**Imaging in Non-Traumatic Neuro-Emergencies - Strategies and Technique**

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Learning objectives

Neurological emergencies have a large number of possibly underlying pathologies.

Stroke is the best-known and probably, with regard to socio-economic impact, most important among these emergencies, but many other pathologic conditions have to be considered.

The aims of this poster are

- to present a clinical-radiological diagnostic pathway in neurological emergencies,
- to summarize key aspects of stroke management, and
- to give an overview of other causes of emergencies than stroke.

Main

Before we discuss imaging …

A neurological emergency can be defined as the sudden loss of motor, sensory, or cognitive functions, and/or the loss of consciousness up to deep coma.

Possible underlying pathologies like metabolic causes (diabetic coma), anaphylactic reactions, or drug abuse will not be considered here; these conditions can mostly be ruled out from the patient's history. We will focus on pathologies that arise within the CNS, its supplying and draining vessels, and directly adjacent tissue.

First step in managing the emergency patient:

Although you are in a hurry and possibly under pressure from the clinical colleagues to start your imaging study, you should try to get as much information about the history and current status as possible:

- **What** exactly occurred **when**?
- Was the onset of symptoms **sudden**, or was there a continuous deterioration?
- Were there any **pre-existing** pathologic conditions?
- What is the **NIHSS score** [1]?
• If an inflammatory process has to be considered: Immunological situation (transplant patient, HIV infection)? Any trips to foreign countries? Animal contact (pets/professional)?

The radiologist has to work in very close cooperation with the referring colleagues, probably from the fields of neurology or internal medicine, to gather as much helpful information as possible as fast as possible and to adjust the exam protocol accordingly.

**Stroke:**

About one fifth of all strokes are caused by intracerebral or subarachnoid hemorrhage, the vast majority is caused by occlusion of a brain-supplying vessel.

"Time is Brain" - stroke is not an event, but a process. In the early phase, the infarct core, i.e., the volume of definitely infarcted and unrecoverable tissue, is still smaller than the volume that is suffering from reduced perfusion (fig. 1). This difference volume between perfusion deficit and diffusion restriction, the diffusion/perfusion-mismatch, is the "tissue at risk," often called the penumbra (fig. 2). This still salvageable tissue - provided that normal perfusion can be restored - is the aim of all therapies.

These key facts determine the questions that an imaging study must answer in order to allow for timely appropriate therapy. At the end of an imaging study (which should ideally be performed in not more than 15-20 minutes), you should know

• whether there is intracerebral hemorrhage,
• which portion of which vessel is occluded,
• the extent of infarct core and possibly salvageable tissue.

When you have gathered enough information to answer these questions, you can select the optimal therapy.

**CT or MRI?**

Before you consider which imaging modality should be used, you should discuss with the referring clinician where the imaging study should be performed. It has been shown that a CT imaging study in a county or community hospital with subsequent transport to a stroke centre adds a delay of about half an hour before intra-arterial thrombolysis begins in comparison to a direct transport to a centre where both diagnosis and therapy are performed [2].

At first glance, there are several arguments in favour of CT:
• CT scanners are widely available; even in tertiary care centres, MRI may not be available 24/7, while emergency CT is usually in operation around the clock.
• A patient in clinically critical condition is easier to monitor in a CT scanner in comparison to MRI where special equipment is required.
• Hemorrhage, esp. small subarachnoid bleeding, is easier to detect in CT.

Still, we prefer MRI in an emergency situation for various reasons:

• Although the concept of the ischemic penumbra can be applied to CT perfusion studies [3], it is often helpful to have a diffusion-weighted series (DWI), esp. for posterior fossa strokes and in inflammatory processes (fig. 3). Furthermore, DWI covers the whole brain which is still not standard in CT perfusion.
• Certain pathologies (e.g., cervical artery dissection) are easier to detect in MRI.
• A complete CT workup in stroke requires a considerable amount of contrast agent for both the CT angiography (CTA) and the CT perfusion studies. If you go on with intra-arterial thrombolysis, the total dose in the - usually elderly - patients may require additional care (hydration). Intracranial time-of-flight angiography allows visualizing a vessel occlusion without administration of contrast agent.
• Susceptibility-weighted imaging (SWI) - not yet generally available - directly shows the intravascular thrombus as well as stasis in the distal vascular areas and the de-oxygenated blood in veins draining an infarcted area.

Our stroke protocol

This is the protocol used at our institution in the work-up of acute stroke. It is meant as a recommendation; feel free to adjust it to your personal needs:

• **Diffusion-weighted axial series**
  Always the first - after one minute, we already have a basic idea of what to expect.
• **T2-weighted axial series**
  Helps to differentiate "shine-through effects" in the DWI and is helpful in wake-up strokes: If we don’t know the exact time of onset of a stroke, and if we have a clear diffusion restriction, the T2-series helps to decide whether we still go for interventional therapy: If we don’t see a demarcated infarct area in the T2-images, it is justified to assume that this event has been rather recent.
• **Intracranial ToF-angiography**
  To visualize the circle of Willis. Occlusions of larger vessels (up to M3-segments of the middle cerebral artery) are immediately visible.
• **Contrast-enhanced T2*-weighted angiography from the aortic arch to the circle of Willis**
  Important to determine whether there is proximal occlusion (e.g., of the internal carotid artery) or dissection (typical "flame sign" [4]) and whether there are conditions that may make intra-arterial access difficult (stenosis, kinking/coiling of cervical vessels)

• **Whole-brain perfusion study**
  To determine the extent of the penumbra in comparison to DWI

• (SWI if T2 suggests hemorrhage)

This protocol takes about 15 minutes; in cases where the clinical and imaging findings make i.v.-thrombolysis or bridging [5] the appropriate therapy, the i.v.-line is inserted and therapy started before we perform the last MR studies.

**Fig. 4 and Fig. 5: 68-year-old patient with sudden onset of aphasia and right-sided hemiparesis.**

**Fig. 4:** Emergency MRI shortly after midnight shows small diffusion restriction (top) and a perfusion reduction that includes the complete territory of the superior branch of the left middle cerebral artery (bottom).

**Fig. 5:** Control CT at ten in the morning after successful intra-arterial thrombolysis: A small insular infarct area that corresponds to the diffusion restriction in the initial MRI is discernible. The rest of the left MCA territory is intact as perfusion was restored in time.

**Horses and zebras**

"When you hear hoof beats, think horses, not zebras" is an old rule in differential diagnosis. Stroke is a major, but by far not the only cause of neurological emergencies.

**Hemorrhage** as a cause of stroke symptoms was already mentioned. Discuss with your neurosurgeons whether the search for the cause of bleeding should be incorporated in the primary imaging study. In cases of subarachnoid hemorrhage where immediate intra-arterial DSA is planned anyway, it may not be necessary to perform CTA or ToF-angiography. If you decide to perform a contrast-enhanced study, however, make sure to include 2D- or 3D-reformations or you might miss the diagnosis although it is in your pictures.

>>> **Fig. 6 and Fig. 7: 50-year-old patient with fast clinical deterioration from dizziness to coma.**
Fig. 6: CT study in an outside institution. Frontal bilateral subarachnoid hemorrhage. (The sagittal reformatted contrast-enhanced images were created later at our institution.) There is hyperattenuation in the frontal superior sagittal sinus and a filling deficit in the same area in the contrast series. The diagnosis was missed; the patient was referred for "angiography in subarachnoid hemorrhage to rule out aneurysm."

Fig. 7: The venous phase of the intra-arterial DSA and the MR study confirm the diagnosis of superior sagittal sinus thrombosis with typical hemorrhage due to reduced venous outflow.

**Inflammation** is best diagnosed in MRI. You won't miss a 3-cm-abscess in CT, but small parenchymal lesions in encephalitis are better detected with MRI. DWI is mandatory in these cases for the detection of such small lesions, the differentiation between abscess and cystic tumour, or the delineation of ventriculitis.

>>> Fig. 8: Multi-focal encephalitis. The Gd-enhanced T1-weighted images show only slight enhancement; in the diffusion-weighted images, multiple lesions are clearly discernible.

>>> Fig. 9: Ventriculitis. The contrast-enhanced series shows ependymal enhancement; a small level of inflammatory debris is discernible in the enlarged temporal horn. DWI more clearly shows the difference between debris (bright signal at the bottom indicates restricted diffusion due to hypercellularity) and "entrapped" CSF.

**Tumours** may become clinically apparent with neurologic deficits or epileptic seizures. Again, MRI is the method of choice; if you fully exploit the possibilities, you can get a lot of information already in the initial study: Anatomic delineation and infiltration of adjacent structures anyway, but additional studies like perfusion and/or spectroscopy may be helpful in histologic grading. Further studies like functional MRI or fibre tracking that some neurosurgeons like to have for an operation are better performed in an elective second session.

Patients suffering from **coma of unclear origin** will probably get a CT study as the first-line exam to rule out hemorrhage or basilar artery thrombosis. If the study yields a result negative for these conditions, you should add a perfusion study: Subtle status epilepticus may go undetected in the emergency room as EEG is not routinely used in this situation; perfusion CT, however, helps to identify this condition as it shows regional hyperperfusion [6].
An emergency study of the head in CT or MRI must be sufficient to rule out **venous sinus thrombosis**. Always cover a volume from the C2-level (to evaluate the proximal internal jugular vein) to the vertex - the superior sagittal sinus must be fully visible in a contrast-enhanced series (cf. fig. 6 and 7).

**Images for this section:**

**Fig. 1:** With decreasing perfusion, cells first go from function to "maintenance mode", but they are still alive. If perfusion is restored in time, they become fully functional again.
**Stroke – Defining tissue at risk**

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<th>Perfusion defect</th>
<th>Diffusion defect</th>
<th>Ischemic penumbra</th>
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<td>CBF ↓</td>
<td>irreversible ischemia (infarct core)</td>
<td>reversible ischemia (tissue at risk, target for therapy)</td>
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<td>CBV ↓</td>
<td>MTT ↑</td>
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**Fig. 2:** Clinical example to illustrate the penumbra concept. (Details in the text.)
Unrestricted diffusion - free extracellular space

Restricted diffusion - narrowing of extracellular space...
**Fig. 3:** The same phenomenon, two different causes. Diffusion restriction in inflammation is mostly due to hypercellularity that restricts the free water diffusion in the extracellular space. In stroke, this restriction is caused by volume augmentation of the cells due to cytotoxic edema.
**Fig. 4:** Left middle cerebral artery stroke with diffusion/perfusion-mismatch. (Details in the text.)

**Fig. 5:** Same patient as fig. 4. Control CT after i.a.-thrombolysis. (Details in the text.)
Fig. 6: Missed diagnosis of superior sagittal sinus thrombosis. (Details in the text.)
Fig. 7: Superior sagittal sinus thrombosis in DSA and MRI. (Details in the text.)
**Fig. 8:** Multi-focal encephalitis. In the contrast-enhanced T1-weighted images (top), only slight enhancement is visible. The DWI series (bottom), however, shows multiple lesions.
Fig. 9: Ventriculitis. DWI allows to discern inflammatory debris from CSF in the enlarged ventricle. (Details in the text.)
References


Personal Information