A. CT or MRI in the acute ischemic stroke?

Poster No.: A-273  
Congress: ECR 2010  
Type: Invited Speaker  
Topic: Neuro - Without Subtopic  
Authors: G. Krumina; Riga/LV  
Keywords: Early signs acute stroke, Non contrast CT, MRI  
Keywords: Neuroradiology brain, Neuroradiology peripheral nerve, Neuroradiology spine  
DOI: 10.1594/ecr2010/A-273

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

1. To recognize and describe the early subtle signs of acute ischemic stroke and its evaluation in follow-up studies at non contrast CT and MRI
2. To learn and discuss the comparative value of CT and MRI in the early diagnostic of ischemic stroke
3. To understand the general principles of patient's selection for thrombolysis based on imaging criteria.
Introduction

Acute brain ischemia constitutes more than 85% of all strokes and is worldwide a leading cause of death and disability (1,2,3). Although there is general agreement that thrombolysis of stroke patients is effective, the major issue is the case selection. There is a great potential for effectiveness but also important risk of the harm: thrombolytic-induced breakup of thrombi could cause new strokes and myocardial damage, intracerebral hemorrhage, and brain edema (4). Clinical benefit was demonstrated for intravenous thrombolysis in patients with acute stroke who were selected on the basis of imaging criteria (5). To extend the therapeutic window, improve efficacy, and limit complications, improved imaging techniques should address four essential issues: 1) presence of hemorrhage; 2) presence of an intravascular thrombus that can be treated with thrombolysis or thrombectomy; 3) presence and size of a core of irreversibly infarcted tissue; and 4) presence of hypoperfused tissue at risk (3), called by Rowley as four Ps—parenchyma, pipes, perfusion, and penumbra. (6). The role of CT and MRI in the evaluation of early stroke signs of brain parenchyma and intravascular thrombi will be highlighted in this lecture.

Non contrast CT

Non contrast CT (NCCT) of the brain is still the primary imaging modality used in the exclusion of lesions that mimic stroke: intracranial hemorrhage, subdural hematoma, cerebritis, migraine, tumors, and revelation of early subtle signs of brain infarction. Early signs of cerebral infarction on CT are hypoattenuation of gray matter structures: insular ribbon sign, disappearing basal ganglia sign; mass effect and hyperattenuating arteries. The NCCT sensitivity for early signs of infarction ranges between 45% and 88% depending on the time of examination, examination technique, ie. use of thin slice multidetector CT, nonstandard, variable soft-copy review settings, and experience of interpreter. The patients with transient ischemic attacks or small infarctions can be presumed to exist in the group negative results on the basis of clinical findings (7,8).

The insular ribbon sign

The insula or island of Reil is an "island" of cortex that lies at the base of the sylvian fissure, overlying the extreme capsule and claustrum. The insular ribbon refers to the island of Reil, extreme capsule, and claustrum. The insular ribbon sign is the loss of gray-white interface definition, reflects cytotoxic edema and relates to specificity of arterial anatomy. The insular ribbon is supplied exclusively by the insular segment of the middle cerebral artery (MCA) and its claustral branches. With interruption of MCA flow, the insular ribbon becomes the region most distal from the anterior and posterior cerebral collateral circulations. Consequently, the insular ribbon becomes a watershed arterial
zone. This factor, in combination with the fact that the winding opercular segments of the MCA are potentially likely places for emboli to lodge, most likely explains the high frequency of early involvement of the insular regions (9, 10).

Fig.: Fig 1. a,b. Schematic depiction (a) and axial non contrast CT image (b), obtained in a 73-year-old woman 2 hours after the onset of left hemiparesis, shows hypoattenuation and obscuration of the posterior part of the right lentiform nucleus (white arrow) and a loss of gray matter-white matter definition in the lateral margins of the right insula (black arrows).

References: MR, CT and US centre, Medical Academy of Latvia - Riga/LV

Fig 1. a,b. Schematic depiction (a) and axial non contrast CT image (b), obtained in a 73-year-old woman 2 hours after the onset of left hemiparesis, shows hypoattenuation and obscuration of the posterior part of the right lentiform nucleus (white arrow) and a loss of gray matter-white matter definition in the lateral margins of the right insula (black arrows).

Early hypoattenuation on CT in patients with acute stroke represents cytotoxic edema secondary to failure of ion pumps in response to inadequate supply of adenosine triphosphate. The attenuation in Hounsfield units (HU) is directly proportional to the degree of cytotoxic edema. An increase in tissue water content by 1% results in a decrease in parenchymal attenuation by 2.5-HU. In an animal model of MCA stroke the mean attenuation decreased from 50.0 to 48.4 HU at 1 hour and to 42.5 HU at 4 hours.
Fig.: Fig.2.a,b. Insular ribbon sign on the left in axial non contrast CT images, obtained in a 58-year-old man (a) 3 and (b) 26 hours after the onset of symptoms.

References: MR, CT and US centre, Medical Academy of Latvia - Riga/LV

Fig.2.a,b. Insular ribbon sign on the left in axial non contrast CT images, obtained in a 58-year-old man (a) 3 and (b) 26 hours after the onset of symptoms.

Clinically two types of insular stroke have been distinguished: the minor and the major insular stroke. Insular lesion is defined as major if more than two thirds of the length of the insula is involved. Smaller insular lesions is termed minor. Truly isolated insular stroke without involvement of other MCA territories is rare. The major insular stroke is associated with poor functional outcome and neurologic abnormalities, including somatosensory, neuropsychological, language, auditory processing, vestibular, and swallowing disorders. A role of insula infarction in causing cerebrogenic arrhythmia and sudden cardiac death has also been suggested (11)
Fig.: Fig 3. a,b. Major insular stroke, (a) schematic depiction of location and (b) axial non contrast CT image, obtained in a 62-year-old woman 6 hours after the onset of hemiparesis.

References: MR, CT and US centre, Medical Academy of Latvia - Riga/LV

Fig 3. a,b. Major insular stroke, (a) schematic depiction of location and (b) axial non contrast CT image, obtained in a 62-year-old woman 6 hours after the onset of hemiparesis.
Fig.: Fig. 4. a, b, c, d, e, f. Axial non contrast CT images. Growth of major insular stroke in a 75-year-old male patient 4 (a, b, c) and 96 hours (d, e, f) after the onset of symptoms. Persistent hyperdense MCA sign (a, d, arrows) and signs of secondary hemorrhagic transformation (f, arrows) are seen.

References: MR, CT and US centre, Medical Academy of Latvia - Riga/LV

Fig. 4. a, b, c, d, e, f. Axial non contrast CT images. Growth of major insular stroke in a 75-year-old male patient 4 (a, b, c) and 96 hours (d, e, f) after the onset of symptoms. Persistent hyperdense MCA sign (a, d, arrows) and signs of secondary hemorrhagic transformation (f, arrows) are seen.

The disappearing basal ganglia sign
The disappearing basal ganglia sign is caused by MCA occlusion proximally to lenticulostriate arteries. Involvement of the lenticulostriate territory indicates that a proximal M1 occlusion must have been present. The basal ganglia becomes isoattenuating to the adjacent white matter structures - the internal capsule and external capsule.

Fig.: Fig.5. a, b. Postmortem specimen (a) and axial non contrast CT image (b) in acute stroke patient with disappearing basal ganglia sign caused by MCA M1 segment occlusion.


Fig.5. a, b. Postmortem specimen (a) and axial non contrast CT image (b) in acute stroke patient with disappearing basal ganglia sign caused by MCA M1 segment occlusion.

The visibility of infarcts can be improved by setting of variable window width and center levels to accent the gray-white matter contrast.
Fig.: Fig.6.a,b,c. Axial non contrast CT images, obtained in a 58-year-old woman 6 hours after the onset of neurologic symptoms. Hyperdense MCA sign (a). Standard settings:(window width, 80 HU; center level, 20 HU (b), variable soft-copy settings: window width, 8 HU; center level, 32 HU chosen to accentuate the gray matter and white matter interface, there is markedly increased conspicuity of the gray matter hypoattenuation at the left posterior putamen and caudate head (c).

References: MR, CT and US centre, Medical Academy of Latvia - Riga/LV

Fig.6.a,b,c. Axial non contrast CT images, obtained in a 58-year-old woman 6 hours after the onset of neurologic symptoms. Hyperdense MCA sign (a). Standard settings:(window width, 80 HU; center level, 20 HU (b), variable soft-copy settings: window width, 8 HU; center level, 32 HU chosen to accentuate the gray matter and white matter interface, there is markedly increased conspicuity of the gray matter hypoattenuation at the left posterior putamen and caudate head (c).

Early mass effect

Early mass effect includes the narrowing of sylvian fissure or loss of cortical sulci.
**Fig.** Fig. 7. a,b,c. Axial non contrast CT images, obtained in a 74-year-old woman 4 hours after the onset of neurologic symptoms. Hyperdense dot sign in sylvian fissure (a), loss of cortical sulci (b), narrowing of sylvian fissure (c).

**References:** MR, CT and US centre, Medical Academy of Latvia - Riga/LV

Fig. 7. a,b,c. Axial non contrast CT images, obtained in a 74-year-old woman 4 hours after the onset of neurologic symptoms. Hyperdense dot sign in sylvian fissure (a), loss of cortical sulci (b), narrowing of sylvian fissure (c).

**ASPECTS standardised quantitative CT grading system**

ASPECTS (Alberta Stroke Program Early CT Score) is standardised quantitative CT grading system in acute anterior circulation ischemic stroke used in many cardiovascular centers and stroke trials: NINDS( National Institute of Neurological Disorders and Stroke ), ACCESS( Acute Cerebral CT Evaluation of Stroke Study), ECSS II( European-Australian Acute Stroke Study) etc (12, 13). The ASPECTS value is calculated from two standard CT scans, one at the level of thalamus and basal ganglia, and one just rostral to the ganglionic structures. The score divides the MCA territory into 10 regions of interest. Each area is compared with the opposite side.
Fig.: Fig.8.a,b. Schematic territory division in standard CT scans at the level of thalamus and basal ganglia (a), and just rostral to the ganglionic structures (b).


Fig.8.a,b. Schematic territory division in standard CT scans at the level of thalamus and basal ganglia (a), and just rostral to the ganglionic structures (b).

The territory of the MCA is granted 10 points. The normal CT scan has an ASPECTS value of 10 points. 1 point is subtracted for an area of early ischaemic change, such as focal swelling, or parenchymal hypoattenuation, for each of defined regions. A score of 0 indicates diffuse ischemia throughout the territory of the middle cerebral artery. The baseline ASPECTS value predicts response to intraarterial and intravenous thrombolysis, functional outcome and incidence of secondary hemorrhage within 3 and 6 hours of stroke onset. In an individual with an ASPECTS value of 7 or less, the risk of secondary hemorrhage with intravenous tissue plasminogen activator alteplase is 14 times higher that in patients with score >7 (14). ASPECTS score >7 corresponds well with the "less than one third MCA" rule.

**The hyperdense artery sign**
The hyperdense artery sign (HAS) represents stasis of flow due to arterial thrombus, most frequently seen in MCA. HAS is an indirect marker of acute infarction. It depends on the timing between the onset of symptoms and the CT, and also section thickness. It has been reported that HAS in MCA is present in 75% of the infarctions in the first 90 minutes and in 15% from hour 12 to hour 24. False positive hyperattenuating MCA signs have been documented in patients with calcified atherosclerosis or high hematocrit levels. The calcifications typically demonstrate a higher density than thrombus. The hyperattenuating appearance of the MCA caused by calcification will persist on follow-up CT scans. The increased attenuation of the vessel has been distinguished from atherosclerotic plaque on the basis of correlative angiograms and follow-up CT scans that demonstrate resolution following recanalization. In most cases, the hyperdense MCA sign disappears within a few days or after thrombolytic therapy, confirming its direct relation to thromboembolic occlusion, which resolves after recanalization.

Hyperdense middle cerebral artery sign (HMCAS) disappearance has prognostic value in patients treated with intravenous thrombolysis. Kharitonova T et al, 2009 studied 1905 stroke patients with HMCAS on admission CT and on follow-up CT scans 22-36 h after thrombolysis. Authors found that HMCAS disappeared after intravenous thrombolysis in about half of cases and these patients had twice as good outcomes compared with those with persistent HMCAS. The prognosis in patients with MCA occlusion that persists after intravenous thrombolysis is poor, which may indicate the need for an alternative treatment approach to this subgroup (15).

Measurements of thrombus composition and volumes can be used as a predictor of thrombolytic efficiency. Thin-section NCCT can provide a measure of thrombus composition based on HU. Thrombus HUs were measured by NCCT using polyethylene tubes as a model of intracranial arteries and in patients with acute ischemic stroke before thrombolysis (16). A study concluded that the HUs of platelet-rich thrombi are lower than those of erythrocyte-rich thrombi. Thrombi with lower HU counts were resistant to thrombolytics.

The presence or absence of hyperdense artery sign (HAS) on NCCT can predict also the thrombus volume. Thrombus volumes are significantly larger in patients with HAS than in those without ICA and M1 occlusions (17). The recanalization rate following intravenous (IV) recombinant tissue-type plasminogen activator (rtPA) treatment in patients with HAS is known to be low. The thrombus can be successfully lysed in only approximately a quarter of these patients treated with IV rtPA (18). The recanalization rate is further decreased in the occlusion of the proximal arteries (ICA terminus and MCA) due to their larger thrombus volume. Rapid estimation of the thrombus location and volume at the time of initial evaluation may be helpful in determining treatment technique in candidates for thrombolysis. For example, in patients whose thrombus is thought to be large and/or located in the large artery, additional treatment with mechanical thrombectomy or intra-arterial treatment for IV thrombolysis may be considered from the beginning (18).
Two types of MCA occlusion have been distinguished: Proximal occlusion caused by thromboembolism within MCA M1 segment and distal occlusion caused by thromboembolism within MCA M2, M3 segments.

Fig.: Fig. 9. a, b. Axial non contrast CT images: proximal MCA (a) and distal MCA occlusion (b).

References: MR, CT and US centre, Medical Academy of Latvia - Riga/LV

Fig. 9. a, b. Axial non contrast CT images: proximal MCA (a) and distal MCA occlusion (b).

This division has prognostic value. It has been reported that proximal hyperdense vessel sign is associated with poor short- and long-term prognosis in patients with ischemic stroke. In the study by Barber et al (19) was shown that patients who showed a proximal hyperattenuating MCA sign after acute stroke and who received intravenous tissue plasminogen activator were either dead or dependent after 3 months. Authors suggested that intravenous thrombolysis is ineffective in cases of proximal MCA occlusion. Contrary, the patients with a distal hyperattenuating MCA sign were independent in 64% of cases.

An association between a hyperattenuating MCA sign and the location of infarction has also been found. Patients with a proximal hyperattenuating MCA sign developed cortical and larger deep MCA infarctions more often. A hyperattenuating middle cerebral artery...
sign has been reported to be associated with a larger volume of infarction at follow-up CT (20).

**Fig.**: Fig.10.a,b,c,d,e,f. Axial non contrast CT images, obtained in a 81-year-old male 5 (a,b,c) and 30 hours (d,e,f) after the onset of neurologic symptoms. Persistent hyperdense MCA sign (a, d), development of large cortical and deep infarction (d, e, f).

**References:** MR, CT and US centre, Medical Academy of Latvia - Riga/LV

Fig.10.a,b,c,d,e,f. Axial non contrast CT images, obtained in a 81-year-old male 5 (a,b,c) and 30 hours (d,e,f) after the onset of neurologic symptoms. Persistent hyperdense MCA sign (a, d), development of large cortical and deep infarction (d, e, f).

Early worsening of acute stroke within 48-72 hours is common (19-37%) and is associated with increased mortality. The underlying mechanisms are failure of collaterals, clot progression, recurrent stroke, raised intracranial pressure, seizures, and hemorrhagic transformation. The radiological predictors of early neurological
deterioration in acute ischaemic stroke are extent of hypodensity more than 1/3 in the territory of MCA, hyperdense MCA sign and brain swelling on CT at 24 hours (21).

**Fig.**: Fig.11.a,b,c,d. Axial non contrast CT images, obtained in a 82-year-old female 3 (a,b) and 72 hours (c, d) after the onset of symptoms. Hyperdense MCA sign (a), development of cortical and large deep infarction with hemorrhagic transformation (c, d).

**References**: MR, CT and US centre, Medical Academy of Latvia - Riga/LV

Fig.11.a,b,c,d. Axial non contrast CT images, obtained in a 82-year-old female 3 (a,b) and 72 hours (c, d) after the onset of symptoms. Hyperdense MCA sign (a), development of cortical and large deep infarction with hemorrhagic transformation (c, d).
The MCA dot sign

The MCA dot sign is a relatively recent added symptom on non contrast CT in the setting of suspected acute stroke. It is a punctate focus of hyperattenuation located in the sylvian fissure and is seen on a noncontrast CT. To be properly applied, the MCA dot sign should have a higher attenuation than any other visible vessel. MCA dot sign correlate angiographically with M2 or M3 branch vessel clot. The sign appears when this high-attenuation structure is viewed in cross section, since the occluded vessel courses in a plane perpendicular to the transverse plane of imaging. While normal vessels on a noncontrast study are expected to display soft-tissue attenuation, thromboembolus and the occluded vessel will have increased attenuation. This hyperattenuation along the course of the MCA has been correlated microscopically with blood clots that demonstrate accumulation of erythrocytes, fibrin, and cellular debris.

![Fig. 12. a,b. Axial non contrast CT (a) and 3D reformation image of CTA (b), obtained in a 82-year-old female 4 hours after onset of symptoms. Hyperdense MCA dot sign (a), occlusion of the distal M1 segment of the MCA, thrombus in M2 branch vessel (b).](image)

**References:** MR, CT and US centre, Medical Academy of Latvia - Riga/LV

Fig. 12. a,b. Axial non contrast CT (a) and 3D reformation image of CTA (b), obtained in a 82-year-old female 4 hours after onset of symptoms. Hyperdense MCA dot sign (a), occlusion of the distal M1 segment of the MCA, thrombus in M2 branch vessel (b).

MCA dot sign is important for the thrombolytic therapy selection and prognosis. The patients with MCA dot sign are good candidates for thrombolysis.

The hyperdense posterior artery sign
The hyperdense posterior artery sign (HPCA) is a CT marker of acute ischemia in the PCA territory. The hyperdensity is seen within the ambient cistern, medial to the tentorium cerebelli, is typically visualized in 1 or 2 adjacent slices and can extend into the quadrigeminal cistern. This sign is detected with good interobserver reliability in more than one third of all patients with PCA ischemia, suiting the incidence of the HMCAS in MCA stroke. The HPCA is often associated with thalamic infarction, large PCA territory ischemia, more severe neurological symptomatology, and a higher risk of hemorrhagic transformation. Therefore, this sign may not only be helpful in the early diagnosis of PCA infarction but might also act as a prognostic marker in acute PCA territory ischemic stroke (22).

Fig.: Fig.13.a,b. Axial non contrast CT images, obtained in a 64-year-old female 7 hours after the onset of symptoms: hyperdense posterior artery sign (a, black arrow ), corresponding large PCA territory ischemia (b).

References: MR, CT and US centre, Medical Academy of Latvia - Riga/LV

Fig.13.a,b. Axial non contrast CT images, obtained in a 64-year-old female 7 hours after the onset of symptoms: hyperdense posterior artery sign (a, black arrow ), corresponding large PCA territory ischemia (b).

CT angiography
CT angiography (CTA) is essential for evaluating of intra- and extracranial vessels, intravascular thrombi, and thrombolytic therapy guiding. CTA is being used increasingly to reveal the origin of infarction and the site of cerebral artery occlusion. CTA allows detailed assessment of the intra- and extracranial vasculature with thin-section multiplanar views that is comparable to that of digital subtraction angiography and MR angiography in terms of the detection rate of major vessel lesions and occlusions.

**Fig.:** Fig.14.a,b,c,d,e,f,g. Axial NCCT images (a,b) and CTA (c,d,e,f,g), obtained in a 30-year-old female 3 (a) and 96 hours (b) after the onset of neurologic symptoms: sudden onset of headache, dizziness, dysarthria, disorientation. The patient experienced slight whiplash head-neck trauma during aerobic exercises one week ago. a - no pathological signs, b - thalamic infarction. CTA performed 96 hours after
onset of symptoms: on source images on the level of C3 (c) and C2 (d) increased external diameter of vertebral arteries, narrowed functional lumen is surrounded by intramural hematoma are seen. On CTA CR and vessel analysis (e,f,g) vertebral artery with tapered, narrowed lumen at V2 on the right and V3 on the left are seen. The final diagnosis in this case is thalamic infarction due to bilateral vertebral arteries dissections.

References: MR, CT and US centre, Medical Academy of Latvia - Riga/LV

Fig.14.a,b,c,d,e,f,g. Axial NCCT images (a,b) and CTA (c,d,e,f,g), obtained in a 30-year-old female 3 (a) and 96 hours (b) after the onset of neurologic symptoms: sudden onset of headache, dizziness, dysarthria, disorientation. The patient experienced slight whiplash head-neck trauma during aerobic exercises one week ago. a - no patholical signs, b - thalamic infarction. CTA performed 96 hours after onset of symptoms: on source images on the level of C3 (c) and C2 (d) increased external diameter of vertebral arteries, narrowed functional lumen is surrounded by intramural hematoma are seen. On CTA CR and vessel analysis (e,f,g) vertebral artery with tapered, narrowed lumen at V2 on the right and V3 on the left are seen. The final diagnosis in this case is thalamic infarction due to bilateral vertebral arteries dissections.

MRI findings in acute stroke

MRI findings in acute stroke are hyperintense zones on T2 FSE and FLAIR; sulcal effacement, mass effect; loss of arterial flow voids on T2 FSE; intravascular hyperintensity on FLAIR, stasis of contrast media within vessels in affected territories; abnormal blooming and parenchymal black dots due to hemorrhage on T2* GRE. T2* relaxation refers to decay of transverse magnetization caused by a combination of spin-spin relaxation and magnetic field inhomogeneity. T2* relaxation is seen only with GRE imaging because transverse relaxation caused by magnetic field inhomogeneities is eliminated by the 180° pulse at spin-echo imaging. T2* relaxation forms the basis for susceptibility-weighted imaging which exploits the magnetic susceptibility differences of the blood and of iron and calcification in various tissues (23). The morphologic changes at MR imaging relate to cellular swelling. Many early findings are analogous to those seen on CT images. For instance, the distinction between gray matter structures and adjacent white matter structures can be lost on T2-weighted MR images (owing to increased signal intensity in white matter structures) in a manner similar to the loss of the gray matter-white matter distinction seen on CT images. On the other hand, loss of MR imaging flow voids and stasis of contrast material within arteries subserving an infarcted territory does not directly reflect the presence of thrombus itself (as is the case with the hyperattenuating artery CT sign) but instead reflects stasis of flow distal to a thrombus. Despite the greater sensitivity of conventional MR images compared with CT images in the 1st few hours, false-negative MR studies can be seen within the 1st few hours if diffusion-weighted sequence is not performed. CT and DWI performed with the same delay after onset of ischemic stroke resulted in significant differences in diagnostic accuracy. DWI gives good interrater homogeneity and has a substantially better sensitivity and accuracy than CT even if the raters have limited experience (24, 25, 26).
Fig.: Fig.15.a,b,c,d,e. Axial NCCT images (a,b), MRI (c , d) and MRA (e), obtained in a 78-year-old female patient 4 hours after the onset of neurologic symptoms: hemiparesis and disorientation. Hyperdense artery sign(a, arrow), subtle disappearing basal ganglia sign (b, arrow), stasis of flow in ICA on T2 FSE (c, arrow) and 3D TOF non contrast MRA (e, arrow), axial DW MRI (b = 1,000 sec/mm²) shows high signal intensity areas consistent the restricted water diffusion in early infarction zones.

References: MR, CT and US centre, Medical Academy of Latvia - Riga/LV
Fig.: Fig.16.a,b,c MRI (a,b) and MRA (c) 6 hours after onset of symptoms, obtained in a 53-year-old male patient with early neurological worsening: disorientation and hemiparesis; a - restricted water diffusion on DWI (b = 1,000 sec/mm²) in infarcted area; b - cortical brain swelling (upper arrow) and parenchymal black lines due to hemorrhage in basal ganglia (lower arrow) on T2* GRE; c - stasis of flow in ICA on 3D TOF non contrast MRA.

References: MR, CT and US centre, Medical Academy of Latvia - Riga/LV
Fig.16.a,b,c MRI (a,b) and MRA (c) 6 hours after onset of symptoms, obtained in a 53-year-old male patient with early neurological worsening: disorientation and hemiparesis; a - restricted water diffusion on DWI (b = 1,000 sec/mm²) in infarcted area; b - cortical brain swelling (upper arrow) and parenchymal black lines due to hemorrhage in basal ganglia (lower arrow) on T2* GRE; c - stasis of flow in ICA on 3D TOF non contrast MRA.
Fig.: Fig. 17.a,b,c,d,e. Axial NCCT images (a,b), DW MRI (c) and non contrast 3D MRA of brachiocephalic vessels- axial source image (d) and MIP in coronal plane (e) obtained in a 47-year-old female 3 hours after sudden onset of headache, disorientation and hemiparesis. Hyperdense artery sign(a), no pathological in brain parenchyma signs on NCCT (b), high signal intensity in the MCA zone (c), narrowed functional lumen is surrounded by intramural hematoma ( d, arrow), two dissections in the extracranial part of ICA, which, obviously, were the source of thrombembolic cerebral ischemic infarction (e, arrows).

References: MR, CT and US centre, Medical Academy of Latvia - Riga/LV
hemiparesis. Hyperdense artery sign (a), no pathological in brain parenchyma signs on NCCT (b), high signal intensity in the MCA zone (c), narrowed functional lumen is surrounded by intramural hematoma (d, arrow), two dissections in the extracranial part of ICA, which, obviously, were the source of thrombembolic cerebral ischemic infarction (e, arrows).

To correct estimate the infarct age DWI signal cannot be used alone. It's important to examine DWI in comparison with ADC maps. The signal intensity on DWI is influenced initially by the changes in water diffusion that are associated with cytotoxic edema secondary to ischemia and later by the T2 relaxation time that is associated principally with vasogenic edema. In focal ischemia, the degree of ADC reduction has been reported to depend on both the anatomic location and the duration of ischemia. It is demonstrated in an animal stroke model that the apparent diffusion coefficient (ADC) decreased by approximately 30%-50% within 30 minutes after onset of focal ischemia.
Fig.: Fig.18 a,b,c,d,e. MRI (a, c, d) and MRA (e) obtained in a 82-year-old female patient with disorientation and slight hemiparesis 6 hours after onset of symptoms. Axial DW MR image (b = 1,000 sec/mm²) shows area of high signal intensity consistent with infarction (a), ADC map showed decrease of approximately 53% in ADC, indicating restricted water diffusion (consistent with early infarction) in this region compared with that of normal brain (b), axial FLAIR image (c) and T2FSE shows local cortical swelling, non contrast axial 3D TOF MRA shows stenosis of A1 segment of ACA more than 50%.

References: MR, CT and US centre, Medical Academy of Latvia - Riga/LV
Fig.18 a,b,c,d,e. MRI (a, c, d) and MRA (e) obtained in a 82-year-old female patient with disorientation and slight hemiparesis 6 hours after onset of symptoms. Axial DW MR image (b = 1,000 sec/mm²) shows area of high signal intensity consistent with infarction
(a), ADC map showed decrease of approximately 53% in ADC, indicating restricted water diffusion (consistent with early infarction) in this region compared with that of normal brain (b), axial FLAIR image (c) and T2FSE shows local cortical swelling, non contrast axial 3D TOF MRA shows stenosis of A1 segment of ACA more than 50%.

**DW MRI in TIA**

Recently, a new definition of transient ischemic attack (TIA) has been proposed by neurologists based on the duration of symptoms and DWI findings. In patients presenting with a TIA, an MRI has discriminative value in predicting the risk of new TIA or stroke. TIA-related DWI abnormalities are associated with prolonged duration of TIA and disturbance of higher brain function. More sustained and extensive ischemia may contribute to DWI abnormalities in patients with TIA. The absence of a DWI lesion at baseline is associated with a high risk for subsequent TIA at 12 months but a small risk of future stroke. TIA patients with DWI abnormalities at baseline have a higher risk of future stroke (27,28,29).

Comparative values of NCCT and MRI in the evaluation of early ischemic stroke signs in brain parenchyma are summarized in the subsequent table (abridged from de Lucas M et al, RadioGraphics 2008; 28:1675).

<table>
<thead>
<tr>
<th>CT</th>
<th>MRI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Widespread Access</td>
<td>Less widely available than CT outside of major stroke centers</td>
</tr>
<tr>
<td>Greater acquisition speed</td>
<td>Contraindications (eg, electronic implants, patient intolerance, medical instability)</td>
</tr>
<tr>
<td>Highly sensitive for the exclusion or confirmation of hemorrhage</td>
<td>Gradient-echo imaging superior to CT for the detection of acute hemorrhage</td>
</tr>
<tr>
<td>Radiation dose</td>
<td>Diffusion-weighted imaging more sensitive than nonenhanced CT for the early detection of acute ischemia</td>
</tr>
<tr>
<td></td>
<td>Much more sensitive than nonenhanced CT for the detection of acute stroke</td>
</tr>
<tr>
<td></td>
<td>No radiation dose</td>
</tr>
</tbody>
</table>
**Fig. 0:** Fig 1. a,b. Schematic depiction (a) and axial non contrast CT image (b), obtained in a 73-year-old woman 2 hours after the onset of left hemiparesis, shows hypoattenuation and obscuration of the posterior part of the right lentiform nucleus (white arrow) and a loss of gray matter-white matter definition in the lateral margins of the right insula (black arrows).

© MR, CT and US centre, Medical Academy of Latvia - Riga/LV
Fig. 0: Fig.2.a,b. Insular ribbon sign on the left in axial non contrast CT images, obtained in a 58-year-old man (a) 3 and (b) 26 hours after the onset of symptoms.

© MR, CT and US centre, Medical Academy of Latvia - Riga/LV
**Fig. 0:** Fig 3. a,b. Major insular stroke, (a) schematic depiction of location and (b) axial non contrast CT image, obtained in a 62-year-old woman 6 hours after the onset of hemiparesis.

© MR, CT and US centre, Medical Academy of Latvia - Riga/LV
Fig. 0: Fig. 4. a,b,c,d,e,f. Axial non contrast CT images. Growth of major insular stroke in a 75-year-old male patient 4 (a,b,c) and 96 hours (d,e,f) after the onset of symptoms. Persistent hyperdense MCA sign (a, d, arrows) and signs of secondary hemorrhagic transformation (f, arrows) are seen.

© MR, CT and US centre, Medical Academy of Latvia - Riga/LV
Fig. 5. a, b. Postmortem specimen (a) and axial non contrast CT image (b) in acute stroke patient with disappearing basal ganglia sign caused by MCA M1 segment occlusion.


Fig. 6. a,b,c. Axial non contrast CT images, obtained in a 58-year-old woman 6 hours after the onset of neurologic symptoms. Hyperdense MCA sign (a). Standard settings:(window width, 80 HU; center level, 20 HU (b), variable soft-copy settings: window width, 8 HU; center level, 32 HU chosen to accentuate the gray matter and
white matter interface, there is markedly increased conspicuity of the gray matter hypoattenuation at the left posterior putamen and caudate head (c).

© MR, CT and US centre, Medical Academy of Latvia - Riga/LV

**Fig. 0:** Fig. 7. a,b,c. Axial non contrast CT images, obtained in a 74-year-old woman 4 hours after the onset of neurologic symptoms. Hyperdense dot sign in sylvian fissure (a), loss of cortical sulci (b), narrowing of sylvian fissure (c).

© MR, CT and US centre, Medical Academy of Latvia - Riga/LV
**Fig. 0:** Fig.8.a,b. Schematic territory division in standard CT scans at the level of thalamus and basal ganglia (a), and just rostral to the ganglionic structures (b).

**Fig. 0:** Fig. 9. Fig. 7. a,b. Axial non contrast CT images: proximal MCA (a) and distal MCA occlusion (b).

© MR, CT and US centre, Medical Academy of Latvia - Riga/LV
**Fig. 0:** Fig.10.a,b,c,d,e,f. Axial non contrast CT images, obtained in a 81-year-old male 5 (a,b,c) and 30 hours (d,e,f) after the onset of neurologic symptoms. Persistent hyperdense MCA sign (a, d), development of large cortical and deep infarction (d, e, f).

© MR, CT and US centre, Medical Academy of Latvia - Riga/LV
Fig. 0: Fig.11.a,b,c,d. Axial non contrast CT images, obtained in a 82-year-old female 3 (a,b) and 72 hours (c, d) after the onset of symptoms. Hyperdense MCA sign (a), development of cortical and large deep infarction with hemorrhagic transformation (c, d).

© MR, CT and US centre, Medical Academy of Latvia - Riga/LV
Fig. 0: Fig. 12. a,b. Axial non contrast CT (a) and 3D reformation image of CTA (b), obtained in a 82-year-old female 4 hours after onset of symptoms. Hyperdense MCA dot sign (a), occlusion of the distal M1 segment of the MCA, thrombus in M2 branch vessel (b).

© MR, CT and US centre, Medical Academy of Latvia - Riga/LV
**Fig. 0:** Fig.13.a,b. Axial non contrast CT images, obtained in a 64-year-old female 7 hours after the onset of symptoms: hyperdense posterior artery sign (a, black arrow), corresponding large PCA territory ischemia (b).

© MR, CT and US centre, Medical Academy of Latvia - Riga/LV
**Fig. 0:** Fig.14.a,b,c,d,e,f,g. Axial NCCT images (a,b) and CTA (c,d,e,f,g), obtained in a 30-year-old female 3 (a) and 96 hours (b) after the onset of neurologic symptoms: sudden onset of headache, dizziness, dysarthria, disorientation. The patient experienced slight whiplash head-neck trauma during aerobic exercises one week ago. a - no patholical signs, b - thalamic infarction. CTA performed 96 hours after onset of symptoms: on source images on the level of C3 (c) and C2 (d) increased external diameter of vertebral arteries, narrowed functional lumen is surrounded by intramural hematoma are seen. On CTA CR and vessel analysis (e,f,g) vertebral artery with tapered, narrowed lumen at V2 on the right and V3 on the left are seen. The final diagnosis in this case is thalamic infarction due to bilateral vertebral arteries dissections.

© MR, CT and US centre, Medical Academy of Latvia - Riga/LV
Fig. 0: Fig.15.a,b,c,d,e. Axial NCCT images (a,b), MRI (c, d) and MRA (e), obtained in a 78-year-old female patient 4 hours after the onset of neurologic symptoms: hemiparesis and disorientation. Hyperdense artery sign(a, arrow), subtle disappearing basal ganglia sign (b, arrow), stasis of flow in ICA on T2 FSE (c, arrow) and 3D TOF non contrast MRA (e, arrow), axial DW MRI (b = 1,000 sec/mm²) shows high signal intensity areas consistent the restricted water diffusion in early infarction zones.

© MR, CT and US centre, Medical Academy of Latvia - Riga/LV
Fig. 0: Fig.16.a,b,c MRI (a,b) and MRA (c) 6 hours after onset of symptoms, obtained in a 53-year-old male patient with early neurological worsening: disorientation and hemiparesis; a - restricted water diffusion on DWI (b = 1,000 sec/mm²) in infarcted area; b- cortical brain swelling (upper arrow) and parenchymal black lines due to hemorrhage in basal ganglia (lower arrow) on T2* GRE; c - stasis of flow in ICA on 3D TOF non contrast MRA.

© MR, CT and US centre, Medical Academy of Latvia - Riga/LV
Fig. 0: Fig. 17.a,b,c,d,e. Axial NCCT images (a,b), DW MRI (c) and non contrast 3D MRA of brachiocephalic vessels- axial source image (d) and MIP in coronal plane (e) obtained in a 47-year-old female 3 hours after sudden onset of headache, disorientation and hemiparesis. Hyperdense artery sign(a), no pathological in brain parenchyma signs on NCCT (b), high signal intensity in the MCA zone (c), narrowed functional lumen is surrounded by intramural hematoma (d, arrow), two dissections in the extracranial part of ICA, which, obviously, were the source of thrombembolic cerebral ischemic infarction (e, arrows).

© MR, CT and US centre, Medical Academy of Latvia - Riga/LV
Fig. 0: Fig. 18 a,b,c,d,e. MRI (a, c, d) and MRA (e) obtained in a 82-year-old female patient with disorientation and slight hemiparesis 6 hours after onset of symptoms. Axial DW MR image ($b = 1,000$ sec/mm$^2$) shows area of high signal intensity consistent with infarction (a), ADC map showed decrease of approximately 53% in ADC, indicating restricted water diffusion (consistent with early infarction) in this region compared with that of normal brain (b), axial FLAIR image (c) and T2FSE shows local cortical swelling, non contrast axial 3D TOF MRA shows stenosis of A1 segment of ACA more than 50%.

© MR, CT and US centre, Medical Academy of Latvia - Riga/LV
References

14. Barber PA, Demchuk AM, Zhang J, et al. Validity and reliability of a quantitative computed tomography score in predicting outcome of
hyperacute stroke before thrombolytic therapy. ASPECTS Study Group.
Alberta Stroke Programme Early CT Score. Lancet. 2000;355:1670-4

Thomassen L, Wahlgren N. Disappearing hyperdense middle cerebral artery
sign in ischaemic stroke patients treated with intravenous thrombolysis:
clinical course and prognostic significance. J Neurol Neurosurg Psychiatry
2009;80:273-27

thrombolytic efficacy in acute ischemic stroke using thin-section noncontrast
CT. Neurology, American Academy of Neurology, 2006;67:1846-1848

17. Kim EY., Yoo E., Choi HY., Lee JW., Heo JH. Thrombus Volume
Comparison between Patients with and without Hyperattenuated Artery Sign

administration of recombinant tissue plasminogen activator as assessed by
pre- and post-thrombolytic angiography in acute ischemic stroke patients.
Stroke 2007;38:192-93

"dot" sign: a CT marker of acute ischemia. Stroke 2001;32:84-88

angiographic correlates of early computed tomography signs in acute

21. Thanvi B, Treadwell S, Robinson T. Early neurological deterioration in acute
ischaemic stroke: predictors, mechanisms and management. Postgrad Med
J 2008;84:412-417

R, Thron A. The Hyperdense Posterior Cerebral Artery Sign. A Computed
Tomography Marker of Acute Ischemia in the Posterior Cerebral Artery
 Territory. Stroke. 2006;37:399-403

Principles, Techniques, and Applications of T2*-based MR Imaging and Its
Special Applications Radiographics September 2009 29:1433-1449

24. Fiebach JB, Schellinger PD, Jansen O, Meyer M, Wilde P, Bender J,
Schramm P, Jöttler E, Oehler J, Hartmann M, Hähnel S, Knauth M, Hacke
W, Sartor K. CT and diffusion-weighted MR imaging in randomized order:
diffusion-weighted imaging results in higher accuracy and lower interrater
variability in the diagnosis of hyperacute ischemic stroke. Stroke. 2002; 33:
2206-2210

stroke. RadioGraphics 2006; 26(suppl 1): S75-S95


27. Boulanger JM, Coutts SB, Eliasziw M, Subramaniam S, Scott J, Demchuk
AM. Diffusion-Weighted Imaging-Negative Patients With Transient Ischemic
Attack Are at Risk of Recurrent Transient Events. Stroke. 2007;38:2367

Personal Information

Gaida Kr#mi#a MD,PhD.

Neuroradiologist, Professor

Chief of Department of Radiology Riga Stradinš University

Director of the Program of Postgraduate Education

gaida.krumina@apollo.lv