Current and particular imaging aspects in ischemic stroke of the middle cerebral artery

Poster No.: C-1171
Congress: ECR 2014
Type: Educational Exhibit
Authors: E. V. Popa¹, M. Buzoianu¹, G. Popa¹, A. Flintoaca-Filip², I. G. Lupescu¹;¹Bucharest/RO, ²Bucuresti/RO
Keywords: Ischaemia / Infarction, Haemorrhage, Education and training, Diagnostic procedure, MR-Diffusion/Perfusion, MR, CT, Neuroradiology brain
DOI: 10.1594/ecr2014/C-1171

Any information contained in this pdf file is automatically generated from digital material submitted to EPOS by third parties in the form of scientific presentations. References to any names, marks, products, or services of third parties or hypertext links to third-party sites or information are provided solely as a convenience to you and do not in any way constitute or imply ECR's endorsement, sponsorship or recommendation of the third party, information, product or service. ECR is not responsible for the content of these pages and does not make any representations regarding the content or accuracy of material in this file.

As per copyright regulations, any unauthorised use of the material or parts thereof as well as commercial reproduction or multiple distribution by any traditional or electronically based reproduction/publication method is strictly prohibited.

You agree to defend, indemnify, and hold ECR harmless from and against any and all claims, damages, costs, and expenses, including attorneys’ fees, arising from or related to your use of these pages.

Please note: Links to movies, ppt slideshows and any other multimedia files are not available in the pdf version of presentations.

www.myESR.org
Learning objectives

The aims of our study are:

- to review and describe the common and particular signs in different phases of ischemic stroke involving the middle cerebral artery (MCA) territory;
- to list and describe the main complications of the ischemic stroke;
- to discuss and illustrate the differential diagnosis.
Background

Definition
Stroke is classically characterized as a neurological deficit attributed to an acute focal injury of the central nervous system (CNS) of a vascular cause, including:
- cerebral infarction,
- intracerebral hemorrhage or
- subarachnoid hemorrhage.
Ischemic stroke, as a consequence of neuronal cell death, represents an episode of neurological dysfunction caused by infarction attributable to ischemia (1).

Epidemiology
Stroke is the third leading cause of death in the United States (US) producing serious, long-term disability. Currently the highest rates are found in countries like Bulgaria, Romania, and Hungary.
It may occur at any age, more common in the elderly.
Sex rate: males have slightly higher age-adjusted rates. Race rate: blacks have a very high stroke mortality (2, 3).
Many studies show that the most common stroke subtype occurs in MCA territory followed by small-vessel stroke and brain stem infarction. In the US, about 15% of strokes are hemorrhagic and 85% ischemic (out of which 25% are due to small vessel disease-lacunar strokes, 25% are thromboembolic and the remainder large vessel disease) (4).

Middle cerebral artery- anatomy (5, 6, 7):
The largest terminal branch of the internal carotid artery (ICA)
The surgical nomenclature identifies four subdivisions

1. Horizontal/sphenoidal part (M1) from the termination of ICA to the bi/trifurcation
2. Insular part (M2) runs in the lateral (Sylvian) fissure;
3. Opercular segments (M3)
4. Cortical branches (M4)
The MCA supplies much of the lateral surface of the brain and is the larger of the of the two terminal branches of the ICA.

Vascular Territory (Fig. 1 on page 8)
**Cortical branches** supply most of the lateral surface of the cerebral hemispheres except for convexity and inferior temporal gyrus.
Penetrating branches:

- Medial lenticulo-striate arteries (from the proximal segment):
  - Medial basal ganglia, caudate nucleus;
  - Internal capsule.

- Lateral lenticulo-striate arteries:
  - Lateral putamen, caudate nucleus;
  - External capsule

Etiology of stroke

**Stroke in adults**<sup>(8)</sup>:

- Atherosclerosis of extracranial arteries that supply blood to the brain
- Hypertension and atherosclerosis
- Arterial embolism
- CNS vasculitis

**Stroke in children and young adults**<sup>(8)</sup>:

- Congenital or acquired heart conditions,
- Hematologic and disorders,
- Vasculopathies, and
- Drug ingestion.

**Neonatal stroke**<sup>(9)</sup>:

- Maternal causes: autoimmune disorders, coagulation disorders, congenital heart disease, diabetes, trauma;
- Placental causes: thrombosis, placental abruption, placental infection, chorioamnionitis;
- Congenital blood disorders;
- Systemic or CNS infection.

Pathophysiology

The three main mechanisms causing ischemic strokes are:
- Thrombosis,
- Embolism and
- Global ischemia (hypotensive) stroke<sup>(10)</sup>.

Cellular changes following stroke are synthetized in **Fig. 2 on page 8**.

**After the first three days (acute phase), reabsorption and then chronic stages follow (Fig. 3 on page 9).**
Classification of ischemic stroke in the MCA vascular territory

- depending on dimension of the vessel\(^{(11)}\):
  1. affecting large arteries:
     - Superficial territory;
     - Deep territory;
     - Entire territory;
  2. affecting small arteries: Lacunar infarction

- depending on evolution in time:
  - Acute;
  - Subacute;
  - Chronic.

Clinical features

Some of the more common symptoms of stroke include hemiparesis, monoparesis, or (rarely) quadripareisis, hemisensory deficits, monocular/ binocular visual loss, visual field deficits, diplopia, dysarthria, facial droop, ataxia, vertigo (rarely in isolation), aphasia\(^{(4)}\).

Diagnosis is based on:
- a detailed history of events plus a physical examination,
- imaging: including CT scanning and/or MRI,
- it is very important to identify the type of stroke and the underlying cause.

Most attacks happen suddenly, develop rapidly and damage the brain within minutes, sometimes continuing to worsen from several hours to a day or two as a steadily enlarging area, as the brain dies \textit{(stroke in evolution)}\(^{(12)}\).

Imaging procedures

Computed Tomography

CT is widely accessible, convenient, has a short imaging time and can be fast and easily performed in severely ill patients who are dependent on support and monitoring devices. The key role of this examination is to rule out hemorrhage (Fig. 4 on page 10) or other possible mimics of stroke. (neoplasms, arteriovenous malformations), as well as detection of ischemic signs of established infarction\(^{(1)}\).

The early signs of cerebral infarction are\(^{(1)}\):

- hyperattenuating arteries (Fig. 5 on page 11);
- hypoattenuation of gray matter strctures (loss of gray-white matter interface (Fig. 6 on page 12), obscuration of the lentiform nucleus (Fig. 7 on page 13));
- loss of cortical sulci.

\[\text{Page 5 of 60}\]
CT evaluation:

- **nonenhanced CT**: exclusion of hemorrhage, calcifications
- **CT angiography**: able to better, and more easily, demonstrate the difference between calcified and fibrous plaques
- **perfusion CT** has been shown to improve detection of acute infarction (Fig. 8 on page 14, Fig. 9 on page 15)
  - the site of vascular occlusion;
  - the infarct core, and salvageable brain tissue;
  - help to assess the degree of collateral circulation.

**Magnetic Resonance Imaging**

Despite the greater sensitivity of conventional MR images compared to CT images, false-negative MR studies can be seen within the 1-st few hours if **diffusion-weighted (DWI)** or perfusion-weighted sequences are not performed.

- **DWI-MRI** is the technique of choice for detection of hyperacute cerebral ischemia (in the first six hours). In many cases of hyperacute stroke in which hyperintense signal is already present on T2-weighted images, DW sequence better defines the size of the affected tissue\(^{(1,13)}\) (Fig. 10 on page 16).
- **Perfusion-weighted imaging (PWI)** provides information on the hemodynamic status of the affected tissue. In hyperacute stroke, the tissue with abnormal perfusion is larger than the DWI lesions, therefore, PWI help identify "tissue at risk" - the so-called ischemic penumbra\(^{(14)}\).
- **Diffusion Tensor Imaging (DTI)** has opened new possibilities of imaging early stages of Wallerian Degeneration. DTI detects changes of water diffusion in the fiber tracts within the first 2 weeks after stroke, at a time when T2-weighted images and maps of the orientationally averaged diffusivity do not reveal obvious changes\(^{(15)}\).

**Differential diagnosis**

Various entities can mimic stroke, such as:

- brain tumor,
- metabolic disorder,
- infection,
- demyelination,
- intoxication,
- traumatic injury\(^{(16)}\).

**Management and prognostic**

In ischemic stroke can be used the following categories of medications:

- anticoagulation, reperfusion, antiplatelet agents, neuroprotective.
• for a stroke in evolution, anticoagulants (e.g. heparin) may be given,
• if the stroke is caused by a blood clot: streptokinase or tissue plasminogen
  activator, are given within three hours after stroke’s onset.
• to reduce swelling and increased pressure may be given drugs such as
  mannitol, or rarely, corticosteroids (17).

Complications include:

A. Clinical outcome:

• **neurologic deterioration** with manifested complications within 24 hours of
  the stroke episode;
• **neuromuscular dysfunction** causing disabilities such as apraxia, pain
  syndromes, limb spasticity, and incontinence;
• **cognitive impairment** (memory loss, dysfunction in reasoning, in speaking,
  and problem-solving, or in extreme cases, dementia, anosognosia, aphasia,
  apraxia);
• **psychiatric disturbances** (depression, anxiety, emotional instability, crisis
  reaction, and post stroke fatigue) (4,12).

B. Imaging of complications after stroke:

• evolving stroke;
• mass effect;
• midline shift;
• hemorrhagic transformation;
• involvement of other vascular territories.
Images for this section:

![Vascular territories of MCA](https://www.radiologyassistant.nl)

**Fig. 1:** Vascular territories of MCA

© www.radiologyassistant.nl (modified)
Fig. 2: Cellular events induced by ischemia: In the vessels lumen, ischemia leads to blood clotting, platelet aggregation and cytokine release. Proinflammatory signals lead to production of cytokines and chemokines. Disruption of neuronal-microglial interaction and increases in extracellular glutamate (Glu) also contribute to the proinflammatory response.

Fig. 3: Acute phase (the first three days): Ionic changes result in necrosis of brain tissue. Reabsorption phase: 5 days after MCA occlusion macrophages produce reabsorption of the necrotic debris in the affected area. Chronic phase (2 weeks-2 months): Restoration of the blood-brain barrier, resolution of vasogenic edema, and cleaning up of necrotic tissue. Calcification and deposition of blood products (hemosiderin) may be seen.

© http://pt851.wikidot.com/stroke-cell-bio (modified)
**Fig. 4:** NECT: Acute hemorrhagic stroke: hyperdense area in the left lenticular nucleus and internal capsule. Right chronic infarction in the capsulo-lenticulo-caudate.

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
Fig. 5: NECT. Hyperdense left MCA.

F, 82 years old, developed loss of consciousness and sudden right hemiplegia.

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
**Fig. 6:** NECT: Acute ischemic stroke in the right temporo-insular region (a,b)- loss of grey-white matter definition, "insular ribbon sign". On the left, chronic infaction in the temporo-parietal area (arrowhead).

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
**Fig. 7:** NECT: obscuration of the lenticular nucleus (arrows).

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
Fig. 8: TTP-time to peak; CBF- cerebral blood flow; CBV-cerebral blood volume;

© Imaging of stroke and cerebral ischemia, John R. Hesselink, MD, FACR
**Fig. 9:** NECT show no significant abnormality. Perfusion CT map showing cerebral blood flow (CBF) reveals region of decreased perfusion in the fronto-opercular area (a). All color maps are coded red for higher values and blue for lower values. Perfusion CT map showing cerebral blood volume (CBV) shows relative symmetric maintenance of blood volume (b). Both mean transit time (MTT) map (c) and T max map (d) show prolongation within the affected region corresponding to the one shown in image a. Time to peak map (TTP) show relatively increased values in the affected are. The abnormalities described suggest an acute ischemic stroke in the right fronto-opercular area.

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania
Fig. 10: MRI examination. Acute ischemic stroke in the right lenticular nucleus: hyperintense signal on T2/Flair (a,b), discrete hipointense on T1wi(c), water restriction on DWI/ADC (d,e).

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
Findings and procedure details

TECHNIQUES
Computed tomography (CT). Nonenhanced and/or enhanced CT head regarding the unenhanced CT findings (CT angiography, perfusion CT).
Magnetic resonance imaging (MRI). Brain MRI protocols consisted of nonenhanced T1, T2, FLAIR, susceptibility-weighted (SWI) and diffusion weighted sequences.

IMAGING FINDINGS

Hyperacute ischemic stroke (the first 6 hours)
CT findings
CT changes obtained within the first 6 hours of cerebral ischemia are very often subtle. It is a general consensus regarding the idea that a normal CT appearance of the brain cannot exclude the presence of ischemic stroke in the first 6 hours.
If middle cerebral artery (MCA) is involved, it has been stated that up to 75% of the cases will have abnormal CT findings within the first 3 hour \(^{(18)}\).
The main task of CT examinations is to rule out cranial hemorrhage and other brain pathologies like tumors, malformation and hemorrhage.

The important CT findings during the early stages of cerebral ischemia can be classified as follows:
A. **Mass effect** for example narrowing of the Sylvian fissure or loss of cortical sulci.
B. **Hypoattenuating appearance of gray matter structures**: gray matter structure becoming isodense to adjacent white matter structures or, in essence, blurring of the gray matter-white matter junction (Fig. 12 on page 25, Fig. 13 on page 26). Examples: the insular ribbon sign (Fig. 11 on page 25), obscured lentiform nucleus (Fig. 13 on page 26, Fig. 14 on page 27).
C. Presence of one or more **hyperattenuating arteries** reflecting an arterial thrombus—most frequently seen in MCA (~50% of the patients) (Fig. 13 on page 26).
Any combination of these findings may be present, or all may be absent \(^{(1,13)}\).

**CT angiography** can depict the occlusion site, the grade of collateral blood flow, and help characterize carotid atherosclerotic disease \(^{(19)}\).

**Perfusion CT** delineates the ischemic tissue (penumbra) by showing increased mean transit time with decreased cerebral blood flow (CBF) and normal or increased cerebral blood volume (CBV), whereas infarcted tissue manifests with markedly decreased CBF and decreased CBV \(^{(17)}\) (see Fig. 9 on page ).

MR Imaging
DWI revolutionized MR assessment of early stroke phases. Based on the detection of microscopic movement of water molecules, DWI is extremely sensitive to minimal changes of movement and minimal increases of water concentration in the brain tissue.

- **High signal intensity on Diffusion-weighted MR imaging (DWI)** with a correspondent low intensity on Apparent diffusion coefficient (ADC) map confirms that restricted diffusion of water is present in the ischemic area. These findings can be observed within minutes after stroke onset at a time when T2-weighted images still show a normal appearance. Water restriction on ADC map can be seen within the first minutes until the ninth day after ictus onset (Fig. 16 on page 29, Fig. 17 on page 30).

- In addition, in many cases of hyperacute stroke in which hyperintense signal is already present on T2/ Flair-weighted images, diffusion-weighted imaging better defines the size of the ischemic region.

Of the conventional spin-echo MR sequences, Flair images are the most sensitive, helping to identify intravascular abnormalities (20), followed by T2-weighted images and T1-weighted images.

**Magnetic resonance angiography** is a set of techniques used to determine etiology of stroke and assess vascular flow dynamics. In early stages they help evaluate the severity of stenosis or occlusion (Fig. 18 on page 31), as well as collateral flow (21).

**Acute ischemic stroke (>6 hours to 3 days)**

**CT Findings**

*CT aspects change gradually over time* due to the fact ischemic stroke is a dynamic process. All early ischemic changes can be present anytime up to 72 hour regardless of the initial presence of CT findings.

- **Hypodense** area detected by previously CT may gradually expand, involving both gray and white matter of the brain.

  The commonest findings during this stage include:

  - low density of the basal ganglia (Fig. 13 on page 26, Fig. 15 on page 28)
  - sulcal effacement, as a result of brain swelling
  - increasing mass effect
  - wedge-shaped low density area involving gray and white matter (Fig. 13 on page 26)

**MR imaging**

Standard MRI images (T2 and T1) are used for detecting vasogenic edema, present in the acute phase of stroke and seen from 24 hours to several days after stroke onset.

The main changes seen in this stage are:
- **DWI**: bright signal, with decreased ADC values in the same areas (Fig. 16 on page 29, Fig. 17 on page 30, Fig. 18 on page 31, Fig. 19 on page 32).
- **FLAIR** and **T2WI**: hyperintense signal with loss of gray-white matter interfaces; **T1WI**: hypointense signal, **T2WI**: hyperintense signal with loss of gray-white matter interfaces; FLAIR images are more sensitive as compared to conventional T1 and T2 weighted images.

- **Increasing mass effect** identified as sulcal effacement, gyral edema;
- **Gradient recalled echo (GRE) /SWI** sequence are useful for the detection of blood products.
- **Meningeal enhancement** adjacent to infarct in the first 24 hours, later adds the gyral enhancement, demonstrated after the administration of Gadolinium. These findings suggest that blood-brain barrier was damaged.

**Subacute ischemic stroke (3 days up to 4 weeks)**

**CT findings**
- **Brain swelling** and **mass effect** will gradually increase within the first week followed by gradual improvement beginning from second week upward (Fig. 20 on page 33, Fig. 23 on page 36).
- **Hypodensity area** remains during this phase (Fig. 21 on page 34, Fig. 23 on page 36, Fig. 24 on page 37, Fig. 25 on page 38).
- Risk of **hemorrhagic transformation** is greater in the first week after the ictus (Fig. 35 on page 48).
- **Luxury perfusion (Fig. 22 on page 35):**
  - is non-specific (may occur in trauma, tumors, alcoholism, sickle cell anemia, diabetic ketoacidosis, meningoencephalitis, or a hyperperfusion syndrome)
  - is related to dysfunctional autoregulation.
  - on CECT appears as an area of enhancement at the margin of the infarct (22).

- **CT fogging effect (Fig. 26 on page 39):**
  - can occur between 2-6 weeks
  - the hypodensed infarcted lesion "disappear", probably due to resolution of edema in the infarcted area.
  - such "disappeared infarct" will reappear in later phase in a form of tissue cavitation (chronic infraction) (23).

**MRI findings**
The first week:
increasing parenchymal enhancement, sometimes to a great extent ("pseudotumoral")
gradually decreasing mass effect;

1 to 4 weeks:
- **Decreasing of edema** and mass effect;
- Moderate parenchymal, gyral, enhancement;
- **High signal intensity on T2** and low signal intensity in T1 images- the affected areas appear in this period well circumscribed
- **Hemorrhagic transformation** can occur any time in this phase, as patchy high signal intensity lesions on T1 WI (Fig. 36 on page 49).

**Chronic Stage (after 4 weeks)**

**CT findings**
- **encephalomalacia**: hypoattenuating, well defined area (density similar to CSF) accompanied by widened sulci and ex vacuo dilatation of ipsilateral ventricle (Fig. 27 on page 41).
- after affecting the superficial territory, focal cortical atrophy can be seen (Fig. 28 on page 40);
- chronic lacunar infarct appears as a hypodense lesion, similar to CSF density, <15 mm diameter (Fig. 29 on page 42, Fig. 30 on page 43). Differential diagnosis with perivascular space should be made. If it is close to the ventricular system, it can cause focal "ex vacuo" dilatation of the ventricle (24).

**MR imaging**
- Large MCA chronic infract: porencephalic cavity as a well-defined area, corresponding to a vascular territory, with homogenous, similar to CSF signal; the cavity may communicate with the ventricles and/or the subarachnoid space (Fig. 31 on page 44, Fig. 32 on page 45).
- **Gliosis** surrounding the encephalomalacic lesion can be better identified on FLAIR and Proton density images;
- "ex vacuo" dilatation of ipsilateral ventricle;
- **hemosiderin depositions**: hypointense signal on T2 GRE WI;
- chronic lacunar infarct (Fig. 33 on page 46) has hypeintense signal on T2/Flair sequences and isointense to mildly hypointense on T1WI(24).

**Wallerian degeneration (Fig. 34 on page 47)** - four stages were defined:

- In the first month after onset of ictus occur degeneration of myelin protein fragments
- 4-14 weeks later lipids remain intact but further destruction of myelin protein fragments that were already degenerated happen;
- > 14 weeks gliosis replaces the degenerated axons and myelin sheaths along with myelin lipid breakdown;
- (months to years) atrophy of the white matter tracts (25).

**MRI characteristics of Wallerian degeneration** (25)

<table>
<thead>
<tr>
<th>Stage</th>
<th>T1</th>
<th>T2</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>no changes</td>
<td>no changes</td>
</tr>
<tr>
<td>II</td>
<td>hyperintense</td>
<td>hypointense</td>
</tr>
<tr>
<td>III</td>
<td>hypointense</td>
<td>hyperintense</td>
</tr>
<tr>
<td>IV</td>
<td>brainstem atrophy +/- hypointensity</td>
<td>brainstem atrophy</td>
</tr>
</tbody>
</table>

**COMPLICATIONS**

1) **Hemorrhagic transformation (HT)** (27)

- minor petechial bleeding (hemorrhagic infarct)
- major mass-producing hemorrhage, parenchymal hematoma (Fig. 35 on page 48).

**Petechial hemorrhage:**

**CT**

- increased attenuation in grey matter.
- sometimes mimics normal grey matter density due to the "fogging effect".

**MRI:** SWI > T2 GE are more sensitive than CT to identify early hemorrhagic conversion (Fig. 36 on page 49).

2) **Vasogenic brain edema with Midline shift (MLS)** is a serious complication in hemispheric stroke (Fig. 37 on page 50).

- The following space-occupying effect can lead to MLS, cerebral herniation, and even death.
- Quantification of MLS can predict cerebral herniation and subsequent death at early time-points, even before clinical deterioration becomes apparent (4).

3) **Cortical laminar necrosis** (cortical pseudolaminar necrosis or simply laminar necrosis), seen in the late subacute stage, is an uncontrolled cell death in the cerebral
cortex accompanied by gliosis and deposition of fat-laden macrophages, in a band-like pattern, with a relative preservation of cells immediately adjacent to the meninges\(^{(10)}\).  

**NECT**: gyriform hyperdensity in the affected area.

**MRI:**
- ribbon of intrinsic high T1 signal (after 2 weeks)
- gyriform intra-cortical hypersignal on Flair images in the corresponding territory in addition to blooming seen on T2*GRE.

4) **Involvement of other vascular territories (Fig. 38 on page 51)** in embolic brain infarcts.

**DIFFERENTIAL DIAGNOSIS**

- cerebral tumor - **low-grade astrocytoma (Fig. 39 on page 52):**
  - do not respect a specific vascular territory;
  - no obvious changes in appearance in the next days;
  - higher ADC values.

- **encephalitis:**
  - do not affect a specific vascular territory;
  - imaging dynamic changes are different to stroke;
  - no changes in ADC values in the first days after onset.

- **cerebral infarction following venous sinus thrombosis (Fig. 40 on page 53):**
  - area of infarction on Flair/T2 wi;
  - MRA demonstrates absence of flow in the affected sinus\(^{(7)}\);

- abnormally **hyperdense intracranial vessels** in cases with high hematocrit: arteries and veins appear hyperdense on NECT

**WHAT WOULD CLINICIANS WANT TO KNOW?**

- the anatomic localization of stroke;
- the "volume" of cerebral tissue irreversibly affected;
- the site of occlusion;
- to rule out hemorrhage/ inflammatory disease/tumor.
**Fig. 11:** NECT: Hypodense area involving gray and white matter in the left temporo-parietal cortex extended to the ipsilateral insula ("insular ribbon sign" - arrow).

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
**Fig. 12:** I-CT examination. Acute ischemic stroke: discrete hipodense temporo-insular right area, with loss of grey-white matter definition.

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
Fig. 13: NECT. Acute ischemic stroke: right hyperdense MCA sign (a); hypodense fronto-temporo-insular right area, with loss of grey-white matter interface, affecting the cortico-subcortical (a), and deep territory of the right MCA (b)-arrow.

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
**Fig. 14:** NECT. Acute ischemic stroke involving the deep territory of the CMA: Hypodense area affecting the posterior limb of the internal capsule and partially the lentiform nucleus.

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
**Fig. 15:** NECT: Discrete hypodense area, 10 mm diameter, in the right lenticular nucleus—acute lacunar ischemic stroke.

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
Fig. 16: MRI evaluation: hyperintense signal on T2-WI/Flair images (a,b), hypointense signal on T1WI (c), with loss of gray-white matter definition in the corresponding area, discrete mass effect- left Sylvian fissure narrowed as compared to the right one. Water restriction on DWI sequence/ADC map (d,e) confirms the diagnosis of acute ischemic stroke in the superficial territory of the left MCA.

© Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
**Fig. 17:** MRI examination. Acute ischemic stroke in the temporo-parietal cortex and the capsulo-lenticular structures: hyperintense signal on T2-WI/Flair images (a,b), hypointense signal on T1WI (c), loss of gray-white matter definition in the corresponding area; discrete mass effect on the anterior horn of the right LV, narrowed right Sylvian fissure. Water restriction on DWI sequence/ADC map (d,e) confirms the diagnosis.

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
**Fig. 18**: MRI evaluation. Acute ischemic infarction affecting the deep white matter located cranial to the basal ganglia, having hyperintense signal on T2/Flair wi (a, b) with water restriction DWI (c) and ADC map (d). 3D TOF MRA: low flow signal of the right MCA as compared to the left one (e).

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
**Fig. 19:** MRI examination. Acute lacunar infarction: hyperintense signal (12mm) on T2/Flair wi (a, b), hypointense signal on T1WI (c), in the posterior limb of the internal capsule. Water restriction on DWI sequence/ADC map (d,e) confirms the diagnosis.

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
Fig. 20: NECT. Early subacute MCA ischemia: hypodense area involving gray and white matter of the superior left temporal cortex with loss of gyration (arrow).

© Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
**Fig. 21:** II NECT, five days later. Early subacute stage: extension of the right hypodense temporo-insular area, with well defined margins.

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
Fig. 22: NECT: Well circumscribed, predominantly hypointense area affecting the right temporo-parietal lobes (a) CECT: Enhancement at the margins of the infarct are seen, corresponding to luxury perfusion (b).

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
Fig. 23: NECT. Hypodense area affecting cortico-subcortical and deep vascular territory of the right MCA; discrete mass effect on the anterior horn of the right LV. No midline shift is visible.

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
Fig. 24: NECT: Hypodense, well defined small lesion (12 mm), in the anterior limb of the internal capsule- subacute lacunar ischemic stroke.

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
**Fig. 25:** NECT: hypoattenuating area of the right internal capsule, expanding in the lenticular nucleus.

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
Fig. 26: NECT: First CT- acute ischemic stroke: hipodense area in the right temporal lobe; right hyperdense MCA sign. 10 days later- subacute stage, the affected area seems pseudonormalised perhaps due to gyriform hyperdensity that impose differential diagnosis between luxury perfusion and subarachnoid hemorrhage. One year later- chronic stage: loss of brain tissue in the corresponding area.

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania
**Fig. 28:** NECT. Loss of cortico-subcortical brain tissue in the temporo-insulo-opercular right area. "Ex vacuo" dilatation of the posterior horn of the ipsilateral lateral ventricle (a,b- arrows).

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
Fig. 27: Loss of cortico-subcortical brain tissue in the left fronto-opercular cortex, extended in the capsulo-lenticular area. Dilatation of the ipsilateral LV (arrows).

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
Fig. 29: NECT. Pseudolacunar infarction: Small oval lesion, isodense with the CSF located in the right capsulo-lenticulo-caudate.

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
**Fig. 30:** NECT: Chronic lacunar infarction: hypodense, well defined small lesion, with density similar to CSF, in the lenticular nucleus.

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
Fig. 31: MRI examination. Loss of cortico-subcortical brain tissue in the temporo-insulo-opercular right area with low signal in FLAIR sequences (b), high signal on T2- FSE (a), similar to CSF signal, with no water restriction DWI/ADC map (c,d); evidence of gliosis surrounding the lesion (b- arrow) on Flair image. "Ex vacuo" dilatation of right posterior part of the lateral ventricle (a,b- arrowheads).

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
**Fig. 32:** MRI examination. Loss of cortico-subcortical brain tissue in the corresponding area: low signal on FLAIR sequence (b), high signal on T2- FSE (a), similar to CSF signal, with no water restriction DWI/ADC map (c,d); evidence of gliosis surrounding the lesion (b- arrow).

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
Fig. 33: MRI examination: Chronic lacunar infarctions: Small lesions capsulo-lenticular bilaterally with hyperintense signal on T2-wi and Flair, hypointense T1-wi; no water diffusion on DWI/ ADC map.

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
Fig. 34: MRI examination. Chronic right MCA infarction: signal similar to CSF on T1/T2/Flair wi; gliosis surrounding the lesion (arrows). The right cortico-spinal tract appears in hypersignal on T2/FLAIR sequences (i, j). Atrophy of the right cerebral peduncle is present (not shown)- Wallerian degeneration, stage IV.

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
Fig. 35: NECT. First CT: Hyperacute ischemic stroke of the right temporal lobe and insula (a,b)- loss of grey-white matter definition and "insular ribbon sign". 48 hours later: Early subacute stage- important extension in the fronto-parietal lobes with hemorrhagic transformation and effusion in the left LV. Midline shift visible (arrowheads). On the left, chronic infaction in the temporo-parietal area (arrowhead).

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
**Fig. 36:** MRI examination. Early subacute ischemia in superficial and deep left MCA territory (hyperintense signal on T2/FLAIR sequences, hypointense on T1-wi, restricted diffusion of water on DWI/ ADC map) Spots of methemoglobin (a, c) and hemosiderin deposition (c,f). Decreased flow in the left MCA and ICA (g).

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
**Fig. 37:** NECT. Subacute ischemic stroke: hypodense area affecting entire right MCA territory, extended to the caudate nucleus; midline shift with approx. 10 mm to left.

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
Fig. 38: NECT. Infarction in the left MCA and ACA territories - subacute stage.

© Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
Fig. 39: MRI examination. Astrocitoma: large, well circumscribed inhomogenous lesion surrounded by edema, cortico-subcortical, extended to capsulo-lenticular structures, with hyperintense signal on T2-wi (a)/ Flair (b), hypointense signal on T1-wi (c); nodular enhancement after administering Gadolinium.

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
Fig. 40: MRI examination. Hyperintense signal in the left temporo-lenticular area on T2 WI associating thrombosis of the straight sinus and the left lateral sinus identified on gadolinium-enhanced T1 sequence.

© Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
Fig. 41: MRI examination. Acute ischemic stroke affecting the vascular territory supplied by perforating arteries from MCA (capsulo-lenticular structures), penetrating arteries from ACA (Heubner artery- the head of the caudate). Chronic watershed infarction in the left frontal anterior border zone (arrowheads). Leucoaraiosis in the parieto-occipital regions.

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
**Fig. 42:** MRI examination. Acute ischemic infarction in the internal capsule: hyperintense signal on T2-WI/ Flair images (a,b), hypointense signal on T1WI (c). Water restriction on DWI sequence (d) with low values on ADC map (e) (arrows) Bilateral chronic infarction surrounded by gliosis.

© Radiology and Medical Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
Conclusion

MRI with a multimodality approach is highly sensitive to detect early changes in stroke:

- DWI and SWI (T2\*): detection of brain ischemia vs hemorrhage
- DWI and PWI: evaluation of the ischemic penumbra
- MRA: vessel occlusion
- SWI (T2\*): hemorrhagic risk

Given the access-related limitations of MRI, unenhanced CT is the most common imaging study used to exclude hemorrhage in the acute patient, identify early signs seen after the ictus onset and the vascular lesion responsible for the neurologic deficit.

- CT perfusion allow to evaluate the ischemic core and the ischemic penumbra
- CT angiography permit to evaluate the vessel status and the occluded vessel
**Personal information**

Dr. Elisabeta Valeria Popa  
Department of Radiology and Medical Imaging  
Fundeni Clinical Institute, Bucharest, Romania.

Sos. Fundeni nr.258, Bucharest, Romania  
E-mail: elisa_braila@yahoo.com,

Prof. Dr. Ioana G. Lupescu  
Head of Radiology and Imaging Department of Fundeni Clinical Institute, Bucharest, Romania  
University of Medicine and Pharmacy "Carol Davila" Bucharest  
mail: ilupescu@gmail.com
References

4. Comparison of Clinical Characteristics and Functional Outcomes of Ischemic Stroke in Different Vascular Territories, Yee Sien NG.
5. Imaging of Stroke: Part 2, Pathophysiology at the Molecular and Cellular Levels and Corresponding Imaging Changes, AJR:198, January 2012.
23. The Essential Can Be Invisible to the Eyes: The "Fogging Effect"Phenomenon in the Subacute Stage of Ischemic Stroke.