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Fig. 1a Unenhanced axial SE T1-W MR image of the brain taken at the level of the pons.

Fig. 1b Axial FSE T2-W MR image of the brain taken at the level of the midbrain.

Fig. 1c Enhanced axial SE T1-W MR image taken at the same level as Fig. 1a.

Fig. 2 Unenhanced axial CT scan taken using soft tissue window settings.

CASE PRESENTATION

A 36-year-old Korean man presented with one episode of loss of consciousness. He had foam coming out of his mouth and he was clenching his fists. Physical examination was essentially normal. Awake and drowsy electro-encephalography were also normal.

Magnetic resonance (MR) and computed tomography (CT) scans of the brain were performed (Figs. 1a-c & 2). What do these show and what is the diagnosis?
IMAGE INTERPRETATION
MR imaging of the brain showed multiple conglomerated round areas in the were left temporal and occipital lobes. These lesions were low signal intensity on both the T1- and T2-weighted images. Surrounding high signal intensity areas on T2-weighted images represented encephalomalacia (Fig. 1b). There was no significant enhancement of these lesions after intravenous gadopentetate dimeglumine administration (Fig. 1c). CT scan of the brain confirmed the presence of large calcific nodules in the temporal and occipital lobes (Fig. 2).

DIAGNOSIS
Chronic cerebral paragonimiasis.

CLINICAL COURSE
The patient was lost to follow-up. He was sent back to Korea by his company for further investigations and treatment.

DISCUSSION
Paragonimiasis is an infestation caused by a lung fluke, of which the most important is Paragonimus westermani. Endemic areas are East and Southeast Asia, West Africa, and Latin America (1). Human infection occurs by ingestion of the uncooked freshwater crab or crayfish infected with the metacercariae of the fluke. The metacercariae excyst in the small intestine and the larvae penetrate the intestinal wall, the peritoneum and the diaphragm, finally reaching the pleural cavity and lung parenchyma in 2 to 8 weeks. The lung is the main habitat of the adult fluke. Ectopic infection of the brain results from the larvae bypassing the lungs and migrating to the intracranial cavity. This probably occurs through the perivascular spaces in the foramina of the skull base, where the larvae penetrates the meninges and invades the brain substance. Cerebral paragonimiasis is found in approximately 1% of patients with pulmonary paragonimiasis (1). The infection usually involves the posterior portion of one cerebral hemisphere. The cerebellum, brainstem and spinal canal are uncommon sites of involvement.

The acute phase of cerebral paragonimiasis manifests as an exudative aseptic inflammation. When the lesion is formed in the subarachnoid area, local meningocele may occur. Congestion, vasculitis and capillary rupture causing infarction, haemorrhage and necrosis may also occur as a result of the migrating worm, usually in the subarachnoid region. In rare cases, ependymal and periventricular invasion can cause choroiditis, which appears as a thickened enhancing choroid plexus together with periventricular enhancing lesions on MR imaging (1,2). In time, granulomatous lesions are formed around the adult Paragonimus. The granulomatous lesions may appear as abscess-like cystic lesions or solid granulomas. Commonly, the granulomas are multiple, conglomerated, interconnected and are located around a focus.

In the early active phase, the most common and characteristic MR imaging finding is conglomerated, multiple ring-shaped enhancing lesions resembling a “grape cluster” with variable degrees of surrounding oedema. This feature strongly suggests early active cerebral paragonimiasis, especially in endemic areas (1-3). Lesions may also be small, solitary and ring-enhancing. In this situation, differentiation from other granulomas, such as tuberculoma or cysticercosis in the degenerating stage or a cerebral abscess, is not possible. Localised foci of haemorrhage may occur. CT readily detects foci of acute haemorrhage which presents as an area of high attenuation. This may however be overlooked by MR imaging. On MR imaging, the subacute or chronic stage of haemorrhage appears as areas of high or low signal intensity on both T1- and T2-weighted images (1-3). Paragonimiasis should therefore be considered as a possible cause of cerebral haemorrhage and infarction in endemic areas (1). In the chronic stage, liquefactive necrosis and fibrinous gliosis occur around the granulomas, resulting in subcortical cerebromalacia, cortical atrophy, secondary ventricular dilatation and porencephaly. Almost all the granulomas shrink and invariably become calcified. On MR imaging, localised cerebromalacia is seen around the calcifications. The calcifications may have several patterns: punctate and amorphous, spotty round nodular, solitary round cystic, conglomerated multiple round, or oval cystic (so-called “soap bubble,” appearance) on the skull radiograph and CT (1). “Soap bubble” calcification is considered a specific sign for the disease. On MR imaging, the calcifications are seen as areas of signal void or hypointense nodules, and occasionally have an “egg shell appearance” with central content of low or high signal intensity.

In addition to neuroimaging findings, recognition of other findings may point to the clinico-radiological diagnosis of cerebral paragonimiasis. Patients with cerebral paragonimiasis may present with headache, epilepsy, hemiparesis, hyperaesthesia, blurred vision, diplopia, homonymous hemianopsia and meningitic symptoms. In the acute phase, there may be pulmonary symptoms, chest radiograph abnormalities, leucocytosis and eosinophilia in pulmonary paragonimiasis, positive reaction to intradermal test, increased serum immunoglobulin, and cerebrospinal fluid (CSF) findings of elevated protein levels and eosinophilia. A nitroty test by ELISA for paragonimus...
specific antibody (Ig G) in CSF is highly sensitive and specific during early active cerebral paragonimiasis, but the test is usually negative in patients with chronic calcifications\(^2\,^3\).

Due to the rarity of paragonimiasis in Singapore, the diagnosis may be overlooked as a cause of epilepsy. Imaging of this patient demonstrates typical features of chronic cerebral paragonimiasis. In the differential diagnosis, remote considerations of brain tumour and arteriovenous malformation may be made. However, the former would be associated with mass effect and vasogenic oedema, with or without enhancement. Encephalomalacia and calcifications are not a feature, unless there had been prior treatment, e.g. following radiotherapy. Dense conglomerate calcifications are however atypical\(^6\). A normal flow voids in relation to the feeding arteries and draining veins may be demonstrated in large arteriovenous malformations. These may be associated with high flow aneurysms and venous ectasia but conglomerate calcifications are usually absent\(^6\).

REFERENCES

ABSTRACT
A 36-year-old Korean man presented with a history of epilepsy. MR imaging of the brain revealed multiple conglomerated round nodules that were hypointense on both T1-and-T2 weighted images. These were located at the left temporal and occipital lobes and had surrounding encephalomalacia. CT scan confirmed the presence of large calcified nodules in the corresponding regions. These imaging findings were typical of chronic cerebral paragonimiasis. The clinical, CT and MR features of cerebral paragonimiasis are reviewed.

Keywords: Brain infection, cerebral paragonimiasis, magnetic resonance imaging, computed tomography