Question 1. Regarding imaging techniques supporting the diagnosis of Creutzfeldt-Jakob disease (CJD), which is highly sensitive and specific, and currently widely in use?
(a) Dynamic susceptibility contrast magnetic resonance (MR) imaging.
(b) Diffusion-weighted MR imaging.
(c) Positron emission tomography/computed tomography.
(d) Susceptibility-weighted MR imaging.

Question 2. Common MR imaging patterns of sporadic CJD include:
(a) Bilateral symmetric T2 hyperintense signal abnormalities in the mesial temporal lobes and hippocampi.
(b) Diffuse T2 hyperintensities in the subcortical and deep white matter of the parietal and occipital lobes, with or without microhaemorrhages.
(c) Asymmetric areas of restricted diffusion in the cerebral cortex and/or basal ganglia regions.
(d) Symmetric restricted diffusion in the dorsomedial thalami.

Question 3. A probable CJD is diagnosed based on:
(a) Detection of scrapie prion protein on biopsy.
(b) Positive electroencephalography (EEG) and MR imaging findings on a clinical background of rapidly progressive dementia and myoclonus.
(c) Equivocal MR imaging and EEG results, with the presence of 14-3-3 protein in cerebrospinal fluid (CSF) in a patient without dementia.
(d) Rapidly progressive dementia in less than two years with pyramidal signs but negative MR imaging, CSF analysis and EEG results.

Question 4. Regarding CJD:
(a) It is caused by abnormal formation and accumulation of scrapie prion protein in brain tissue.
(b) There are several subtypes and variant CJD is the best understood subtype thus far.
(c) It is rapidly progressive in clinical course and invariably fatal regardless of treatment.
(d) Visual disturbance is an uncommon clinical presentation.

Question 5. Regarding the differential diagnosis of CJD:
(a) MR imaging is the key to differentiate it from hypoglycaemic encephalopathy.
(b) Encephalitis usually spares the limbic system, unlike CJD.
(c) In a postictal state, transient signal changes can usually be seen in the hippocampi and corpus callosum.
(d) Wernicke’s encephalopathy can also be seen in non-alcoholic individuals.