

# Ischemic stroke/PGS/diagnosis

## Definition

The following division is possible in most cases only with knowledge of the subsequent development of the condition. We start the treatment at the moment when we often cannot categorize the stroke.

### Complete Stroke:

rapidly developing clinical signs of focal cerebral palsy, lasting more than 24 hours or leading to death, unless clinical, laboratory, and basic imaging tests indicate another cause of neurologic deficit.

### *Transient ischemic attack (TIA):*

rapidly developing clinical signs of focal cerebral palsy or impaired monocular vision that typically last less than 1 hour, but no longer than 24 hours, unless clinical, laboratory, and imaging tests indicate another cause of neurological deficit.

## Clinical picture

Sudden development of focal neurological symptoms of central origin manifesting according to the territory of the affected cerebral artery (weakness up to paralysis and/or impaired sensitivity of half of the body, impaired symbolic functions, deviation of the head and eyeballs, visual paresis, visual field excursions, diplopia, sudden dizziness or sudden fall in connection with previous central neurological symptoms, amaurosis, coordination disorder or other symptoms depending on the location of the lesion.)

Lacunar infarcts are mainly manifested by isolated motor and/or sensory deficit, atactic hemiparesis, dysarthria, "clumsy hand". Rarely, ischemic stroke is manifested by headache, initial vomiting, impaired consciousness, even more rarely is the initial symptom epileptic paroxysm.

Minor and Major Stroke Symptoms

## Causes

A number of diseases can etiologically contribute to the occlusion or narrowing of a cerebral vessel with reduced blood flow and subsequent ischemia of part of the brain tissue (Tab. 1).

*'Tab. 1 - Overview of the causes of ischemic stroke*

The most common causes of ischemic stroke'
<ul style="list-style-type: none"><li>▪ <b>Atherosclerosis</b> with involvement of the neck or intracranial arteries</li><li>▪ <b>Embolization</b> (cardiac or other central source)</li><li>▪ <b>Paradoxical embolization</b> (open foramen ovale)</li><li>▪ <b>Microangiopathy</b> of perforating arterioles</li><li>▪ <i>'Dissection</i> of the carotid artery</li><li>▪ <b>Thrombophilic conditions</b></li><li>▪ <b>Thrombosis</b> cerebral venous shunt</li><li>▪ <b>Vasculitis and angiopathy</b></li><li>▪ <b>Vasospasm</b> in subarachnoid hemorrhage</li><li>▪ <b>Pressure of vascular structures</b> during intracranial expansion</li><li>▪ <b>Infection</b></li><li>▪ <b>Genetic diseases</b></li></ul>

## Classification

### According to etiopathogenesis

- *Cardioembolization* - atrial fibrillation, condition after myocardial infarction, thrombus in the left ventricle or left atrium, akinetic segment of the left ventricle with aneurysm, [ [dilated cardiomyopathy]], valve replacements, myxoma atrial, infective endocarditis.
- *Disease of large arteries* - macroangiopathic defect with malacia size over 1.5 cm, mostly in patients with carotid stenosis over 50 % and finding infarction in the ipsilateral corticosubcortical area or subcortical area, by the mechanism of embolization or hypoperfusion .
- *Small artery disease* (lacunar infarcts) – ischemia in the subcortical region, typically in the basal ganglia, thalamus, capsula interna or brainstem up to a size of 1-1.5 cm.
- "Other cause" - collagenoses, vasculitis, non-inflammatory vasculopathy (fibromuscular dysplasia, Moya moya,

amyloid angiopathy) and others.

### According to the affected artery

#### a. cerebri anterior' - hemiparesis with predominance on the lower limb

- On the left – transcortical motor aphasia, behavioral disorders – apathy, abulia or loss of inhibitions, ideomotor apraxia within the disconnection syndrome
- On the right – motor or spatial neglect, behavioral disorders

**a. cerebri media'** - it depends on the extent of the involvement of the artery, symptoms of involvement of the frontal, parietal and temporal lobes the dominant symptoms are hemiparesis to hemiplegia, central involvement of the facial nerve, hemisensitive defects, homonymous disturbances of the visual field and visual paralysis to the other side. A lesion in the area of speech dominance leads to aphasia and apraxia, agnosia, a lesion secondary to a disorder of spatial perception.

- Occlusion of the main superior anterior branch - involvement of the frontal lobe, faciobrachially accentuated hemiparesis, visual palsy, motor aphasia and apraxia
- Closure of the main inferior posterior branch - mainly sensory deficit, homonymous visual field defects, possibly sensory aphasia x neglect according to affected hemisphere.
- Closure of the main trunk - involvement of the basal ganglia, internal capsule and hemiparesis up to plegia and sensitivity disorders on one half of the body, often with a dramatic progression of findings.
- Closures of the lenticostriate and striate arteries lead to lacunar infarcts with involvement of the basal ganglia and internal capsule, with sensorimotor impairment and eventually. with extrapyramidal motor manifestations.

#### a. cerebri posterior'

- Closure of the segment before a. communicans posterior – sensorimotor hemiparesis and hemianopsia
- Closure of the segment behind a. communicans posterior - left: hemianopsia, neuropsychological deficit (alexia without agraphia, constructional apraxia, agraphia, acalculia), transcortical sensory aphasia, with a right-sided defect: spatial perception disorder, visual neglect to the left, prosopagnosia

**a. basilaris'** - circulation disorders in this area are manifested either by the involvement of the encircling or perforating arterioles or by a significant deficit when closing the entire trunk. In the clinical picture, there are various combinations of involvement of the brainstem, cerebellum, thalamus, temporal and occipital lobes. In the area of brainstem involvement, in addition to limb paresis and sensory disturbances, it is necessary to pay attention to symptoms such as dysphonia, palsy of the soft palate, hiccups, impaired algic and thermal sensitivity in the face, hypo to ageusia, central vestibular syndrome, tinnitus, paresis of horizontal or vertical vision and other disorders of oculomotor innervation (subjectively perceived often as diplopia, sometimes with oscillopsia of one of the double images).

## Investigation and treatment of acute ischemic stroke

In the optimal case, treatment should take place in specialized workplaces - in a stroke unit or stroke beds, with provision of an intensive care unit (ICU).

Ischemic stroke is an acute condition, therefore after the patient's arrival at the hospital, it primarily requires classic care and examination/assurance of basic vital functions. See the algorithm diagram of the investigation procedure in Chapter 2, emphasizing the timely performance of blood sampling, especially coagulation testing, as waiting for the results of late samples could frustrate subsequent efforts for accurate diagnosis and timely thrombolytic therapy.

Throughout the investigation, it should be kept in mind that as a result of a decrease in cerebral blood flow below 20 ml/100 g tissue/min, function has died out in a certain area and it is possible that the integrity of the cell has not yet been damaged to a greater extent. We therefore assume that part of the tissue is in a state of ischemic penumbra, in which we can provide assistance to return local circulation and oxygenation.

The basic treatment of the patient is followed by a specific examination procedure and a therapeutic algorithm according to the examination results (diagram).

**The essential point of the anamnesis is the time indication of the onset of focal symptoms! We find out the time when the patient was last without difficulties. If the patient wakes up in the morning at 7 o'clock with already developed neurological symptoms, and before that he was last without problems in the evening, he is most likely already outside the therapeutic window for thrombolytic treatment.**

In addition to a specific examination procedure and possible application of thrombolytic treatment (see diagram), overall care must be provided as for any patient with an acute neurological disease:

- Elevated position of the head and trunk 30-40 degrees.
- Correction of oxygen saturation below 96 % - with glasses or a mask, possibly. by supporting ventilation
- Careful correction of blood pressure (BP) above 220/120 (caution: limit for thrombolysis 185/110)
- Correction of blood glucose above 10 mmol/l with insulin

- Body temperature correction above 37.5 °C
- Elimination of the risk of vomiting and aspiration of prokinetics, antiemetics, possibly. by nasogastric tube - prevention of thromboembolic disease by bandages of the lower limbs

If it is not possible to treat the patient with thrombolytic therapy, immediate initiation of antiplatelet therapy, administration of ACE inhibitors (if there is no tendency to hypotension) and statins is indicated.

#### Note:

*urgent carotid endarterectomy of symptomatic hemodynamically significant stenosis within 6 hours of the onset of neurological symptoms with a negative native CT of the brain*

**Tab. 2 - NIHSS - Stroke Scale**

Investigation	Score	Investigation	Score
<b>Level of Consciousness</b>		<b>Motor - lower limb</b>	
Vigilant	0	(lying on back, hold at 30°)	
Subdued (awakenable by mild stimulation)	1	Holds over 5 s	0
Sleepy (repeated or strong stimulation)	2	Drops to middle position before 5 s	1
Reflex movements/no response	3	Fall on the bed within 5 s	2
		No performance	3
<b>Answers - name month and age</b>		<b>Ataxia of the limbs</b>	
Both correct	0	None	0
One right	1	Present on one limb	1
None correctly	2	Present on two limbs	2
<b>Following instructions</b>		<b>Sensitivity</b>	
He hears both correctly	0	Normal	0
He will listen to one correctly	1	Slightly reduced	1
Both wrong	2	Severe to total loss	2
<b>Field of vision</b>		<b>Neglect</b>	
No vision loss	0	None	0
Partial hemianopsia	1	Visual, tactile or auditory	1
Complete hemianopsia	2	More than 1 modality/deep lethargy	2
<b>Pauses of n. facialis</b>		<b>Dysarthria</b>	
None	0	None	0
Light (NL groove smoothing)	1	Mild to moderate (understandable)	1
Partial (slight drop)	2	hard (unintelligible)	2
Complete	3		
<b>Motor - upper limb</b>		<b>Speech</b>	
(45° lying on the back, possibly 90° sitting)	0	Normal	0
Holds over 10 s	1	Mild impairment (dysnomia, paraphrase, mild aphasia)	1
Drop 10 s ago	2	Moderate phatic disorder	2
Only effort against gravity	3	Unable to speak x global aphasia	3
No performance			
<b>Overall Score:</b>			
Optimum:			
Pessimum: 20 (some points are mutually exclusive in increasing negative value)			

'Tab. 3 – Overview of contraindications to thrombolytic treatment

Main contraindications of thrombolytic treatment'
<ul style="list-style-type: none"><li>■ Mild symptoms (NIHSS &lt; 4) or rapidly improving symptoms</li><li>■ Signs of bleeding on brain CT</li><li>■ Seizure symptoms of CMP or suspected subarachnoid hemorrhage</li><li>■ Serious head injury in the last 3 months</li><li>■ Serious surgery or trauma in the previous 3 months</li><li>■ GIT or urinary tract bleeding in the previous 3 weeks</li><li>■ Difficult to correct blood pressure &gt; 185/110 mmHg</li><li>■ Glucose level &lt; 50 mg/100 ml or &gt; 400 mg/100 ml</li><li>■ Arterial puncture in a non-compressible place or lumbar puncture in the previous week</li><li>■ Plate count &lt; 100,000</li><li>■ Heparin therapy – effective – for increased APTT</li><li>■ Current anticoagulation treatment – with INR &gt; 1.7</li></ul>

## Subacute phase of ischemic stroke - recommended examinations

internal and cardiology examination

transthoracic and transesophageal echocardiography

Holter monitoring ECG and blood pressure

special laboratory examination

- Thrombophilic conditions (examination of major coagulation, protein C and S, factor II, V, APC resistance, lupus anticoagulant, antiphospholipid antibodies).
- Metabolic disorders (investigation of MRHFR mutation, homocysteine – treatment with folate + vit.B1, B6).

special sonographic examination (microembolization, determination of cerebrovascular reserve capacity)

SPECT of the brain using the HMPAO method, including determination of cerebrovascular reserve capacity

## Secondary prevention of ischemic stroke

- **Antiplatelet therapy** - acetylsalicylic acid alone, or in a more effective combination with dipyridamole with controlled release, in case of intolerance and in other specific cases treatment with clopidogrel
- **ACE inhibitors** - if there is no tendency to hypotension
- **Statins** - low doses even in the case of a normal lipidogram
- **Anticoagulation therapy** - strictly individualized in patients with a high risk of reembolization - heparin, LMWH. Warfarin sodium – full anticoagulation treatment – start as soon as possible in TIA or minor stroke, in case of smaller ischemic lesion without expansive manifestations after 1–2 weeks, after 4–6 weeks in extensive ischemic lesion with expansive behavior and perifocal edema.
- **Carotid endarterectomy** or endovascular intervention - the indication falls within the competence of specialized workplaces, TIA-RIND- minor stroke with an interval of days - weeks, for more extensive lesions an interval of > 6 weeks.