Subacute Infarction

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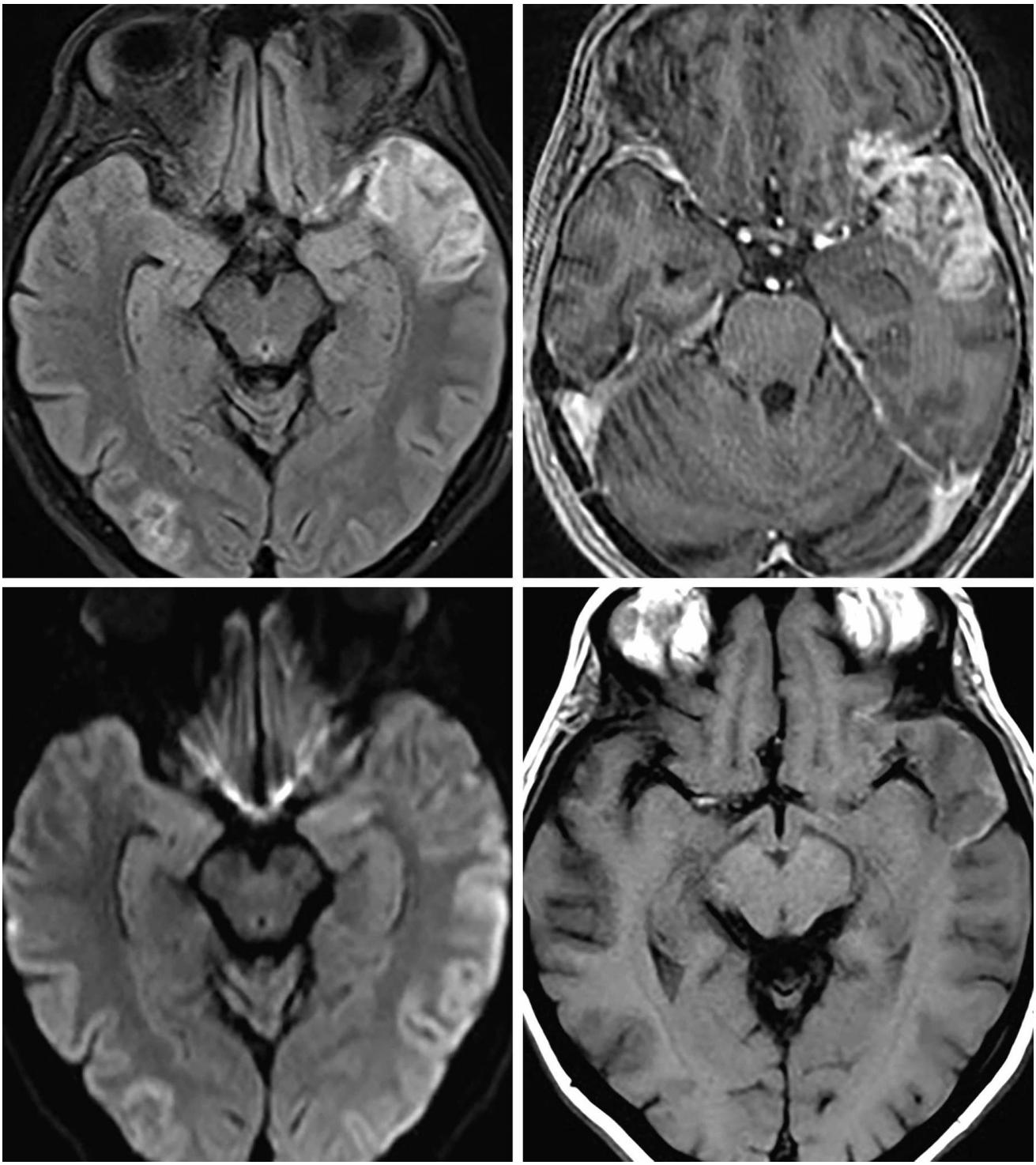

Figure 1: Masslike, cortical, FLAIR-hyperintense signal (top left) with heterogeneous enhancement (top right) in the anterior left temporal lobe and right occipital lobe. (Bottom Left) No evidence of reduced diffusivity

on DWI. This presentation is often challenging and requires an appropriate history to make the correct diagnosis, because subacute infarction is commonly mistaken for a neoplasm. (Bottom Right) The faint T1 hyperintensity in the cortex can be a clue to cortical laminar necrosis of subacute infarct.

Description

- Interrupted flow to brain resulting in cerebral ischemia or infarction with variable neurologic deficits

Pathology

- Critical disturbance in cerebral blood flow (CBF)
  - Severely ischemic core
    - CBF < (6–8 mL)/(100 g/min)
  - Oxygen depletion, energy failure, terminal depolarization, ion homeostasis failure
- Evolution from ischemia to infarct
- Ischemic penumbra is theoretically salvageable tissue

Clinical Features

- Symptoms
  - Focal acute neurologic deficit
  - Paresis, aphasia, altered mental status
- Age
  - Usually older adults
- Gender
  - Slightly more common in men
- Prognosis
  - Second most common cause of death worldwide

Imaging

- General
  - Abnormalities within particular arterial distributions

    - Rarely, an acute ischemic stroke can demonstrate an ill-defined cerebral lesion with mass effect, with or without parenchymal enhancement
    - Subacute infarction is more likely to mimic a brain tumor
        - Often demonstrates contrast enhancement, particularly in involved areas of gray matter
        - DWI may be falsely negative at the point of ADC sequence pseudonormalization
- Modality specific
    - CT
        - Loss of gray–white differentiation
        - Hyperdense thrombosed vessel sign (commonly middle cerebral artery)
        - Insular ribbon sign—blurring of the insular gray–white junction with hypoattenuation
        - Gyral swelling and sulcal effacement
        - Hemorrhagic transformation (15%–45%)
    - Perfusion CT
        - Infarcted tissue: prolonged mean transit time, decreased CBF and cerebral blood volume
        - Ischemic penumbra: similar to infarcted tissue but will have near-normal or even increased cerebral blood volume
    - CT angiography
        - Confirm occlusions, stenoses, and collateral flow
    - MRI
        - T1WI and T2WI
            - Variable with phase and hemorrhagic transformation
        - DWI
            - Restricted diffusion lasting 7 to 10 days
        - PWI
            - Similar to perfusion CT

        - Contrast
            - Variable enhancement
                - Hyperacute: intravascular enhancement
                - Acute: meningeal enhancement
                - Subacute: parenchymal enhancement, particularly in the involved gray matter (either cortex or basal ganglia)
    - MR angiography
        - Identify occlusions, stenoses, and status of collateral flow
    - MRS
        - Elevated lactate and decreased *N*-acetyl-aspartate
- Imaging recommendations
    - Nonenhanced CT as initial study to exclude hemorrhage or focal mass
    - If available, obtain perfusion CT and CT angiography for potential intravascular treatment
    - If uncertain or need confirmation, obtain MRI with contrast (including DWI, GRE, and possibly PWI)
- Mimic
    - When in the subacute phase, demonstrating ring enhancement, infarction can be difficult to distinguish from tumor on imaging. Subacute lacunar infarcts, in particular, often have the appearance of small metastases. Diffusion abnormalities of subacute infarct, vascular territory of involvement, or sequelae of other previous infarcts may help to indicate infarct over tumor, but sometimes follow-up MRI is the best next step in evaluation.

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