Aspiring medical professionals should know and differentiate between the various stroke symptoms of ischaemic stroke. Furthermore, they must be familiar with the basics of treatment to be able to respond effectively in an emergency. This article thoroughly and compactly presents the knowledge required for this, and is optimal both for preparing for the exam and clinical application.

Anatomical and Pathophysiological Considerations of Stroke

A stroke is a cerebrovascular accident or CVA which is characterized by loss of adequate blood supply to the brain. Ischaemic stroke is defined as a circulatory disorder of a specific region of the brain. This illness is quite common in our society and manifests in many different causes and symptoms. As it is very vulnerable tissue, the brain must be quickly and adequately treated to minimize any long-term damage.

Circulatory disorders in the brain are generally referred to as “strokes”. This term is quite vague and encompasses different symptoms. Primarily we differentiate between cerebral ischaemia and haemorrhagic infarctions, which form as a result of haemorrhage.
Cerebral blood supply

An ischaemic stroke results in minimal neuronal circulation, and subsequent failure of the neurons. In order to understand this illness, its effects and its classifications, you should be very familiar with the anatomy behind the supply of blood to the brain.

Essentially the brain is supplied by two circulatory systems, anterior and posterior circulation. The AA. Carotidinterna forms the carotid artery system. At the base of the brain, the A. carotid divides in the so-called carotid T into the A. cerebri media and the A. cerebri anterior and thereby forms an anterior circulation. A. cerebri media supplies the majority of the white brain matter, portions of the frontal, temporal and parietal lobe, and the basal ganglia. The A. cerebri anterior ensures circulation of the structures near the median and the margo superior cerebri.

The posterior circulation (= vertebrobasilar artery system) forms from the merging of the paired Aa. vertebrales into the unpaired A. basilaris. This passes cranially along the front of the brain stem and branches into the Aa. cerebriposteriores at the so-called head of the basilaris artery. The posterior circulation is thus responsible for circulation of the brain stem, the cerebellum (via the A. basilaris and its branches) and the occipital cortex (via the Aa. cerebriposteriores).
Because the brain, as an essential bodily organ, must constantly be well perfused, an anastomotic system, referred to as the **circulus arteriosus Willisi (circle of willis)**, exists between the anterior and posterior circulations. Terminal branches of vertebral and internal carotid arteries anastomose to form a circular blood vessel termed as Circle of Willis. Furthermore, the common variants must later be considered during radiological assessment of the functionality of the cranial arteries. It is thus a common occurrence for the posterior cranial arteries to stem out from the **A. carotis interna**.

**Tip**: If you memorize the anatomical course of the cranial arteries with the respective supply areas, the clinical symptoms of vascular occlusion can often be deduced directly.

**Patterns of damage for circulatory disorders**

If relevant circulatory disorders are present in the cranial arteries, various patterns of damage may arise:

**Embolic infarctions**
If an embolus is carried into the brain by the bloodstream, this typically leads to the occlusion of mid-sized arteries with a cuneiform, so-called territorial infarction.

Watershed cerebral infarctions

A fall in blood pressure results in a perfusion deficit in the distal portions of an artery. This, in turn, causes an infarction area in the border zone with the neighboring artery system. This type of infarction is relatively rare thanks to the good anastomisation.

Local arteriosclerotic infarctions

Long-existing arteriosclerotic risk factors can locally cause occlusion of small border zone arteries. This results in lacunar infarctions (infarction area < 15 mm), chiefly in the brain stem, the white matter and in the region of the basal ganglia.

A brief look at cellular pathophysiology helps in understanding the eventual therapeutic measures. The neuronal metabolism consists of the structural metabolism that maintains the cell’s neural life, which induces the tangible function of the neural system. Should decreased blood flow into the brain occur, the neurons first adjust the functional metabolism, resulting in neurological failures. Should the neuronal blood supply continue to sink, the structural metabolism and cells undergo irreversible damage.

Epidemiology, Risk Factors and Risk Illnesses of Stroke

Strokes are relatively common in Germany, and the risk of having one increases greatly with age alone. Younger adults have a risk of about 1 : 5000, and this risk becomes 1 : 30 for people over 80 yrs of age. The frequency becomes clear when the risk factors are considered.

Along with age, arterial hypertension and smoking are special risk factors. All factors that
increase the likelihood of *arteriosclerotic arterial* changes increase the risk of a stroke. As with *coronary heart disease* and peripheral arterial occlusive disease, metabolic syndrome is also especially dangerous when it comes to a stroke.

**Risk factors for an Ischemic Stroke:**

- Hypertension
- Diabetes
- Tobacco
- Heart disease (atrial fib, valvular disease, cardiomyopathy)
- Hypercholesterolemia
- Age

**Risk factors of genetic conditions:**

- CADASIL
- Sickle cell disease
- Hypercoagulable states (cancer, pregnancy, oral contraceptives, antiphospholipid syndrome, protein C deficiency)

**Common Causes of Stroke**

The most common are embolic infarctions (approx. 50 %), followed by lacunar infarctions (approx. 30 %). Embolic infarctions can further be divided into cardio-embolic and arterio-arterial embolic strokes.

**Cardio-embolic causes of Stroke**

Cardiac embolisms are responsible for about 25 % of all strokes. Atrial fibrillation is most often present, which affects approximately 10 % of all people of advanced age. It can often go unnoticed and only to be noticed as the result of a stroke.

If a **patent foramen ovale (= PFO)** is present, a venous embolus can paradoxically transgress into the artery and embolise into the brain. An additional **atrial septal aneurysm** increases the risk of stroke.

A more uncommon cardio-embolic cause is **endocarditis**, which can result in (often multiple) infections embolisms with the risk of a septic brain abscess. Embolisms in other organs must also be considered with endocarditis.
Macroangiopathic (arterio-arterial embolic) causes of Stroke

**Carotid artery stenosis**

Sources of embolism are primarily stenoses of the **Aa.carotid communis** and the **Aa. carotid internae (carotid artery stenosis)**. Carotid artery stenoses usually form due to the turbulent blood stream profiles in the area of the **carotid bifurcation**, directly at the terminal point into the **carotid artery**. The **macroangiopathic** changes always arise over many years, so that collateral circulation that have formed parallel, hemodynamically relevant stenosis.

Based on the macroangiopathy, a thrombus is removed after embolising into the brain. Rarely does a complete occlusion of the carotid arteries lead to an ischaemia. Along with the classic embolism pattern with territorial infarction areas, carotid artery stenosis can also lead to watershed infarctions in conjunction with cardiac insufficiency.

Carotid artery stenoses can either be asymptomatic or symptomatic. One typical symptom is amaurosis fugax, which describes reversible impaired vision due to a circulatory disorder of the **A. ophthalmica**. If carotid artery stenosis is symptomatic, there is a much greater risk of manifest ischaemic stroke.

**Dissection**

Along with **carotid artery stenosis**, dissection must be mentioned as another macroangiopathic cause for ischaemic stroke. Either spontaneously or as a result of trauma, it results in bleeding into the vascular wall, causing a shift of the vascular lumen. Carotid artery stenosis near the base of the skull or the **A. vertebrais** during the flow along the **upper cervical vertebra** is especially predestined.

Dissection is one of the most common causes of stroke in younger people. Older people
appear to be somewhat protected because of their often stiffer vascular walls due to simultaneously present arteriosclerosis.

Microangiopathic Stroke

A long-existing cardiovascular risk profile can result in cerebral microangiopathy. Local thrombotic processes are responsible for the formation of lacunar infarctions. Since microangiopathy rarely selects a special vessel, but rather entails a principal vascular disease, lacunar infarctions do not occur only due to cerebral microangiopathy, there are other causes too. In conjunction with a subsequent cognitive impairment or even dementia, this is referred to asBinswanger’s disease (= subcortical arteriosclerotic encephalopathy, vascular dementia).

More Uncommon Causes of Stroke

There are many uncommon causes of stroke that must especially be considered when either the aetiology cannot be explained in any other way, or the patient does not fit into the typical “stroke group” because of their young age. The main possibilities to consider here are antiphospholipid syndrome, vasculitis, coagulopathy, neoplasia or genetic factors.

The following table should provide a rough overview of possible uncommon causes of strokes.

<table>
<thead>
<tr>
<th>Aetiological group</th>
<th>Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inflammation</td>
<td>Vasculitis, antiphospholipid antibody syndrome</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>Heart valve reconstruction, aneurysms, cardiomyopathy, patent foramen ovale, endocarditis</td>
</tr>
<tr>
<td>Haematology</td>
<td>Coagulopathy (e.g. APC resistance, thrombophilia), polycythaemia vera, essential thrombocythaemia</td>
</tr>
<tr>
<td>Genetics</td>
<td>M. Fabry, CADASIL (Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts and Leukoencephalopathy)</td>
</tr>
<tr>
<td>Trauma</td>
<td>Dissection</td>
</tr>
<tr>
<td>Iatrogenic</td>
<td>Air or fat embolisms after central venous catheter, osteosyntheses</td>
</tr>
<tr>
<td>Paraneoplasia</td>
<td></td>
</tr>
<tr>
<td>Psychogenic</td>
<td></td>
</tr>
<tr>
<td>Seizure</td>
<td>Migraine, epilepsy</td>
</tr>
</tbody>
</table>

Differential Diagnostically Special Forms of Stroke

Cerebral vein thrombosis

Strictly speaking, ischaemic stroke is not present with cerebral vein thrombosis, but rather a drainage occlusion. Due to the disrupted drainage and subsequent increase in pressure, however, secondary ischaemic lesions with intracerebral haemorrhages can form.

Typical risk factors are:

- Pregnancy
- Oral contraception
Congenitally increased likelihood of coagulation, such as the “Factor V Leiden mutation”.

This illness is relatively rare, with warning signs of the presence of cerebral vein thrombosis typically including headaches, potential risk factors (see above), epileptic attacks or unconsciousness combined with focal neurological deficits. The symptoms are far more protracted as opposed to pure arterial ischaemia. Highly effective anticoagulation should occur during treatment.

Vasculitis

Vasculitis has no specific cause, especially in young patients. Vasculitis is marked by inflammation of blood vessels leading to ischemia in the downstream artery system with corresponding symptomatic failures. Vasculitides can systemically affect different organs and thus do not cause any purely neurological symptoms. Some vasculitides, however, regularly affect the central nervous system:

- **Primary CNS vasculitis**: This vasculitis is entirely present in the CNS and causes a range of symptoms over multiple lesions.
- **Temporal arteritis**: When primarily older people report (also pressure-sensitive) pain when chewing or loss of vision, temporal arteritis must always be considered. Steroids are prescribed on suspicion to prevent irreversible loss of vision.
- **Takayasu’s arteritis**: Mainly affects larger vessels, including the aortal outflow points. With symptoms such as Vertigo, syncopal or vision impairments.
- **Vasculitides of smaller vessels** (e.g. granulomatosis with Wegener’s polyangiitis) or accompanying vasculitides alongside collagenosis (e.g. systemic lupus erythematosus) can affect the central nervous system.

Spinal Ischaemia

In rare cases, arterial ischaemia can affect the spinal central nervous system. The spinal cord is azygously supplied with blood ventrally, and doubly supplied dorsally. The most important (and often asked about) "anterior spinal artery syndrome" usually arises in the thoracic spinal cord, and thus affects the entire anterior spinal cord and presents paraparesis (or -plegia).

It may occur naturally quite suddenly with spinal ischemia. However, more frequently, anterior spinal artery syndrome arises during aortal dissections or iatrogenically during operations in the thoracic aorta.

The Symptoms of Different Strokes

Following an ischaemic stroke, a certain neuronal group of cells loses its functional capabilities. Essentially any extent or localization of damage is possible. Every symptom of damage to the CNS is thus fundamentally possible due to an ischaemic stroke (which also indicates the extensive differential diagnostic possibilities).

The description of sudden symptoms is the most important aspect of anamnesis. The anamnesis must also include the exact point in time at which the symptoms began. Based on the progress over time, various stages of stroke are defined in accordance to the manual from the “Deutsche Gesellschaft für Neurologie”: 
<table>
<thead>
<tr>
<th>Stage</th>
<th>Clinical description</th>
<th>Reversibility of the symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Asymptomatic findings</td>
<td></td>
</tr>
<tr>
<td>IIa, IIb</td>
<td>Transitory ischaemic attacks (TIA) minor stroke</td>
<td>Complete regression of symptoms within 1 hour, complete regression of symptoms within 7 days</td>
</tr>
<tr>
<td>III</td>
<td>Progressive ischaemia with progressive advancement of the symptoms over days</td>
<td>Only partial regression of symptoms possible</td>
</tr>
<tr>
<td>IV</td>
<td>Complete stroke</td>
<td>Irreversible damage without regression</td>
</tr>
</tbody>
</table>

Larger infarct areas often entail cortical defects. One example of this is **aphasia** for cortical infarctions of the left hemisphere, and neglect of the left half of the body (= left-sided neglect) for cortical infarctions of the right hemisphere.

**The affected vascular territory determines the symptoms**

Despite the principal variety of symptoms, there are chronically recurring typical damage patterns that frequently appear in the clinic. They are also often asked about in exams. You should also memorize these symptoms:

**Ischaemia in the A. cerebri media**

Territorial infarctions in the system of the **A. cerebri media** are very common. Usually, this results in **contralateral brachiofacial hemiparesis** with **hemihypesthesia**. Because the sensory and motoric language areas and many cognitive networks are located in the supply area of the **A. cerebri media**, cortical signs like aphasia or neglect may arise.

An affected oculomotor system can result in gaze movement toward the lesion. The visual pathway can be disrupted by **homonymous hemianopsia**. A. cerebri media is the artery most occluded in stroke.

**Ischaemia in the A. cerebri anterior**

The **A. cerebri anterior** supplies the frontal lobes and areas that control legs and contralateral hemiparesis of the legs occurs accordingly. Central bladder incontinence may also arise, as the upper bladder control centre is located near the **margo superior cerebri**. The frontal lobe function can also be visibly impaired, as reflected in drive disorders or an unstable emotional state. Akinetic mutism can occur on the affection of both the anterior cerebral territories.

**Ischaemia in the A. cerebri posterior**

**Homonymous hemianopsia** forms contralaterally. In addition to the **occipital lobe**, the **A. cerebri posterior** also supplies the **thalamus** and the **capsula inerna**. Proximal occlusions may cause unconsciousness or hemiparesis. If the entire visual cortex suffers from insufficient supply, the patients suffer from cortical blindness that they often do not perceive.

This is referred to as “Anton-Babinski syndrome”. The symptoms are color blindness, failure to see to-and-fro movements, verbak dyslexia and hallucinations. Posterior infarctions are usually painful!

**Ischaemia in the A. vertebralis and A. basilaris**
A. vertebrais and A. basilaris supply the posterior 2/5th of the cerebrum, part of cerebellum and the brain stem. Ischaemia in these arteries may result in following morbidities.

Territorial cerebellar infarctions entail impaired motor function. Ataxia in extremities or the trunk, intention tremor, dysarthria or nystagmus is typical. Balance disorders may provoke central vertigo.

Many neuronal qualities for many areas of the body are anatomically bundled into the smallest area in the brainstem, and marked symptoms can arise accordingly quickly. So-called “alternans syndromes” are typical. This describes unilateral brainstem lesions that exhibit a range of symptoms. The cranial nerves are affected ipsilaterally, the extremities and trunk contralaterally. Alongside hemi- and tetraparesis, dysarthria, central oculomotor disorders and dysphagia, there are also special forms that will be discussed later in the text.

**Carotid artery stenosis**

A. ophthalmica is a branch of carotid artery. If stenosis occurs in this artery, then visual defects such as dark curtain hung over eyes may appear which is self-limiting in nature ending after a certain period. This condition is known as amaurosis fugax with an expression of retinal TIA.

**Dissection of the vessels supplying the brain**

Patients report pain in the throat area. Horner’s syndrome is often discovered during the clinical examination, and this entails ptosis, miosis and enophthalmos. Of course, insufficient supply to the respectively subsequent artery system can occur with similar consequences.

**Lacunar infarctions of the A. cerebri media branches**

Lacunar infarctions often affect the basal ganglia. The relay of all motor pathways occurs in the basal ganglia. Unlike with territorial ischemia of the A. cerebri media, complete contralateral hemiparesis or hemihypesthesia is found in the clinical examination. However, usually, only one neuronal quality, such as motor skills, is impaired due to the small infarction area.

**An overview of the various brain-stem syndromes**

**Wallenberg syndrome**

Ischaemia in the supply area of the A. cerebelli inferior posterior (= an outflow point of the A. vertebrais, so-called PICA) results in a dorsolateral medulla oblongata infarction. Ipsilaterally this causes Horner’s syndrome, paresis of the lower cerebral nerves, analgesia and thermanaesthesia in the face, and hemiataxia. Analgesia and thermanaesthesia occur contralaterally in the trunk and extremities (= dissociated sensory loss).
Locked-in syndrome

An extensive brain-stem lesion, e.g. due to thrombosis in the A. basilaris, forms and voluntary motor function is no longer possible due to damage to the pyramidal tracts and cerebral nerve nuclei. The cerebrum remains undamaged and the patients are entirely alert and able to be contacted.

Top-of-the-basilar syndrome

Ischaemia is present at the bifurcation point of the A. basilaris into both Aa. cerebri posteriores. Bilateral damage to the thalamus results in a comatose clinical course.

Weber’ syndrome

This results in the ipsilateral failure of the N. oculomotorius with contralateral hemiparesis.

Foville’s syndrome

This results in facioplegia along with internuclear ophthalmoplegia with contralateral hemiparesis.

Review Questions

The correct answers can be found after the references.

1. Which of the following factors is least likely to be aetiopathogenetically involved in the occurrence of an ischaemic stroke?

   A. Paroxysmal atrial fibrillation
   B. Arterial hypertension
   C. Vasculitis
   D. Status post heart valve replacement
   E. Status post thrombophlebitis

2. Upon the emergency admission of a patient you record the following: Patient
conscious, somewhat agitated, somewhat oriented, no gaze paresis, right-sided
(anamnestically acutely occurring) hemiparesis magnitude 3/5, right-sided
difficulty opening mouth, no sensory disturbances, no neuropsychological
impairments.

What is the most likely pathological background?

A. Glioma, left hemisphere
B. Cerebral abscess, right hemisphere
C. Aneurysm in the A. communicans anterior
D. Cerebral vein thrombosis
E. Cerebral microangiopathy

3. An 80 year-old female patient reports impaired vision with blindness in the
right eye, which persisted for about 2 - 5 minutes the previous day. She was
then able to see again. Current neurological results are ordinary. Which
combination of diagnostics and pathology is the most likely to explain the
patient’s symptoms?

A. Cranial MRI with evidence of Anton-Babinski syndrome.
B. Doppler sonography with evidence of Anton-Babinski syndrome.
C. Deduction of optically evoked potentials with evidence of a central conduction
disturbance, e.g. stemming from acute inflammatory processes.
D. Doppler sonography of the vessels supplying the brain with evidence of stenotic
vascular changes.
E. Blood pressure measurement in both arms with evidence of subclavian steal
syndrome.

4. A 54 year-old female patient reports right-sided hemiparesis, primarily of the
leg, with bladder dysfunction. Where is there most likely a cerebral
impairment?

A. The middle cerebral artery, left.
B. The middle cerebral artery, right.
C. The area of the margo superior cerebri, left.
D. The area of the margo superior cerebri, right.
E. The right cerebellar peduncle.

References


doi:10.1161/01.str.0000054262.69831.24

Correct answers: 1E, 2E, 3D, 4C

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