Position Statement

Practice guidelines of vascular Doppler Echo 2022 of the Argentine Federation of Cardiology. FAC Peripheral Vascular Disease and Stroke Committee. Upgrade 2025 focalized on atherosclerotic plaque and dolichoarteriopathies.

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ABSTRACT

Vascular Doppler ultrasound is now considered worldwide as the first-line non-invasive technique for evaluating all vascular territories of the body, with the advantages of extremely high sensitivity and specificity, as well as positive and negative predictive values. Currently, vascular ultrasound is a subspecialty of significant importance due to its contribution to clinical practice in diagnosis, decision-making, and therapeutic guidance.

This update aims to expand the characterization of atherosclerotic plaques and include the evaluation of a quite common finding: tortuosity, kinking, and coiling.

INTRODUCTION

Doppler vascular ultrasound is now considered the first choice noninvasive technique throughout the world, for the evaluation of all vascular territories of the body, with the advantages of being highly sensitive and specific, as well as its positive and negative predictive values. Currently, vascular echography is a very important subspecialty due to its contribution to clinics for diagnosis, decision-making and therapeutic guidance.

It is operator-dependent. Being an "observer of flow and image" is key for a good vascular echography specialist, because flows and images "speak", and provide invaluable information that enables a great diagnostic approach.

To study all vascular territories there are other excellent techniques, such as multislice CT, magnetic resonance angiography, PET scan, capillaroscopy, conventional angiography, etc. The Committee on Peripheral Vascular Diseases and Strokes of FAC presents this update of the Practical Guideline for Doppler Vascular Echocardiograms, published in 2022, to turn it into another tool for cardiologists that have been or are being trained in this exciting and constantly evolving subspecialty, offering practical considerations on the principles and practice of vascular echocardiography. It was centered on the characterization of carotid plaque and dolichoarteriopathies.

Dr. Adrián H. D'Ovidio

CHARACTERIZATION OF CAROTID PLAQUES

This update is addressed to enhancing the characterization of atherosclerotic plaques and adding the evaluation of a very frequent finding such as tortuosities, kinks and coils. The rest of what is written in the 2022 guidelines has not changed, so we suggest to keep taking them into account always.

By making a study on Doppler Echocardiogram of Neck Vessels, it has been suggested already in the guidelines published by the Committee on Peripheral Vascular Diseases and Stroke, and also published in the Journal of the Sociedad de Imágenes *(Imaging Society)* of the Sociedad Interamericana de Cardiología *(Inter-American Society of Cardiology)*, to use five steps to organize the evaluation of patients who undergo the study and to prevent key omissions.[1,2]

The 5 steps are:

1. Presence and number
2. Location and extension
3. Characterization
4. Hemodynamic significance
5. Conclusions.

The first approach is through direct view with 2-dimensional images. Intima-media is characterized by a double

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line with defined intima-lumen and media-adventitia interfaces. EIM is the distance between two acoustic interfaces. Atheromatous carotid plaque (CP) is defined as a focal structure extending at least 0.5 mm toward arterial lumen and/or measuring more than 50% of the surrounding EIM and/or presenting an EIM>1.5 mm.[3,4,5,6,7,8,9,10,11]

To characterize an atherosclerotic plaque, several classifications have been described and used over the years, based on the different characteristics of plaques.

Characterizing is knowing clearly which are the characteristics of atherosclerotic plaques that make them more or less dangerous, with risk of stroke, and also being able to evaluate its evolution over time.

As a fundamental concept, we should always take into account that atherosclerotic plaques are a "living" element, not an accumulation of fat that developed within a vessel, but a richly vascularized structure, that changes significantly over time, that can become more stable or unstable with the presence or absence of optimal medical treatment (lifestyle and medications).

CHARACTERIZATION

- ***Echogenicity:*** defined comparing the plaque's echogenicity with those of adjacent structures (blood, muscle, vessel adventitia and bone), and classified into:
  - *Hyperechoic or echolucent:* darker, that is to say, of echogenicity similar to blood and less echogenic than the sternocleidomastoid muscle. Intraplaque echolucent areas correspond mainly to bleeding by neovascularization; the latter is the key culprit for plaque instability.
  - *Isoechoic:* echogenicity close to that of the muscle.
  - *Hyperechoic:* lighter than adjacent muscle.
  - *Calcified:* very echogenic, creating acoustic shadow due to calcium deposits. Echogenicity is comparable to that of bone.
- ***Echotexture:*** plaque texture is classified into homogeneous or heterogeneous.
  - *Homogeneous:* uniform in low and high echoic levels.
  - *Heterogeneous:* a mixture of high, medium and low echoic levels.
- ***Surface:*** it is classified into three classes, taking into account the surface toward the vessel lumen into:
  - *Smooth:* irregularities of less than 0.4 mm of depth.
  - *Irregular:* from 0.4 to 2 mm of depth.
  - *Ulcerated:* crater greater than 2 mm of depth and 2 mm of width.
- Area, volume, length and height of plaque.
- Evidence of embolic activity.
- Changes in plaque appearance over time.
- Plaque mobility.
- Color flow and pulsed-wave Doppler (laminar, turbulent, normal and pathological wave morphology).

It is known that the mechanisms by which an atherosclerotic plaque is more "dangerous" in relation to plaque instability are its size (plaque height greater or lesser than 3 mm and plaque area greater or lesser than 0.39 cm2), its volume, the presence of large lipid core, thin fibrous layer (less than 200 μm), the presence of neovascularization, related to intraplaque bleeding, the presence of ruptures, ulcerations, thrombosis, etc.

All these characteristics entail the key concept of "plaque vulnerability", that is related to its characterization and not with hemodynamic significance.

The known mechanisms generating carotid plaque instability are:

- Inflammation
- Oxidative stress
- Endothelial dysfunction
- Intraplaque bleeding (related to neovascularization)
- Lipid metabolism
- Vascular remodeling
- Calcification
- Plaque rupture and ulceration.

The fibrous layer, the thinner it is, the more chances it presents to undergo erosions or just break, generating brain embolisms, ulcerations or plaque thrombosis. It is interesting to take into account that plaques in coronary arteries suffer erosions of its capsule more frequently than carotid arteries. The point of greatest danger of rupture is around 65 μm, while in the carotid plaques, the point of greatest rupture danger is around 200 μm. This is related to differences in flow velocity and shear stress, which are much greater in the carotid arteries in regard to coronary arteries. Noninvasive image modalities (ultrasound, CT, MRI) and intravascular techniques (intravascular ultrasound, optical coherence tomography and near-infrared spectroscopy) have played an essential role in characterization and detection of unstable carotid plaques. Structural phenotypes

TABLE 1.
Classical classification (Grey-Weale)[6]

Type	Description
1	Predominantly echolucent with thin fibrous layer
2	Substantially echolucent plaque with small areas of increased echogenicity
3	Predominantly echolucent plaque, with one or more echolucent areas (<25%)
4	Uniformly echogenic

TABLE 2.
European consensus 1997

Type	Description
1	Uniformly echolucent, no need for an echogenic cap to be present
2	Predominantly echolucent and <50% of echogenic material
3	Predominantly echogenic plaque, and <50% of echolucent material
4	Uniformly echogenic
5	Not classifiable (extensive calcification)

TABLE 3.
Classifications of Stary et al. 1995[14] and Cai et al. 2002[15]

Conventional classification (Stary et al. 1995) [14]		AHA MRI (Cai et al. 2002) [15]	
Type I	Initial lesion with foam cells	**Type I-II**	Almost normal myointimal thickness, with no calcification
Type II	Fatty streak	**Type III**	Diffuse myointimal thickening or small eccentric plaque with no calcification
Type III	Preatheroma (intermediate lesion) with extracellular lipid pool	**Type IV-V**	Plaque with necrotic lipid core, atheroma with echolucent lipid core, surrounded by fibrous tissue, with possible calcification
Type IV	Atheroma with echolucent lipid core	**Type VI**	Complex plaque with possible surface defect, with bleeding or thrombus
Type V	Fibroatheroma	**Type VII**	Calcified plaque
Type VI	Complicated plaque (possible surface lesion, hemorrhage or thrombus)	**Type VIII**	Fibrotic plaque with no lipid core, with possible small calcifications
Type VII	Calcified plaque		
Type VIII	Fibrotic plaque with no lipid core		

TABLE 4.
Classification of Johry et al.

Grade 0: absence of plaques, myointimal thickness <1.5 mm
Grade I: protruding (focal thickening of vessel wall, myointimal thickness <1.5 mm
Grade II: protruding or diffuse (increased myointimal thickness ≥1.5 mm), plaque thickness 1.5 to 2.4 mm.
Grade III: protruding or diffuse (≥2.5 mm)

TABLE 5.
Plaque-RADS classification (Saba et al. 2024)[6]

RADS-Score 1	Absence of plaques. Normal study.
RADS-Score 2 **Plaque height ≤ 3 mm**	Myointimal thickening Necrotic core, rich in lipids Calcifications
RADS-Score 3a **Plaque height ≥ 3 mm**	Thick fibrous layer + necrotic core, rich in lipids
RADS-Score 3b **Plaque height ≥ 3 mm**	Thin fibrous layer + necrotic core, rich in lipids
RADS-Score 3c **Plaque height ≥ 3 mm**	Ulcerated plaque
RADS-Score 4a	Intraplaque bleeding
RADS-Score 4b	Plaque rupture
RADS-Score 4c	Endoluminal thrombosis
Complementary characteristics	Calcifications Neovascularization Inflammation Positive remodeling Plaque load Stenosis progression
Modifiers	Calcifications Stents Endarterectomy

of carotid plaque, including its location and amount, affect plaque biomechanics, contributing to the risk of rupture.[13,14,15,16,17]

Here, we should remember that when we discuss hemodynamic significance, it means the capacity of vessel diameter reduction that this plaque generates to reduce tissue perfusion, being applied to any territory of the organism, and in the specific case of carotid and vertebral plaques, the risk of ischemic stroke.[1,12,13]

"All hemodynamically significant plaques are vulnerable, but not all vulnerable plaques are hemodynamically significant."[1]

Within classifications, we have classical ones, like the one by Gray et al., about echogenicity of plaques, graded from I through IV, with hypoechoic plaques being the most dangerous and related to stroke.

There are different forms described in literature to classify carotid plaques based on plaque echogenicity, its homogeneity, myointimal thickness, and whether they protrude or not: Gray-Whale's classification *(Table 1),* the description of atherosclerotic plaques according to European consensuses *(Table 2),* and the AHA classification *(Table 3)*.[6,9,14,15]

Later, within the framework of the Guidelines of the American Society of Echocardiography (ASE), Johry et al. started highlighting the height of the plaque (more or less than 2.5 mm of height) and its rupture, significantly contributing, along with classical classifications, to identify plaques with greater potential for rupture or embolization (plaque vulnerability) *(Table 4)*.[16]

In February 2024, Saba et al. published the RADS-Score classification for atherosclerotic plaques, based on echocardiography tests, MRI and anatomical pathology, which is very important to know, and that his Committee strongly recommends for use, making a position statement, as it is practical and validated. It does not invalidate, naturally, those previously described, which remain useful, but they are included in this new classification *(Table 5) (Figures 1 through 9)*.[18]

The risk of stroke of type 3 plaques is moderate. The risk of stroke of type 4 plaques of the RADS classification is high.

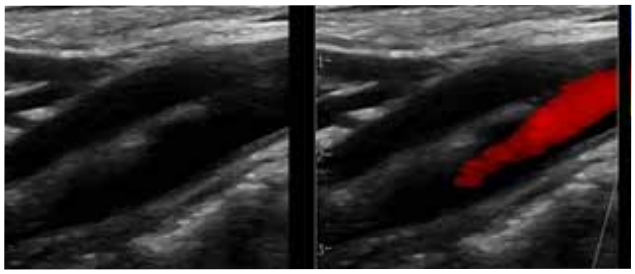

FIGURE 1.
Rads-Score 1, normal study. Absence of atherosclerotic plaques

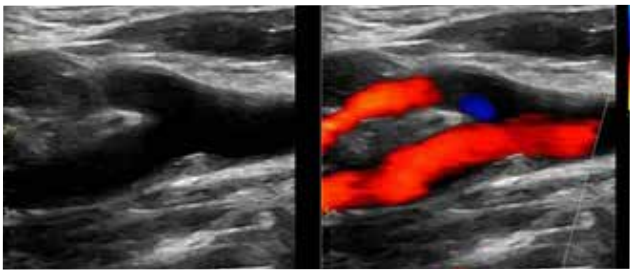

FIGURE 2.
Rads-score 2, myointimal thickening, necrotic core, rich in lipids, calcification

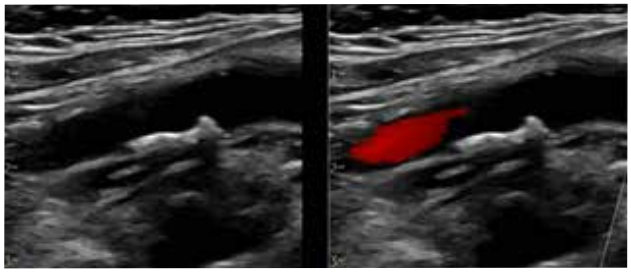

FIGURE 3.
Rads-score 2, myointimal thickening, necrotic core, rich in lipids, calcification

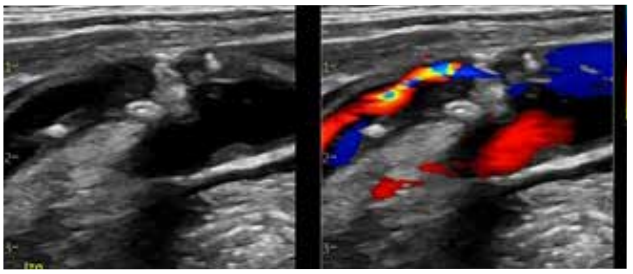

FIGURE 4.
Rads-score 3a (plaque height ≥3 mm). Thick fibrous layer + necrotic core, rich in lipids

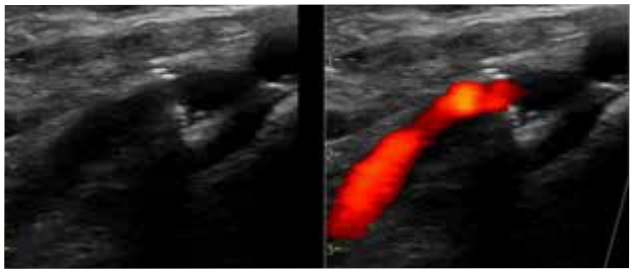

FIGURE 5.
Rads-score 3b (plaque height ≥3 mm). Thin fibrous layer + necrotic core, rich in lipids

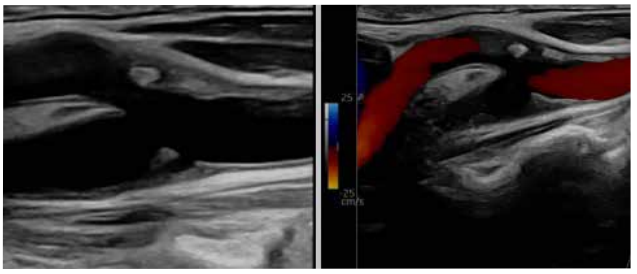

FIGURE 6.
Rads-score 3c (plaque height ≥3 mm), ulcerated plaque

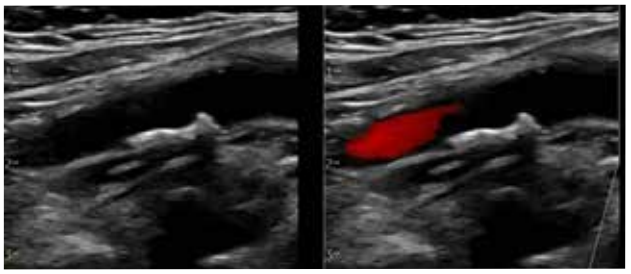

FIGURE 7.
Rads-score 4a, regardless of height. Intraplaque bleeding

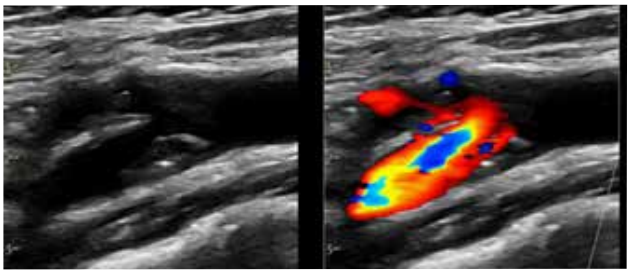

FIGURE 8.
Rads-score 4b, regardless of height. Ruptured fibrous layer

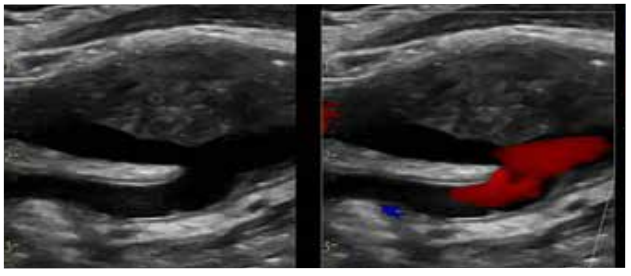

FIGURE 9
Rads-score 4c, regardless of height. Endoluminal thrombosis

TABLE 6.
Classification of Togay

Tortuosity	Elongation or undulation in S or C in ICA trajectory
Mild kinking	ICA kinking with angle ≥ 60°
Moderate kinking	ICA kinking with angle 30-60°
Severe kinking	ICA kinking with angle < 30°
Coiling	Elongation or redundancy of ICA, leading to circular configuration (360°).

This classification has (and this should be included in reports) supplementary characteristics *(Figures 10 through 12):*

- Calcifications
- Neovascularization
- Inflammation
- Positive remodeling
- Plaque load
- Stenosis progression
- "Limited" diagnostic study
- Stents
- Endarterectomy.

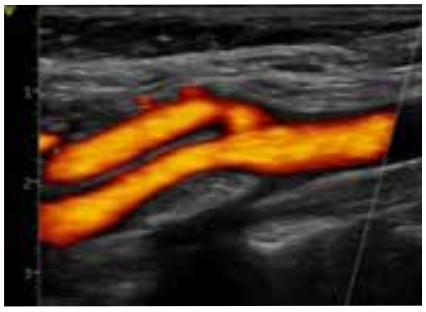

FIGURE 10.
Calcifications

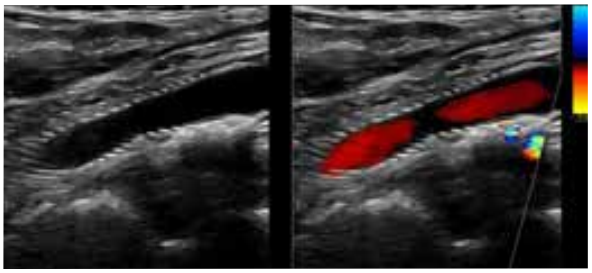

FIGURE 11.
Stent

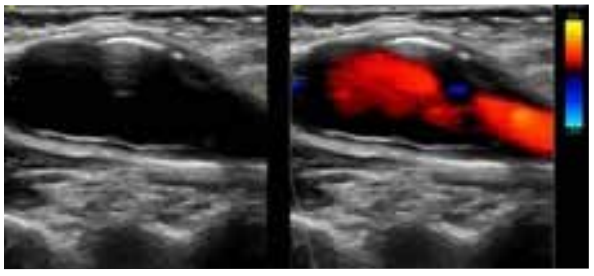

FIGURE 12.
Endarterectomy

This last classification is practical, guiding, it includes the other classifications and gives them value again, it is validated and it allows, with an adequate characterization of plaques, to detect those that present greater risk of stroke for patients. It is a therapy guide, with prognostic value, and for follow-up.[18,19,20]

CONCLUSIONS

This update of our Guidelines of Practice for Doppler Vascular Echocardiography of the Argentine Federation of Cardiology, focused on the characterization of the carotid plaque, were motivated by the publication of the new Plaque-RADS classification, comprehending all previous classification, and it is practical for daily use, it has a message, it estimates the vulnerability of the plaque, and it allows for a uniform language, it is validated, so a routine use is suggested.

CAROTID DOLICHOARTERIOPATHIES

Introduction

The morphological variations in the trajectory and geometry of carotid vessels are called dolichoarteriopathies (DAP). In literature, there are different synonyms to describe tortuosities, kinks and coilings, such as elongations, undulations, loops, spirals or winding.

The cervical portion of the internal carotid artery has two fixation points, specifically in its bifurcation and at the base of the skull (fibrous ring of the temporal bone). The dolichoarteriopathies of the internal carotid artery may occur if the vessel size is larger than the distance between these two points.[21]

This entity was initially described in the 18th century, in autopsy specimens, until it was related to bleeding complications during otorhinolaryngological surgeries, and in the mid-20th century, it was related to cerebral ischemic events. Since then, the knowledge on this pathology has enhanced, and it has been found to be not rare, but in most cases to be asymptomatic.[22]

The common carotid artery (CCA) and the initial portion of the internal carotid artery (ICA) emerge from the third aortic arch. The distal portion of the ICA emerges from the cranial portion of the left dorsal aorta, and these structures make up a "loop". All these process occur in the embryonic pharynx. In the fifth month of intrauterine life, the carotid arteries are normally bended, and when the fetal neck takes shape, the heart descends into the mediastinum,

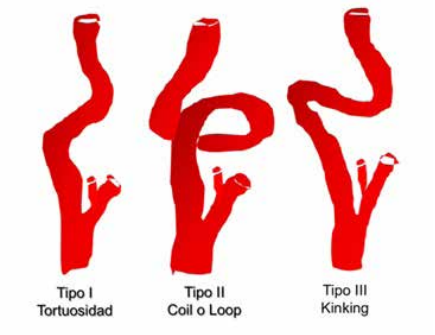

FIGURE 13.
Classification of Weibels and Fields

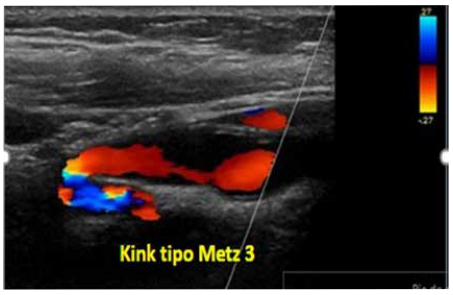

FIGURE 14.
Kink of the Metz 3 type

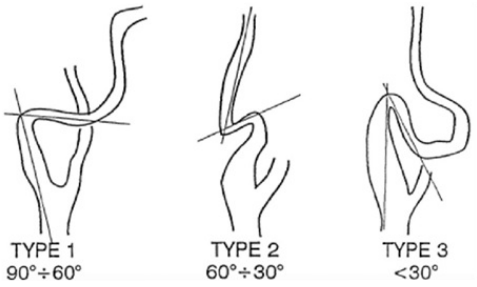

FIGURE 15.
Metz classification

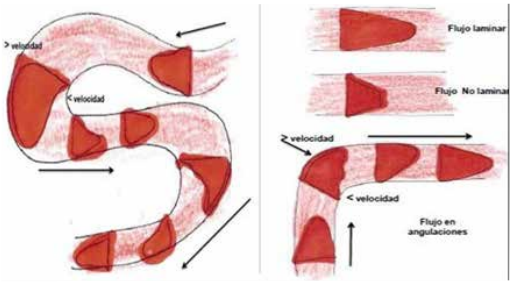

FIGURE 16.
Kink hemodynamics

and the supra-aortic vascular structures stretch and become straight. The alterations in cardiac descent or vascular elongation would explain the origin of dolichoarteriopathies (persistence of embryonic anatomy). The proximal portion of the internal carotid artery is drawn from the third aortic arch, and the rest of the artery, from the left dorsal aorta.[23]

The prevalence of the general population is 1.3%, in patients with history of TIA it is 20%, and in the patients with arteriographies made due to severe neurological symptoms, the finding of carotid anomalies ranges between 4 and 58%, according to other authors, and up to 30% of cases with Doppler echocardiogram.[24,25,26]

DAPs are more prevalent in women and elderly patients, particularly in those older than 60 years, and could be both uni and bilateral, affecting the common carotid (CCA), internal (ICA) and external carotid arteries (ECA), but most frequently ICA, particularly from the left side.[27,28] Tortuosities and kinks are more frequent than coiling, and the most affected artery is the ICA, then the CCA, the ECA, and finally the vertebral one.

CLASSIFICATION

Weibel and Fields *(Figure 13)*[29]

Type 1: tortuosity: it is defined as non-rectilinear trajectory of an artery with angles of more than 90°.

Type 2: loop: angulation of an artery in 360° over its transversal axis (circular configuration).

Type 3: kinking: by definition, it is the inflection of two or more segments of one artery with internal angle equal or less than 90° *(Figure 14).*

Metz et al. classified kinking into three grades of severity, depending on the vessel angulation over its axis: grade 1, curvature over its axis between 90° and 60°; grade 2, between 60° and 30°; and grade 3, less than 30° *(Figure 15).*[30]

In year 2025, Togay et al. published a classification with the modified criteria proposed by Weibel-Felds and Metz, based on exploration by Duplex color ultrasound *(Table 7).*[31]

ETIOLOGY

Embryology theory

It is based on these anatomical anomalies being observed by different methods of diagnosis in fetuses, children and teenagers.[31,32,33,34] Due to persistence of the embryological status of ICA (with kink since intrauterine life) by failed absorption in the third aortic arch, or due to asynchronous development of the carotid arteries and the skeletal system in the process of growth, the arteries would adopt a winding trajectory.[23,24] In a study made in Argentina with tests made with Doppler echocardiogram, almost the same prevalence of DAP was observed in children and adults.[26]

Acquired DAP theory

In relation to his, hypertension would produce alterations in the wall, favoring its weakening, with the subsequent kinking of the artery, while other authors proposed the importance of the relationship between arterial ageing and degenerative alterations in artery walls, as elasticity loss along with action by hemodynamic forces would lead to an increase in preexisting elongation or angulation.[35,36,37,38,39] Also, an association between atheromatosis

and carotid kinks has been described, or with anatomopathological patterns with arterial media thickening by cystic necrosis or hyperplasia, elastic fragmentation and fibrosis; in some cases with histological characteristics of classical abdominal aortic aneurysm (AAA) (thinning of tunica media underlying the atherosclerotic plaque), suggesting a common base of connective tissue alterations underlying both pathologies.[40,41]

There is a hypothesis that tissue inflammation of aponeurosis in the area surrounding the carotid artery would produce artery retraction.[42] Others claim that DAP could be due to kyphosis and lordosis that would deviate the carotid axis.[24] La Barbera proposed a hypothesis by which extracranial ICA is considered a segment of transition between an elastic vessel (CCA) and a muscular one (intracranial ICA), so the elastic and muscular tissues of the ICA in patients with DAP is substituted by lax connective tissue, configuring a metaplasia of the tunica media.[43]

Although still controversial, most authors propose that the main cause is the acquired one, associated with atheromatosis, an advanced age and hypertension.

The pathophysiology of DAP is controversial, and the association with traditional risk factors is not clear, since although correlated, they do not depend on the latter.[44]

In general, it is considered a multifactorial disease, with a genetic predisposition, associated to cardiovascular risk factors, causing arterial wall thickening and significant hemodynamic disorder, which may change blood flow within a vessel and entail an infrequent cause for stroke.[45]

Kinking is a very important cause, as it is not rare, and along with dissections, it represents the most likely cause for cryptogenic stroke.[46,47]

PATHOPHYSIOLOGY

Hemodynamics of kinks

Tortuous vessels can have an effect over velocities.

A flow that runs through a non-rectilinear circuit, such as in DAP, experiences both centrifugal and viscous forces on the vessel walls, and the result of these forces is a secondary flow in the shape of 2 helicoid vortices (turbulence).

In the case of parabolic flow, the flow in the center of the vessel experiences the highest velocity, and thus develops the greatest strength. These vortices will cause the high-velocity flow to move toward the far wall of the vessel. If the velocities in curvatures exceed 170 cm/sec, we should think that besides the curvature and its turbulence, the presence of significant atheroma could be suspected *(Figure 16).*

Atherosclerotic/thrombotic mechanism

There would be an atherosclerotic or thromboembolic mechanism, as endothelial lesions have been described, mainly microulcerations secondary to flow alterations in the curvature sites, as well as the concomitant presence of atheroma plaque with formation of platelet thrombi and distal embolization. The last point is crucial in the prognosis and management of DAP.[48] HTN and age would play a key role with an increase of endoluminal pressure and parietal pressure.

Hemodynamic mechanism

The other pathophysiological mechanism proposed is the hemodynamic one, by which there is an inverse relationship between the kink angle and flow reduction; the more acute, the more severe.

When the kink is very marked (internal angle <90°), neurological ischemic symptoms may occur if there is blood flow reduction because of kink.

It may be triggered with changes in the head and neck, by what is called dynamic obstruction. For example, in supine position or when getting up from bed, and even it has been proven arteriographically and with oculoplethysmography, that rotation maneuvers in the neck could reduce carotid flow.

The hemodynamic mechanism becomes more important the greater the kink. There are experimental studies showing that blood flow can be reduced to less than 40% with 60° kink in ICA, and 60% with 30° kink. The tests made in patients showed a marked increase in pressure drop when the torsion angle was less than 30°. The marked kink induced an average decrease of 15.5% in blood pressure.[49]

Diagnosis and clinical implications

Most DAPs are asymptomatic, but they could be suspected by auscultation of carotid murmurs or the presence of hyperpulsatility in the neck, palpable masses, dysphagia, dysphonia or feeling of cervical foreign element.

DAPs may cause hypoperfusion or cerebral ischemia with hemispheric syndromes (TIA, stroke, amaurosis) leading to encephalopathy, vertigo, diplopy, TIA and infarction.[48] The prevalence of cerebrovascular symptoms in patients with DAP ranges between 15 and 23%.[50] They can be exacerbated with movements of the head or neck turning or stretching. Reproduction or demonstration with Doppler echocardiogram of neck vessels, transcranial Doppler ultrasound or angiography is a clear indication of surgery.

The addition of risk factors to the presence of DAP may increase the risk of vascular events.[38] Iwai-Takano et al. observed an association between DAP and ageing, HTN and gender, but not with dyslipidemia, diabetes or smoking.[51]

Its clinical course seems to be benign, but on occasions is associated to cerebral ischemic phenomena, in isolation or when associated to atherosclerotic lesions of carotid bifurcation, so a better knowledge on its natural evolution would allow improving treatment indications.

In general, it is considered a multifactorial disease, with a genetic predisposition associated with cardiovascular risk factors, which leads to arterial wall thickening, and significant hemodynamic disorder, which may change blood flow within a vessel and entail an infrequent cause of cerebral vascular event.

These anomalous configurations, according to the degree of severity, may significantly reduce blood supply to

the brain, which may overlap to conditions like atherosclerosis, hypertension and ageing, which would produce significant hemodynamic symptoms and alterations.[52,53]

When there is clinical suspicion that neurological deficit could be the consequence of a morphological alteration in the carotid artery, it is essential to rule out the rest of causes for cerebral pathologies (arrhythmia, orthostatic hypotension, epilepsy, labyrinthine alterations, etc.), mainly if symptoms are not focal. A kink with no atherosclerotic plaque (frequent entity) is a very rare cause of stroke.

In adult patients with cerebrovascular ischemia with symptoms, internal carotid kinks are associated frequently with atheromatosis of the carotid bifurcation. When these two lesions are combined in symptomatic patients, surgical treatment is indicated, during which endarterectomy will be carried out in the atheromatous plaque, and kink correction will be conducted.[54]

Also, kinks without severe plaque but symptomatic by dynamic obstruction have indication of surgery. Surgical techniques consist of resection and reanastomosis of tortuous ICA, or use of patch, reimplantation of ICA, grafts with GSV or prosthesis, or artery pexy to the muscle.

To this date, there are few studies assessing the results of endovascular treatment in carotid kinking, and the recommendations are limited to patients in very high surgical risk and in post-endarterectomy restenosis.[55]

Elongations are relatively frequent in children. It is advised to rule out this type of pathology when neurological location appears, although there is no study recommending surgical intervention in asymptomatic children.[56]

IMAGING DIAGNOSIS

Doppler ultrasonography

To study DAPS, the gold standard seems to be ultrasonography.[57,58] It is possible to observe focal parietal thickening, and turbulent flow with velocity increase.[59]

Besides, it is useful to evaluate significant concomitant atheromas.

Erroneous diagnoses may arise (carotid artery pseudo-occlusion or "interruption" of its flow) during neck vessel Doppler echocardiogram, as often the vessel does not appear in a single plane. The transducer should be manipulated in different positions and change the steer constantly to observe the vessel trajectory. Occasionally, vessel ascent may appear as not rectilinear in the short axis.

In the echographic presence of DAP, mainly kinking, it is advisable to determine the kinking, based on the mentioned classifications. It is essential to detect the presence of atheromas within the kink, and in case of observing them, velocities should be measured before the kink, within the kink, and in the kink outlet. Togay-Isikay observed in a study on 345 echocardiograms on neck vessels that peak systolic velocity (PSV) is greater in the kink than in the coils and tortuosities, being greater in the kinks, at the origin of ICA than in the kink outlet.[31] Likewise, they stated that the relatively high PSV, the angle correction of the sample while the study was made at kink level, it is often not optimal and produces an overestimation of flow velocities.

With Doppler echocardiogram, continuous changes can be observed in the direction of the blood column and in its velocities. PSV in the kink greater than 170 cm/sec support the diagnosis of hemodynamic compromise secondary to ICA kink.

In the presence of coils, it is possible to observe a sign similar to the Yin-Yang of aneurysms.

Other methods

Digital subtraction angiography (DSA), CT angiography, SPECT and MRI

Digital subtraction angiography (DSA) provides hemodynamic data of DAP and their morphology, but cannot determine the pathological changes at the level of the ICA wall.[50] CT angiography and SPECT allow studying ICA tortuosity morphology with greater definition and alterations secondary to perfusion, and evaluating subtle anomalies, such as microaneurysms. CT 3D reconstructions seem more efficient at the time of classifying the morphological variations of ICA and detecting these anomalies.[60,61,62,63] MRI is a good choice for DAP diagnosis.[64]

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