1. Ventricular cystic neurocysticercosis.
Fig. 1a is a T2-weighted axial scan showing a large, smooth-edged, well-defined cyst in the frontal horn of the left lateral ventricle, together with further smaller cysts in the anterior interhemispheric fissure. There is secondary hydrocephalus. Figures 1b and 1c are T1-weighted coronal scans, pre and post gadolium, respectively. The signal intensity of the cysts is similar to that of CSF and shows no enhancement. These features, together with the clue of her foreign travel, lead to the correct diagnosis of the cystic form of neurocysticercosis. Do not let the fact she is a vegetarian put you off. Contamination of salads through poor horticultural and culinary practices is common.

Humans are accidental hosts. Taenia Solium eggs from the pork tapeworm hatch in the stomach and the larvae burrow through the gut wall and disseminate via the circulation, particularly to the brain. The larvae may encyst. A host reaction may lead to a granulomatous inflammatory reaction. Seizures and focal neurological abnormalities are most likely to occur at this stage. The lesion may then disappear completely, or leave a calcified remnant. Note: calcified lesions are commonly seen in the asymptomatic population in endemic areas. Their presence should not automatically be assumed to be the cause of a patient’s epilepsy. The hydrocephalus sometimes seen with the ventricular forms may be intermittent due to cyst mobility.

Further Reading

2. He had an epileptic fit. He has an astrocytic hamartoma of his optic disc. The appearance has been likened to a mulberry, on account of the glistening, yellowy surface. It is most commonly seen in patients with tuberous sclerosis, although it also occurs in neurofibromatosis and in normal people.

3. (a) True  (b) True  (c) False  (d) False  (e) False

Early on there is usually no wasting, even in a very weak muscle. The electrophysiological correlate is a normal compound muscle action potential. Muscle cramp, fatigue and twitching are frequent features and occur particularly on prolonged use of the limbs. Anti-GM1 antibodies are seen in only 50% of cases – their presence is useful, but absence unhelpful. Intravenous immunoglobulin typically leads to an improvement in a few weeks which then tapers over the following months. In contrast to chronic inflammatory demyelinating polyneuropathy, both plasma exchange and steroids are ineffective in the majority of patients and indeed may produce a dramatic deterioration.

Further Reading