State of the art CT and MR imaging and assessment of carotid artery disease: the reporting - a consensus document by the European Society of Cardiovascular Radiology (ESCR)

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List of Abbreviations

^{18}F: 18- fluorodeoxyglucose
AHA: American Heart Association
CCA: common carotid artery
CMPR: curved multi planar reformations
CT: computed tomography
CTA: computed tomography angiography
CUBE:
DIR/QIR:
EI: eccentrity index
FC: fibrous cap
FSD: flow-sensitive dephasing
ICA: internal carotid artery

IDR: iodine delivery rate
IPH: intraplaque hemorrhage
LPNC: lipid-rich necrotic core
MIP: maximum intensity projection
MR: magnetic resonance
MRA: magnetic resonance angiography
MSDE: motion-sensitized driven-equilibrium
PD: proton density
PET: positon emission tomography
PFD: perivascular fat density
SNR: signal-to-noise ratio
SPACE: sampling perfection with application optimized contrasts using different flip angle evolutions
T1W: T1-weighted
TOF: time of flight
US: ultrasound
USPIO: ultrasmall superparamagnetic iron oxide
VR: volume rendering
SCA: superior cerebellar artery
AICA: anterior inferior cerebellar artery
PICA: posterior inferior cerebellar artery

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Abstract

The European Society of Cardiovascular Radiology (ESCR) is the European specialist society of cardiac and vascular Imaging. As such this society's highest priority is the continuous improvement, development and standardization of education, training, and best medical practice, based on experience and evidence. The present intra-society consensus is based on the existing scientific evidence and on the individual experience of the members of the ESCR writing group on Carotid diseases, the members of the ESCR guidelines committee as well as of the members of the executive committee of the ESCR. The recommendations published herein reflects the evidence-based society opinion of ESCR. The purpose of this second document is to discuss suggestions for standardized reporting based on the accompanying consensus document part I.

1. Introduction and purpose of this document

In the last 20 years, several new evidence have been added on the understanding of the physiopathology of the carotid-related stroke occurrence, by introducing the concept of the carotid artery vulnerability related to the plaque's features. At the same time in the last decades a significant evolution in the imaging techniques has occurred by routinely allowing not only the degree of stenosis quantification but also the assessment of the plaque composition and the detection of the features of vulnerability.

The European Society of Cardiovascular Radiology (ESCR) is the European specialist society of cardiac and vascular Imaging. As such this society's highest priority is the

continuous improvement, development and standardization of education, training, and best medical practice, based on experience and evidence. The present intra-society consensus is based on the existing scientific evidence and on the individual experience of the members of the ESCR writing group on Carotid diseases, the members of the ESCR guidelines committee as well as of the members of the executive committee of the ESCR. The recommendations published herein reflects the evidence-based society opinion of ESCR. The purpose of this second document is to discuss suggestions for standardized reporting based on the accompanying consensus document part I.

2. Standardized reporting

ESCR Consensus Statement

In reporting carotid artery atherosclerotic disease, assessment of stenosis severity should be complemented with information regarding plaque morphology and plaque composition. Some imaging biomarkers, described in the accompanying part I document, which are not yet validated by official guidelines should be reported and interpreted with caution.

In this section we will explain how and what information should be included in the report for the assessment of carotid artery pathology. We classified the information as [**Mandatory**] and [***Optional***]. Some information is modality-specific (e.g. only MR). For broad application, this document does not include advanced approaches that are not considered standard or proprietary technologies.

In general terms, a carotid imaging CT/MR report has three main components: 1) description of presence and degree of stenosis 2) description of the different plaque components and 3) description of vessel characteristics.

Table 1 provides a proposal for structured reporting applicable for both CT- and MR-examinations. A glossary of commonly used terms is also added as **supplemental material**.

2.1. Degree of stenosis [**Mandatory, CT/MR**]

As previously stated, we recommend the NASCET method to quantify ICA stenosis. The narrowest luminal diameter at the level of the stenosis must be used for calculation of stenosis severity. Measurements should be performed in a plane perpendicular to the vessel long-axis. As such, this implies construction of a CMPR. Standard, axial, sagittal or coronal planes are not applicable. Also, the used reference diameter distal from the stenosis should be a patent vessel segment.

The fact that the NASCET method uses the normal distal ICA for calculation of the stenosis percentage has some practical implications that one should be aware of. First, caution should be taken in cases of near-occlusion with post-stenotic underfilling [1,2]. Therefore, the NASCET collaborators recommend assessing near-occlusion first, as in such cases a collapsed/underfilled distal ICA used for percentage calculation will result in an underrated stenosis severity (**Figure 1**). As such, it is an error to report a near-occlusion as 99% stenosis as in this specific situation the distal portion of the ICA may not be used as a denominator for the quantification of the degree of stenosis [1,2].

Also, merely reporting of the degree of luminal stenosis can result in underestimation of disease as the luminal diameter can be maintained through positive remodeling of the vessel wall with outward growth rather than inward growth but without luminal compromise. This is in the NASCET method further illustrated by the anatomy of the carotid artery at the level of the bulb, as the normal outward bulge of the lumen at the carotid bulb may be lost due to the presence of disease but still not cause any measured stenosis given the location of the reference diameter (**Figure 2**). Consequently, the presence of positive/negative remodeling should always be reported as well.

Finally, special care should be applied on the identification of a possible tandem stenosis, defined as the simultaneous presence of a significant carotid bifurcation stenosis with a ≥50% identifiable stenosis in any downstream distal cerebral artery.

2.2. Plaque components & morphology

An overview of the different plaque components, their clinical significance and preferred imaging modality is given in **Table 2**.

2.2.1. Type of Plaque [**Mandatory**, CT]

The fundamental CT analysis of the type of the plaque is based on the attenuation value. According to the HU attenuation values it should be described as low-attenuated (< 60 HU), mixed (between 60 HU and 130 HU) or calcified (> 130 HU) plaques (**Figure 3**).

2.2.2. Plaque composition: Calcification [**Mandatory, CT**].

The presence, and morphology of calcification should be described. The severity should be visually assessed and broadly described as absent, mild, moderate, severe (grade 0, 1, 2 or 3). The presence of a positive "rim sign" (**Figure 4**), defined as the presence of an adventitial calcification (< 2-mm thick) with internal soft plaque (≥ 2-mm thickness), should be reported because of its potential association with IPH.
Dual energy CT with post-processing using 80-100 keV provides the most accurate estimates of calcification size, as compared to histology[3] [**Optional**].

2.2.3. Plaque composition: IPH [**Mandatory, MR**]

With MR, the identification of IPH is possible using different approaches. The simplest approach is the detection of focal regions of T1 hyperintensity within the carotid plaque that is 1.5x greater than the adjacent sternocleidomastoid muscle, attributable to the strong T1-shortening effect of methemoglobin generated from erythrocyte degradation (**Figure 5**) [4]. It is also possible to use the time-of-flight (TOF) for the IPH detection [5] due to its inherent T1 contrast. Limitations include susceptibility to flow artifacts, particularly in tortuous segments, low sensitivity to IPH, and inability to differentiate juxtaluminal IPH from ulceration [6].
CTA is not yet considered a robust method to detect the presence of IPH but some authors suggest that some specific features are significantly associated with the presence of IPH in CTA: presence of the napkin sign [7]; positive rim sign [8] and presence of internal low attenuation (< 25 HU or 30 HU according to different groups) [9,10].

2.2.4. Presence of remodeling [**Mandatory, CT/MR**]

Remodeling should be classified as outward (positive), neutral, or inward (negative) [**Mandatory**]. It is also possible to add the plaque remodeling ratio (RR) determined, according to Hardie [11], by 1) measurement of the outside vessel circumference of the extracranial internal carotid artery at the point of maximal luminal stenosis, and 2) divided by the outside vessel circumference at a region unaffected by atherosclerotic disease [**Optional**]

The eccentric or concentric morphology of the plaque must be at least visually evaluated [**Mandatory**]. Alternatively, it can be expressed quantitatively with the eccentricity index (EI) of the plaque calculated as the ratio between the maximum thickness and minimum thickness at the point of maximum stenosis [12] [**Optional**]

2.2.5. Plaque ancillary findings: thrombus (floating) [**Mandatory**]

Intraluminal thrombus is an additional marker highly associated with stroke [13] that can be detected on CTA as a donut sign (**Figure 6**) [14]. Another definition is the presence of filling defect outlined by lumen contrast that is visualized on multiplanar reformats.

2.2.6. Plaque ancillary findings: Carotid Web [**Mandatory**]

A carotid web appears as a shelf-like projection into the lumen of the proximal cervical internal carotid artery without evidence of calcification (**Figure 7**) [15]. This is a proposed stroke mechanism that may underlie cryptogenic stroke, particularly in younger patients without vascular risk factors[16].

2.2.7. Plaque composition: Lipid-rich necrotic core [**Optional, MR**]

As, previously stated, CT cannot assess the presence of LRNC but can identify the presence of low-density areas that could represent LRNC. Basic assessment involves measuring attenuation density in a ROI in the plaque. More advanced assessment involves the calculation of the volume of plaque subcomponents.

MR T2 weighted imaging can be used to detect the presence of LRNC [17]. Direct assessment of LRNC can also be done in patients undergoing contrast administration using a post-contrast T1w scan. CE-MRA followed by post-CE vessel wall imaging in patients without contraindication will improve detection and quantification of the LRNC.

2.2.8. Status of the fibrous cap [**Optional, MR**]

When assessing the carotid plaque with high-resolution MRI, information on the fibrous cap status should be given by categorizing as thick (normal), thin or fissured.

2.2.9. Plaque inflammation [**Optional, CT/MR**]

This is currently not a parameter required in standard CT / MR reports.
It is possible to include information with CT, regarding the PFD measured as the attenuation value of the pericarotid fat. It should be measured as a circular / elliptic ROI obtained close to the point of maximum stenosis [**Optional**].
Ultrasmall superparamagnetic particles of iron oxide enhanced MRI has been shown to identify macrophage infiltration [18].
PET/CT or PET/MR can be used to study plaque inflammation or biological activity, along with plaque characterization in a single examination [19]. However, only 18F-FDG PET is to date routinely used in clinical practice, and when assessing small structures, the sensitivity and specificity can be low due to spatial resolution and often suboptimal lesion-to-background ratio. New tracers that are being developed are warranted to enhance the role of PET/CT and PET/MR in clinical practice.

2.2.10. Plaque Neovascularization [**Optional**]

It is possible to include information about plaque neovascularization from CT, by giving the contrast plaque enhancement (CPE) value by measuring the attenuation value of the plaque with a region of interest (ROI) on pre- and post-contrast scans. It is important to avoid

areas of calcium and beam hardening because of the high attenuation value and to exactly compare with the same ROI on the pre- and post-contrast imaging.

Using MR, the detection and quantification of neovascularization is possible using the DCE-MR (K^{trans} - volume transfer coefficient method). Alternatively, gadofosveset-enhanced MRI can be used to visualize plaque microvasculature without the need to use pharmacokinetic modeling[20].

2.2.11. Plaque burden – distribution and subcomponents [**Optional**]

Knowledge of the location and distribution of plaques can be helpful in the pre-procedural workup of a patient with carotid artery disease. In particular, plaque length measurement (longitudinal extension) and the relationship of a targeted plaque with the carotid bifurcation may provide better insights regarding the optimal surgical approach or the possibility to deploy a stent. This information can be easily obtained using CPR reconstructions along the trajectory of interest with centerline measurements, usually performed on CT but also possible with MR. Moreover, CT can calculate the volume of carotid artery plaque and determine the volume of plaque sub-components, according to attenuation density threshold.

With MR, efficient 3D large coverage black-blood MR may be better suited for this purpose. With commercially available software it is possible to obtain a fast visualization of spatial localization of the tissue components.

While this information is not currently a parameter required in standard CT / MR reports due to the lack of standardization and required post-processing, it may be implemented on a case-by-case basis. Also, it's reasonable to think that with the current ongoing evidence related to the impact of the plaque burden and the development of advanced software packages, this parameter could also be incorporated in future radiological report of the carotid arteries[21].

2.3. Vessel morphology [all sections **Mandatory**]

Most atherosclerotic plaques occur at the carotid bifurcation, involving the distal CCA, the bifurcation and extending to the proximal ICA. Nevertheless, a detailed analysis should be performed of the complete trajectory of the carotid arteries from their origin until their intracranial segments.

2.3.1. Definition of aortic arch and supra-aortic vessel **branching** anatomy

The description of the aortic arch (including the presence of calcification, aneurysm, thrombi and/or penetrating atherosclerotic ulcers (PAU)) as well as **branching** anatomy of supra-aortic vessels and the presence of subclavian artery pathology is fundamental given the potential implications for carotid artery treatment.

2.3.2. Common carotid artery **status**

Presence and quantification of the severity of plaque (> 50%) in the common carotid artery (CCA) should be reported by describing the type (calcified, low-attenuated, mixed).

2.3.3. ICA Vessel tortuosity

As previously the vessel tortuosity can be classified according to the modified criteria of Weibel-Fields and Metz [22–24], which describe the course as tortuous (elongated), kinked (mild, moderate, severe) or coiled when applicable (**see also Table 4, part I**). This abnormal morphology can be found in all segments of the common carotid artery (CCA) and ICA and should be commented on in both CT- and MR reports.

2.3.4. Carotid arteries – distal extracranial segment and intracranial segments

Description of any distal plaques should be reported in particular for the potential impact on surgical procedures. The presence of a significant distal carotid artery plaque should be defined as a “tandem” plaque [25]. While the presence of a tandem lesion infrequently alters the surgeon's decision to perform an endarterectomy, detecting tandem stenoses may have important implications for long-term medical management in symptomatic patients [25][26].

2.3.5. Vertebral arteries/ Basilar and other vessels

Description of anatomy from the subclavian origin through to the basilar artery as well as pathological stenosis. Also, the SCA-AICA-PICA should be commented in the report when absence of these vessels is noted.

2.3.6. Circle of Willis

Analysis of the circle of Willis should be included. The key information is 1) anatomy with presence of variants; 2) exclusion of aneurysms; and 3) identification of atherosclerotic (or other type e.g. inflammatory) lesions in the main arteries.

2.4. Carotid differential diagnosis

During the assessment of supra-aortic vessels, a differential diagnosis hypothesis can be formulated when typical features are detected. The definition of these multiple entities is out of the scope of this paper and only some key elements are further described here.

2.4.1. Carotid dissection

Carotid dissection is responsible for 20 % of ischemic stroke in young adults under 45 years and for about 2 % of ischemic strokes overall [27]. In MR, the detection of this condition is usually straightforward as the blood within the layers (intima and media) has a hyperintense signal visible with T1W-fat-sat sequences (**Figure 8**) [28]. In CTA, it is challenging to distinguish the presence of blood degradation products based on the mere attenuation density. Other features that should be identified are: 1) localization, as usually carotid dissection does NOT normally involve the bifurcation that is the usual location of atherosclerosis; 2) morphology of the occlusion because the occlusion (or sub-occlusion) usually shows a "flame" sign that is not typical in the atherosclerotic process. In the case of previous dissection, a pseudo-aneurysm can occur, caused by a weakened wall due to the absence of the intima. However, some pitfalls can be encountered such as flow-related enhancement in arteries and veins that can simulate intramural hematoma and special care regarding the technical acquisition is fundamental [29].

2.4.2. FMD

Fibromuscular dysplasia (FMD) is an idiopathic, non-inflammatory and non-atherosclerotic disease with a prevalence between 0.3 and 3 % in the cervico-encephalic arteries. The string-of-beads aspect is highly suggestive of FMD (**Figure 9**)[30]. Another frequent finding suggestive of an FMD diagnosis is the presence of a "web-like" defect at the origin of the internal carotid artery[31].

2.4.3. Vasculitis

Carotid vasculitis can be defined as the inflammation of carotid artery walls with or without necrosis, leading to stenosis or occlusion of the lumen[32]. Vasculitis may be associated with systemic connective tissue disorders or may be secondary to infection, malignancy, drugs, or radiation therapy. For a correct diagnosis, relevant laboratory tests should also be assessed. The 2012 Chapel Hill Consensus Conference defined different types of vasculitis in terms of (a) the size of the involved arteries and (b) associated pathologic lesions. The most frequent vasculitis involving carotid arteries are the Takayasu arteritis and the Giant cell arteritis[33] [34]. However, other conditions (e.g. infection, syphilis, tuberculosis, drugs) could also cause vasculitis.

With CTA / MRA imaging, signs of carotid vasculitis are vessel wall thickening (mostly concentric representing a key parameter in the differential diagnosis) and contrast enhancement. Usually there is no preference for involvement of the carotid bifurcation (different to atherosclerotic disease). In case of active vasculitis contrast enhancement of the thickened vessel wall may be seen in both CT and MR. Some authors suggest to use T2-weighted TSE imaging for edema detection in mural inflammation but this is less sensitive and more vulnerable to artifacts compared to T1-weighted, fat-suppressed, contrast-enhanced, black blood imaging [35].

2.4.4. Ancillary information

The analysis should be completed with assessment of the other visualized structures [**Mandatory**]. All pathological findings should be reported at the end of the report as ancillary findings.

3. Discussion and conclusion

The use of standardized and structured reporting for radiology examinations has gained significant momentum in recent years. However, it may come as a surprise that this concept is not new, as Perston Hickey proclaimed almost 100 years ago that “reports filed in hospitals should be scientifically accurate and should follow and accepted nomenclature, so at to have value in a statistical sense”[36].

This formalized approach to deliver the information contained in imaging datasets following a pre-defined template has many advantages. As a standard model sharing the same language, it is a means to avoid errors of interpretation, as the reports will be mostly void of personal preferences in the way they deliver information [37]. Also, it allows the reporting radiologist to follow a step-by-step process, assuring that all relevant information is covered and as such reducing important omissions. This is especially important when radiologist are less experienced in the pathology at hand, especially when performing examination on call after normal office hours [38].

It is therefore that the ESCR has promoted structured reporting in its publications [39] and has a joint venture with specialized companies to promote this among the cardiovascular imaging community [40]. This part II document on carotid imaging presents a further effort to stabndardize the reporting of carotid pathology in CT and MR-examinations, and las left room for inclusion of upcoming technologies.

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