QUESTION 65

A 68yo man presents with an altered conscious state. Three days prior to presentation he had an episode of ataxia and vertigo with residual imbalance. On the day prior to presentation he developed an acute horizontal diplopia, which persists. He is brought to hospital after a sudden deterioration in his level of consciousness.

The most likely diagnosis is:

A. Pontine haemorrhage
B. Paraneoplastic syndrome
C. Acute disseminated encephalomyelitis (ADEM)
D. Multiple cardioembolic strokes
E. Basilar artery thrombosis

This patient presents with a stepwise decline in neurological function suggestive of crescendo TIAs. A pontine haemorrhage may cause the signs listed in the question but the onset would be sudden rather than over a few days.

ADEM is usually associated with constitutional symptoms and some degree of encephalopathy though neurological signs can vary.

Paraneoplastic syndromes tend to be subacute, developing over weeks to months, or even chronic, over months to years.

Multiple cardioembolic strokes is possible but less likely than a single lesion, especially as there is no known history of AF.

The clinical features described are consistent with a posterior circulation stroke – ataxia and vertigo suggest cerebellar or spinocerebellar tract problems. Horizontal diplopia indicates a problem with the horizontal gaze centre.

BASILAR ARTERY THROMBOSIS

- Basilar artery formed at pontomedullary junction by the 2 vertebral arteries
- Gives off a number of branches to the cerebellum and pons
- Terminal branch is the PCA which supplies the midbrain and thalamus and parts of the temporal and occipital lobes
- Atheroma occurs more often at the origin and termination of the basilar artery
- Clinical picture varies depending on the site of the occlusion and the presence of retrograde flow
- May be features involving the corticospinal, corticobulbar, ascending sensory tracts and cranial nerve nuclei
- In complete basilar artery occlusion there will be bilateral sensory and motor signs as well as cranial nerve and cerebellar dysfunction
- Generally the proximal and distal parts of the basilar artery are occluded by emboli while the mid-section is usually atherothrombotic
Clinical

- A stuttering and progressive course of symptoms is seen in patients with atherosclerotic occlusion
- A lot of patients will have TIAs for several days to weeks prior to the occlusion
- Most common heralding symptoms include:
  o Hemiparesis, facial paresis or quadraparesis
  o Dysarthria
  o Vertigo, nausea and vomiting
  o Headache
  o Visual disturbances
  o Altered consciousness
- If occlusion due to emboli then the presentation is of sudden onset of severe motor and bulbar symptoms with reduced consciousness
- Eye signs reflect involvement of vertical gaze centre of midbrain, abducens nucleus, horizontal gaze centre and/or medial longitudinal fasciculus resulting in:
  o Ipsilateral abducens palsy
  o Ipsilateral conjugate gaze palsy
  o Internuclear ophthalmoplegia
  o One-and-a-half syndrome (ipsilateral conjugate gaze palsy and internuclear ophthalmoplegia)
  o Skewed deviation
  o Ocular bobbing (due to lesion in pons)
- Other pontine signs are:
  o Ataxia
  o Facial weakness
  o Dysarthria
  o Dysphagia
  o Hearing loss
- Locked-in syndrome is due to occlusion of the proximal and middle segments of the basilar artery \( \rightarrow \) ischaemia of basis pontis but tegmentum of pons spared \( \rightarrow \) quadriplegia, consciousness preserved
- Top-of-the-basilar syndrome is due to ischaemia of the upper brainstem and diencephalon (usually due to embolus) \( \rightarrow \) reduced consciousness, visual symptoms (such as hallucinations or blindness), 3rd nerve palsy, papillary abnormalities
- Important to recognise heralding signs of occlusion as complete occlusion carries poor prognosis