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Lecture [3]

IMAGING OF STROKE

Learning objectives:

1. To understand the role of CT and MR in patients with stroke
2. To define early signs of infarction on CT and MR
3. To identify patients with tissue at risk for guidance in selecting the appropriate therapy

Definition: acute neurological deficit of presumed vascular origin lasting more than 24 hours

Classification:

1) Thromboembolic (70% of strokes)
2) Primary cerebral hemorrhage (15% of strokes)
3) Other causes (15% of strokes) e.g., acute hypotension (watershed infarct), vasculitis …etc.

THROMBOEMBOLIC STROKE

Pathophysiology:

- **0–2 hours:** reduced perfusion
- **2–6 hours:** intracellular neuronal grey matter swelling (cytotoxic edema)
- **> 6 hours:** vasogenic white matter edema (blood–brain barrier breakdown)

Territorial involvement

- 50%: MCA
- 20%: lacunar
- 20%: vertebrobasilar system
- 10%: anterior cranial artery

Imaging features in acute infarction

Thromboembolic stroke in the first 6 hours

CT (non-contrast)

- Normal (40%).
- Hyperdense MCA sign (intraluminal thrombus)
- Loss of definition of lentiform nucleus
- Loss of definition of insular cortex
- Sulcal effacement

MRI (T1WI and T2WI)

- Normal or non-specific changes in the early stages.
- Absent flow void in a major occluded vessel is sometimes seen

MRI (DWI)

- Most accurate test for ischemia in the first 6 hours
- Acute stroke appears bright on DWI (restricted diffusion of water)
- Acute stroke appears dark on the corresponding apparent diffusion coefficients (ADC) map image
- High DWI signal persists for around 5 days

MR spectroscopy

- Low N-acetyl aspartate
- High lactate peak

CT/MRI perfusion

- Perfusion will demonstrate infarction and area is at risk **(penumbra).**

Established infarction [>6 hours]

CT

- Vasogenic white matter edema develops
- Infarct becomes better defined [hypodense area] and exerts mass effect
- Hemorrhagic transformation occurs in around 10% of cases

MRI

- High signal on T2WI
- Small petechial hemorrhages are commonly seen
- Contrast enhancement is typical beyond 48 hours (owing to opening of collateral vessels) in the cortex and overlying leptomeninges

Imaging features in chronic stroke

Encephalomalacia, well-defined area of brain atrophy with enlargement of adjacent CSF spaces

LACUNAR INFARCTION

- Caused by occlusion of small deep penetrating end arteries, e.g. lenticulostriate branches of MCA or pontine perforators of the basilar artery
- Small (< 1 cm^2) areas of infarction in deep structures, e.g. basal ganglia, internal capsule, thalamus, pons, deep white matter
- Common in patients with diabetes or hypertension

Imaging features

CT: Small, well-defined areas of low attenuation
MRI: Small areas of high signal on T2WI with bright areas in the acute stages on DWI

VERTEBROBASILAR INFARCTION

Clinically presents with cerebellar signs and cranial nerve palsies.

Imaging features

CT and MRI

- Infarcts correspond to the vascular territory supplied by the occluded vessel
- Specific imaging features are the same as for the anterior circulation
- Basilar artery thrombosis is a rare but important variant of vertebrobasilar infarction
    - Bilateral occipital lobe and thalamic infarcts
    - 90% mortality if untreated
    - Treated with systemic thrombolysis (within 3 hours)
    - CT (non-contrast) may show **hyperdense clot in the basilar artery**

WATERSHED INFARCT

- Caused by an episode of profound hypotension, e.g. from cardiac arrest
- Affects regions of the brain located between major vascular territories, e.g. between ACA and MCA (anterior watershed) or between MCA and PCA (posterior watershed)
- Causes symmetrical deep cerebral white matter infarcts

VENOUS INFARCTION

- Accounts for 1–2% of all strokes
- Commonest in children and young adults
- Thrombotic occlusion of a dural venous sinus mainly superior sagittal sinus
- Main causes include idiopathic in 25%, Dehydration (in neonates), pregnancy, oral contraceptive pill ...etc.

Imaging features

Non-contrast CT

- Hyperdense thrombus in a dural sinus: **'delta' sign**, seen in about 25% of cases
- Hemorrhagic infarction at underlying grey–white matter junction
- multifocal areas of hemorrhagic infarction that do not correspond to an arterial distribution

Contrast-enhanced CT or CT venography

- Modality of choice
- Non-enhancing thrombosed sinus: **'empty delta' sign**, seen in about 30% of case

MRI

- Loss of normal flow void in the occluded sinus
- Direct visualization of clot within the sinus
- Adjacent subcortical cerebral edema or infarction appears high signal on T2WI
- Low signal areas within this represent hemorrhage

Magnetic resonance venography (MRV)

- Provides complimentary information
- Sinus thrombosis appears as a segment of signal loss

PRIMARY CEREBRAL HEMORRHAGE

Imaging features

CT

- Focal hyperdense area (60–80 HU) in the acute stage, which slowly decreases in size over several weeks becoming isodense with brain parenchyma eventually becoming a low-density area of encephalomalacia

Main causes

- Hypertensive bleed Rupture microaneurysms.
- Vascular malformations
- Coagulopathy

MRI

- **T2 gradient echo** is the most sensitive sequence for blood breakdown products
- **0–2 days:** low T1, low T2, deoxyhemoglobin
- **3–7 days:** high T1, low T2, methemoglobin (intracellular)
- **7–14 days:** high T1, high T2, methemoglobin (extracellular)
- **Chronic:** low T1, low T2, hemosiderin