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Carotid Dolichoarteriopathies: A Comprehensive Overview

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Abstract: Carotid dolichoarteriopathies (CDA) represent a group of morphological abnormalities, with changes in the geometry and tortuosity of the carotid arteries. They were described in 1925 for the first time and were classified in three types according to the angle of torsion, in tortuosity (angle > 90°), coiling (S-shaped curve or loop) and kinking (angle < 90°). The pathophysiology of CDA is controversial and includes congenital mechanisms, genetic factors, connective tissue diseases, acquired mechanisms which can be associated with but not dependent on cardiovascular risk factors, and anomalies of the cervical spine. CDA have been associated with different cardiovascular and cerebrovascular events secondary to hemodynamic abnormalities, thromboembolism and cerebrovascular insufficiency and ischemia. However, the evidence is limited and for some authors they are more of a curiosity than a real predictor of ischemic events. Other studies support the clinical value of the diagnosis and follow-up of CDA and their understanding not only by internists, cardiologists and neurologists, but also by surgeons and otolaryngologists. Several authors proposed different therapeutic strategies to correct CDA, including surgical procedures. However, the indications and management approaches are controversial, and further randomized, multicenter, prospective studies are required to determine the most appropriate course of action. Until then, imaging techniques remain the basis for the etiologic diagnosis of cerebrovascular adverse events when all other causes have been excluded, and close clinical monitoring and follow-up of patients remain key strategies for the prevention of secondary events.

Key words: dolichoarteriopathies; tortuosity; carotid arteries; vessel abnormalities; cerebrovascular disease; cardiovascular risk factors

1. Introduction

Atherosclerosis is the most common cause of extracranial carotid artery disease and one of the leading causes of cerebrovascular disease (CVD) and death. [1, 2] It is the predominant etiology of CVD in the West. [3] However, other non-atherosclerotic causes, such as fibrodysplasia, Takayasu disease, and aortic dissection, among others, are associated with CVD. [1, 4] This group includes dolichoarteriopathies (from the Greek δόλιχος, dolichos, "abnormally long"), which

have recently received the attention of specialists. Carotid dolichoarteriopathies (CDA) have been associated with a wide spectrum of CVDs, such as stroke and transient ischemic attack (TIA). [5] However, their actual clinical significance remains unclear. [1] In this paper, we review the main aspects of CDA, focusing on its pathophysiology, diagnosis, prognosis, and treatment.

2. Methods

A non-systematic review was conducted, selecting the most significant works related to this topic. The search engines PubMed, Scielo, and Google Scholar were used, using the following keywords: dolichoartheriopathies, carotid dolichoartheriopathies, arterial tortuosity, dolichoectasia, and their Spanish equivalents.

3. Results

3.1 Definition, classification, and epidemiology

CDAs are an anatomical anomaly consisting of a change in the geometric arrangement that causes coiling, kinking, and tortuosity of the carotid vessels along their course. [1, 5] Some authors include them within the group of dolichoectasias (elongation, tortuosity, and/or dilation of the vessels). [6] CDAs were first described by Kelly in 1925 [7] and classified by Weibel and Fields [8] (Figure 1) into three groups:

- Type I or Tortuosity: Any elongation or undulation in the S or C shape of the carotid artery, with an angulation greater than 90°.
- Type II or Coiling: Elongation or redundancy of the internal carotid artery, resulting in an exaggerated S-curve or a circular configuration, acquiring a loop-like morphology (a 360° angulation of the artery about its transverse axis).
- Type III or Kinking: Angulation of one or more segments of the artery, with an angle less than 90°.

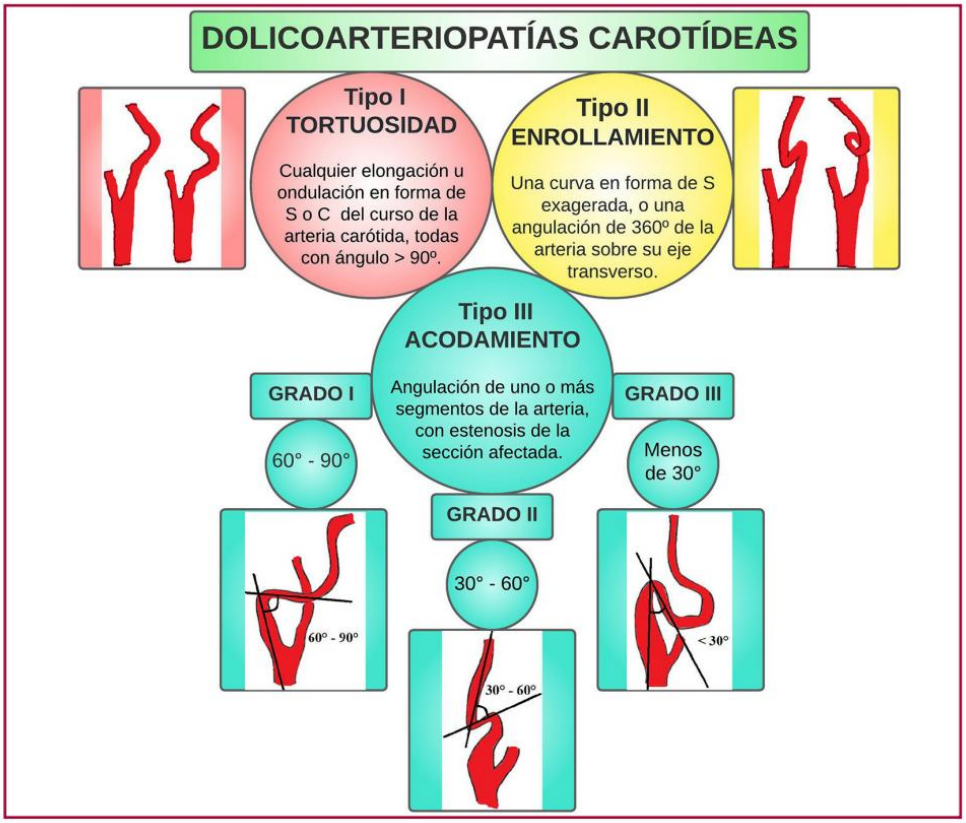

Figure 1. Schematic of the classification of carotid dolichoarteriopathies, combining the Weibel-Fields and Metz classifications. The illustration of the Weibel-Fields classification was modified from reference #41. The illustration of the Metz classification was modified from reference #5.

Metz et al. subclassified kinks based on the severity of the angulation: Group I (acute angle measured between the two segments forming the torsion with an amplitude between 90° and 60°, Figure 2, Panel A), Group II (between 60° and 30°, Figure 2, Panel B), and Group III (less than 30°, Figure 2, Panel C). [9]

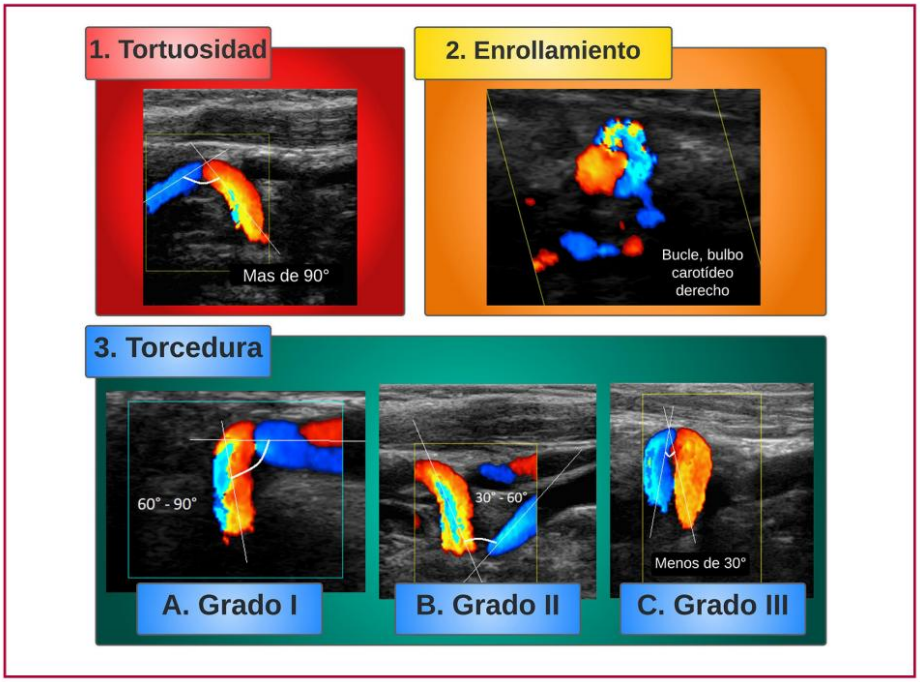

Figure 2. Images of different types of carotid dolichoarteriopathies obtained with color Doppler echocardiography. The angle between each part of the artery is drawn with white lines and described in the corresponding image.

CDA is a common entity, with a prevalence of 10-45%. [10] Beigelman et al. [1] and Ghilardi et al. [11] diagnosed CDA in 13.3-31% of cases, while in studies of hospitalized patients, the prevalence ranged from 13.5% to 58%. [6] CDA is more prevalent in women and elderly patients, especially those over 60 years of age, and can be either uni- or bilateral, affecting the common carotid artery (CCA), internal artery (ICA), and external artery (ECA), but more frequently the ICA, especially on the left side. [12, 13] Tortuosities and kinks are more common than coils. [1, 12]

3.2 Pathophysiology

Several mechanisms have been implicated in the development of CDA, although controversy remains. A key question is whether it is a congenital or acquired phenomenon, and whether it is related to atherosclerosis or other cardiovascular risk factors. Some studies shed light on this issue.

A. Embryological mechanism

The cerebral vasculature begins to develop early in the embryo, during the third week of intrauterine life, through the formation of the aortic arches (AA). [13, 14] There are six pairs of AAs that connect the ventral aorta (VA) with the dorsal aorta (DA). [14, 15] Each AA arises from the bulbus arteriosus and runs within its corresponding pharyngeal arch to terminate in the DA. [14] The first and second arches disintegrate, and their nascent roots from the VA and DA persist as incipient ICA and ECA. The ICA integrates with the third AA bilaterally, whereas the ventral aortic root between the third and fourth arches persists as the CCA. [15] There is considerable variability in the course and position of the CCA and carotid bifurcations. Elongation of the CCA and ICA leads to tortuosity and kinking. [16] The position of the carotid bifurcation reflects the degree of embryological migration of the ECA and is variable. [16] Huber et al. located the carotid bifurcation at the level of C4-C5 in 48% of 658 cases, and C3-C4 in 34%. [17] Cases of bifurcation from T3 to C2 have been described. [18] In children, C2 to C3 was observed in 40%, and C3 to C4 in 40%. [19] Carney et al. found tortuosities

in 5 of 20 fetuses between 5 months and full term. [20] Harrison et al. suggested that the development of the carotid arteries and the skeletal system may be asynchronous, generating alterations and tortuosity in the course of the artery. [21] Beigelman et al. studied a population of 885 people (4.5 hours postnatal - 90 years), and divided them into a control group of 245 people (children and adolescents up to 15 years), and another of 640 individuals (16 - 90 years), in whom color Doppler echo of neck vessels had been requested due to clinical suspicion of atherosclerosis. The incidence of coils and kinks was similar in both groups. The presence of atheromatous plaques in the tortuosity was only observed in 3 patients in the second group. The authors found a lack of association with other cardiovascular risk factors, and of the location of carotid plaques with CDA. [1]

B. Genetic and molecular disorders

Voevoda et al. studied 61 families whose children had been previously diagnosed with ICA CDA using color duplex Doppler ultrasound. The group consisted of 100 individuals with this disorder. The control group (n = 245) was formed from a DNA bank based on a population-based approach. They found an association between the A80807T polymorphism of the Sp4 transcription factor gene and CDA. [22] Zaidi et al. reported the case of a consanguineous Kurdish family whose child presented with severe elongation and tortuosity of the aorta, carotid arteries, and other arteries, including other abnormalities (loose skin, joint hypermobility, hernias, and facial features resembling Ehler-Danlos syndrome [EDS]). They found homozygosity of the 20q13 locus in the affected child. [23] Arslan et al. observed significantly higher levels of matrix metalloproteinase (MMP) type 2 in patients with CDA compared to the control group. MMP-12 expression was higher in those with atheromatous plaques than in those without atherosclerosis. [24]

C. Connective tissue diseases (CTD)

Foiadelli et al. reported the case of 7 children with EDS, aged 3 to 13 years, with CDA and variable clinical manifestations. [25] Welby et al. studied 286 patients divided into control and CTD groups. The presence of CDA was 44% in the latter group and 16% in the controls ($p < 0.001$). Coils were more prevalent. CDA was seen in 88% of cases with Marfan syndrome, 63% for Loeys-Dietz syndrome, 42% for neurofibromatosis type 1, and 19% for EDS, both vascular and nonvascular types. [26]

D. Association with other congenital diseases

According to Paltseva et al., CDAs have higher elastin levels, but with fiber fragmentation, increased MMP-9 expression, and decreased smooth muscle actin expression. [27] Ballotta et al. analyzed 78 carotid arteries with CDAs and found typical and atypical patterns of fibromuscular dysplasia (FMD). [28] Sethi et al. found a significantly higher prevalence of S-shaped CDAs in patients with FMD. [29]

Ballota et al. studied 43 patients with asymptomatic abdominal aortic aneurysms (AAAs) and CDAs, and found degenerative dysplastic changes in the tunica media in all carotid samples. In some cases, these changes overlapped with atherosclerotic intimal lesions. The histological features of classic AAA (thinning of the tunica media underlying the atherosclerotic plaque) were observed in almost all aortic wall samples, suggesting a common basis of connective tissue alterations underlying both pathologies. [30]

E. Acquired mechanisms

Harrison et al. argue that CDA could be due to kyphosis or lordosis that deviate the carotid axis. [21] For Etheredge et al., inflammation of the pericarotid tissues causes the carotid arteries to retract and deviate, developing tortuosities. [31]

Ghilardi et al. and Del Corso et al. describe a high prevalence of high blood pressure (HBP) and atherosclerosis in patients with CDA. [11, 32] However, both studies lack a normal group of participants, and address only a population selected for vascular pathology, in which a predominance of such cardiovascular risk factors is expected. For Khasiyev et

al., because CDAs also show dilation, compensatory external remodeling in response to atherosclerosis may play a role in this phenotype. [6] Carotid dilation may also reflect non-atherosclerotic connective tissue weakness. Pancera et al. found a statistically significant association between kinking and hypertension, and also between the presence of hypertension and a greater degree of kinking flexion. [33] Oliviero et al. studied the prevalence of CDA in patients with hypertension and diabetes. The prevalence of CDA was higher in the hypertension group than in the diabetes and control groups, and was also associated with the length of time the patients were hypertensive. [34] Eccentric forces associated with hypertension, and possibly arterial aging, accompanied by the development of stiffness, may play a role in ICA dilation and, therefore, in the development of CDA. [6]

Wang et al. studied the relationship between body mass index (BMI) and CDA. They analyzed a total of 513 patients, all without CTD, using the tortuosity index (TI), expressed as a percentage, calculated as a ratio of the actual ICA length (LR) to the length measured from the carotid bifurcation to the skull base, called cord length (CL):

$$TI = [(LR/CL) - 1]*100$$

Male sex and BMI were significantly correlated with TI. Each BMI point increased the risk of developing CDA by 1.59 times. [35] Dilba et al. used the same index in a population from the *Plaque At RISK* cohort. They concluded that age, obesity, and hypercholesterolemia are associated with higher TI. Furthermore, they found a higher prevalence and severity of CDA in the left ICA. Elevated BMI and CDA may be due to high abdominal pressure. Increased abdominal pressure can push the diaphragm upward, and with it, the entire mediastinum. This would elevate the carotid arteries, which, being surrounded by loose connective tissue, can slip and develop tortuosity. Because the brachiocephalic artery absorbs some of the lifting force, the right ICA develops less severe tortuosity than the left. Thus, repeated and prolonged exposure to periods of high and low flow results in stress that affects arterial remodeling, causing tortuosity. [36]

Derubertis et al. observed a higher prevalence of CDA in patients previously treated with radiation therapy. Radiation can cause retraction and alteration of connective tissue, producing tortuosity of the artery. [37] Saba et al. studied a population of 124 patients with ICA dissection diagnosed by computed tomography (CT) or magnetic resonance imaging (MRI). They observed a statistically significant association between dissection and kinking and coiling. [38] Barbour et al. found a significant association between spontaneous arterial dissection and ICA tortuosity, especially if the torsion is bilateral. [39] Some authors suggest that hemodynamic alterations can cause endothelial rupture and damage. The resulting inflammation can alter vessel structure, and inflammatory agents can lead to arterial dissection. [6]

3.3 Diagnosis

CDA can be diagnosed using a wide range of imaging techniques. The gold standard appears to be ultrasonography. [1, 2, 6] Turbulent blood flow within tortuous vessels can be studied with Doppler scanning. [40] Di Pino et al. studied the prevalence of CDA in the ICA using color Doppler echocardiography in 2,856 subjects (0 - 96 years), demonstrating peak prevalence at the age extremes (< 21 years and > 60 years). CDA was detected in 9.9% of participants. This study was one of the first to use ultrasonography in a large cohort. [41] Uchino et al. reported a case of CDA diagnosed by computed tomography (CT) angiography and single-photon emission computed tomography (SPECT). Both allowed for a more detailed study of the morphology of ICA tortuosity and secondary perfusion abnormalities. [42] In other studies, CT has been able to assess fine abnormalities such as microaneurysms, and 3D CT reconstructions appear to be more effective in classifying morphological variations in the ICA and detecting these abnormalities. [43 - 45] Balevi et al. found a prevalence of CDA of 40.3% in the general population using contrast-enhanced magnetic resonance angiography (MR angiography). [12] Tomiya et al. performed MR angiography in 13 patients, obtaining clear images of CDA. [46] Yu et al. are of the opinion that MRI is a good option for the diagnosis of CDA. Digital subtraction angiography (DSA), the gold

standard for the diagnosis of cervical and intracranial vascular diseases, can provide hemodynamic data of CDA and study its morphology, but it cannot determine the pathological changes in the ICA wall. [40] It would function as a complement in the study of CDA rather than a useful imaging technique for diagnosis. To reach a correct diagnosis, all these techniques are valid and not mutually exclusive, and should be chosen in order of greater complexity and invasiveness.

3.4 Clinical implication

The clinical implications of CDA are controversial. There is limited evidence to support the association between CDA and CVD. [6] Yin et al. observed a higher incidence of white matter lesions in patients with CDA compared to controls. The severity of these lesions was directly related to the number of kinks. [47] Other authors maintain that ischemic disease is more common in people with CDA and other cardiovascular risk factors. Pancera et al. found a significant association between kinks and TIA. Hypertension and CDA could be additive risk factors in the pathophysiology of TIA. [33] Oliviero et al. demonstrated that the presence of carotid kinks in hypertensive subjects cannot be considered an additional risk factor for ischemic events. [48] However, as Pancera et al. state, the addition of risk factors to the presence of CDA may increase the risk of vascular events. [33] Iwai-Takano et al. observed an association between CDA and aging, hypertension, and gender, but not with dyslipidemia, diabetes, or smoking. [49]

Those who support the danger of CDA argue that the curves in the ICA, in addition to narrowing the artery and causing endothelial damage, can produce turbulence in blood flow that could lead to a prothrombotic state. For others, such as Balevi et al., CDAs are more of a curiosity than a true predictor of ischemic events. [12] Valvano et al. argue for the absence of an association between CDA and CVD. [50] CDA can cause cerebral hypoperfusion, leading to encephalopathy, vertigo, diplopia, TIA, and infarction. [40] The prevalence of cerebrovascular symptoms in patients with CDA varies between 15 and 23%. [51] Coiling is not considered a risk factor for ischemic events because of its weak association with symptoms, unlike kinking, which appears to be more symptomatic due to transient hypotension during sleep or sudden extreme movement of the head and neck. [40] Other observed alterations are visual impairment such as amaurosis fugax, uveitis, retinitis and macular dystrophy. [52] In severe cases, CDA may produce mass effect, leading to dysphagia, dyspnea, narrowing of the upper airway and obstructive apnea, and, more rarely, pulsatile tinnitus and mesolingual spasm. [40, 53, 54]

Regarding the relationship between CDA and atherosclerosis, in the Northern Manhattan Study (NOMAS), Khasiyev et al. studied the relationship between CDA and atherosclerosis biomarkers in 558 participants. CDA correlated with diastolic blood pressure and large aortic root diameter, but not with other measures of atherosclerosis. Determining the risk of vascular events associated with this non-atherosclerotic phenotype may aid in better risk stratification for individuals with CDA. [55]

Beigelman et al. studied the genesis of neurological complications related to CDA. Sixty patients with CDA without atherosclerosis underwent head rotation testing and color Doppler imaging of the ICA and ophthalmic arteries, and hemodynamics were assessed in the latter. The results suggested that CDA is not a cause of neurological disturbances or symptoms, since no events were recorded during the study, and a significant reduction in ophthalmic artery velocities was observed in only 3 of 60 cases. [56]

Wang et al. analyzed pressure variations caused by CDA in 12 patients using DSA. They measured the kinking angle using rotational angiography and calculated blood pressure at the proximal, intrakinking, and distal sites of the ICA using a microcatheter. These results were compared with two simulations: a numerical simulation, using a geometric model of a tortuous ICA constructed using software; and an in vitro flow simulation, using a silicone tube subjected to different degrees of torsion, through which water flowed driven by a peristaltic pump. The software simulation showed a linear drop

in flow pressure with decreasing kinking angle. The minimum pressure drop was at a kinking angle of 180°, and the maximum at 30°. However, a reversal occurred between 30° and 20°: increasing the degree of kinking led to a smaller drop in pressure. The same was true for in vitro simulation: when the flow rate was constant, the pressure drop decreased with increasing torsion angle, but increased when the angle increased from 20° to 30°. However, these findings did not correspond to clinical observations: tests performed on patients showed a marked increase in pressure drop when the torsion angle was less than 30°; the marked kinking induced an average decrease of 15.5% in blood pressure. In clinical measurements, the development of pressure drop reversal was not observed at angles greater than 20° - 30°. This was attributed to excessive elongation and stenosis of the ICA in cases of severe torsion. And although the pressure drop changed gradually in simulations, it decreased rapidly in clinical measurements when the angle was less than 45°, but was more gradual at higher angles. This behavior could be caused by the fluid characteristics (blood is a non-Newtonian fluid) and the elastic walls of the ICA. Both factors create a "protective range" that may contribute to the autoregulation mechanism of blood flow. [57] This work was one of the first to evaluate these aspects of CDA by comparing clinical and artificial observations with direct measurements of ICA pressures. These findings show that alterations in blood pressure and blood flow may be responsible for the interruption of cerebral perfusion, resulting ischemia, and, therefore, cerebrovascular events.

Some authors observed a relationship between CDA and aneurysms in the context of CTD. [26] The positive predictive value of the combination of aortic aneurysm and CDA associated with CTD was 95.4%, with a specificity of 98.6%. A higher TI of the aorta and vertebral arteries was an independent predictor of arterial dissection in patients with Marfan syndrome. Thus, CDAs allow us to suspect the presence of aneurysms of great clinical significance in patients with CTD. [6, 26, 40]

CDAs have been associated with increased morbidity and mortality in head and neck surgical procedures. This risk was already recognized in the works of Weibel and Fields [8] and Metz [9]. Nayak et al. emphasize the importance of studying the morphology of the carotid vessels before performing surgical procedures in the pharyngeal area, as they can be complicated by vessel injury, bleeding, and, in severe cases, death due to massive hemorrhage. [58] Therefore, their exhaustive study is imperative before performing any procedure in the cervical region.

3.5 Treatment

Despite the controversy surrounding the clinical implications of CDA, some therapeutic indications have been developed for treating them based on the degree of hemodynamic compromise of the tortuosity, among other parameters. Gavrilenko et al. [59] proposed treating CDA in the following conditions:

(1) ICA stenosis of 60% or more, with atherosclerotic plaques and any degree of cerebrovascular insufficiency; or,

(2) ICA stenosis of less than 60% with atherosclerotic plaques, moderate to severe cerebrovascular insufficiency, combined with tortuosity, linear blood flow of 100 cm/s or more, and turbulent blood flow.

Grego et al. [60] proposed specific cases in which treatment is justified, such as:

a. TIA (hemispheric symptoms);

b. Asymptomatic patients with a kinking angle less than 30°, with contralateral carotid occlusion;

c. Patients with non-hemispheric symptoms, after ruling out other possible neurological or non-neurological causes with positive results from the following studies: 1) Doppler ultrasound of neck vessels with increased velocities, 2) CT and cerebral MRI with ischemic lesions in the ipsilateral hemisphere, and 3) reversal of circulatory flow in the anterior cerebral artery and its reduction in the middle cerebral artery, in association with head rotation and flexion-extension maneuvers.

Other authors have proposed initiating treatment in cases with an ICA/common carotid artery ratio greater than 2 or blood flow velocity greater than 180 cm/sec [61, 62]. However, the actual indication for treatment of CDA remains controversial. Most agree on intervention in cases of symptomatic CDA after ruling out any other cause.

Invasive techniques have been proposed to correct CDA, with successful results (fixation of the ICA to the digastric muscle, end-to-end anastomosis, end-to-side reimplantation, carotid patch endarterectomy, carotid bypass and angioplasty, and stenting). [40] However, despite the success of these techniques, the appropriate treatment remains controversial. Further studies are needed to determine the real effectiveness of the surgical approach to CDA.

4. Conclusion

CDA is a common entity with a multifactorial etiology and limited, but not insignificant, evidence of cerebrovascular involvement. Further studies are needed to establish the usefulness of therapeutic strategies to improve its prognosis. Strict monitoring and closer follow-up of patients, with a comprehensive assessment of thromboembolic risk, are prudent measures to prevent future events.

Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

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