Stroke/ PGS

Introduction

Circulatory failure is the second to third largest cause of death in most industrialized countries. The incidence of strokes (ischemic and hemorrhagic together) in the Czech Republic is twice as high as in other medically developed countries, it is around **300 cases** per **100 000 inhabitants** per year. Ischemic strokes account for **80–85 %** of all cerebrovascular accidents (CVA). Hemorrhage, regardless of the cause, accounts for about 20% of total CVA, **10–15 %** for spontaneous intracerebral hemorrhage and the remaining 5% is due to spontaneous subarachnoid or intraventricular hemorrhage. Therapeutic options for CVA are currently undergoing significant changes, which, due to the acute nature of the condition, must be constantly monitored and supplemented according to the latest findings. In addition to the diagnostic progress given mainly by neurological applications of new imaging methods, the possibilities of timely and effective treatment have also significantly expanded.

Ischemic stroke

Definition

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

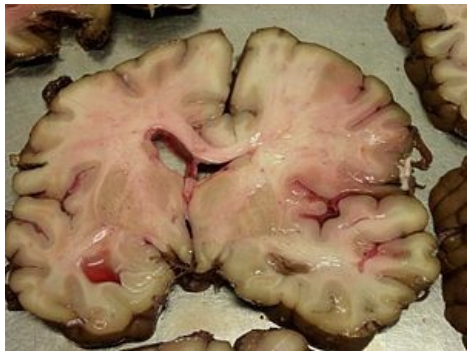
Completed stroke:

Rapidly developing clinical signs of focal brain disease lasting more than 24 hours or leading to death, unless clinical, laboratory and basic imaging tests suggest another cause of neurological deficit.

Transient ischemic attack (TIA):

Rapidly developing clinical signs of focal brain disease or monocular vision impairment, which typically last less than 1 hour, but not more than 24 hours, unless clinical, laboratory and imaging tests suggest another cause of neurological deficit.

Clinical picture



Acute stroke

Sudden development of focal neurological symptoms of central origin manifested according to the territory of the affected cerebral artery (weakness to paralysis and / or sensitivity of the body, symbolic function, deviation of the head and eyeballs, visual paresis, visual field loss, diplopia, sudden dizziness or sudden fall in conjunction with previous central neurological symptoms, amaurosis, incoordination, or other symptoms depending on the location of the lesion.)

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

Small and large symptoms of stroke

Causes

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

Tab. 1 - Overview of the causes of ischemic stroke

The most common causes of ischemic stroke
▪ **Atherosclerosis** with carotid or intracranial artery disease ▪ **Embolization** (cardiac or other central source) ▪ **Paradoxical embolization** (open foramen ovale) ▪ **Microangiopathy** of perforating arterioles ▪ Carotid artery **dissection** ▪ **Thrombophilic conditions** ▪ **Thrombosis** of cerebral venous canal ▪ **Vasculitis and angiopathy** ▪ **Vasospasm** in subarachnoid hemorrhage ▪ **Oppression of vascular structures** during intracranial expansion ▪ **Infection** ▪ **Genetic diseases**

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, atrial myxoma, infectious endocarditis.
- *Large arterial disease* - a macroangiopathic defect with a malacia size over 1.5 cm, mostly in patients with carotid stenosis over 50% and finding a heart attack in the unilateral corticocortical area or subcortical area, by the mechanism of embolization or hypoperfusion.
- *Diseases of small arteries* (lacunar infarcts) - ischemia in the subcortical area, typically in the basal ganglia, thalamus, internal capsule or brainstem up to 1-1.5 cm in size.
- *Another cause* - collagenosis, vasculitis, non-inflammatory vasculopathy (fibromuscular dysplasia, Moyamoya, amyloid angiopathy) and others.

According to the affected artery

a. cerebri anterior - hemiparesis with a predominance of the lower limb

- Left - transcortical motor aphasia, behavioral disorders - apathy, abulia or loss of inhibitions, ideomotor apraxia within the disconnection syndrome
- Right - motor or spatial neglect, behavioral disorders

a. cerebri media - depends on the extent of arterial involvement, symptoms of frontal, parietal and temporal lobe involvement predominant symptoms are hemiparesis to hemiplegia, central facial nerve involvement, hemi-sensitive defects, homonymous visual field disorders and visual paralysis to the other side. Lesions in the area of speech dominance lead to aphasia and apraxia, agnosis, lesions bilateral to impair spatial perception.

- Closure of the main upper anterior branch - frontal lobe involvement, faciobrachial accentuated hemiparesis, visual paralysis, motor aphasia and apraxia
- Closure of the main lower posterior branch - mostly sensitive deficit, homonymous field of view defects, or sensory aphasia x neglect according to the affected hemisphere.
- Closure of the main trunk - involvement of the basal ganglia, internal capsule and hemiparesis to plegia and sensory disorders in one half of the body, often with dramatic progression of the finding.
- Lenticular and strial artery occlusions lead to lacunar infarcts with basal ganglia and internal capsule involvement, sensorimotor involvement and event with extrapyramidal motor manifestations.

a posterior cerebral artery

- Segment closure in front of posterior communicative artery - sensorimotor hemiparesis and hemianopsia
- Segment closure behind the posterior communicator a .

a. basilaris - circulatory disorders in this area are manifested either by involvement of the surrounding or perforating arterioles or a significant deficit in the closure of the entire strain. In the clinical picture, there are various combinations of brainstem, cerebellum, thalamus, temporal and occipital lobe disorders. From the area of brainstem involvement, in addition to limb paresis and sensory disorders, it is necessary to pay attention to symptoms such as dysphonia, soft palsy, hiccups, algic and thermal sensory disorders in the face, hypo to ageusia, central vestibular syndrome, tinnitus, horizontal or vertical paresis and other ocular innervation disorders (subjectively often perceived as diplopia sometimes with oscillopsia of one of the duplicate images).

Examination and treatment of acute ischemic stroke

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

Ischemic insult is an acute condition, so after the patient's arrival in the hospital, it primarily requires classical care and examination / maintenance of basic vital functions. There is an emphasis on timely blood sampling, especially coagulation testing, as waiting for late sampling results could frustrate subsequent efforts for accurate diagnosis and early thrombolytic treatment.

Throughout the examination, it should be borne in mind that due to a decrease in cerebral blood flow below 20 ml / 100 g tissue / min, it has become extinct in a certain area of function and it is possible that cell integrity has not been impaired to a greater extent. We assume that part of the tissue is in a state of ischemic penumbra, in which we can provide help to return local circulation and oxygenation.

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

The crucial point of the anamnesis is the time of the onset of focal symptoms! We find out the time when the patient was last without problems. If the patient wakes up at 7 o'clock in the morning with already developed neurological symptoms, and has previously had no problems in the evening, he is probably already out of the therapeutic window for thrombolytic treatment.

In addition to the specific examination procedure and the possible application of thrombolytic therapy (see diagram), general care should be provided as for any patient with acute neurological disease:

- Elevated position of the head and torso 30-40 degrees.
- Oxygen saturation correction below 96% - glasses or mask, event. ventilation support
- Careful correction of blood pressure (BP) above 220/120 (attention: limit for thrombolysis 185/110)
- Glycemic correction above 10 mmol / l insulin
- Body temperature correction above 37.5 ° C
- Elimination of the risk of vomiting and aspiration of prokinetics, antiemetics, event. nasogastric tube - prevention of thromboembolic disease by lower limb bandages

If the patient cannot be treated with thrombolytic therapy, immediate initiation of antiplatelet therapy, ACE inhibitors (if not prone to hypotension) and statins are indicated.

Note:

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

Tab. 2 - NIHSS - stroke scale

Examination	Score	Examination	Score
Level of consciousness Vigilant Muted (awakened by mild stimulation) Sleeping (repeated or strong stimulation) Reflex movements / no answer	0 1 2 3	**Motor - Lower limb** (lying on your back, holding at 30 °) Holds for over 5 seconds It drops to the middle position 5 s ago Fall on the bed within 5 s No performance	0 1 2 3
Answers - Name, month and age Both right One right None correct	0 1 2	**Ataxia of the limbs** None Present on one limb Present on two limbs	0 1 2
Fulfillment of instructions He will listen to both correctly He will listen to one correctly Both incorrectly	0 1 2	**Sensitivity** Normal Slightly reduced Heavy to complete loss	0 1 2
Field of view No vision loss Partial hemianopsia Complete hemianopsia	0 1 2	**Neglect** None Visual, tactile or auditory More than 1 modality / deep inertia	0 1 2
Facial paralysis None Light (NL groove smoothing) Partial (slight decrease) Complete	0 1 2 3	**Dysarthria** None Mild to moderate (understandable) Difficult (incomprehensible)	0 1 2
Motorized - upper limb (45° lying on your back, possibly 90° sitting) Holds for more than 10 seconds Decline 10 s ago Only efforts against gravity No performance	0 1 2 3	**Speech** Normal Mild disorder (dysnomia, paraphrase, mild aphasia) Moderate fatal disorder Unable to speak x global aphasia	0 1 2 3
Total Score: Optimum: Pessimum: 20 (some points are mutually exclusive increasing the negative value)			

Tab. 3 - Overview of contraindications to thrombolytic therapy

The main contraindications to thrombolytic treatment
▪ Non-severe symptoms (NIHSS <4) or rapidly improving symptoms ▪ Signs of bleeding on brain CT ▪ Seizures of stroke or subarachnoid hemorrhage suspicion ▪ Serious head injury in the last 3 months ▪ Severe surgery or trauma in the previous 3 months ▪ Bleeding into the GIT or urinary tract in the previous 3 weeks ▪ Difficult to correct blood pressure> 185/110 mmHg ▪ Glucose level <50 mg / 100 ml or> 400 mg / 100 ml ▪ Artificial puncture in an incompressible place or lumbar puncture in the previous week ▪ Number of plates <100,000 ▪ Heparin therapy - effective - in increasing APTT ▪ Current anticoagulant therapy - with INR> 1.7

Subacute phase of ischemic stroke - recommended examinations

internal and cardiological examination

transthoracic and transesophageal echocardiography

Holter ECG and blood pressure monitoring

special laboratory examination

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

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

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

Secondary prevention of ischemic stroke

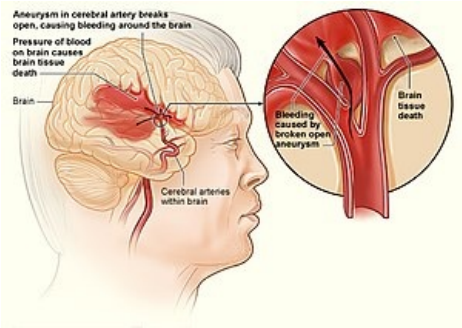
- **Antiplatelet therapy** - acetylsalicylic acid alone or in a more effective combination with dipyridamole controlled release, intolerance and in other specific cases treatment with clopidogrel
- **ACE inhibitors** - if there is no tendency to hypotension
- **Statins** - low doses even in the case of a normal lipidogram
- **Anticoagulant therapy** - strictly individual in patients at high risk of re-embolization - heparin, LMWH . Warfarinum natricum - full anticoagulant therapy - initiation as soon as possible in TIA or minor stroke, in a small-scale ischemic lesion without expansive manifestations after 1-2 weeks, after 4-6 weeks in a large 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 larger lesions an interval> 6 weeks.

Hemorrhagic stroke

Hemorrhagic CVAs are divided into two large, partially co-occurring groups: intracerebral hemorrhage and subarachnoid or intraventricular hemorrhage.

Definition

Bleeding into brain tissue, possibly with associated ventricular bleeding or subarachnoid bleeding .



Hemorrhagic stroke

Clinical picture

Same as ischemic stroke or finding very rapidly progressing to loss of consciousness or early onset of epileptic manifestations. Clear resolution is only possible on the basis of an imaging examination.

Causes

Tab. 4 - Overview of the most common causes of spontaneous intracerebral hemorrhage

The most common causes
▪ Small vessel disease ▪ Artificial hypocoagulation (anticoagulation treatment) ▪ Amyloid angiopathy ▪ Cavernous angioma ▪ Arteriovenous malformations ▪ Bleeding into an expansive lesion ▪ Bleeding manifestations of infections ▪ Hemorrhagic transformation of ischemic infarction ▪ Intracranial venous thrombosis

Classification

By location:

typical central bleeding - "hypertonic" intracerebral bleeding based on small vessel disease complex;

atypical lobar hemorrhage - intracerebral hemorrhage based on the above causes (Tab.4).

Examination procedure and treatment

The same algorithm as in any acute neurological patient or in a patient with impaired consciousness, possibly as in patients with intracranial hypertension syndrome, with emphasis on early CT of the brain and subsequent transport to the ICU (intensive care unit) care regardless of the type of bleeding and any good clinical condition of the patient.

In the case of atypical hemorrhage, it is necessary to consult the neurosurgical department with regard to further investigation of the causes and consideration of a possible neurosurgical intervention (decompression, ventricular drainage). In younger patients without risk factors, it is necessary (even with a delay) to exclude the source of bleeding at any location (MR brain, MR angio, DSA, CT angio).

Treatment of spontaneous intracerebral hemorrhage and prevention of its progression takes place only in the relevant ICU.

- blood pressure control (correction of systolic blood pressure above 180 in hypertensive patients, optimally up to 160)
- correction of hypocoagulation disorder (application of vitamin K, frozen plasma, recombinant activated plasma factor VII).
- reduction of intracranial pressure - antiedematous therapy - mannitol
- analgesia / relaxation, artificial lung ventilation - permissive hypocapnia
- barbiturate coma
- hypothermia
- elimination of intracranial pressure fluctuations - laxatives, antitussives
- consideration of an indication for decompression craniectomy

Subarachnoid hemorrhage

Definition

Blood penetration into the leptomeningeal space, ie the intermeningeal space between the pia mater and the arachnoid. It can be combined with parenchymal, which either secondarily penetrated into the cerebral sheaths, or, conversely, was formed secondarily by blood penetration into the cerebral parenchyma.

Clinical picture

Sudden headache (in seconds to minutes) is the only sign of subarachnoid hemorrhage in 20%. There are also nausea and vomiting, focal neurological deficit with the possibility of rapid progression of qualitative and quantitative disorders of consciousness, meningeal syndrome (often up to about 6 hours after the event). Bleeding often occurs during increased physical exertion. In clinical practice, the classification according to WFNS (World Federation of Neurological Surgeons) or Hunt and Hesse is used (Table 6).

Tab 6. - Overview of classifications for scaling the clinical picture of subarachnoid hemorrhage

WFNS classification			**Hunt and Hesse classification**	
Degree	GCS	Deposit finding	Degree	Clinical signs
I	15	No	I	Mild headaches, meningism
II	14-13	No	II	Severe headaches, meningism, disorders of cranial nerve V
III	14-13	Yes	III	Somnolence, mild focal symptoms
IV	12-7	Yes/ no	IV	Stupor, hemiparesis, vegetative disorders
V	6-3	Yes / no	V	Coma

Causes

Tab 5. Overview of the most common causes of subarachnoid hemorrhage

The most common causes
Aneurysm - 85%
Perimesencephalic bleeding - 10%
Rupture of the atherosclerotic vascular wall
Arterial dissection
Fungal aneurysms

Examination procedure and treatment

The same algorithm as in any acute neurological patient (Chapter 2) or in a patient with impaired consciousness (Chapter 3) or as patients with intracranial hypertension syndrome (Chapter 4?), With emphasis on **early brain CT**, which has up to 98% sensitivity in during the first 12 hours, for older bleeding, **brain MR** is recommended. If the CT finding is negative but subarachnoid hemorrhage is suspected from the clinical picture,**lumbar puncture** is required.

Positivity of CT (MR) findings or positivity of lumbar puncture examination are indications for consultation of the neurosurgical department, because causal treatment and prevention of rebleeding is possible only through neurosurgery or interventional neuroradiological procedures. For this reason, the next examination procedure (DSA) should only be performed at a workplace capable of these procedures. Therefore, it is necessary to ensure the transport of the patient by ambulance accompanied by a doctor. Depending on the overall condition, depending on the GCS, patient transport is indicated in some cases only after the intensivist has previously secured and stabilized vital functions.

Causal treatment is fully within the competence of neurosurgery and / or interventional neuroradiology by performing open surgery (clipping) and / or endovascular techniques (coiling).

Further prevention of complications of subarachnoid hemorrhage - can take place in the department of neurointensive care. (3H therapy, transcranial doppler monitoring, prevention and treatment of vasospasms (nimodipine iv / po, papaverine iv), laxatives, antitussives).

Links

Related articles

- ws: Cévní mozkové příhody/PGS
- Brain ischemia

Links and literature

- European Stroke Initiative, Recommendations 2008, Ischaemic Stroke,Prophylaxis and Treatment
- Doporučený postup pro diagnostiku a léčbu pacientů s mozkovým infarktem, TIA, Cerebrovaskulární sekce České neurologické společnosti.

External links

- www.cmp.cz (http://www.cmp.cz/jnp/cz/index.html)