late twentieth century. The quality of the available evidence on CNO treatment studies is insufficient, with the majority of studies employing retrospective, single-center designs with small samples. Consequently, the results of these studies may not be applicable to all patients with CNO due to the variations in the baseline patient characteristics, surgeon proficiency, and the perioperative care provided. Several studies have also indicated a correlation between different CNO subtypes and clinical prognosis (36,53,69,79,101,107,109); however, the evidence is limited and inconsistent, which poses challenges in drawing definitive conclusions. Large-scale, prospective, multicenter, clinical studies are, therefore, needed in the future.

Conclusions

The mechanisms underlying CNO stroke primarily include hypoperfusion and embolism. DSA and CTA can effectively diagnose most cases of CNO based on feature interpretation. Additionally, ultrasound measurement of distal flow velocity and PC-MRI assessment of ICA blood flow hold promise for identifying CNO. Diagnostic errors in CNO may arise from anatomical variations (alterations in the circle of Willis or congenital ICA hypoplasia) and device-related factors (imaging posture, edge effects, and measurement inaccuracies). The perception of low stroke risk in patients with CNO remains controversial. Current guidelines advocate for BMT as the primary treatment option for CNO. Although numerous studies have suggested the potential benefits of revascularization, the supporting evidence is insufficient. Prospective, large-scale, and multinational clinical trials should be conducted to establish the most effective treatment strategies for patients with various CNO subtypes.

This article presents a comprehensive review of the latest research on the CNO mechanism, imaging diagnosis, and treatment prognosis, offering valuable insights into the current state and controversies of CNO diagnosis and treatment. However, some limitations should be noted. First, the studies included in this review exhibit heterogeneity, which precludes the derivation of further quantitative results. Second, PubMed was the only database searched, and due to the lack of a unified terminology for CNO, a small number of relevant reports might have been missed.

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Footnote

Reporting Checklist: The authors have completed the Narrative Review reporting checklist. Available at https://qims.amegroups.com/article/view/10.21037/qims-24-1037/rc

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://qims.amegroups.com/article/view/10.21037/qims-24-1037/coif). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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