Herpes Simplex Encephalitis

Case Studies | October 14, 2013
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**Case History:** A 65-year-old patient with history of altered behavior and memory loss for 15 days

Figure 1: T2WI image showing abnormal hyperintensity in medial temporal lobes
Figures 2-3: Axial FLAIR image showing abnormal hyperintensity in medial temporal lobes
Figure 3
Figures 4-5: Coronal FLAIR image showing abnormal hyperintensity in medial temporal lobes
Herpes Simplex Encephalitis

Discussion: Herpes simplex encephalitis

Diagnosis: Herpes simplex encephalitis

Discussion: Herpes simplex (HSV) encephalitis is the most common cause of fatal sporadic viral encephalitis and has characteristic imaging findings.

Two sub types are recognized which differ in demographics, virus and pattern of involvement:
1. Neonatal herpes encephalitis
2. Childhood and adult herpes encephalitis

Childhood and adult herpes encephalitis is usually due to HSV-1 (90 percent) with the rest due to HSV-2. There is no particular age, sex or seasonal predilection.

Presentation is unfortunately relatively non-specific consisting of fever, headaches, focal neurological deficits, seizures, and altered or decreased level of consciousness.

Diagnosis is established with PCR of CSF, although the combination of clinical scenario, CSF demonstrating pleocytosis and elevated protein, and appropriate imaging is usually highly suggestive and permits commencement of treatment.

In the adult immunocompetent patient, the pattern is quite characteristic, involving the medial temporal lobes, insular cortex and inferolateral frontal lobes. The basal ganglia are typically spared, helping to distinguish it from a middle cerebral artery infarct.

In immunocompromised patients, involvement can be more diffuse, and more likely to involve the brainstem. It is important to appreciate that the pattern of involvement in children and adults is different to that of neonatal HSV encephalitis.

CT: Early diagnosis is difficult and a 'normal' CT scan should not dissuade from the diagnosis. If findings are present, they typically consist of subtle low density within the anterior and medial parts
of the temporal lobed and the island of Reil (insular cortex). If scanned later then the changes may become more obvious and even progress to hemorrhage. Contrast enhancement is uncommon during the first week of the disease. Thereafter patchy low level enhancement may be seen.

MRI: Affected areas however have a similar appearance, in terms of signal characteristics:

- T1
  - May show general edema in affected region
  - If complicated by sub acute hemorrhage there may be areas of hyper intense signal
- T1 C+ (Gd)
  - Enhancement is usually absent early on
  - Later enhancement is variable in pattern
  - Gyral enhancement
  - Leptomeningeal enhancement
  - Ring enhancement
  - Diffuse enhancement
- T2
  - Hyperintensity of affected white matter and cortex
  - More established haemorrhagic components may the hypo intense
- DWI / ADC
  - More sensitive than T2 weighted images
  - Restricted diffusion is common due to cytotoxic oedema
  - Beware of T2 shine through due to vasogenic oedema
- GE / SWI: May demonstrate blooming if haemorrhagic (rare in neonates, common in older patients)

Mortality ranges dramatically depending on how early treatment is instituted. Even in patients who are young and otherwise well, and only lethargic still have a mortality of 25 percent. Older patients or those comatose at the time treatment is started invariably have a much poorer outcome. Overall mortality is over 70 percent with only 2.5 percent of affected patients every fully recovering.

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