Ischemic Stroke: Systematic Vascular Imaging from Head to Heart

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Learning objectives

Learning objectives:

1. To know the different etiologies of strokes
2. To showcase typical imaging findings of all etiologies
3. To know key points of evaluation for each etiology
Background

Stroke

Stroke accounted for 11.8% of total deaths worldwide and is the second most common single cause of death in Europe, with great socio-economical impact, and constitutes a large part of emergency room (ER) presentations[1]. Stroke is caused by sudden insufficient blood flow to brain parenchyma and it can be divided into ischemic (which accounts for 85% of strokes) and hemorrhagic (which accounts for 15%).

Ischemic stroke can be caused by occlusion of either small vessels in the brain or large arteries feeding the brain. Roughly 25% of strokes are due to large vessel disease, small vessel disease, and cardioembolism, respectively, yet 25% remain of unknown origin[2].

Imaging studies are essential for diagnosis, with brain computed tomography (CT) being performed most frequently. Treatment consists of reestablishing physiological blood flow, either by thrombolysis (tPA) or mechanical endovascular thrombectomy, in the most timely manner. The latest recommendations state that "first picture to puncture time should be less than 90 min."[3]. With that in mind, the use of imaging in the ER means most often NECT with subsequent CTA.

Thorough imaging studies are nonetheless essential for further management of the patient, with the identification of the cause of stroke being paramount.

Imaging Studies

Ultrasound is useful both for evaluating the carotid arteries and the heart in stroke patients. It is a first-line modality both for screening and diagnostic evaluation of carotid disease and its use is increasing. However, despite being non-irradiating, it suffers from low accuracy and has low inter-observer agreement in characterizing plaques. Contrast Enhanced Ultrasound offers higher potential, but it has low ER availability as of writing [4].

Digital Subtraction Angiography (DSA) is the gold standard of vascular imaging, having the unique advantage of offering the possibility of direct treatment alongside diagnosis. However, it has the lowest availability, highest costs and highest procedure risk of any diagnostic procedure[5]. Its use is therapeutic rather than diagnostic.
Magnetic Resonance Angiography (MRA) offers highly detailed information about both vessel and parenchymal characteristics and is able to describe specific characteristics of plaques with potential future use [6]. Its use in the ER is limited due to availability and time constraints (both the time needed for the examination and the time needed to ensure the patient's condition is stable, MR-safe and the ability to cooperate the examination). Lastly, its non-iradiating character is of particular use in patients with potential multiple examinations [7].

CT Angiography (CTA) is the most used vascular study in stroke patients. It is highly available, as virtually all CTs can perform adequate CTAs; it is fast, with scan times being under a minute, and with patients already being positioned for the head CT; it is thorough, having the possibility of extending the scan area to include areas of interest with relative ease (i.e. the heart for suspicion of cardiac emboli); it is highly detailed, offering information about stenoses, plaque morphology, parietal characteristics, and vessel morphology - information helpful to neurointerventionists when planning an endovascular procedure [4, 8]. Moreover, there are developments of modified CTA protocols, such as the multiphase CT, that can offer useful information on collaterals. Current recommendations are that ischemic stroke patients be investigated with NECT and CTA[9].
Findings and procedure details

**Procedure Details:**

We reviewed the cases of patients with previously confirmed ischemic stroke of the past 2 years of the Department of Radiology and Medical Imaging of the University Emergency Hospital of Bucharest, selecting eloquent aspects for each pathology.

The scanners used were a 16-row CT for emergencies, a 64-row CT for follow-up and a 1.5Tesla MRI.

**Findings:**

We divide findings according to the mechanism of stroke as seen in Fig. 1 on page 9, inspired by TOAST classification [10].

**Penetrating artery disease**

Lacunar ischaemic stroke is defined as a stroke that is attributable to a recent small infarct <1.5 (or some say 2) cm diameter in the white matter, basal ganglia, pons or brainstem, and is consistent with a lacunar clinical syndrome. The mechanisms proposed are either embolic - occluding lenticulostriate arteries, atheromatous - atheromas in the parent artery extending to cover the penetrating branches, intrinsic small vessel disease with thickening vessel wall and secondary luminal occlusion[11]. Such vascular changes are too discrete to be visualised directly, magnetic resonance imaging managing to detect parenchymal changes [12].

**Cardiac**

Emboli of cardiac origin accounts for about one fifth of ischaemic strokes. Strokes due to cardioembolism are in general severe and prone to early recurrence [2]. The suspicion for a cardioembolic source for stroke is raised with a large vessel occlusion or when strokes occur in multiple vascular territories ( Fig. 2 on page 9 ). Haemorrhagic transformation happens in up to 71% of cardioembolic strokes and as many as 95% of hemorrhagic strokes are cardioembolic. Emboli from the heart are distributed evenly throughout the body, with more than 80% of symptomatic emboli affect the brain.[13].

Imaging of the heart was performed either as an extension on the CTA or subsequently, to determine the source of the stroke.
Atrial Fibrillation (AF)

AF is a powerful risk factor for stroke, independently increasing risk 5-fold throughout all ages, with particular impact at 80 to 89 years of age, where it accounts for 23.5% of strokes [14]. Both atrial and ventricular thrombi have to be distinguished from either contrast filling defects and papillary muscles, both of which have higher HU in contrast studies. Visualization of the cerebral arteries can point out filling defects consistent with emboli. Fig. 3 on page 10

Endocarditis

Infective endocarditis (Fig. 4 on page 11) is complicated by stroke in about 10% of cases. Emboli can be multiple, especially in the case of infection of prosthetic valves and in infections due to aggressive agents, such as Staphylococcus aureus [2]. Serious complications and deaths related to left-sided infective endocarditis are due to either cerebral emboli causing severe neurologic deficits or to intracardiac suppurative complications, which can cause severe valvular regurgitation and perivalvular abscesses[15]. Cardiac imaging can confirm valvular vegetation and the presence of complications (Fig. 5 on page 11).

Atrial Septal Defect

Atrial wall anomalies play a dual role in stroke. On the one hand, the combination of patent foramen ovale and atrial septal aneurysm may provide both an locus for in situ thrombosis and site for right to left embolization[16]. On the other, patients with atrial septal defects have a higher chance of developing AF, especially as they age[17] Fig. 6 on page 12.

Left Ventricular Thrombus Fig. 7 on page 13

Myocardial infarction is associated with ventricular wall immobility and may lead to left ventricular thrombus formation and cardioembolic stroke. The stroke rate from acute myocardial infarction (AMI) is 1%-3% from any AMI and 2%-6% anterior wall MI. The presence of LV Thrombus increases it to 15% at 3 months[18].

Atrial Myxoma
Myxomas are the most common primary heart neoplasm and are generally located in the atria. They have significant association with embolic events. The main differential diagnosis is with thrombi. Myxomas are larger in size (1-15cm), may change position during cardiac cycle and may have contrast enhancement [19].

Atherosclerotic Disease

Carotid atherosclerosis is one of the most common causes of death and disability in the western countries. The main criteria evaluated for risk stratification has been the degree of luminal stenosis, being a well-known risk factor for the development of neurologic. Recent research has concluded that a number of other plaque features contribute to the occurrence of neurologic symptoms, introducing the "vulnerable plaque", responsible for almost half of stroke cases. The main mechanism seems to be that of artery-to-artery embolization [4].

Carotid plaques are typically classified into smooth, irregular or ulcerated (Fig. 9 on page 15), with the last having high clinical significance for neurological symptoms, as much as a four-fold increase. The term smooth cad be used for a plaque with regular luminal morphology, irregular can be used for plaques whose surface fluctuates <1 mm, whereas the term ulceration is reserved for cavities measuring at least 1-2 mm[20]. Each ulcer is characterized by a neck and a base, both of varying sizes and resulting in various shapes, but none of them of particular increased risk. What is correlated with increased complications is plaque hemorrhage, which can be indirectly determined if the plaque presents enhancement [21] Fig. 10 on page 16.

Basically, ulceration can be diagnosed when contrast medium is identified extending beyond the vascular lumen (and within the plaque limits) for at least 1 mm in at least two planes [22] and hemorrhage by observing plaque post-contrast enhancement.

Lastly, measurement of degree of stenosis can be done by following NASCET criteria:

% ICA stenosis = (1 - [narrowest ICA diameter/diameter normal distal cervical ICA]) x 100. One has to keep in mind that measurements need to be done perpendicularly to the vessel axis and mentioning which modality was used in the report. Fig. 11 on page 16

Aortic Atheroma

Aortic atheromas a discrete protrusion of the intimal surface of the vessel >2 mm in thickness, different in appearance from the adjacent intact intimal surface. The presence of protruding atheromas in the aortic arch has been recognized as a potential cause of
cerebral or peripheral embolization in the elderly, with studies showing that protruding aortic atheroma (>4-5 mm) is 3-9 times more common in stroke patients than in healthy [2]. Findings indicate that the risk of ischemic stroke in elderly patients with arch atheromas is more strongly related to the complexity of the lesion than to its size. Complex atheromas present either a mobile component or ulcerations as previously defined[23] (Fig. 12 on page 17).

Other causes

Iatrogenic

Many vascular procedures present a risk of stroke, but few are as serious as the ones that generate fat emboli. Even though it is a rare complication, trauma is responsible for 90% of cases, but also liposuction, burns and cardiopulmonary bypass [24] Fig. 13 on page 18.

Dissection Fig. 15 on page 19

Craniocervical artery dissection is rare, but in young and middle-aged patients, spontaneous craniocervical artery dissection is the cause of up to one-fourth of strokes, with a peak prevalence in the fifth decade of life. As a suggested mechanism, intramural hemorrhage forms through ruptures of the vasa vasorum without intimal tear, especially if the wall is arteriopathic [25].

Arteritis

Lastly, recent CTA studies demonstrated that CTA can easily measure carotid artery wall thickness (CAWT) and found that, by using a 1 mm threshold, there was a 8 fold increase in stroke in patients with thicker (#1 mm) CAWT [7] Fig. 16 on page 20.
Fig. 1: Stroke mechanism inspired by TOAST classification.

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**Fig. 2:** NECT of stroke patients with cardioembolic suggestive patterns: (A) multiple vascular territory lesions; (B) hemorrhagic stroke.

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**Fig. 3:** (A) NECT with multiple ischemic lesions in both anterior and posterior circulation; (B) Volume-rendered (VR) image from a CTA seen from the front shows filling defects in both M1 segment of the right MCA and A1 segment of the right ACA; (C) axial MIP reconstructions showing the same filling defects
Fig. 4: (A) NECT showing acute (red arrow) and chronic (white arrows) ischemic lesions in multiple vascular territories; (B) Cardiac MRI LVOT (left ventricle outflow tract) View SSFP Cine images showing flow void (yellow arrow) consistent with Aortic Regurgitation; (C) Cardiac MRI Aortic Valve View SSFP Cine images showing thickened aortic valve leaflets (green arrows) - findings consistent with endocarditis
Fig. 5: F,50 with bicuspid aortic valve, calcifications and vegetations of the aortic valve and an adjacent aneurysm (red markers) with aortic-like contrast dynamics visible on MPR CT (A, B, C) and on VR (D); surgical piece confirmed perivalvular abscess (E)

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Fig. 6: Cardiac MRI 4CH (Four Chamber) View SSFP Cine Images showing wide communication between the left and right atria (yellow arrow)

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Fig. 7: MPR (A-4CH, B-long axis, C-short axis) chest CT showing apical LV Thrombus (yellow arrows) with partial calcifications in a patient with a history of stroke; (D) CT performed in a stroke patient with suspicion of aortic dissection showing endocardial hypoperfusion at apical, septal and lateral cardiac levels (red arrows) suggesting AMI.

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Fig. 8: (A) Axial CECT showing a hypodense mass adjacent to the interatrial septum; (B) Axial CMR T2 Black Blood images showing the same mass; (C) CMR 4CH SSFP Cine sequential images showing the mass (white arrows) prolapsing through the atrioventricular valve and occupying part of the ventricle (red arrow); (D) surgical specimen of the removed atrial myxoma

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**Fig. 9:** VR internal carotid arteries (ICA) with smooth plaque (A), irregular plaque (B) and ulcerated plaque (D), better seen on MIP MPR (D), where the depth, width and angle of the ulceration can be measured and the fatty nature of the plaque more clearly observed

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**Fig. 10:** (A) NECT showing basal ganglia acute ischemic stroke caused by preocclusive stenosis (green arrow) of the right ICA, with string-like distal flow (red arrows) visible on MIP MPR (B), VR (C) and curved planar reformation (CPR) (D)

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Fig. 11: M, 49 presented with left hyperdense MCA (yellow arrow) on NECT (D); follow-up 13 days later revealed hemorrhagic transformation (red arrow) with midline shift (E); CTA shows the presence of a ulcerated and possible mobile lipidic plaque in the proximal left ICA (A,B,C) - findings are consistent with artery-to-artery embolisation

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**Fig. 12:** M, 70 (A) VR of ulcerated (white arrow) protruding aortic arch atheroma (red arrow) in patient with history of stroke; vascular axis cross-section (B)

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**Fig. 13:** M, 70 after recent coronary artery by-pass grafting (CABG): (A) NECT showing hypodense lesion overlapping the right ICA (red arrows) with associated acute stroke in the right ICA territory; (B) coronal CECT showing lack of opacification of right ICA

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**Fig. 14:** Previously described 48,F with endocarditis (Fig 4) and ischemic stroke (A) presents filling defects of femoral-popliteal bypass graft and distal occlusion on VR (B); intra-graft thrombi (red arrows) can be seen on CPR (C)

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Fig. 15: F, 41 NECT shows hyperdense right ICA compared with the left (A) and acute ischemic changes in the anterior circulation (B); CTA show progressive narrowing of the R ICA in the proximal portion consistent with dissection (C, D) and the presence of collaterals in the right hemisphere (E).

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Fig. 16: M, 39 presented with TIA. On CTA you can distinguish aortic, brachiocephalic and left vertebral wall thickening (red arrows) with contrast enhancement (A vs B and C vs D) and left vertebral artery stenosis at its origin (green arrows) and a descending aortic dissection; Follow up examinations over 9 months show a increase in circulating volume (yellow circle)

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Conclusion

It is possible to investigate most ischemic stroke etiologies using CTA if one keeps in mind the needed parameters necessary to be assessed for each, thus providing an essential tool in secondary prevention.
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