Noninvasive Radiologic Diagnosis of Extracranial Vascular Pathologies

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Fridon Todua
Department of Radiology
National Academy of Sciences of Georgia
Research Institute of Clinical Medicine
I. Javakhishili Tbilisi State University
Tbilisi
Georgia

Editorial Advisor
Michael Okujava
Department of Radiology
Institute of Medical Research
Ilia State University
Tbilisi
Georgia

Department of Neurodiagnostics
and Neuroscience
Research Institute of Clinical Medicine
Tbilisi
Georgia

Dudana Gachechiladze
Department of Radiology
Programme of Caucasus University
Tbilisi
Georgia

Department of Ultrasound Diagnostics
National Medical Academy of Georgia
Research Institute of Clinical Medicine
Tbilisi
Georgia

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In Memoriam: Fridon Todua (1944–2017)

Professor Fridon Todua was the founder of the Research Institute of Clinical Medicine, Tbilisi, Georgia—one of the biggest diagnostic and treatment centers not only in Georgia but in the whole region as well.

Fridon Todua's contribution to the development of radiology and oncology in Georgia was tremendous and he was greatly honored throughout the radiological community.

He was hugely admired as a leader, researcher, and teacher, and was a great inspiration to many.

Fridon Todua was the author of more than 650 scientific works, including 13 monographs. He was the scientific supervisor of more than 85 PhD dissertations.

In recognition of his dedication and commitment, he received a number of awards and honors throughout his career and was widely respected for his work and research. He was the recipient of honorary doctorates and memberships from various universities and scientific academies, scientific societies, and scientific associations around the world.

He also received many other Georgian and foreign awards, among them the Order of Honor of UC for Contribution to Construction the United Europe, the Russian Federation Order of St. Andrew the First-Called, and the Albert Schweitzer Order.

Introduction

Cerebral circulatory disorders are a global medical and social problem; about 4.5 million people annually die from stroke, of whom three-quarters are in developing countries. Even in developed countries, stroke has the third highest mortality rate after Coronary heart disease and cancer. In Western developed countries the average mortality rate of stroke varies in the range of 5–100 per 100,000 people per year, which represents 10–12% of the overall mortality.

According to different studies (Laitinen-Krispijn and Bijl 2000), the standardized annual mortality of stroke in the 45–85 year age group in Western Europe is 326 per 100,000 people, while in Eastern Europe and some regions of Russia this figure is up to 600 per 100,000 people.

It should be noted that in those countries where high-technology diagnostics are widely used for cerebrovascular diseases, the incidence of stroke does not exceed 240–380, while in developing countries in Asia it exceeds 600. Epidemiological statistical analysis shows that the outcome of stoke varies greatly depending on whether modern diagnostic and therapeutic technologies are actively implemented in the management of cerebrovascular diseases.

As the proportion of the aged population increases, the incidence of acute vascular accidents, such as stroke, is increased, as is the incidence of chronic cerebrovascular diseases (leukoaraiosis, Binswanger disease, vascular dementia), creating many medical and social problems.

Hemorrhage resulting from different diseases of the intracranial vessels (aneurysms, arterio-venous malformations) are some of the most important problems in vascular neurology.

It should also be noted that cerebral circulatory disorders are some of the most "expensive" diseases. In developed countries, the overall whole-of-life expenses for one patient with stroke amount to US$55,000–73,000.

At the end of the twentieth century, great advances were made in the study of particular cerebrovascular pathologies, as well as in studies of the brain generally, mainly owing to the invention of neuroimaging diagnostics such as computed tomography (CT), magnetic resonance imaging (MRI), and positron-emission tomography, and their widespread implementation in clinical practice.

Neuroimaging methods give scientists the opportunity to look into the living human brain and study its structure, blood supply, and functional status, even including subtle molecular and chemical processes. These methods help to reveal

small lesions of several millimeters in size (such as tumors, infarctions, or hemorrhages) and observe their dynamics.

The diagnosis (even with the help of up-to-date diagnostic facilities) and the appropriate medical treatment of cerebral circulatory disorders are not possible without exhaustive studies of the causes and mechanisms of these circulatory disorders. Such studies have shown that timely prevention of circulatory risk factors, screening at early stages of the disease, and prompt medical treatment are crucially important.

The European Society of Cardiology, the European Society of Atherosclerosis, and the European Society of Arterial Hypertension recommend taking measures for the prevention of cerebral circulation disorders, targeting risk factors such as arterial hypertension, carotid artery damage, cardiac pathologies, hypercholesterolemia, smoking, physical inactivity, and excess weight.

Considering that about 75–78% of men and 55–60% of women in the population have at least one of the above risk factors, it is obvious that the problem needs active involvement aimed at the reduction of vascular risk factors.

The most frequent cause of cerebral circulatory disorders is atherosclerosis of the extracranial arteries, mainly the carotid arteries. It is particularly worth noting that, according to leading clinical centers, approximately 30–35% of the population has asymptomatic (latent) stenosis of the carotid arteries, which, in addition to certain other factors, may cause carotid system disease, leading to acute cerebrovascular disorders with, further, the necrosis of neuroglial tissues.

As extracranial arterial pathologies play an undisputed role in the development of cerebrovascular disorders, it is most important to use simple, non-invasive, safe, and easily reproducible diagnostic methods for these pathologies, thus making it possible to perform effective surgery to prevent atherosclerotic or thromboembolic arterial damage. As well as X-ray contrast angiography, which is widely used, such diagnostic methods include: duplex scanning in color and power Doppler modes, transcranial Doppler sonography, computed angiography, and magnetic resonance angiography; these methods are used in both clinical practice and scientific studies.

The above methods have been revolutionary for vascular neurology, allowing investigators to study vascular topography, the lumen, and the vascular walls in vivo, as well as to detect the exact location of pathological lesions, atheromatous plaques, or thrombi, and determine their hemodynamic significance. With the help of modern ultrasound equipment and computed or magnetic resonance angiography, it is possible to diagnose a hemodynamically significant stenosis of the carotid artery with practically 100% accuracy and to plan the optimal tactics for medical treatment.

The undisputed advantage of duplex scanning and color Doppler, in comparison with other methods, lies in the ability of these methods to accurately assess the structure and surface of atheromatous plaques. The latest modifications of ultrasound equipment can detect, in 97–98% of cases, potentially embologenic atheromatous plaques. The detection of such plaques is particularly important in modern vascular neurology, given their role in the pathogenesis of local cerebrovascular disorders and multi-infarct dementia.

Transcranial Doppler sonography is very important for the detection and assessment of cerebrovascular disorders, allowing investigators to study hemodynamic parameters in the Willis arteries, to assess collateral circulation and reserves, and to detect such features as stenosis, arterial and venous malformations, and aneurysms

in the intracranial vessels. With the help of specific software, it is also possible to monitor microemboli in the Willis arteries and to study cerebral venous hemodynamics during different anomalous occurrences (such as high intracranial pressure, sinus thrombosis, and cerebral thrombophlebitis).

Circulatory complications can be avoided through the timely and adequate treatment of the above vascular conditions; such treatment is not possible without efficient diagnostic equipment.

Thanks to the new generation of CT and magnetic resonance angiography devices, there have been great advances in the non-invasive diagnosis of various anomalies, pathological conditions, and malformations of the intracranial vessels. These devices help, in practically 100% of cases, to detect cerebral vascular conditions such as saccular and fusiform aneurysms and arterial or venous malformations, and the devices also show their location, topography, contact with surrounding structures, size, shape, and nourishing and draining vessels. Special three-dimensional (3D) reconstruction software shows clear pictures of the vessels.

In high-risk patients (those with conditions such as ischemic heart disease, arterial hypertension, and diagnosed stenosis of the carotid arteries), non-invasive investigations are used to detect asymptomatic stenosis, in order to study the structure, hemodynamic importance, and embologenic potential of any atherosclerotic plaque. The world's leading clinics recommend that targeted ultrasound studies (color Doppler, transcranial Doppler) of the extracranial arteries be done twice per year in high-risk patients. In special cases, screening also includes CT or MRI and magnetic resonance or computed angiography, while in patients with previously diagnosed cerebrovascular diseases, cerebral CT, MRI, or radionuclide perfusion is recommended.

Recent global epidemiological studies of cardiovascular conditions clearly show that the real possibility of reducing the spread of vascular neurological diseases lies with the use of diagnostic and treatment facilities for the early detection of cerebrovascular disorders.

However, although the above-mentioned modern non-invasive radiological examinations are employed in both the treatment and prevention of transient ischemic attacks and acute stroke, such therapeutic measures cannot be employed for completed strokes or established necrotic lesions following an infarction.

Accordingly, non-invasive neurological imaging of cerebrovascular conditions is regarded as valid for conditions such as transient ischemic attacks, but rather, the prevention of risk factors, while clinical and diagnostic assessment of the disease is based on angiography rather than on neuroimaging.

Currently, there is no doubt that the most radical therapeutic and preventive measures—such as carotid endarterectomy and endovascular correction of the lumen—for cerebrovascular conditions have developed efficiently as the result of the employment of high-technology neuroimaging and angiography, which allow the assessment of both the structural and functional status of the brain.

At the Research Institute of Clinical Medicine (Tbilisi, Georgia), patients with different cerebrovascular and circulatory conditions are examined using the latest-generation equipment.

The equipment used for MRI is Magnetom Avanto and Aera (Siemens) 1.5T and Magnetom Verio and Skyra (Siemens Erlangen, Germany), with a 3.0-T magnetic field.

The following sequence parameters are used for 3T: T1w-TR 300 ms, TE 15 ms, T2w-TR 6000 ms, TE 117 ms, PDw (Proton-density weighting)-TR 300 ms, TE-15 ms, turbo inversion recovery magnitude (TIRM)-TR 6000 ms, TE 93 ms, fluid-attenuated inversion recovery (FLAIR)-TR 6000 ms, TE 93 ms, T1 1600 ms, GE-TR-863 ms, TE 15 ms, fa (flip angle) 55°, TIRM fat-suppressed-MT-TR 1800 ms, TE 32 ms, T1 110 ms, TIRM fat-suppressed-TR 4000 ms, TE 48 ms, TI 110 ms, multi echo-TR 2400 ms, and TE 26, 58, 90, and 125 ms.

Magnetic resonance angiography of the intracranial arteries was performed with time of flight (TOF) sequence fl3d-multiple-trv- TR-21 ms, TE 3.60.4 ms, fa 40°, while for the extracranial arteries, the sequence used was TOF-fl2d-trav-sat-TR-21 ms, TE 3.60 ms, FOV 200 mm, fa 70°, and for venous sinuses, the sequence used was TOF-fl2d-cor-TR-32 ms, TE 14 ms, fa 60°.

The following parameters were used for 1.5-T systems: T1se TR-500 ms, TE 8.1, T2tse FOV 230, SE 5(19), TR-4500 ms, TE 101 ms, PD-T2tse TR 2840, TIRM (FLAIR) FOV 230, SE 5(19), TR 9000 ms, TE 111 ms; TIRM fat-suppressed-MT-TR 1800 ms, TE 32 ms, T1 110 ms, TIRM fat-suppressed-TR 4500 ms, TE 48 ms, TI 110 ms.

For contrast angiography (with gadolinium) of the extracranial vessels, the following sequence was used: FOV 313, TR 36.9, TE 1.39 ms.

For intravenous contrast, we used 20 ml of Magnevist (Bayer Healthcare Pharmaceuticals, Whippany, NJ) 5% solution (1 ml contains 469 mg of gadopentetate dimeglumine). Projection pictures were made according to MIP (Maximum intensity projection) and MPR (Multiplanar reconstruction) algorithms.

Cerebral CT and multislice CT angiography of the extra- and intracranial arteries were performed with a multislice tomograph (Aquilion One 640, Toshiba (Otawarashi, Tochigi, Japan)); Siemens tomograph Definition edge 384 sl and a SOMATOM Sensation Cardiac 64 s (Erlangen Germany), with 0.5-, 2-, and 4-mm axial sections. We used 50–100 ml of intravenous Ultravist 370 (Bayer Healthcare Pharmaceuticals, Whippany, NJ) 5% solution (60 ml on average) as contrast. The waiting period for extracranial arteries was 15–18 s.

Further reconstruction of images was performed on a special workstation (Vizard; Siemens, Vitrea fX; Toshiba). MIP (Maximum intensity projection), SSD (surface shaded display), VR (Volume rendering), and MPR reconstructions were used across the axial, coronary, and sagittal axes.

Ultrasound examination of the extracranial vessels was performed on Toshiba devices Aplio 500 and Aplio i800. Examinations were performed with 5–14 MHz and 7–18 MHz multi-frequency linear transducers. The degree of carotid stenosis was assessed based on ECST (European Carotid Surgery Trial) methodology.

Scanning of the intracranial vessels was performed with the Aplio 500 and Aplio i800 devices (Toshiba) at 2–4 MHz frequency. Transcranial emboli were detected with the Nicolett 8080 unit, based on the generally accepted methodology.

Reference

Laitinen-Krispijn S, Bijl RV (2000) Mental disorders and employee sickness absence: the NEMESIS study. Netherlands Mental Health Survey and Incidence Study. Soc Psychiatry Psychiatr Epidemiol 35(2):71–77

Contents

Part I

Vascular Anatomy and Basic Hemodynamics

Anatomy of Cerebral Circulation System

1

Cerebral circulation starts from aortic arch vessels (see the diagram in Fig. 1.1). The first artery coming out of the aortic arch is brachiocephalic trunk (TrB), which then divides into right subclavian and right common carotid arteries (CCA). The other branches are left subclavian and left common carotid arteries. The left CCA originates directly from the aortic arch.

The cervical section of both common carotids follows a similar course. Each vessel passes obliquely upward from behind the sternoclavicular joint to the level of the upper border of the thyroid cartilage. In the lower neck, the two common carotid arteries are separated from each other by the trachea. The left CCA is usually longer than the right CCA. Common carotid arteries proceed to the brain and most often at the upper level of thyroid cartilage (at the third or fourth cervical vertebrae) divide into internal carotid arteries (ICA) and external carotid arteries (ECA). The carotid bifurcation (CB) is an anatomically and surgically important landmark as it is involved in a variety of physiological and pathological processes. The height of the carotid bifurcation is classically defined in relation with vertebral levels and is highly variable across literature. Finally, geometry of CB is a determinant of local blood hemodynamic and wall shear stress, commencing or promoting the process of atherogenesis (Uflacker 2007).

External carotid artery (ECA) provides approximately 1/3 of the blood flow supplied by common carotid artery; ECA also originates symmetrically on the both side, has a relatively short trunk, and divides into several branches (the superior thyroid artery, the lingual artery, the facial artery, the maxillary artery, the occipital and superficial temporal artery, etc.). The first large branch, superior thyroid artery, is easy detectable on ultrasound examination and can be used for differentiation between ECA and ICA.

* Fridon Todua was deceased at the time of publication.

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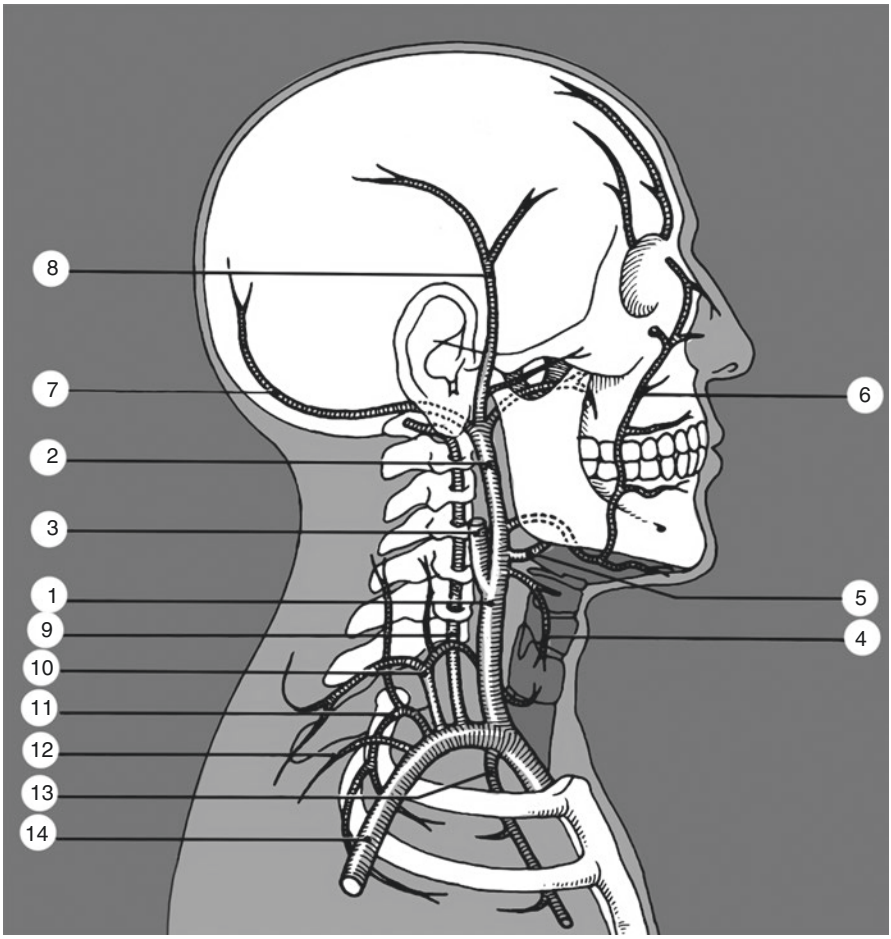

Fig. 1.1 Anatomical overview of the extracranial arteries. 1. common carotid artery; 2. external carotid artery; 3. internal carotid artery; 4. superior thyroid artery; 5. lingual artery; 6. facial artery; 7. occipital artery; 8. superficial temporal artery; 9. vertebral artery; 10. thyrocervical trunk; 11. costocervical trunk; 12. descending scapular artery; 13. internal thoracic artery; 14. subclavian artery

At the carotid bifurcation, the CCA widens, and the dilatation continues into the proximal portion of the ICA. This part is called the carotid sinus. Beyond the carotid sinus, the caliber of the ICA is uniform. In this segment the arterial wall has a number of particularities: Medial layer is relatively thin and adventitia is thick, with multiple elastic fibers and baro- and chemoreceptors.

The carotid sinus contains baroceptors able to detect acute changes in arterial pressure alongside chemoreceptors able to detect acute changes in arterial oxygen. Those receptors communicate with brainstem and through reflexes regulate homeostasis of these vital parameters (Valdueza et al. 2008).

Internal carotid artery (ICA) provides 2/3 of the blood flow supplied by CCA. Its diameter is larger than of ECA. The right and left ICAs develop symmetrically and have lateral or dorsolateral position in relation to the ECA. ICA rises to the scull base without branching.

The ICA has three main segments: cervical, petrous, and intracranial segments.

At the cervical segment, the ICA is almost vertical, from the origin to the carotid canal at the base of the skull. It is closely connected to the jugular vein (JV) and the vagus nerve, which is located between and behind these two vessels, forming a neurovascular bundle.

Intrapetrosal part is located in the pyramidal channel of the temporal bone. There is a vertical and a horizontal portion of the petrous segment of the ICA. In this segment it is bordered with the venous plexus.

The intracranial portion of the ICA may be divided into three segments: the precavernous segment, the cavernous segment, and the supraclinoid segments.

The ICA runs through the carotid canal to the cranial cavity and enters the cavernous sinus, where it forms the curved carotid siphon. The upper part of carotid siphon gives its first branch—ophthalmic artery (OA). After that, the internal carotid artery enters subarachnoid cavity, where it bifurcates into two main branches: middle cerebral artery (MCA) and anterior cerebral artery (ACA).

The MCA originates from the division of the ICA. The MCA has the larger caliber among the arteries of the circle of Willis. MCA is slightly curved and runs laterally (M1 segment). At the lateral cerebral fissure, it has 2–5 branches (M2 segment).

The anterior cerebral artery (ACA) rises from the anterior wall of the ICA. It runs medially (A1 segments), passing over the optic nerve and chiasm and anteriorly in the cerebral fissure (A2 segment). It is connected by the opposite ACA over the optic chiasm through the anterior communicating artery (AComA). In this segment also starts posterior communicating artery (PComA), which connects carotid and vertebrobasilar arterial systems.

Functional particularities of carotid arteries define their histological structure. CCA belongs to so-called elastic-type arteries, which corresponds to its main function—transportation of a larger volume of blood in comparison with the other arterial systems. Elastic artery is a vessel with a large number of collagen and elastin filaments in the tunica media, which gives it the ability to stretch in response to each pulse. Internal carotid artery is a muscular-elastic artery, innervated by a number of cranial, cervical, thoracal, and spinal nerves. Periarterial plexus of internal carotid artery, spreading to intracranial vessels, consists of cervical (mainly superior sympathetic) ganglia (Lasjaunias and Berenstein 1987).

The subclavian artery, like a carotid artery, on the right side emerges from the brachiocephalic trunk and on the left side directly from aortic arch. The vertebral artery (VA), in most of cases, originates at the upper posterior aspect of the first segment of the subclavian artery (SA). VA is divided into four main segments, out of which three are extracranial and one intracranial (Fig. 1.2).

The first segment, so-called V 1 segment of vertebral artery, starts from the subclavian artery and ends before entering the costotransverse channel. Vertebral artery