

2. Stroke (cerebrovascular accident – CVA)

Stroke is defined by WHO as a “neurological deficit of cerebrovascular cause that persists beyond 24 hours or is interrupted by death within 24 hours”. The symptoms stem from the affected part of the brain.

Stroke can be classified according to etiology into **hemorrhagic** (15%) and **ischemic** (85%).

Ischemic stroke may be caused by thrombotic occlusion of a cerebral artery in cerebral atherosclerosis, or less commonly due to embolism (e.g. in left atrial thrombus) or thrombotic occlusion in vasculitis affecting the cerebral arteries. Spontaneous intracranial hemorrhage is caused by rupture of a cerebral artery aneurysm, arteriovenous malformation, tumor, or coagulopathy.

Diagnostic algorithm:

The initial imaging method for stroke is **CT without contrast** to rule out intracranial hemorrhage (i.e., to rule out hemorrhagic stroke).

Freshly coagulated blood is hyperdense compared to brain tissue (70-90 HU vs. 35 HU) and is clearly visible on plain CT.

1. Non-traumatic intracranial hemorrhage

Non-traumatic intracranial hemorrhage is most often represented by **intracerebral hematoma** or **subarachnoid hemorrhage**.

If the **intracerebral hematoma** is located at typical sites for spontaneous intracerebral hemorrhage, no further imaging is required. The decision between surgical or conservative treatment is based on the size, location, and expansive behavior of the hematoma (contralateral shift of midline structures, edema surrounding the hematoma, brain herniation).

Typical sites of spontaneous intracerebral hemorrhage include the area of nucleus lentiformis, thalamus, cerebellum, and brainstem. The most common etiology of spontaneous intracerebral hemorrhage is hypertension. In atypical localizations of intracerebral hematoma, it is appropriate to supplement the investigation with **CT angiography** of cerebral arteries to exclude vascular malformation. A (potential) tumor masked with high density of blood may not be distinguishable by CT angiography. Such a tumor is often diagnosed perioperatively in an attempt to evacuate an intracerebral hematoma.

If a non-traumatic subarachnoid hemorrhage is present on plain CT, **CT angiography of the cerebral arteries** should be performed to demonstrate an aneurysm or arteriovenous malformation as the source of bleeding. In the event of a ruptured aneurysm, in addition to subarachnoid hemorrhage, an intracerebral hematoma may form simultaneously and extend into the ventricular system, causing hemocephalus (intraventricular hemorrhage – IVH). In hemocephalus, the cerebral aqueduct (aqueductus Sylvii) can be blocked by coagulating blood and hemocephalus may develop.

Subarachnoid hemorrhage (SAH) is divided into spontaneous and traumatic. The most common cause of spontaneous SAH is a ruptured aneurysm. It manifests clinically as a sudden, severe pain from full health (patients report the worst pain they have experienced), it may be accompanied by vomiting and impaired consciousness.

2. Cerebral ischemia

Cerebral ischemia may have a completely normal finding on **plain CT** in the first 12 hours. If no bleeding is present on CT, intravenous thrombolysis can be initiated in indicated cases. To verify the vascular occlusion before possible mechanical thrombectomy, it is again necessary to perform **CT angiography** of the cerebral arteries.

In cerebral ischemia of unclear age, the so-called **CT perfusion of the brain** is sometimes performed, which displays the infarct itself (already dead brain tissue) and the surrounding penumbra (ischemized but still viable brain tissue, which can be saved by treatment).

Immediate signs of cerebral ischemia include the relatively rare "dense artery sign" (hyperdense thrombus asymmetrically on the affected side - most often in a.cerebri media). Sometimes there are early signs of ischemia (within 6 hours) such as blurred contours of gray and white matter and blurred contours of cerebral gyri or nucleus lentiformis due to edema. Gradually, in the acute phase (24 hours - 1 week), the ischemic area becomes clearly hypodense due to progressive edema, sometimes a mass effect is expressed (surrounding edema, contralateral shift of midline structures, etc.). In the subacute phase, the edema gradually disappears and the density of the affected area increases, the appearance on CT may even return to normal. In the chronic phase, the affected area is replaced by glia, on CT it is hypodense and atrophic (reduction of brain tissue volume, focal enlargement of subarachnoid spaces or adjacent brain chambers).

In case of clinical doubt in a normal finding on non-contrast CT, ischemia may be confirmed or excluded by non-contrast **brain MRI**. The key sequence for imaging early cerebral ischemia is **DWI** (diffusion-weighted image) - evidence of restricted diffusion in the ischemic area can be observed just minutes after stroke due to inactivity of ion pumps on cell membranes (these contribute to the formation of gradients necessary for diffusion).

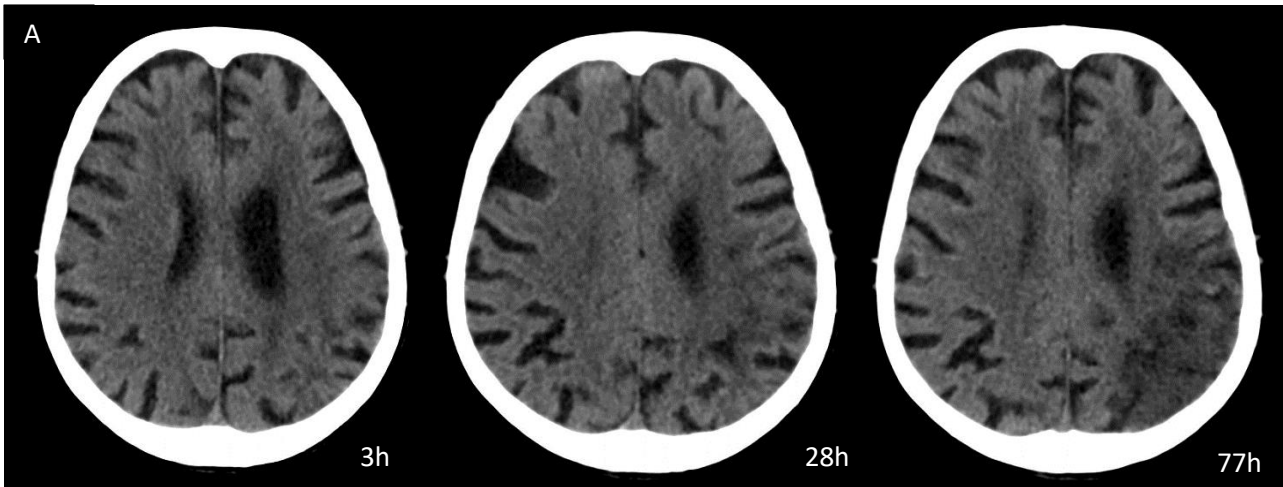


Fig. A - Development of ischemic stroke in the region of the left a. cerebri media on non-contrast CT. No fresh ischemic changes are detectable on CT **3 hours** after the onset of symptoms. After **28 hours**, there are blurred contour of the gyri parietally on the left - the onset of cytotoxic edema. After **77 hours**, developed ischemic changes in the form of hypodense area, blurred gyri and reduction of subarachnoid spaces due to edema caused by ischemia.

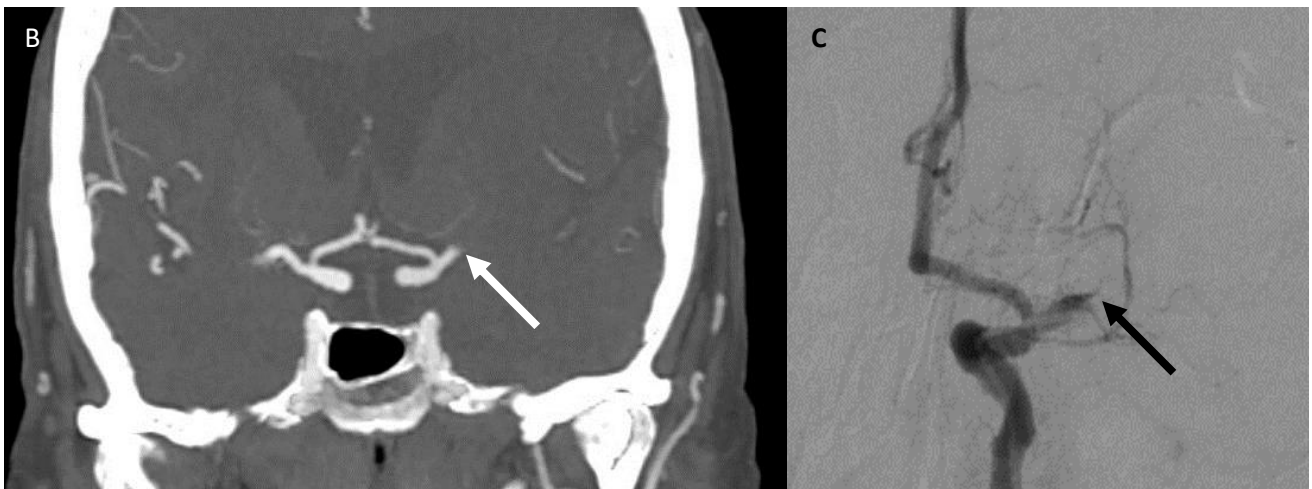
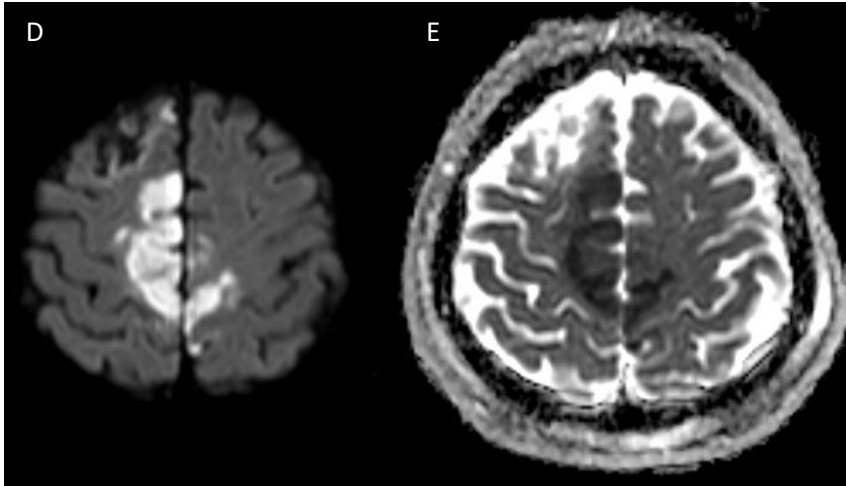
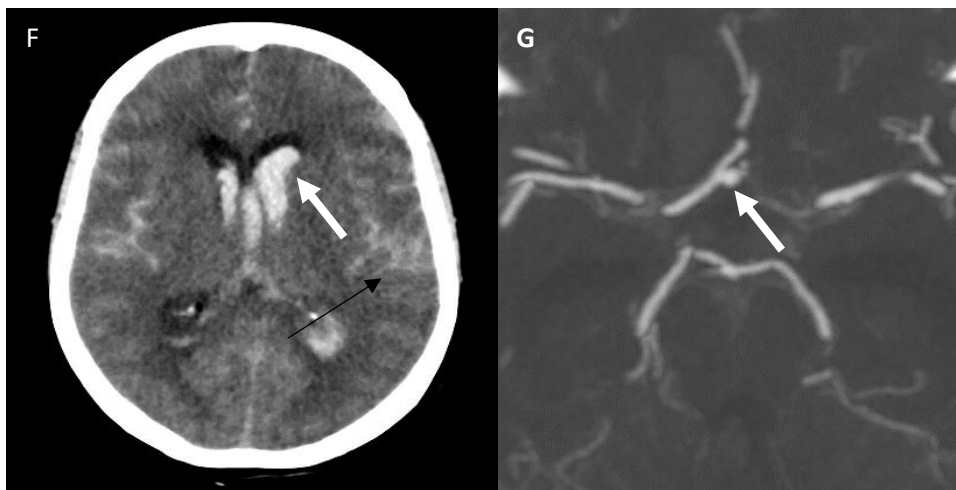


Fig. B - CT angiography, coronal plane – occlusion of a.cerebri media on the left. Mechanical thrombectomy was indicated.

Fig. C – Direct angiography displays corresponding occlusion of left a. cerebri media, image before mechanical thrombectomy. Arrows indicate an amputated stump of a thrombus-closed artery.



MRI displays early ischemia in the region of a.cerebri anterior bilaterally (more extensive on the right). In initial phase the ischemia is characterised by hypersignal restricted diffusion on DWI sequence of MRI - **Fig. E**, which corresponds to low signal regions on ADC maps - **Fig. D**. MRI was indicated because of not specific symptoms and normal finding on CT.



Patient with sudden intense headache with consequent impaired consciousness.

Fig. F – extensive subarachnoidal hemorrhage (**black arrow**) on non-contrast CT with hematoma (not visualised) and hemocephalus (**white arrow**).

Fig. G – CT angiography displays small aneurysm (white arrow) on a.communicans anterior, which was the cause of subarachnoidal hemorrhage.