Acute stroke

Ischaemic stroke

Characteristics

- Stroke is the third most common cause of death in the UK, and the leading cause of disability.
- 80% of strokes are ischaemic
  - Large vessel occlusive atheromatous disease (50%)
  - Small vessel disease of penetrating arteries (25%) = lacunar infarct
  - Cardiogenic emboli (20%)
  - Non-atheromatous causes (5%)
- Ischaemic infarction of the brain may be secondary to thrombosis or embolic disease.
- Transient ischaemic attacks (TIAs) precede a quarter of ischaemic strokes, and over 40% of these are in the 7 days before the stroke. The risk is highest in those patients with carotid stenosis or artial fibrillation.
- The incidence of stroke increases with age, although one in four people who experience a stroke are under 65 yrs.
- Risk factors include hypertension, smoking, diabetes, hyperlipidaemia, atherosclerosis, atrial fibrillation, the oral contraceptive pill and obesity.

Temporal classification

- TIA = transient ischaemic attack. The clinical syndrome lasts less than 24 hours, although in a proportion there may be infarction on cerebral imaging.
- Progressing stroke = stepwise or gradually progressing accumulative neurological deficit evolving over hours or days
- Completed stroke = persistent stable neurological deficit – cerebral infarction as end stage of prolonged ischemia.
  - Thrombolysis therapy has the potential to revolutionise the rapid assessment and treatment of ischaemic strokes **(see Appendix 2**)

Clinical features

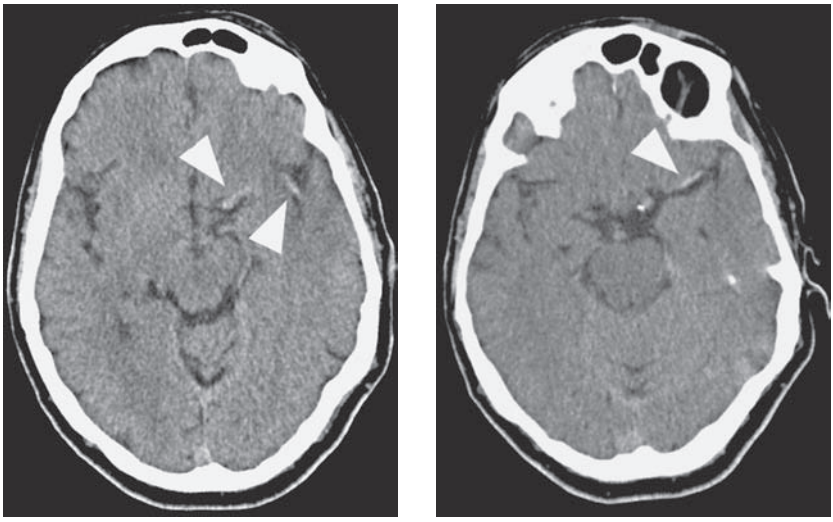
- Spectrum of presentation from mild symptoms and signs, in a well patient, to a moribund comatosed patient.
- Commonly presents with unilateral weakness and/or sensory loss, visual field defect, dysphasia, and inattention/neglect.

- Lacunar infarcts typically present with a purely motor and/or sensory deficit. Features of cortical involvement (visual field defect, dysphasia or inattention/neglect) are absent.
- Posterior circulation infarcts commonly present with vertigo, ataxia, diplopia, dysarthria, dysphasia or bilateral limb signs.
- The neurological deficit can be sudden, often occurring during sleep. This makes the time of onset difficult to ascertain.

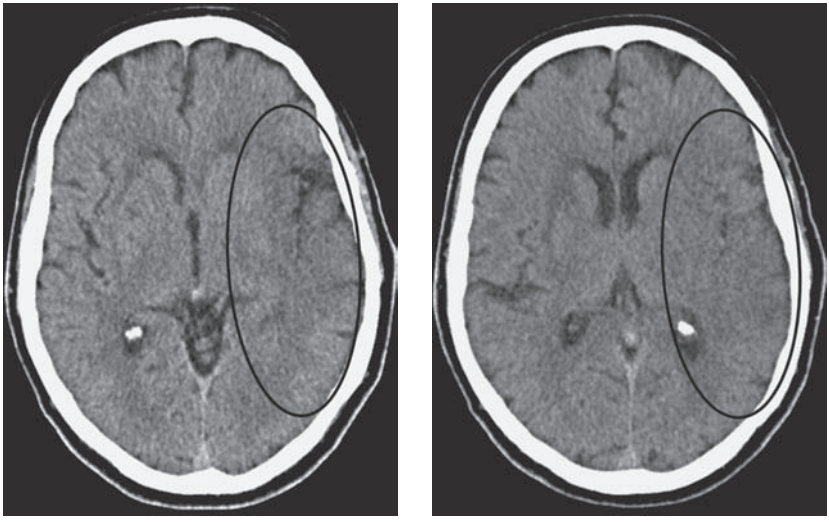
Radiological features

CT features

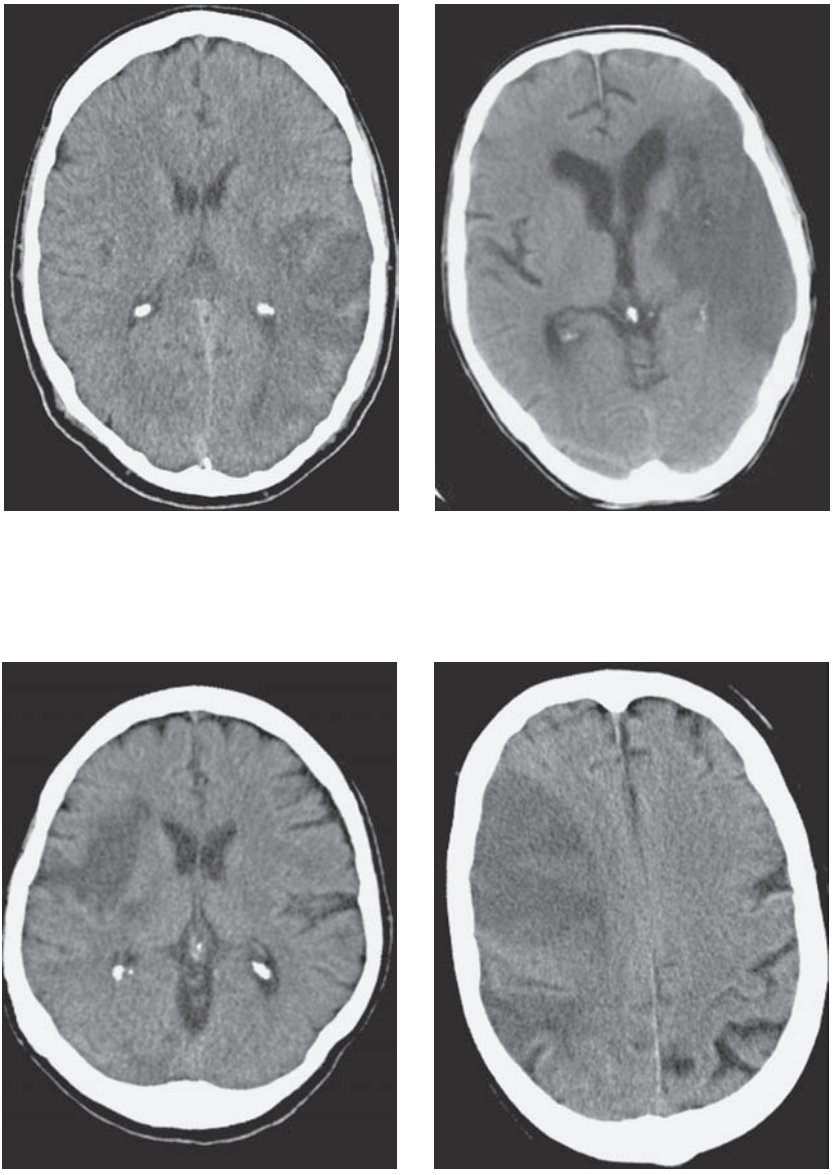
- *Hyperacute infarct (< 12 hours)*:
  - Non-contrast CT may appear normal in up to 60%.
  - However, contrary to general opinion, the CT may be abnormal in up to 75% of patients with MCA infarction, imaged within the first 3 hours.
  - 'Hyperdense MCA' sign represents acute intraluminal thrombus, and is seen in 25–50% of acute MCA occlusions. It is recognised as focal or linear white density within the MCA in the Sylvian fissure. Although not sensitive, it is a relatively specific sign.
  - The normally well-defined lentiform nucleus becomes obscured in 50–80% of acute MCA occlusions.
- *Acute infarction*
  - ⋆ *12–24 hours*
  - Low-density basal ganglia
  - Loss of normal grey/white differentiation secondary to oedema
  - Look for 'the insular ribbon sign' = hypodense extreme capsule no longer distinguishable from insular cortex.
  - Loss of the normal sulcal pattern is suspicious of underlying oedema.
  - ⋆ *1–7 days*
  - Area of hypodensity in a vascular distribution (in 70%) due to cytotoxic oedema
  - Mass effect – local or generalised compression of the ventricles, basal cisterns and midline shift.
  - Haemorrhagic transformation may occur after 2–4 days in up to 70%.
- *Subacute/chronic infarction (> 7 days – months)*
  - Decrease of mass effect and *ex vacuo* dilatation of ventricles.
  - Loss of parenchymal mass, with associated sulcal/ventricular widening, due to encephalomalacia.



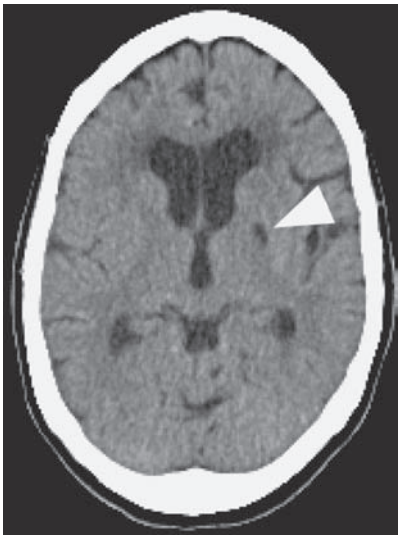
Left MCA territory infarcts: two examples of a hyperdense left MCA due to acute intraluminal thrombus (arrowheads).



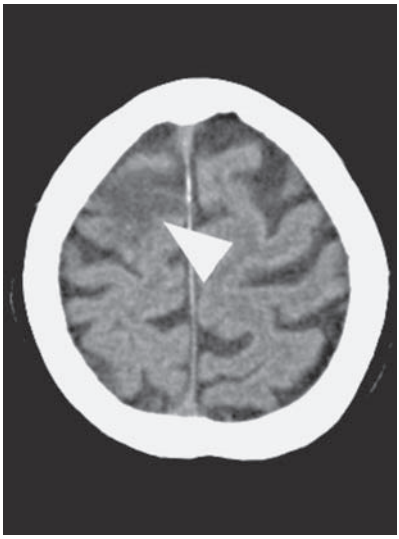
Two examples of early left MCA territory infarction. Note the subtle effacement of grey/white matter differentiation, due to oedema, and the 'insular ribbon sign'.



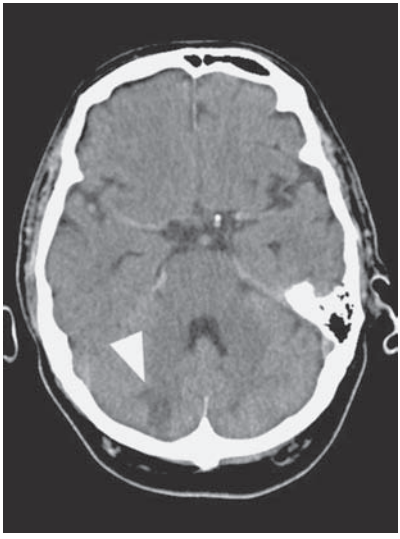
Large areas of hypodensity within the left (top images) and right (bottom images) middle cerebral artery vascular territories, due to cytotoxic oedema.



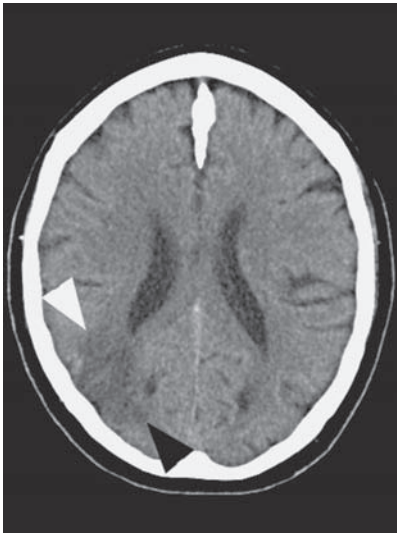
Lacunar infarct left lentiform nucleus (arrowhead).



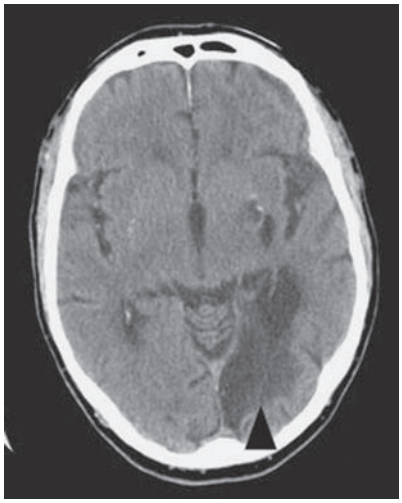
Infarct right superior frontal lobe (arrowhead).



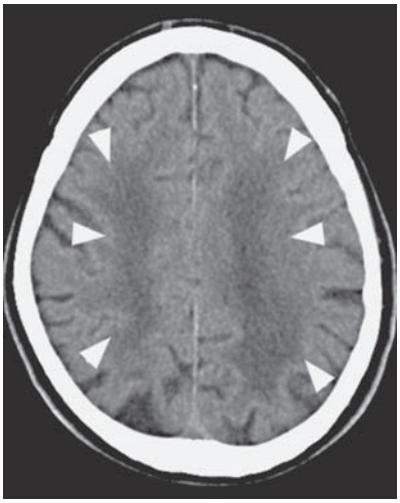
Small cerebellar infarct (arrowhead).



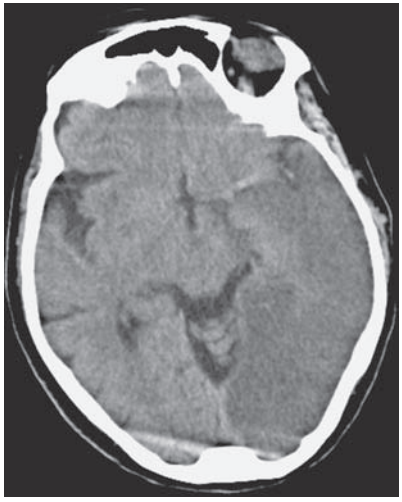
Right posterior watershed infarct. This is an infarct at the 'watershed' between middle and posterior cerebral artery territories (arrowheads).



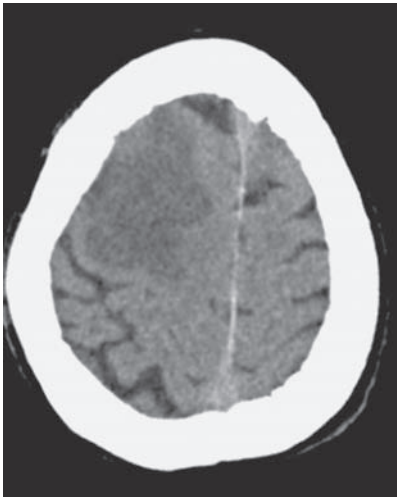
Posterior cerebral artery territory infarct (arrowhead).



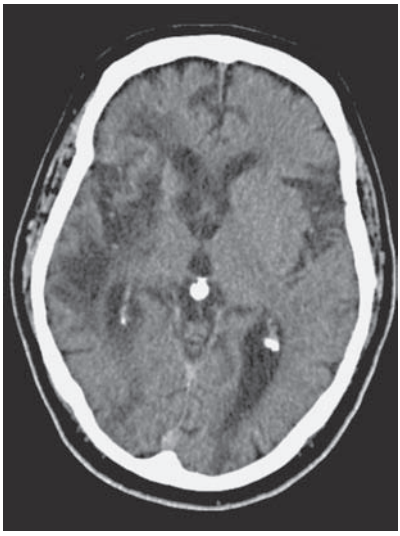
Generalised low attenuation within the deep white matter of both cerebral hemispheres due to small vessel disease (arrowheads).

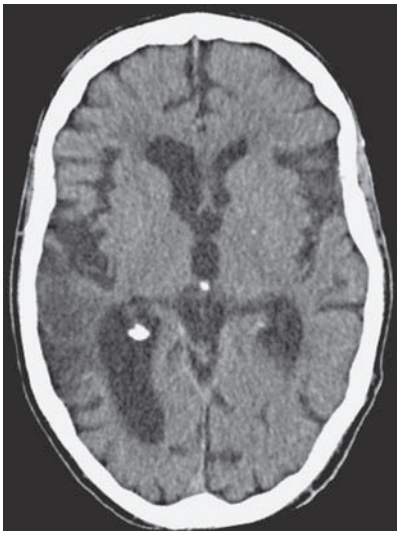


Large infarct involving both the left middle and posterior cerebral artery territories.

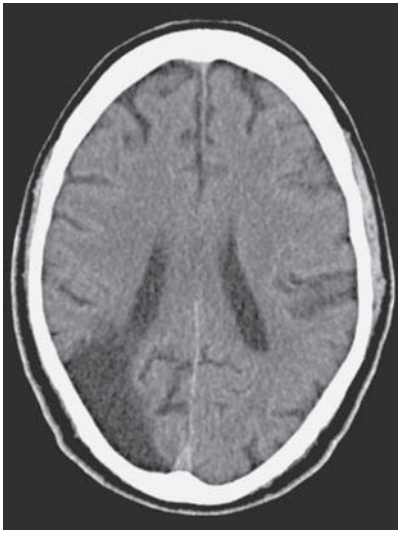


Another example of a right superior frontal lobe infarct.

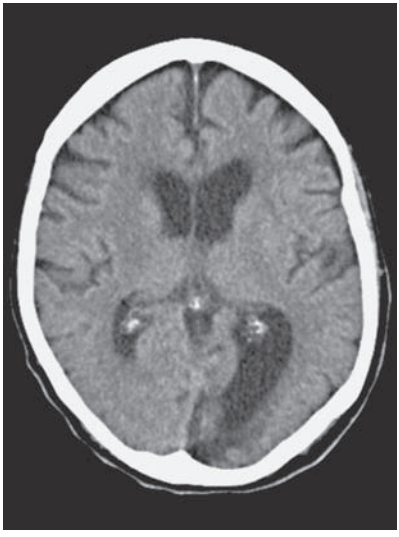




Chronic right MCA territory infarction. The infarcted area is of 'CSF' density due to loss of brain substance, secondary to encephalomalacia, i.e. CSF eventually fills the 'dead' space left following infarction. As a result, there is widening of local sulcal spaces and *ex vacuo* dilatation of adjacent ventricles, in this case the Sylvian fissure and right occipital horn, respectively.



Chronic right posterior watershed infarct.



Chronic left posterior cerebral artery territory infarct with *ex vacuo* dilatation of the left occipital horn.