Hong Kong J Radiol. 2025;28(4):e250-6. Published 10 December 2025 | https://doi.org/10.12809/hkjr2417944

CASE REPORT

Intracranial Neuroendocrine Tumour of Unknown Origin Mimicking Neurocysticercosis: A Case Report

CW Chan, KO Cheung, CY Cheung

Department of Radiology, North District Hospital, Hong Kong SAR, China

INTRODUCTION

Neuroendocrine tumours of the central nervous system are relatively rare entities and most cases are metastases. Primary intracranial neuroendocrine tumour is even rarer, with fewer than a dozen cases reported worldwide.[1-8] Apart from a case report on a 5-year-old child,[9] all reported primary cases have been in adults. The location of lesions reported varies greatly. Reported extra-axial locations include, but are not limited to, the cerebellopontine angle, jugular foramen, cavernous sinus, and skull base. Reported intra-axial locations include the left temporal and parietal lobes, as well as the left cerebellum. We report a case of neuroendocrine tumour of unknown origin with multiple intracranial metastases.

CASE PRESENTATION

A 56-year-old woman with good past health presented to the accident and emergency department with dizziness in October 2021. Initial computed tomography of the brain revealed multiple hyperdense lesions scattered across both cerebral hemispheres, the brainstem, and the cerebellum (Figure 1). Whole-body positron emission

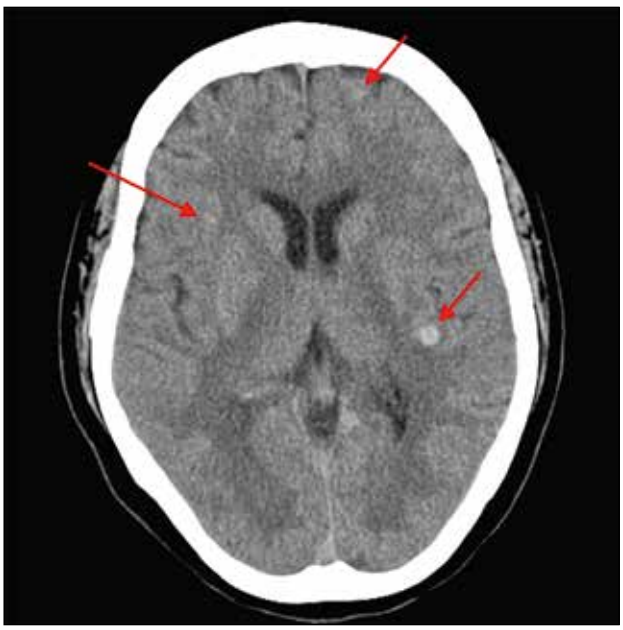

Figure 1. Non-enhanced computed tomography of the brain in axial view, performed in November 2021, showing multiple intra-axial hyperdense lesions (arrows) in the bilateral cerebral hemispheres. Additional lesions are noted in the brainstem and cerebellum (not shown).

Correspondence: *Dr CW Chan, Department of Radiology, North District Hospital, Hong Kong SAR, China*
Email: ccw147@ha.org.hk

Submitted: 3 December 2024; Accepted: 11 April 2025.

Contributors: All authors designed the study. CWC acquired and analysed the data and drafted the manuscript. All authors critically revised the manuscript for important intellectual content. All authors had full access to the data, contributed to the study, approved the final version for publication, and take responsibility for its accuracy and integrity.

Conflicts of Interest: All authors have disclosed no conflicts of interest.

Funding/Support: This study received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Data Availability: All data generated or analysed during the present study are available from the corresponding author on reasonable request.

Ethics Approval: The patient was treated in accordance with the Declaration of Helsinki and provided verbal consent for publication of this case report, including the accompanying clinical images.

tomography–computed tomography (PET-CT) with ^{18}F-fluorodeoxyglucose (^{18}F-FDG) did not reveal any primary malignancy elsewhere that could suggest brain metastases. Contrast-enhanced magnetic resonance imaging (MRI) of the brain was performed to characterise the intracranial lesions (Figure 2). The corresponding cerebral, brainstem, and cerebellar lesions showed T1 hyperintense and T2 heterogeneous mixed signals. Most lesions showed susceptibility artefacts, while some showed a signal on the phase sequence characteristic of calcification. Overall features were suggestive of concurrent haemorrhagic and calcified lesions. Some lesions also showed eccentric nodular enhancement. One 7-mm lesion in the left frontal lobe demonstrated restricted diffusion and a suspicious eccentric scolex (Figure 2b). In view of the previously negative whole-body PET-CT, the possibility of central nervous system infection with neurocysticercosis in different stages was considered a likely possibility. A differential diagnosis of haemorrhagic/calcified brain metastases appeared less likely. Serology testing for *Taenia solium* was negative, but given the radiological appearance of neurocysticercosis, the patient was prescribed a course of albendazole and praziquantel, as well as dexamethasone to minimise cerebral oedema.

Multiple follow-up MRI scans of the brain were performed. Initially, at 1-month post-treatment, some

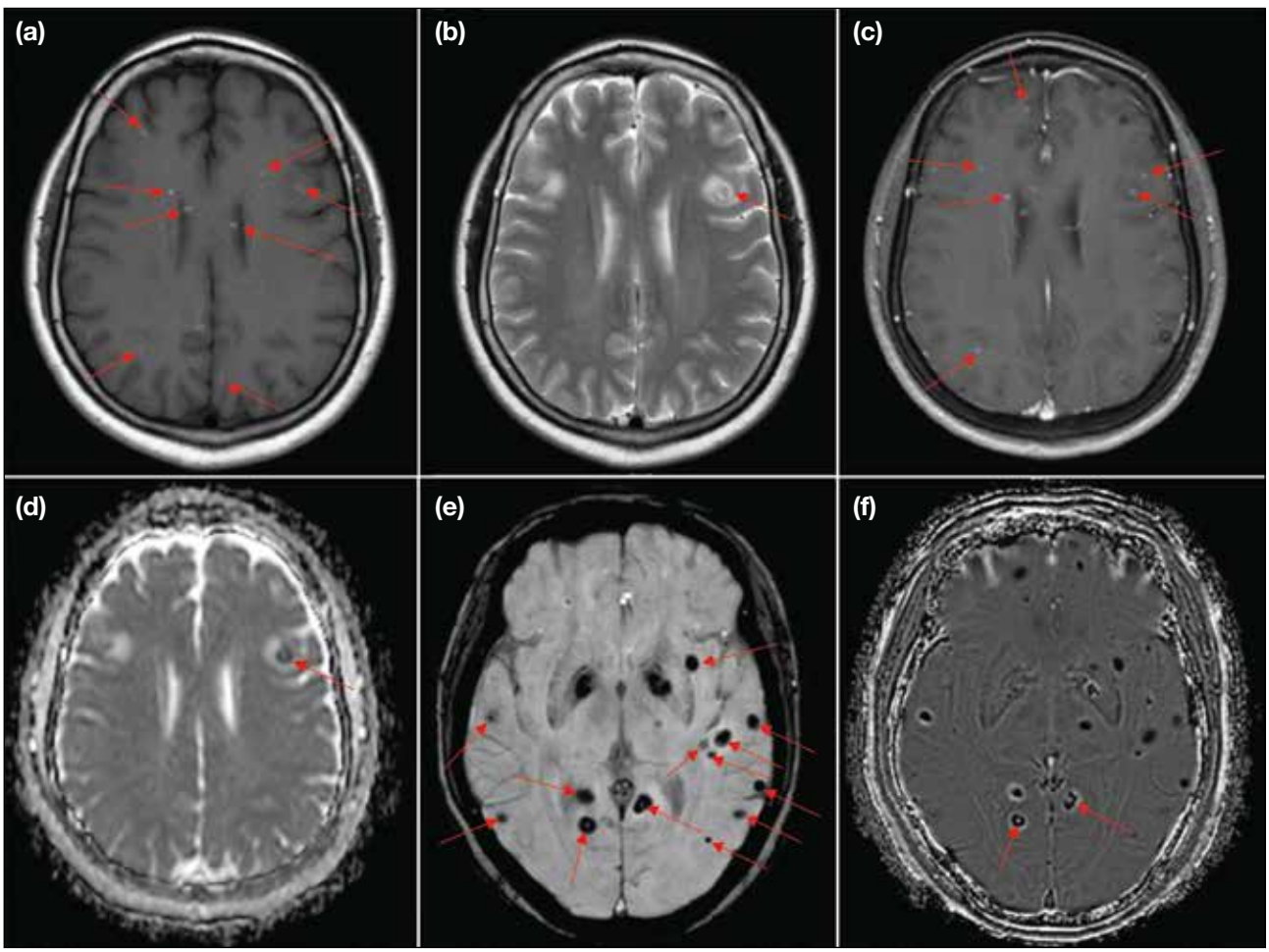

Figure 2. Initial (a) T1-weighted and (b) T2-weighted magnetic resonance imaging confirm multiple intra-axial lesions shows T1 hyperintense and T2 heterogeneous mixed signal, respectively. Some of the lesions show mild adjacent oedema. One of the lesions at left frontal lobe shows suspicious eccentric scolex (arrows). (c) T1-weighted post-contrast image demonstrates nodular enhancement of these lesions (arrows). (d) Apparent diffusion coefficient image shows a lesion (arrow) with peripheral rim-like restricted diffusion in the left frontal lobe. (e) Susceptibility-weighted imaging sequence shows extensive blooming artefacts (arrows). (f) Phase sequence shows some of the lesions (arrows) contain hyperintense signal suggestive of diamagnetic compounds such as calcification. Note the bright spot in the pineal gland (physiological calcification), used as a reference. The hypointense signal is suggestive of paramagnetic and/or superparamagnetic compounds, such as blood products.

lesions (particularly those at the frontal and temporal lobes) showed interval enlargement with an increase in perilesional vasogenic oedema (Figure 3). These findings were thought to be attributable to posttreatment change. A scan at 5 months posttreatment revealed continued progression of some lesions in the bilateral frontal and left inferior parietal lobes (Figure 4a and b), while some lesions in the bilateral temporal lobes had regressed (Figure 4c and d). The overall picture favoured a mixed treatment response.

A further course of antiparasitic treatment was given, assuming the infection was unresolved. Nonetheless, follow-up scan at 16 months after initiation of antiparasitic treatment showed not only persistent lesions, but interval enlargement of some (the largest at the left cerebellar hemisphere; Figure 5), with developing obstructive hydrocephalus. In view of the patient's worsening symptoms of increased intracranial pressure (headache, dizziness and vomiting), as well as imaging findings, the neurosurgical team intervened and left posterior craniotomy was performed for decompression and to excise the left cerebellar lesion. An external ventricular drain was placed. Intraoperative findings noted a large intra-axial tumour at the left cerebellar hemisphere, likely malignant. Pathology confirmed a grade 3 neuroendocrine tumour, with additional comment that a metastatic lesion was likely. A repeated whole-body PET-CT with gallium-68-DOTA-tyr^3-octreotate (Ga-68-DOTATATE) showed multiple hypermetabolic nodules in the brain suggestive of known neuroendocrine tumour (Figure 6), but still no obvious location for a primary malignancy. A preliminary diagnosis was reached of neuroendocrine tumour of unknown origin, with possible primary within the brain. Postoperatively, the patient underwent further follow-up MRI scans that

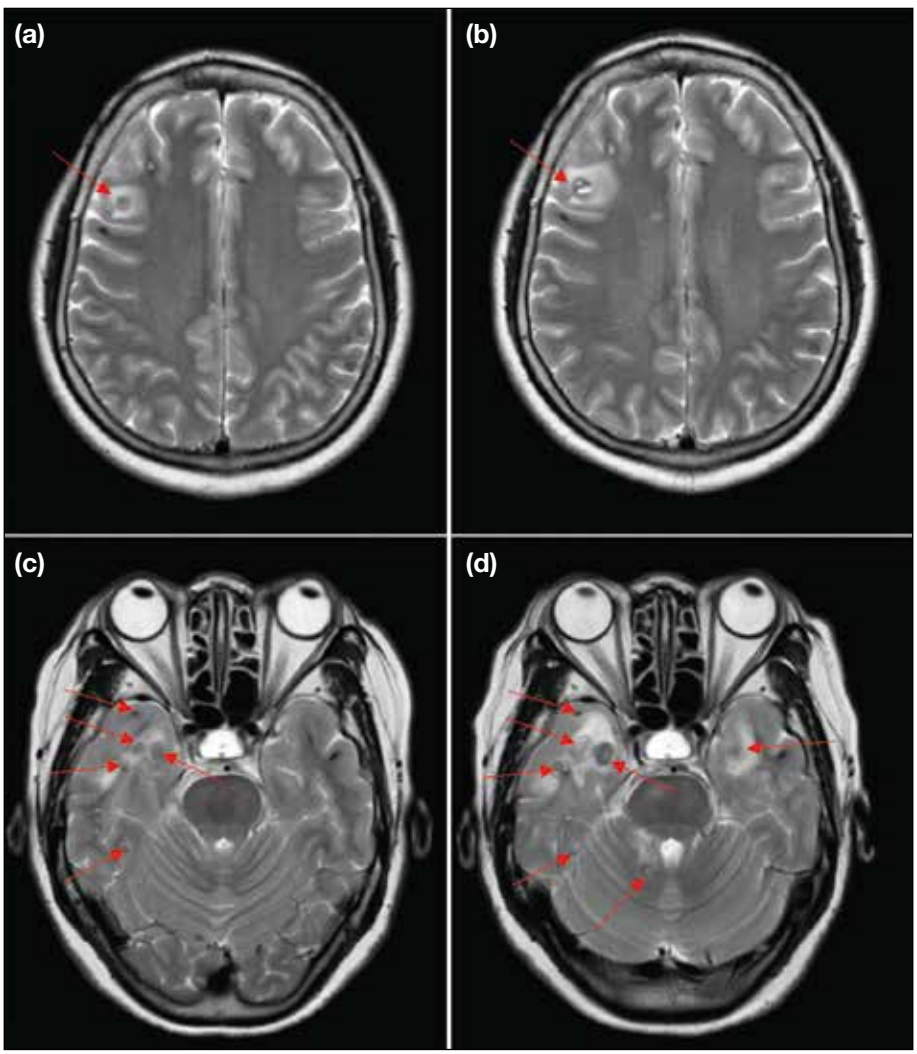

Figure 3. (a) Pre-antiparasitic treatment T2-weighted image (taken in December 2021) shows a right frontal lobe lesion (arrow). (b) One-month posttreatment image (i.e., following antiparasitic treatment in January 2022) shows interval enlargement of the lesion with increased vasogenic oedema (arrow). (c) Pre-antiparasitic treatment T2-weighted image shows multiple lesions in the right temporal lobe (arrows). (d) One-month posttreatment image shows interval progression of the lesions (arrows).

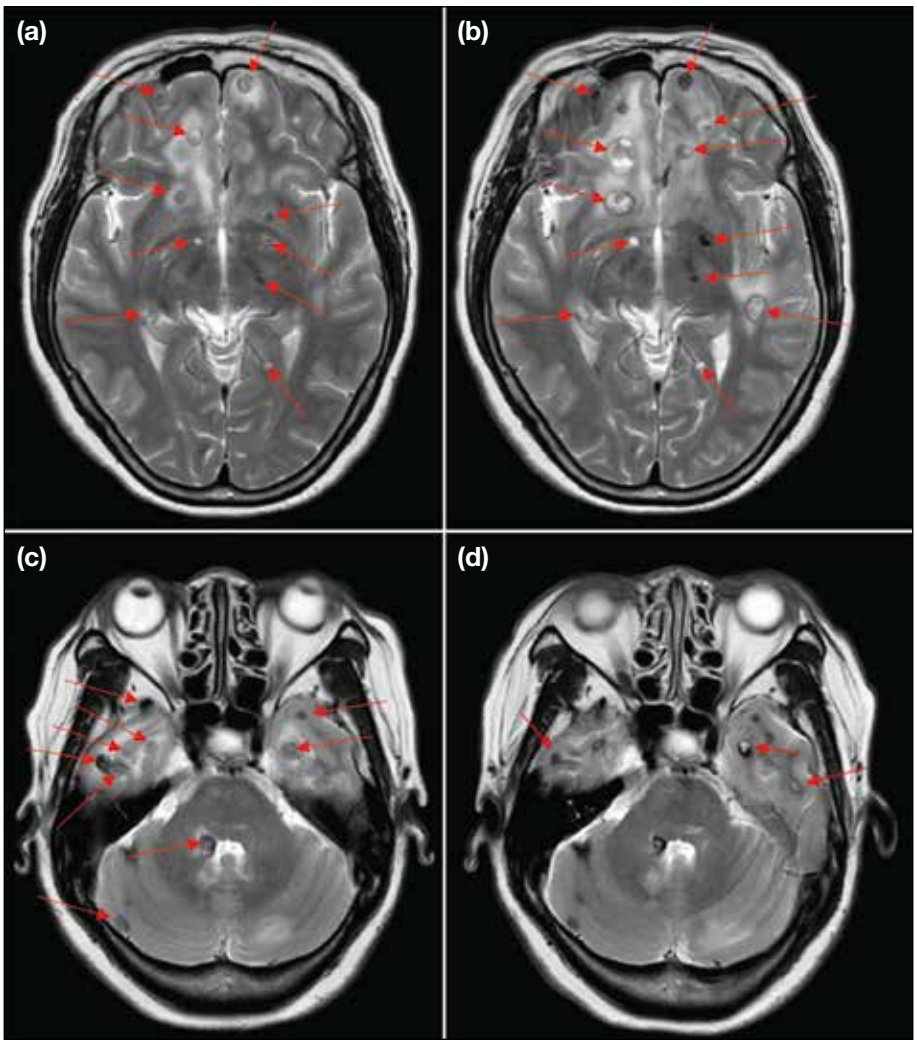

Figure 4. (a) One-month posttreatment T2-weighted image (taken in February 2022) shows multiple lesions in the frontal lobes. (b) Five-month posttreatment image (taken in June 2022) shows interval enlargement of these lesions and a new left inferior parietal lobe lesion. Note the increase in vasogenic oedema. (c) One-month posttreatment T2-weighted image shows multiple lesions in the bilateral temporal lobes and cerebellum (arrows). (d) Five-month posttreatment image shows interval regression of some of the temporal lobe lesions (arrows), with interval reduction in vasogenic oedema.

revealed new suspicious drop metastasis at the C4 level, as well as significant progression of brain metastases and worsening vasogenic oedema (Figure 7). The patient was followed up by the neurosurgery and oncology teams and underwent radiotherapy of the whole brain and the cervical spinal cord as palliative care. At 35 months after the initial presentation, the patient died due to a complication of pneumonia.

DISCUSSION

Neurocysticercosis and neuroendocrine tumour of the brain are two distinct entities that require very different treatment approaches. The patient's presenting signs and symptoms (such as headache, dizziness and seizure) are often non-specific. Serology testing for *Taenia solium*, while specific, is often not sensitive. A negative serology test does not exclude the diagnosis of neurocysticercosis; hence, it was reasonable for our patient to undergo a trial of antiparasitic treatment based on radiological appearance alone.

Imaging plays an important role in guiding the diagnosis as well as treatment in such difficult cases. Nonetheless, as with our case, imaging also has its limitations and can be misguided by disease mimics.

On MRI, neurocysticercosis has varied radiological appearances depending on its four main stages.[10,11] During the vesicular stage, cysts with cerebrospinal fluid (CSF) intensity are often seen, sometimes with an eccentric scolex that may show enhancement. Typically, no surrounding vasogenic oedema is seen at this stage. Intraventricular cysts may be difficult to visualise, and heavily T2-weighted sequences such as FIESTA (fast

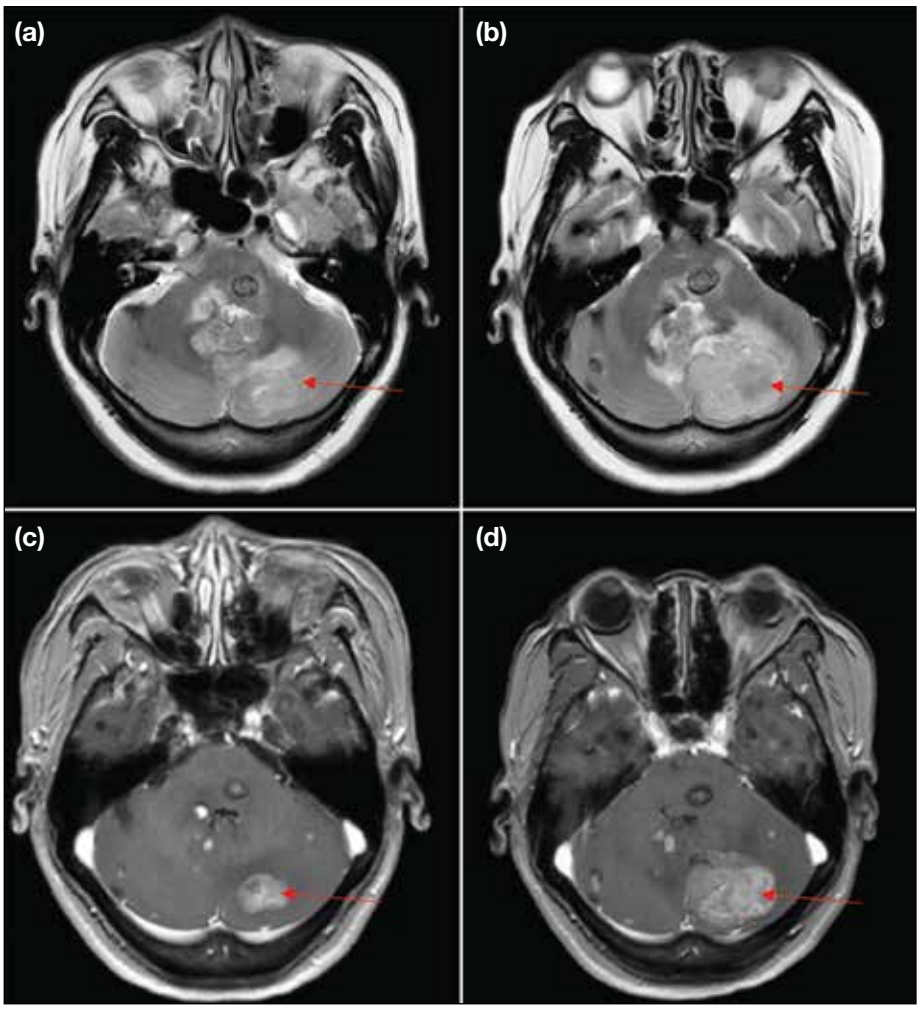

Figure 5. T2-weighted imaging at (a) 5 months posttreatment (taken in June 2022) and (b) 16 months posttreatment (taken in May 2023) shows enlargement of the left cerebellar lesion (arrows). T1-weighted post-contrast images of the same lesion at (c) 5 months posttreatment and (d) 16 months posttreatment (arrows).

imaging employing steady-state acquisition) may help delineate the walls and scolex of neurocysticercosis. In addition, the cystic content may show a slightly lower signal compared with CSF, making them stand out.[12] For our case, the FIESTA sequence was not performed due to limited resources.

During the colloidal vesicular stage, cysts will often contain increased proteinaceous content, leading to T1 and fluid-attenuated inversion recovery hyperintense signal relative to CSF. Thickening and enhancement of the cyst wall, as well as surrounding oedema, may be seen. Some lesions may also show restricted diffusion,[11] as in our case, which further complicates the clinical picture. During the granular nodular stage, the cystic component will resolve, becoming a small enhancing nodule. Contrast enhancement and perilesional oedema will gradually decrease and eventually resolve in the final nodular calcified stage, where calcified nodules are seen.

Neuroendocrine tumour of the brain, whether primary or secondary, can also have variable appearances mimicking other diseases. Among the reported primary cases, MRI appearances ranged from a solid enhancing mass to a cystic mass with a peripheral enhancing component.[1-9]

Spontaneous regression of up to one quarter of neuroendocrine tumours has also been reported, albeit most were extracranial in location, possibly due to host immune response against neoantigens expressed by the tumour.[13] This further increases diagnostic confusion, as in our patient, and led us to interpret the regression of lesions as a partial response to antiparasitic treatment.

Other imaging modalities such as PET scan may

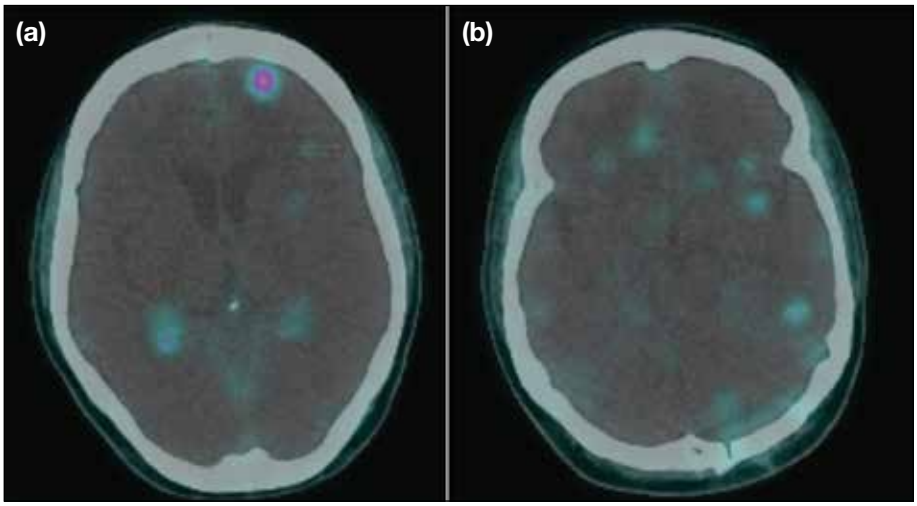

Figure 6. Selected positron emission tomography–computed tomography images using gallium-68-DOTATATE tracer show multiple intra-axial hypermetabolic nodules in the brain and are consistent with known neuroendocrine tumour metastases. No obvious primary malignancy is identified elsewhere.

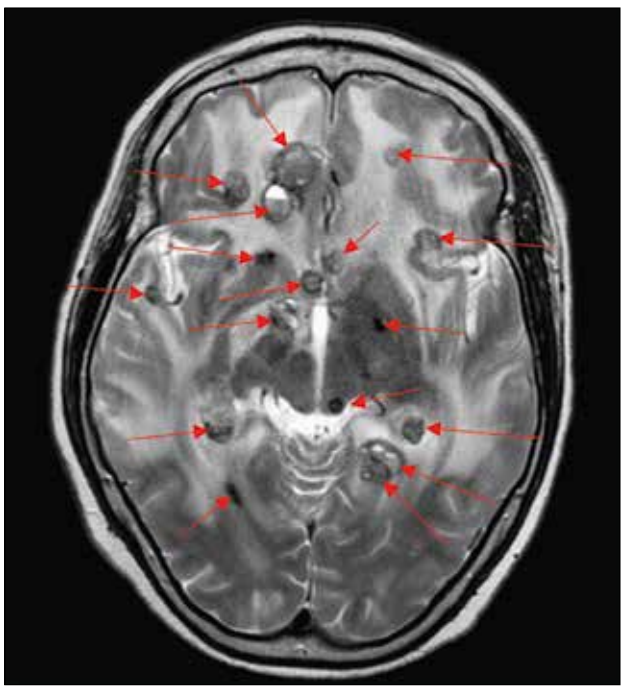

Figure 7. T2-weighted axial image taken in March 2024 shows further disease progression, with interval increase in the size and number of metastatic lesions (arrows), as well as extensive cerebral oedema.

offer more diagnostic clues, but ^{18}F-FDG, which is the most common tracer, may not show uptake in well-differentiated neuroendocrine tumours. On the contrary, Ga-68-DOTATATE has a high sensitivity and specificity in the detection of neuroendocrine tumours.[14] Contrary to ^{18}F-FDG which targets glucose metabolism, Ga-68-DOTATATE targets somatostatin receptors that are usually overexpressed by neuroendocrine tumours. Nonetheless, this tracer is not yet widely available in our region.

With hindsight, there are lessons to be learnt from our patient and improvements to be made, especially in her management. There was an 11-month period (between 5 and 16 months posttreatment) with no imaging follow-up or further workup. There were already significantly enlarging lesions on the 5-month posttreatment scan, and although present, regression of the temporal lesions was subtle. More aggressive follow-up imaging (e.g., within a few months) would have been appropriate.

Furthermore, brain parenchymal haemorrhage, which was already present on her initial MRI scan, is an uncommon finding in neurocysticercosis. Alternative differential diagnoses should have been considered, especially in view of the suboptimal radiological response to antiparasitic treatment. Given the vital location of the enlarging lesions, further investigations such as brain biopsy should also have been considered and offered at an earlier stage.

Although treatment trials with antiparasitic drugs and interval follow-up scans may provide a general idea of the course of the disease, histological diagnosis including excisional biopsy may be the only means by which to confirm a diagnosis.

CONCLUSION

Neurocysticercosis in our region is uncommon, and neuroendocrine tumour of the brain is even rarer. We encountered an atypical presentation of a neuroendocrine

tumour of the brain mimicking neurocysticercosis. A multidisciplinary approach involving the infectious diseases team, as well as neurosurgical and oncological specialists, is necessary to reach definitive diagnosis.

REFERENCES

1. Caro-Osorio E, Perez-Ruano LA, Martinez HR, Rodriguez-Armendariz AG, Lopez-Sotomayor DM. Primary neuroendocrine carcinoma of the cerebellopontine angle: a case report and literature review. Cureus. 2022;14:e27564.
2. Porter DG, Chakrabarty A, McEvoy A, Bradford R. Intracranial carcinoid without evidence of extracranial disease. Neuropathol Appl Neurobiol. 2000;26:298-300.
3. Deshaies EM, Adamo MA, Qian J, DiRisio DA. A carcinoid tumor mimicking an isolated intracranial meningioma. Case report. J Neurosurg. 2004;101:858-60.
4. Ibrahim M, Yousef M, Bohnen N, Eisbruch A, Parmar H. Primary carcinoid tumor of the skull base: case report and review of the literature. J Neuroimaging. 2010;20:390-2.
5. Hakar M, Chandler JP, Bigio EH, Mao Q. Neuroendocrine carcinoma of the pineal parenchyma. The first reported case. J Clin Neurosci. 2017;35:68-70.
6. Liu H, Wang H, Qi X, Yu C. Primary intracranial neuroendocrine tumor: two case reports. World J Surg Oncol. 2016;14:138.
7. Reed CT, Duma N, Halfdanarson T, Buckner J. Primary neuroendocrine carcinoma of the brain. BMJ Case Rep. 2019;12:e230582.
8. Tamura R, Kuroshima Y, Nakamura Y. Primary neuroendocrine tumor in brain. Case Rep Neurol Med. 2014;2014:295253.
9. Stepien N, Haberler C, Theurer S, Schmook M, Lütgendorf-Caucig C, Müllauer L, et al. Unique finding of a primary central nervous system neuroendocrine carcinoma in a 5-year-old child: a case report. Front Neurosci. 2022;16:810645.
10. Teitelbaum GP, Otto RJ, Lin M, Watanabe AT, Stull MA, Manz HJ, et al. MR imaging of neurocysticercosis. AJR Am J Roentgenol. 1989;153:857-66.
11. Santos GT, Leite CC, Machado LR, McKinney AM, Lucato LT. Reduced diffusion in neurocysticercosis: circumstances of appearance and possible natural history implications. AJNR Am J Neuroradiol. 2013;34:310-6.
12. Neyaz Z, Patwari SS, Paliwal VK. Role of FIESTA and SWAN sequences in diagnosis of intraventricular neurocysticercosis. Neurol India. 2012;60:646-7.
13. Amoroso V, Agazzi GM, Roca E, Fazio N, Mosca A, Ravanelli M, et al. Regression of advanced neuroendocrine tumors among patients receiving placebo. Endocr Relat Cancer. 2017;24:L13-6.
14. Yang J, Kan Y, Ge BH, Yuan L, Li C, Zhao W. Diagnostic role of Gallium-68 DOTATOC and Gallium-68 DOTATATE PET in patients with neuroendocrine tumors: a meta-analysis. Acta Radiol. 2014;55:389-98.