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CASE REPORT

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Acute cerebrovascular incident in a young woman: Venous or arterial stroke? – Comparative analysis based on two case reports

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Summary

Background: Cerebrovascular diseases are the most common neurological disorders. Most of them are arterial strokes, mainly ischemic, less often of hemorrhagic origin. Changes in the course of cerebral venous thrombosis are less common causes of acute cerebrovascular events. Clinical and radiological presentation of arterial and venous strokes (especially in emergency head CT) may pose a diagnostic problem because of great resemblance. However, the distinction between arterial and venous stroke is important from a clinical point of view, as it carries implications for the treatment and determinates patient's prognosis.

Case Report: In this article, we present cases of two young women (one with an acute venous infarction, the second with an arterial stroke) who presented with similar both clinical and radiological signs of acute vascular incident in the cerebral cortex. We present main similarities and differences between arterial and venous strokes regarding the etiology, clinical symptoms and radiological appearance in various imaging techniques.

Conclusions: We emphasize that thorough analysis of CT (including cerebral vessels), knowledge of symptoms and additional clinical information (e.g. risk factors) may facilitate correct diagnosis and allow planning further diagnostic imaging studies. We also emphasize the importance of MRI, especially among young people, in the differential diagnosis of venous and arterial infarcts.

Key words: cerebrovascular diseases • arterial stroke • venous stroke

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Background

Cerebrovascular disease is the most common cause of acute neurological events, the majority of which are arterial strokes, mainly ischemic, rarely hemorrhagic. Cerebral venous thrombosis is a rare vascular cause of acute neurological events. Both clinical as well as radiological pictures (particularly in emergency CT images) of arterial and venous strokes may pose significant diagnostic problem due to high similarity. However, differentiation between arterial and venous stroke is important from a clinical point of view, as it influences patient treatment and prognosis.

In this article we discuss cases of two young women (one with a venous and the other with an arterial stroke), who presented with a similar clinical and radiological picture of an acute vascular lesion of cerebral cortex. Described cases are the basis for detailed comparative analysis of venous and arterial strokes.

Case Report

Case 1

A 41-years-old woman was admitted to the hospital emergency department due to a sudden difficulty in speaking and confusion. Neurological examination revealed

sensorimotor aphasia and slight right-sided paresis accompanied by droping of the right mouth corner, without pathological meningeal and pyramidal signs. An emergency CT examination without contrast administration demonstrated a slightly hypodense area, 3.5×3.0×2.5 cm in diameter, in the left temporoparietal region. The lesion encompassed mainly cerebral cortex, to a lesser extent the adjacent white matter and exhibited slight mass effect manifesting as pressure on the trigone of left lateral ventricle and narrowing of sulci in the left temporoparietal area. Careful examination of vessels revealed hyperdensity of the left transverse sinus, sigmoid sinus and vein of Labbe (Figure 1). Diagnosis of cerebral venous thrombosis complicated by venous infarction without hemorrhagic conversion in the left temporoparietal area was suggested based on CT examination.

An MRI study performed on the 5th day showed an edematous area in the left temporoparietal cortex and adjacent white matter, hyperintense on T2-weighted images and FLAIR sequences without signs of restricted diffusion in DWI. Involved cortex exhibited linear signal hyperintensity on T1-weighted images (picture of hemorrhagic necrosis) and linear contrast enhancement (sign of brain-blood barrier damage) (Figure 2). High signal within the transverse sinus, sigmoid sinus and left vein of Labbe was noted in T1- and T2-weighted images a well as in the FLAIR sequence. Following administration of contrast medium filling defects were visible in the lumens of those sinuses, indicating venous thrombosis (Figure 3). MRI picture corresponded to cerebral venous infarction in the course of venous sinus thrombosis.

Doppler ultrasound examination of cervical vessels performed on the 12th day of hospitalization did not reveal signs of jugular vein thrombosis and showed normal picture, morphology and blood flow within carotid arteries.

In the course of hospitalization we acquired additional information regarding long-term use of oral contraception by the patient, which was most likely the cause of cerebral venous thrombosis.

Case 2

A 28-years-old patient was admitted to the hospital emergency department due to a sudden appearance of speech disturbances and weakening of muscle force in the right upper limb. An emergency CT examination revealed normal brain and intracranial fluid spaces. There was no increase in the density of venous sinuses or arteries, which might suggest thrombosis. Since it was the second neurological incident of a TIA type (the previous incident took place 6 months before) patient was admitted to the hospital. On the second day of hospitalization the woman suffered the first focal epileptic attack in her life, which subsequently turned into a bilateral tonic-clonic seizure. A head CT performed after the epileptic attack showed a hypodense area encompassing mainly cerebral cortex in the left temporoparietal region. A minute, oval, hyperdense focus was noted peripherally to the hypodense area, requiring differentiation between a small hemorrhagic focus and cortical venous thrombosis. Moreover, there was a small amount of hyperdense, extravasated blood in the pericerebral space (Figure 4). Due to patient's young age, atypical clinical symptoms (epileptic seizure) and ambiguous radiological picture that required differentiation between arterial ischemic stroke complicated by a small hemorrhage and venous infarction in the course of venous thrombosis patient was ordered a head MRI. Non-contrast MRI was performed on the next day and showed an area in the left temporoparietal cortex hypointense on T1-weighted images, hyperintense on T2-weighted images and FLAIR sequence, exhibiting strong diffusion

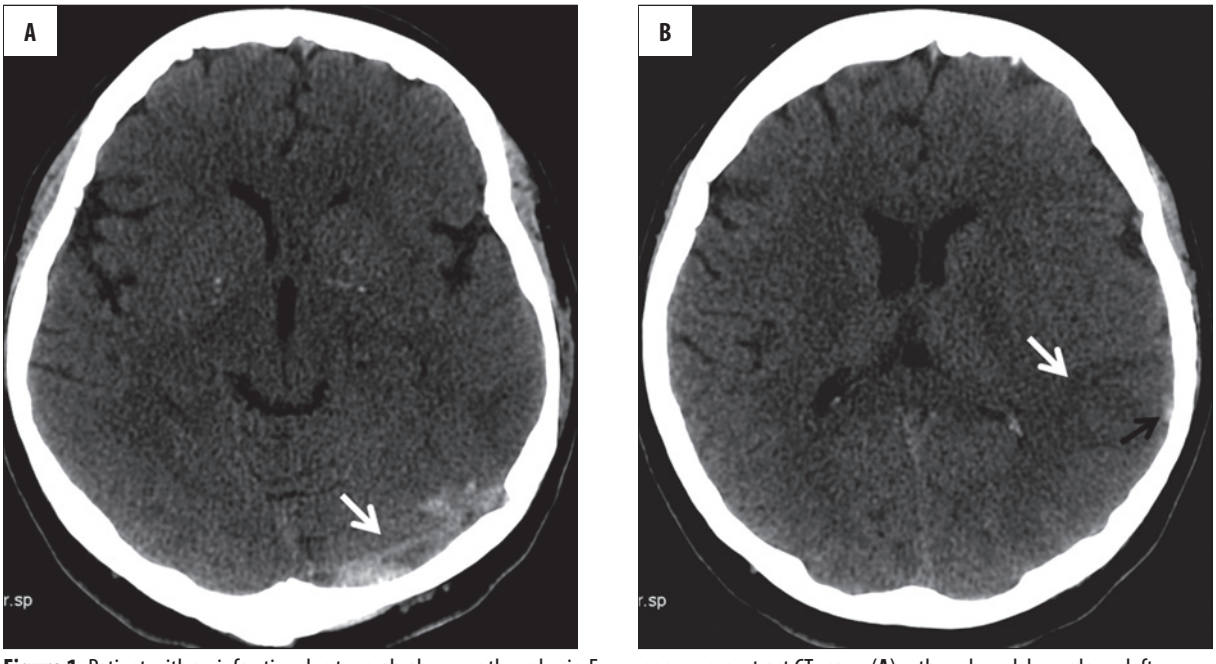


Figure 1. Patient with an infarction due to cerebral venous thrombosis. Emergency non-contrast CT scans. (**A**) – thrombosed, hyperdense left transverse sinus (arrow), (**B**) – hypodense venous infarction within the left temporal cortex (white arrow) and thrombosed hyperdense vein of Labbe (black arrow).

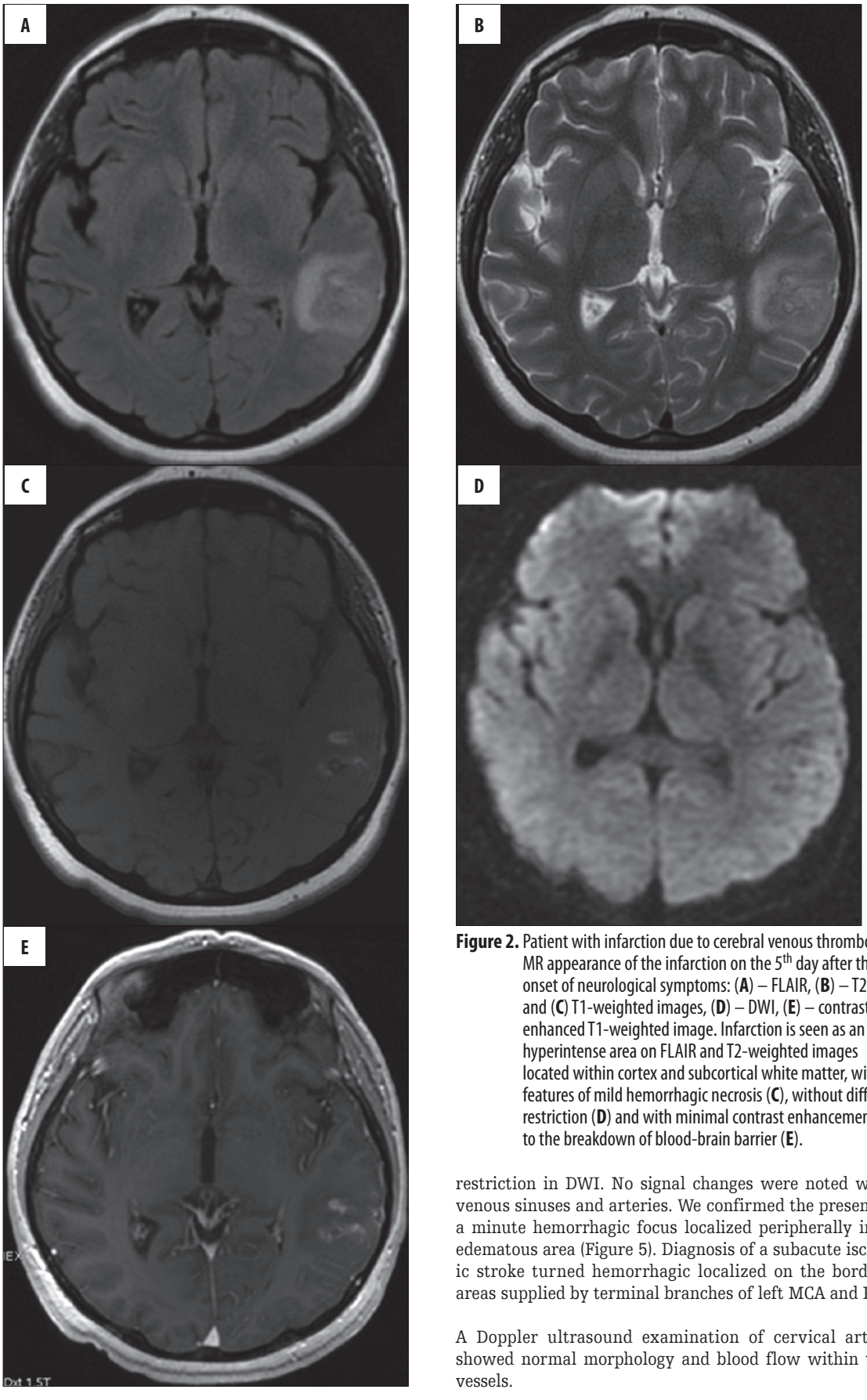


Figure 2. Patient with infarction due to cerebral venous thrombosis. MR appearance of the infarction on the 5th day after the onset of neurological symptoms: (**A**) – FLAIR, (**B**) – T2- and (**C**) T1-weighted images, (**D**) – DWI, (**E**) – contrast enhanced T1-weighted image. Infarction is seen as an hyperintense area on FLAIR and T2-weighted images located within cortex and subcortical white matter, with features of mild hemorrhagic necrosis (**C**), without diffusion restriction (**D**) and with minimal contrast enhancement due to the breakdown of blood-brain barrier (**E**).

restriction in DWI. No signal changes were noted within venous sinuses and arteries. We confirmed the presence of a minute hemorrhagic focus localized peripherally in the edematous area (Figure 5). Diagnosis of a subacute ischemic stroke turned hemorrhagic localized on the border of areas supplied by terminal branches of left MCA and PCA.

A Doppler ultrasound examination of cervical arteries showed normal morphology and blood flow within these vessels.

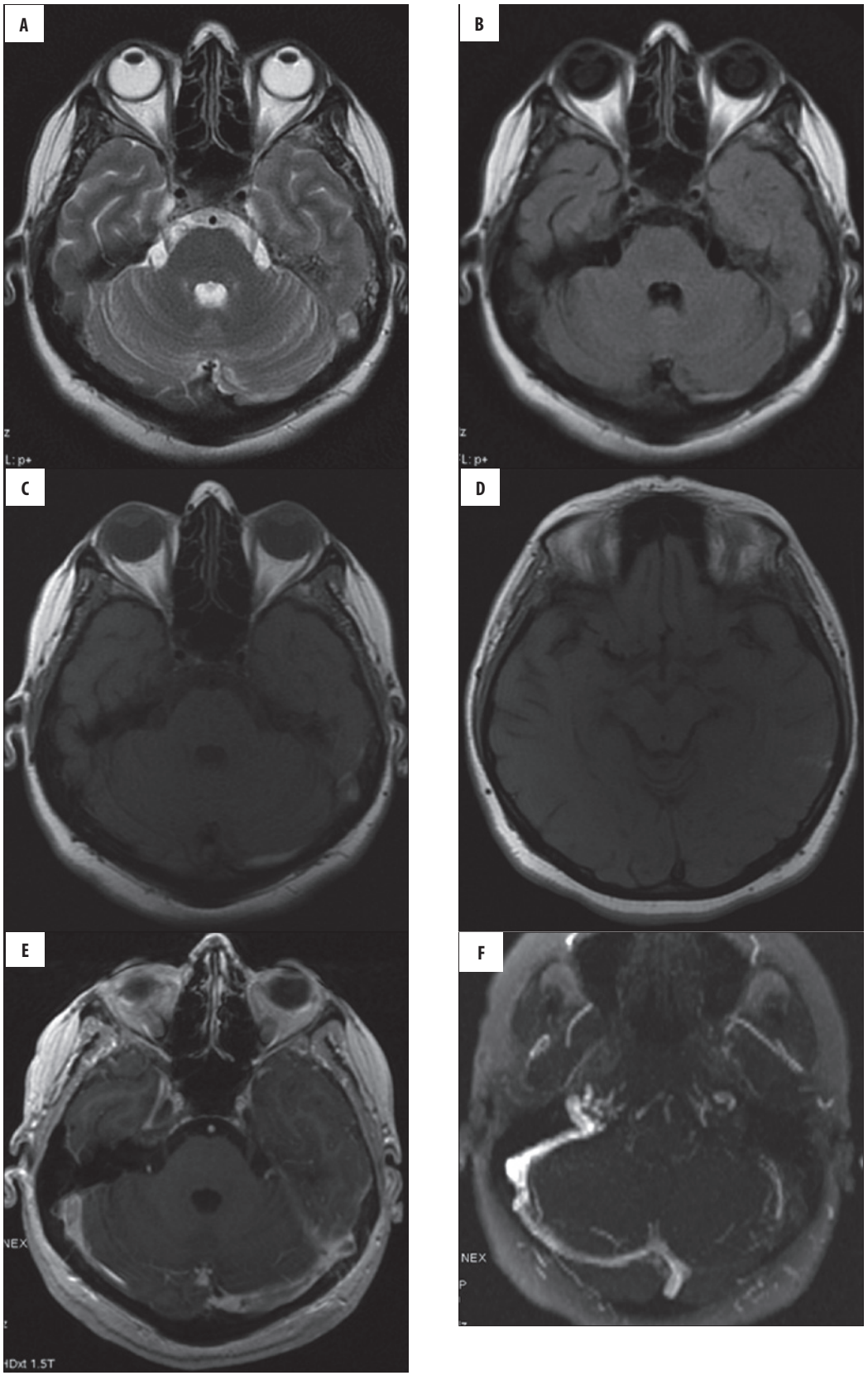


Figure 3. Patient with cerebral venous thrombosis. Changes within cerebral veins in MRI: (**A**) – T2-, (**B**) – FLAIR, (**C, D**) – T1-weighted images, (**E**) – contrast enhanced T1-weighted image, (**F**) – MR venography without contrast injection. Thrombosed left transverse sinus is seen as hyperintense on T1, T2 and FLAIR images (**A–C**), as well as filling defect after contrast injection (**E**) of loss of signal in MR venography (**F**). Thrombosed left vein of Labbe is also visible as hyperintense dot on T1-weighted image (**D**).

Cardiac ultrasound performed in the course of hospitalization showed a defect and aneurysm in the atrial septum, which was considered the immediate cause of embolic ischemic events in this patient.

Discussion

The two cases described above exemplify the difficulty in differentiation between typical ischemic strokes of arterial origin and less common venous infarctions. Venous as well as arterial strokes manifest as acute neurological events that may demonstrate very similar clinical and radiological picture. Despite many similarities, there are also significant differences regarding the etiology, clinical and radiological signs, the knowledge of which makes the diagnosis easier. A radiologist describing an emergency head CT of a patient with acute neurological incident is often the first doctor to undertake differential diagnosis. Below, we present main clinical and radiological elements that should be taken into consideration when differentiating between those two diseases.

Epidemiology

Vascular diseases are the most common disorders of the brain. They mainly affect older population, but in 25% of cases they also occur in patients younger than 55 years old. Most of them are arterial pathologies leading to strokes, more often ischemic (75% of strokes) than hemorrhagic (25%) [1]. Venous infarctions in the course of venous thrombosis (involving sinuses, deep and superficial veins) are much rarer than ischemic strokes of arterial etiology (2.7 per million in general population) and constitute about 0.5–1% of causes of all strokes. They most often occur in young patients, more frequently in women (about 75% of patients) [2].

Etiology

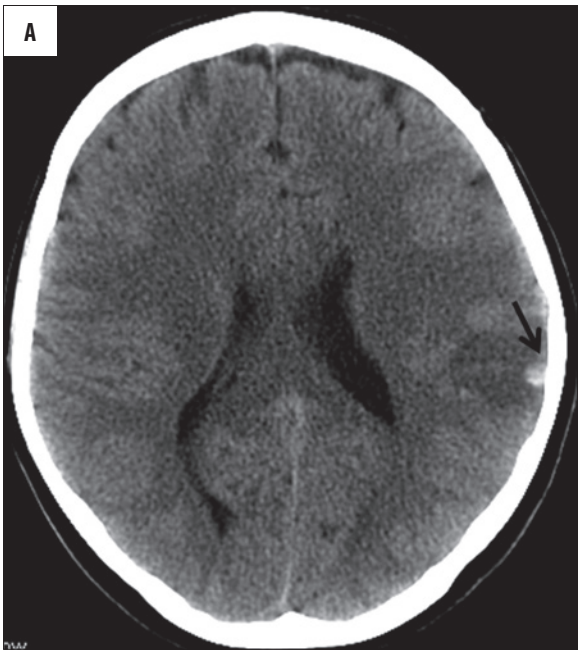
The most common cause of arterial stroke is occlusion of an artery supplying blood to a given area, in 2/3 of cases caused by thrombi and in 1/3 by embolisms. Risk factors for ischemic stroke in the older age group include atherosclerosis, diabetes, hypertension, hypercoagulable states, atrial fibrillation. Genetic (e.g. innate coagulopathies), inflammatory (connective tissue diseases) factors as well as congenital or acquired cardiovascular anomalies [2] play a greater role in young people, as supported by the case of Patient no. 1, in whom arterial occlusion with thrombotic material from the heart was considered the cause of ischemic stroke. It should be emphasized that heart disease constitutes about 18% of all causes of ischemic strokes in patients below 40 years old [3–6]. It is suggested that patent foramen ovale (PFO) may be one of the main causes of stroke of previously undetermined etiology in young adults. Presence of PFO in patients after a vascular cerebral incident increases the risk of recurrence 5-fold or even more if the defect is accompanied by atrial septal aneurysm [7]. Yearly rate of stroke recurrence among patients with PFO is estimated at about 1.5–12% [7]. In case of our patient with atrial septal aneurysm and patent foramen ovale, two ischemic cerebral incidents took place within half a year. Cardiac origin of ischemic stroke is related to thrombus formation in left heart chambers, which atrial septal aneurysm predisposes to. It is also associated with the presence of venoarterial shunt that facilitates transfer of thrombotic material from the venous system into systemic circulation. Thus, thorough cardiac diagnostics should be performed in any case of unexplained cerebral stroke, particularly in young patients.

Venous strokes related to sinus and cerebral venous thrombosis are associated with other etiologies. Inflammatory background, especially suppurative mastoiditis, predominated before introduction of antibiotics, often leading to sigmoid sinus thrombosis. Currently, non-inflammatory causes of cerebral venous thrombosis prevail, such as pregnancy and puerperium, hypercoagulable states (protein S and protein C deficiency), dehydration, oral contraception, direct infiltration of the vein by a tumor, neurosurgical procedures and craniocerebral trauma [8]. Oral contraception is one of the most common causes of cerebral venous thrombosis, as in the case of Patient no. 1. Ethinyl estradiol promotes synthesis of proteins, including plasma clotting factors in the liver and in this mechanism increases the risk of thrombosis in the venous system. Epidemiological studies showed that incidence of cerebral venous thromboembolic disease in women taking oral contraception is 2–6 times greater compared to women who do not use oral contraceptives [9].

Clinical symptoms

Symptoms of stroke vary and depend on the region of ischemia. The most common symptoms of arterial stroke include paresis or hemiparesis, weakening or loss of sensation on one side of the body, motor or sensory aphasia. There may be also sudden, very severe dizziness and headache, cranial nerve symptoms, visual disturbances or memory impairment [3–6]. Patient no. 2 described in this paper initially presented with typical symptoms of arterial ischemic stroke such as aphasia and weakening of muscle force in the right upper limb. Occurrence of epileptic attack on the 2nd day of hospitalization was less typical. However, according to the literature, focal seizures or secondary generalized attacks may take place in the acute phase of arterial ischemic stroke (8.6% of cases), as it was in case of our patient [10].

Venous stroke may present with variable clinical picture, from scantily symptomatic to severe symptoms. Symptoms may change in time, as they depend on the activity of coagulation factors and thrombolysis, time of recanalization and collateral circulation. They are often non-specific and headache is the most common (75–95% of cases) symptom [11]. Among other symptoms, there is also nausea, vomiting, epileptic attacks and symptoms more commonly associated with arterial stroke such as motor and sensory deficits, visual and speech disturbances, as well as impairment of conscience. Patient no. 1 described in this publication presented with symptoms less typical for venous stroke such as aphasia and limb paresis. Clinical symptoms of venous stroke also depend on the location of involved venous sinus. For example, in case of superior sagittal sinus thrombosis headache is the predominating symptom, in transverse sinus thrombosis it is ear ache, in case



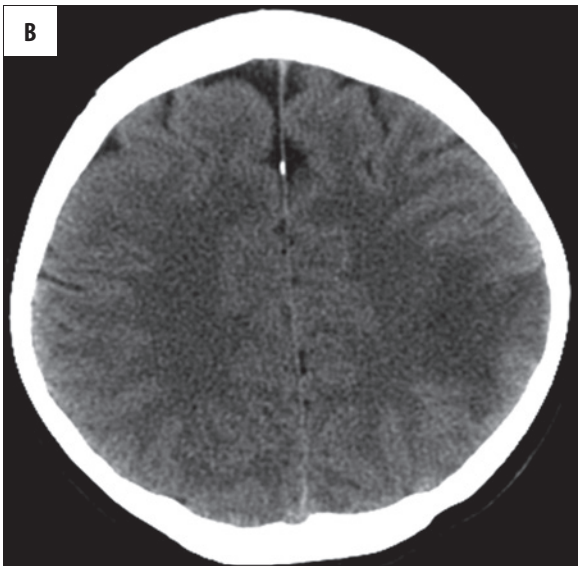


Figure 4. Patient with an arterial ischemic infarction, non-contrast CT images performed on the 2nd day after the onset of neurological symptoms. Infarction is visible as a hypodense lesion within cortex of the left temporo-parietal area (**A, B**). Small peripheral hyperdense dot (arrow) represents an area of hemorrhage not a thrombosed cortical vein (**A**).

of cavernous sinus thrombosis – conjunctival swelling and exophtalmia with local tenderness as well as oculomotor nerve damage, while in cortical vein thrombosis (as in our patient) aphasia, limb paresis or seizures prevail [8].

Prognosis in venous thrombosis is better than in arterial strokes. Regression of thrombotic lesions and symptoms is acquired in 70% of patients [8]. Permanent focal deficits remain in 6–20% of patients, epileptic seizures in 5–11%, and mortality varies from several to about 30%. On the other hand, ischemic stroke is the third most common cause of death in the USA (following cancer and myocardial infarction) and the main cause of severe disability [1]. Half of patients after ischemic stroke suffer from permanent neurological deficits.

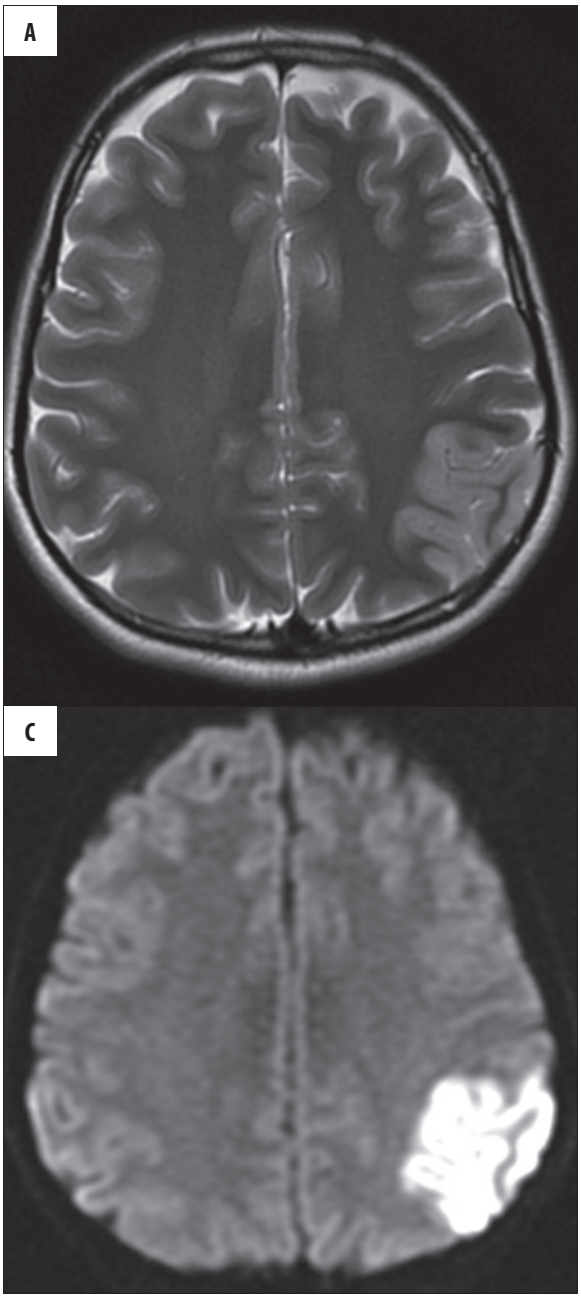
Imaging diagnostics

Imaging diagnostics of venous and arterial strokes is aimed at visualizing brain tissue lesions on one hand and localization of vascular pathology on the other. Emergency CT without contrast is the most commonly performed examination in the course of diagnosis of acute cerebral vascular events. Radiological diagnostics of venous and arterial strokes may be aided by administration of contrast during CT examination, MRI with or without contrast and vascular studies, i.a. CT angiography (CTA) or MR angiography (MRA).

Imaging of brain lesions

Both in acute arterial and venous stroke, a hypodense area of edema accompanied by minute/moderate mass effect (depending on the size of the lesion) appears in CT several hours after the onset of clinical symptoms. The term "infarction" is reserved for irreversible necrotic lesions. As opposed to arterial ischemia, tissue changes in the course of venous thrombosis are more often reversible and more frequently involve primary or secondary hemorrhage (in about 32% of patients) [12,13]. Multifocal, fingerlike, well demarcated hemorrhagic foci located centrally in the edematous region are typical for venous thrombosis. Significant hemorrhagic transformation of ischemic stroke occurs less frequently and is relatively more common in embolic strokes [14], as in the case of Patient no. 2. Characteristic linear (garland-like) contrast enhancement of cerebral cortex is apparent in arterial stroke following administration of contrast medium due to brain-blood barrier damage and local hyperemia (luxury perfusion). Contrast enhancement is usually seen about 3 days after the event. It is most prominent about the 3rd week and disappears about the 3rd month [1]. In venous stroke contrast enhancement is discrete and ensues from damage to blood-brain barrier.

On classic T1- and T2-weighted MR images as well as in FLAIR sequence edematous zones present in similar manner in the course of venous thrombosis and in arterial ischemia: they are hypointense on T1-weighted images and hyperintense on T2-weighted and FLAIR sequences. They may exhibit heterogeneous signal due to partial hemorrhagic transformation (variable signal is visible depending on the products of hemoglobin metabolism). Similar to CT study, a typical pattern of cortical enhancement is observed in MRI following administration of contrast medium in arterial stroke and weak enhancement is seen in venous stroke. DWI sequence is particularly useful for differentiation between arterial and venous stroke. This sequence allows for visualization of water diffusion in extracellular space. In ischemic stroke, which is associated with accumulation of water in intracellular space and increase in cell size (cytotoxic edema) as well as reduction of extracellular space, DWI sequence shows signs of limited water diffusion (restriction) visible as a hyperintense area on DWI images or hypointense area on ADC maps. The area of diffusion restriction in DWI is visible as soon as several minutes following appearance of clinical signs of ischemic



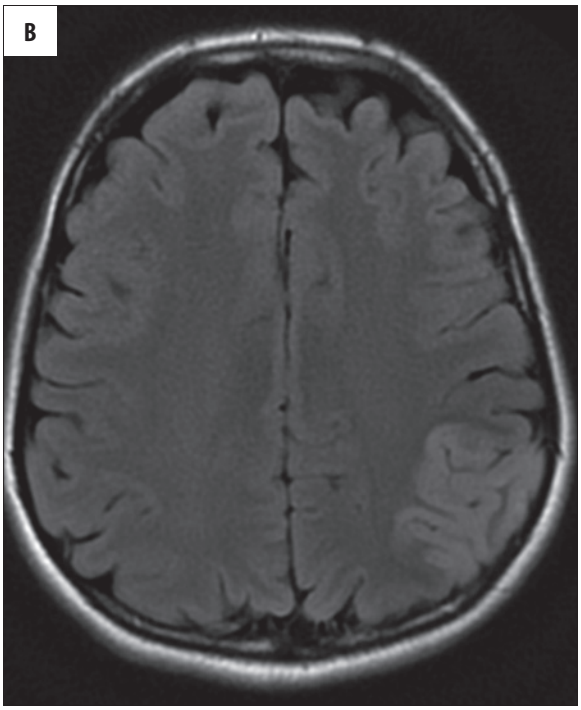


Figure 5. Patient with an arterial ischemic infarction. MR images without contrast injection on the 3rd day after the onset of neurological symptoms: (**A**) – T2-weighted, (**B**) – FLAIR images and (**C**) – DWI. The infarction is seen as a hyperintense lesion on T2-weighted and FLAIR images with strong diffusion restriction (**C**) typical for cytotoxic edema.

stroke. One should remember that after about 10–14 days vasogenic edema predominates, cell size decreases and diffusion restriction is no longer seen in DWI in the area of ischemic stroke. In order to unequivocally verify the presence of acute or subacute ischemic stroke an MRI study with DWI should be performed up to the 10th day from the appearance of clinical symptoms. As opposed to arterial stroke, in venous stroke there is no diffusion restriction or it may involve a limited area [12]. In case of our Patient no. 2, in whom morphology and location of the lesion as well as her age strongly suggested venous thrombosis, the DWI study made it possible to state the diagnosis of arterial ischemic lesion (Figure 5C).

Early diagnostics of ischemic stroke may be aided by CT and MR perfusion techniques. Such techniques enable imaging of ischemic area in an early phase based on reduction of Cerebral Blood Flow (CBF) and Cerebral Blood Volume (CBV) perfusion parameters and elongation of mean transit time (MTT) [13]. They are not routinely used for the diagnosis of ischemia, but are most often ordered before planned intravenous or intraarterial thrombolysis. Since such therapy was not planned in our patient perfusion studies were also not performed.

Assessment of lesion location both in CT and in MRI is another feature that allows for differentiation between venous and arterial stroke. Location of edema in venous thrombosis depends on the site of vascular occlusion. We differentiate between deep venous thrombosis and dural sinus or superficial venous thrombosis. Deep brain structures such as basal ganglia, thalamus, periventricular white matter are drained by single veins (e.g. vein of Galen) and a single sinus rectus. In venous thrombosis of those single vessels bilateral zones of edema symmetrically encompass the region of basal ganglia and both thalami [8]. In sinus and superficial vein thrombosis (superior sagittal sinus, transverse and sigmoid sinuses as well as cortical veins, including the vein of Labbe) signs of edema are noted within cerebral cortex and adjacent white matter and their location does not correspond to the areas of arterial supply [8]. In ischemic stroke related to occlusion of a large artery (ACA, MCA, PCA) typical shape of infarcted area allows for easy and definite diagnosis. Ischemic arterial strokes in the cortical region and at the border of two areas supplied by large vessels (as in Patient no. 2) pose a diagnostic problem requiring differentiation from strokes in the course of superficial vein thrombosis.

Imaging of vascular changes

Diagnostic imaging of arterial and venous strokes also encompasses visualization of vascular changes. In CT examination without contrast both thrombosed veins (especially the sinuses and vein of Labbe) and arteries (most often the main MCA stem) present with increase in density – picture of a hyperdense vessel. Hyperdense MCA image is noted in about 35–50% of occlusion cases, while pathological hyperdensity of venous sinuses is seen in about 25% of cases of venous thrombosis [8]. In Patient no. 1 the picture of hyperdense sigmoid and transverse sinuses as well as the vein of Labbe suggested venous thrombosis complicated by brain infarction (Figure 1). Chronic thrombi become isodense in non-contrast CT after about 14 days.

Picture of thrombosed vessels, particularly sinuses and cortical veins, is even more characteristic in non-contrast MRI. One should suspect thrombosis in MR when high signal appears within the lumen of a vessel. Physiologically, lumens of arterial and venous vessels show lack of signal (signal void) both on T1- and T2-weighted images and in FLAIR sequence due to rapid blood flow through the vessels. A thrombus causes slowing or obstruction of blood flow and loss of signal, which is best and earliest seen on T2-weighted images. With time, thrombus becomes well visible as a hyperintense strip within the vessel lumen on T1-weighted and FLAIR images due to metabolism of hemoglobin, which is transformed into methemoglobin (subacute thrombus after the 3rd day produces high signal in all images) [8,12], as seen in Patient no. 1 (Figure 3A–3D). It should be emphasized that this phenomenon enables visualization of thombi within even small cortical vessels or deep veins, which is difficult to attain in imaging studies.

Absence of contrast in the sinuses (so-called delta sign – loss of contrasting of superior sagittal sinus visible in transverse planes) is noted in about 25–30% of venous thrombosis cases following intravenous administration of contrast medium in CT and MR examinations [8,12]. Loss of full contrasting of left transverse sinus was apparent in Patient no. 1 (Figure 3E).

Arterial and venous phase angiographic studies performed using CTA or MRA are dedicated to assessment of vessel morphology and patency. Within occluded artery or vein in CTA no contrast flow is noted, while MRA shows segmental loss of signal (Figure 3F). CTA examination is more precise than MRA, but burdened with large dose of radiation and requires intravenous administration of iodine contrast, which not indifferent to the organism. CTA is necessary in arterial ischemic stroke before planned intravascular therapy. However, it seems that in suspected acute venous thrombosis venous phase CTA may be omitted, especially in young people, due to the possibility of non-invasive visualization of thrombi even in small-caliber vessels in MR examination (also without contrast). Venous phase CTA is decisively more useful for monitoring chronic thrombi, which do not exhibit such typical high signal on MR images.

Conventional DSA catheter angiography is currently performed only in diagnostically complex cases or when considering intravascular intervention in order to restore arterial flow.

Additionally, as in case of Patient no. 2, Doppler ultrasound studies of cervical vessels are also performed to assess the patency of arteries and jugular veins.

Treatment

Patient with acute stroke of venous or arterial origin requires immediate treatment in the hospital. Therapeutic management must be multidirectional, including causative treatment (reducing the size of thrombus) on one hand and symptomatic treatment (antiedematous, antiseizure, analgetic therapy, etc.) on the other. In venous thrombosis antithrombotic treatment involves administration of low-molecular-weight heparin and oral anticoagulants [8,12]. Complete recanalization is more frequent if thrombosis involves superior sagittal sinus and sinus rectus than in case of transverse sinus or sigmoid sinus thrombi. However, complete recanalization is not necessary for full recovery. Causative treatment in ischemic arterial stroke may be conducted in various ways, beginning with least effective therapy with oral anticoagulants and antiplatelet drugs, intravenous thrombolysis with tissue plasminogen activator (tPA) within 4.5 hours from the beginning of stroke or intraarterial thrombolysis and thrombectomy within 6 hours from the onset of symptoms [1].

Conclusions

One should remember that in young people acute vascular neurological incident might be associated with both arterial and venous stroke. Venous strokes are more common in a young population, particularly among women with accompanying risk factors, e.g. hormonal contraception. Assessment of cerebrum and vessels in CT and MRI studies, even without contrast, is important. Bilateral zones of edema with signs of multifocal hemorrhagic transformation should suggest the diagnosis of venous strokes. In young patients MR should be the second study to aid differentiation between arterial and venous stroke following CT examination. Particularly useful is the DWI sequence that unequivocally confirms ischemic origin of the stroke in the acute or subacute phase as well as T1-, T2-weighted and FLAIR images that enable good visualization of thrombi within vessels, particularly veins (even without contrast). MR examination is indicated especially in young females due to lack of exposure to ionizing radiation and iodine contrast agents used in CT and CT angiography studies.

Radiologist's knowledge of symptoms and additional clinical information (e.g. risk factors) may bring the diagnosis closer and enable planning of further imaging diagnostics. Differentiation between arterial and venous stroke is clinically important, as it influences patient treatment and determines prognosis.

References:

1. Howard AR: Choroby naczyniowe mózgu. In: Brant EW, Helms AC (eds.): Podstawy diagnostyki radiologicznej. Medipage, Warszawa, 2007; tom I: 93–120 [in Polish]
2. Nedeltchev K, der Maur T, Georgiadis D et al: Ischaemic stroke in young adults, predictors of outcome and reccurence. J Neurol Neurosurg Psychiatry, 2005; 76: 191–95
3. Putaala J, Metso A, Metso T et al: Analysis of 1008 consecutive patients aged 15 to 49 with first-ever ischemic stroke: the Helsinki young stroke registry. Stroke, 2009; 40: 1195–203
4. Lee T, Hsu W, Chen C et al: Etiologic study of young ischemic stroke in Taiwan. Stroke, 2002; 33: 1950–55
5. Rasura M, Spalloni A, Ferrari M et al: A case series of young stroke in Rome. Eur J Neurol, 2006; 13: 146–52
6. Carolei A, Marini C, Ferranti E et al: A prospective study of cerebral ischemia in the young. Analysis of pathogenic determinants. The National Research Council Study Group. Stroke, 1993; 24: 362–67
7. Di Tullio M, Sacco RL, Gopal A et al: Patent foramen ovale as a risk factor for cryptogenic stroke. Ann Intern Med, 1992; 117(6): 461–65
8. Leach J L, Fortuna RB, Blaise VJ et al: Imaging of cerebral venous thrombosis: current techniques, spectrum of findings and diagnostic pitfalls. Radiographics, 2006; 26: 19–43
9. Lidegaard Ø, Nielsen LH, Skovlund CW et al: Risk of venous thromboembolism from use of oral contraceptives containing different progestogens and oestrogendoses: Danish cohort study, 2001-9. BMJ, 2011; 343: d6423
10. De Reuck JL: Napady padaczkowe i padaczka związane z udarem mózgu. Neurologia i Neurochirurgia Polska, 2007; 41: 144–49 [in Polish]
11. Perenc A: Ból głowy jako objaw wewnątrzczaszkowej zakrzepicy żylnej, Via Medica, 2011; 13(1–2): 22–27 [in Polish]
12. Bronwyn EH: Zakrzepica żył głębokich mózgu. In: Osborn A et al. (eds.): Diagnostyka obrazowa. Mózgowie. (Diagnostic Imaging. Brain), Medipage, Warszawa, 2012; 1(4): 154–58 [in polish]
13. Walecki J, Bulski T, Sklinda K: Choroby naczyniopochodne ośrodkowego układu nerwowego. In: Walecki J (eds.): Postępy Neuroradiologii. PAN, Warszawa, 2007; 472–97 [in Polish]
14. Gurański K, Podemski R, Ejma M. Ukrwotocznione zawały mózgu. Udar mózgu, 2001; 3(2): 77–84 [in Polish]