Stroke (CVA, Cerebrovascular Accident) — Causes and Symptoms

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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 (CVA) that is characterized by the loss of adequate blood supply to the brain. Ischemic stroke is defined as a circulatory disorder of a specific region of the brain. This illness is quite common and has many different causes and symptoms. As the brain is very vulnerable, loss of blood supply to it 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 ischemia and hemorrhagic infarctions, which result from hemorrhage.
Cerebral blood supply

An ischemic stroke results in poor neuronal circulation, and subsequent failure of the neurons. In order to understand this illness, its effects, and classifications, it is crucial to have knowledge of the anatomy of blood supply to the brain.

Essentially, the brain is supplied by 2 circulatory systems, the anterior and posterior circulations. The **internal carotid artery** forms the **carotid artery system**. At the base of the brain, the carotid artery divides in the so-called ‘carotid T’ into the **middle cerebral artery** and **anterior cerebral artery** and thereby forms an anterior circulation. The **middle cerebral artery** supplies the majority of the white brain matter, portions of the frontal, temporal, and parietal lobes, and the basal ganglia. The **anterior cerebral artery** ensures blood supply to the structures near the median and superior margins of the cerebral hemisphere.

The **posterior circulation** (vertebrobasilar artery system) is formed from the merging of the paired **vertebral artery** into the unpaired **basilar artery**. This passes cranially along the front of the brain stem and branches into the **posterior cerebral artery** at the head of the **basilar artery**. The posterior circulation is thus responsible for blood supply to the brain stem, cerebellum (via the **basilar artery** and its branches), and **occipital cortex** (via the **posterior cerebral artery**).
Because the brain, as an essential body organ, must constantly be well perfused, an anastomotic system, referred to as the **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 (circle of Willis). Furthermore, the common variants must later be considered during the radiological assessment of the functionality of the cranial arteries. It is thus a common occurrence for the posterior cranial arteries to stem from the **internal carotid artery**.

**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 to 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 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 because of the good anastomoses.

**Local arteriosclerotic infarctions**

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

A brief look at cellular pathophysiology helps in understanding 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 decrease in blood flow to the brain occur, the neurons first adjust the functional metabolism, resulting in neurological failures. Should the neuronal blood supply continue to decrease, the structural metabolism and cells undergo irreversible damage.

**Epidemiology, Risk Factors and Risk Illnesses of Stroke**

Strokes are relatively common, 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 important 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 ischemic stroke:

- Hypertension
- Diabetes
- Tobacco
- Heart disease (atrial fibrillation, valvular disease, cardiomyopathy)
- Hypercholesterolemia
- Age
- Pregnancy
- Oral contraceptives

Genetic risk factors for ischemic stroke:

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

Common Causes of Stroke

The most common causes of stroke are embolic infarctions (approx. 50%), followed by lacunar infarctions (approx. 30%). Embolic infarctions can further be divided into cardioembolic 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 approx. 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 is present, a venous embolus can paradoxically transgress into the artery and embolize to the brain. An additional atrial septal aneurysm increases the risk of stroke.

A more uncommon cardioembolic cause is endocarditis, which can result in (often multiple) infectious 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 common carotid and internal carotid arteries (carotid artery stenosis). Carotid artery stenoses usually form due to the turbulent bloodstream profiles in the area of the carotid bifurcation, directly at the terminal point into the carotid artery. Macroangiopathic changes usually arise over many years, so that collateral circulation forms parallel, hemodynamically relevant stenosis.

Based on the macroangiopathy, a thrombus is removed after embolizing to the brain. Rarely does a complete occlusion of the carotid arteries leading to ischemia. 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 ophthalmic artery. If carotid artery stenosis is symptomatic, there is a much greater risk of manifest ischemic stroke.

Dissection

Along with carotid artery stenosis, dissection is another macroangiopathic cause of ischemic 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 vertebral artery during flow along the upper cervical vertebra is common.

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 co-occuring arteriosclerosis.

**Microangiopathic Stroke**

A longstanding 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 entails a principal vascular disease, lacunar infarctions do not occur only due to cerebral microangiopathy. In conjunction with a subsequent cognitive impairment or even dementia, this is referred to as Binswanger’s disease (subcortical arteriosclerotic encephalopathy, vascular dementia).

**More Uncommon Causes of Stroke**

There are many uncommon causes of stroke that must be especially considered when either the etiology cannot be explained in any other way, or if the patient does not fit into the typical ‘stroke group’ because of their young age. These included antiphospholipid syndrome, vasculitis, coagulopathy, neoplasia, or genetic factors.

The following table is an 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., resistance to activated protein C, thrombophilia), polycythemia vera, essential thrombocytemia</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>
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</tbody>
</table>

**Differential Diagnostically Special Forms of Stroke**

**Cerebral vein thrombosis**

Strictly speaking, ischemic stroke is not present with cerebral vein thrombosis, but rather a drainage occlusion. Due to the disrupted drainage and subsequent increase in pressure, secondary ischemic lesions with intracerebral hemorrhages could occur.

**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 ischemia. Effective anticoagulation should be administered during treatment.

**Vasculitis**

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

- **Primary CNS vasculitis**: This vasculitis is entirely present in the CNS and causes a range of symptoms over multiple lesions.
- **Temporal arteritis**: Usually, when older people report pain (also pressure-sensitive) 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, syncope, 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 CNS.

**Spinal Ischaemia**

In rare cases, arterial ischemia can affect spinal innervation. The spinal cord receives azygous (unpaired) blood supply ventrally and paired supply dorsally. The 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 with paraparesis (or -plegia).

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

**The Symptoms of Different Strokes**

Following an ischemic 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 ischemic 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 with 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>Transient ischemic attacks (TIA) minor stroke</td>
<td>Complete regression of symptoms within 1 hour, complete regression of symptoms within 7 days</td>
</tr>
<tr>
<td>---</td>
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</tr>
<tr>
<td>III</td>
<td>Progressive ischemia 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:

**Ischemia in the middle cerebral artery**

Territorial infarctions in the system of the **middle cerebral artery** are very common. Usually, this results in **contralateral brachiofacial hemiparesis** with **hemihypesthesia**. Because the sensory and motor language areas and many cognitive networks are located in the supply area of the **middle cerebral artery**, 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**. The middle cerebral artery is the most frequently occluded artery in stroke.

**Ischemia in the anterior cerebral artery**

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

**Ischemia in the posterior cerebral artery**

**Homonymous hemianopsia** forms contralaterally. In addition to the **occipital lobe**, the **posterior cerebral artery** also supplies the **thalamus** and **internal capsule**. Proximal occlusions may cause unconsciousness or hemiparesis. If the entire visual cortex receives insufficient supply, 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, verbal dyslexia, and hallucinations. Posterior infarctions are usually painful.

**Ischemia in the vertebral and basilar arteries**

The vertebral and basilar arteries supply the posterior 2/5 of the cerebrum, part of the cerebellum, and the brain stem. Ischemia in these arteries may result in morbidities.

Territorial cerebellar infarctions entail impaired motor function. Ataxia in the 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. Accordingly, marked symptoms can arise quickly. Further, ‘Alternans syndromes’ are typical. These are 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

The ophthalmic artery is a branch of the carotid artery. If stenosis occurs in this artery, then visual defects such as a dark curtain hung over the 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 clinical examination, and this entails ptosis, miosis, and enophthalmos. Of course, insufficient supply to the respective artery system can occur with similar consequences.

Lacunar infarctions of the middle cerebral artery 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 middle cerebral artery, complete contralateral hemiparesis or hemihypesthesia is found in 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

Ischemia in the supply area of the posterior inferior cerebellar artery (PICA, an outflow point of the vertebral artery) results in a dorsolateral medulla oblongata infarction. Ipsilaterally, this causes Horner’s syndrome, paresis of the lower cerebral nerves, analgesia and thermanesthesia in the face, and hemiataxia. Analgesia and thermanesthesia 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 basilar artery, occurs and voluntary motor function is no longer possible due to damage to the pyramidal tracts and cerebral nerve nuclei. Here, the cerebrum remains undamaged and patients are alert but unable to move or communicate.

Top-of-the-basilar syndrome

Ischemia is present at the bifurcation point of the basilar artery into both posterior cerebral arteries. Bilateral damage to the thalamus results in a comatose clinical course.

Weber’s syndrome

This results in the ipsilateral failure of the oculomotor nerve with contralateral hemiparesis.

Foville’s syndrome

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

References


