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PICTORIAL ESSAY

Magnetic Resonance Imaging Features of Cerebral Ring-Enhancing Lesions with Different Aetiologies: a Pictorial Essay

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BACKGROUND

Cerebral ring-enhancing lesions are defined as an area of hypodensity (in computed tomography) or hypointensity (in magnetic resonance imaging [MRI]) of brain tissue surrounded by a rim of enhancing tissue after contrast injection. Presentation of diseases varies depending on the site and extent of brain involvement and the aetiology. It is always important to correlate clinical symptoms and any previous imaging as the same imaging appearance can suggest vastly different aetiologies with variation in disease presentation. The aetiologies of cerebral ring-enhancing lesions grossly include infectious, neoplastic, post-treatment, demyelinating, and vascular causes. A series of cases with definitive diagnosis based on clinical, microbiological, or pathological evidence were retrieved from our hospital database and their radiological images reviewed. Multiple aetiologies of cerebral ring-enhancing lesions will be discussed. Imaging features will mainly focus on those shown on MRI.

INFECTIOUS CAUSES

Pyogenic Abscess

Pyogenic abscess is a potentially life-threatening disease entity. Presentation includes septic symptoms, focal neurological deficit depending on region of disease involvement and symptoms of increased intracranial pressure. The disease usually presents rather quickly with rapid deterioration over days. Predisposing factors include those that facilitate haematological spread of pathogens such as infective endocarditis and right to left cardiac shunt with concomitant septic focus in other regions.[1] The most common pathogen is *Streptococcus* species and is identified in 35% to 50% of cerebral pyogenic abscesses.[1] Regarding MRI features, the central part of the lesion will be T1 hypointense and T2 mildly hyperintense with no suppression on fluid-attenuated inversion recovery (FLAIR) sequence. Peripheral ring enhancement should be smooth and thin, with the wall mostly being T1 and T2 hypointense.[2] One of the features of pyogenic abscess is restricted diffusion at the abscess cavity, with high diffusion-weighted imaging (DWI) signal and lower apparent diffusion coefficient signal. This is due to tightly packed cellular content within the abscess cavity. Pyogenic abscess often has marked surrounding vasogenic oedema with mass effect that is significant compared with abscess size (Figure 1). If it ruptures into the ventricles, ventriculitis will ensue in which there will be intraventricular debris at the dependent region with restricted diffusion and enhancement along the ependymal surface of the ventricle (Figure 2). The prognosis is often very poor in such cases.

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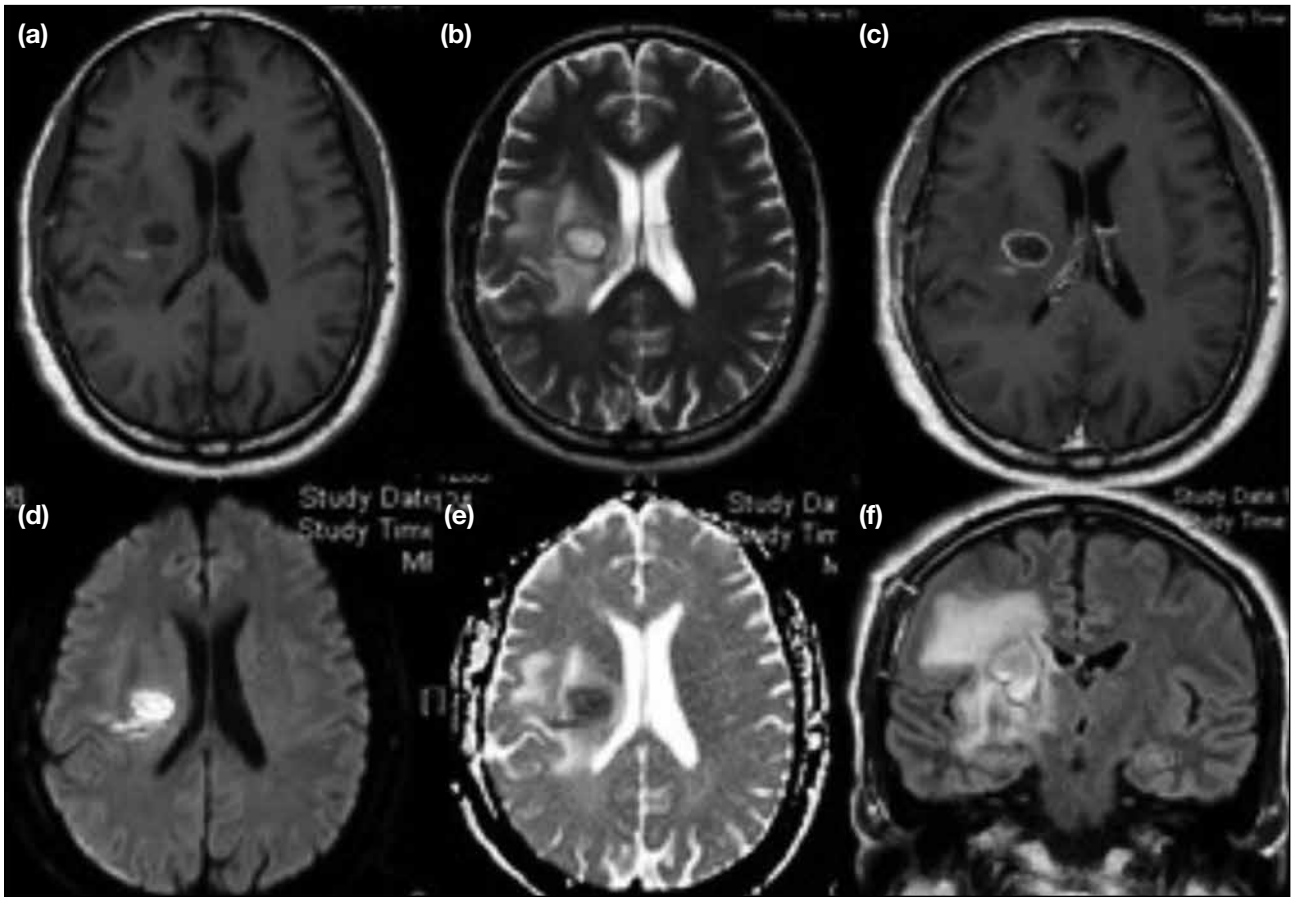

Figure 1. Case of pyogenic abscess at right corona radiata. (a, b) The lesion appears T1 hypointense and T2 hyperintense. The abscess wall appears T2 hypointense. Mild subarachnoid haemorrhage is also seen at the right frontal sulcus. (c) Post-contrast image showing smooth and thin wall peripheral ring enhancement of the abscess. (d, e) Restricted diffusion is seen at the abscess cavity. (f) Fluid-attenuated inversion recovery sequence showing hyperintense signal at the abscess cavity. Marked perilesional oedema is also noted.

Tuberculoma

Tuberculoma is granulation tissue that occurs following central nervous system (CNS) tuberculosis infection. It differs to the much less common tuberculosis abscess that is a true collection of pus.[3] It can occur with or without tuberculous meningitis. When there is concomitant tuberculous meningitis that predominantly affects the basal cistern, the patient may present with cranial nerve palsy. The lesion is usually T1 hypointense and T2 hypointense/isointense. However, T2 hyperintensity may be noted when there is central liquified caseating material.[4] Restricted diffusion is usually not a feature. Enhancement pattern is usually ring-shaped but a mass-like enhancing pattern may sometimes be seen. The presence of concomitant basal cistern leptomeningeal enhancement suggests meningitis with a high probability of CNS tuberculosis as the underlying aetiology (Figure 3).

Fungal Abscess

Fungal cerebral abscess is a rare cause of cerebral ring-enhancing lesion. It usually occurs in immunocompromised individuals such as those prescribed immunosuppressive therapies or having undergone organ transplantation.[5] Morbidity and mortality is high, and diagnosis should be made as soon as possible. The estimated mortality is 85% to 100%.[6] On MRI, the lesions are usually T1 hypointense and T2 hyperintense with no suppression on FLAIR sequence. Fungal abscesses are more likely to be multiple with involvement of the basal ganglia whereas pyogenic abscesses are likely to be solitary and rarely involve the basal ganglia.[6] One characteristic that has been demonstrated in fungal abscess is restricted diffusion of the abscess wall instead of the abscess cavity (Figure 4). In pyogenic abscess, the cavity shows marked restricted diffusion.[6,7]

Cerebral Toxoplasmosis

Cerebral toxoplasmosis is a parasitic infection in the brain with *Toxoplasma gondii*. It is the most common cause of

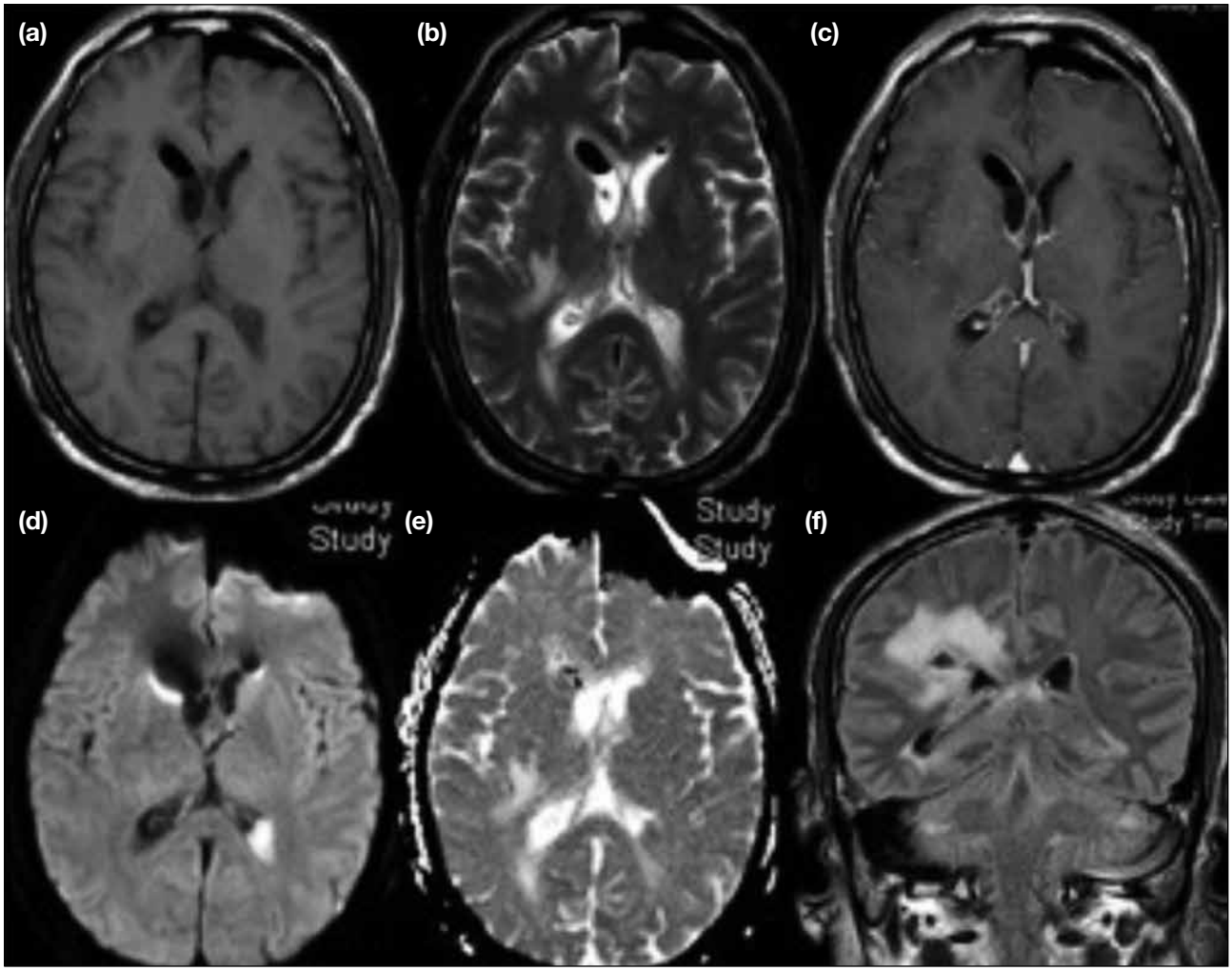

Figure 2. Case of ventriculitis due to ruptured pyogenic abscess. (a, b) Periventricular T2 hyperintensity likely due to transependymal oedema. Bilateral external ventricular drainage catheters are seen. Gaseous content within ventricles is noted. (c) Enhancement along the ependymal surface is noted. (d, e) Restricted diffusion at the left occipital horn due to infected debris. (f) Perilesional oedema around the right parietal abscess and ventricle on fluid-attenuated inversion recovery sequence.

brain abscesses among HIV-positive patients and is also an AIDS-defining illness.[8] In immunocompetent people, infection is often asymptomatic. Cause of infection is most likely due to ingestion of food contaminated by cat faeces that carry the oocytes of the parasites. On MRI, cerebral toxoplasmosis usually appears as multiple small abscesses at the grey-white junction, basal ganglia, and thalami.[9] They are usually T1 isointense to hypointense with variable T2 signal. No definite restricted diffusion within the abscess cavity is seen. Ring enhancement is often seen on contrast injection. The characteristic "eccentric target sign" is highly specific for cerebral toxoplasmosis but is only present in 30% of cases.[10] The lesion has an inner eccentric enhancing core surrounded by a hypointense zone and an outer peripherally enhancing rim, overall giving an eccentric mural nodule appearance (Figure 5).[11]

Neurocysticercosis

Neurocysticercosis is caused by CNS infection with the pork tapeworm *Taenia solium*. The disease is endemic in certain parts of Asia, Africa, and America. The most common presentation in endemic areas is seizure. Other presentations will depend on the site of involvement in the brain. If the lesion is seen within the ventricular system and causes obstruction to cerebrospinal fluid flow, hydrocephalus may develop. The disease is spread by ingestion of food contaminated with *Taenia solium* eggs. Neurocysticercosis develops over four stages. During the vesicular stage, the membrane of the parasite is intact and the parasite is viable. In the colloidal vesicular stage, the parasite dies and the membrane becomes leaky. Significant adjacent oedema around different parasitic lesions is seen during this stage. The granular nodular stage is reached when the extent of oedema decreases.

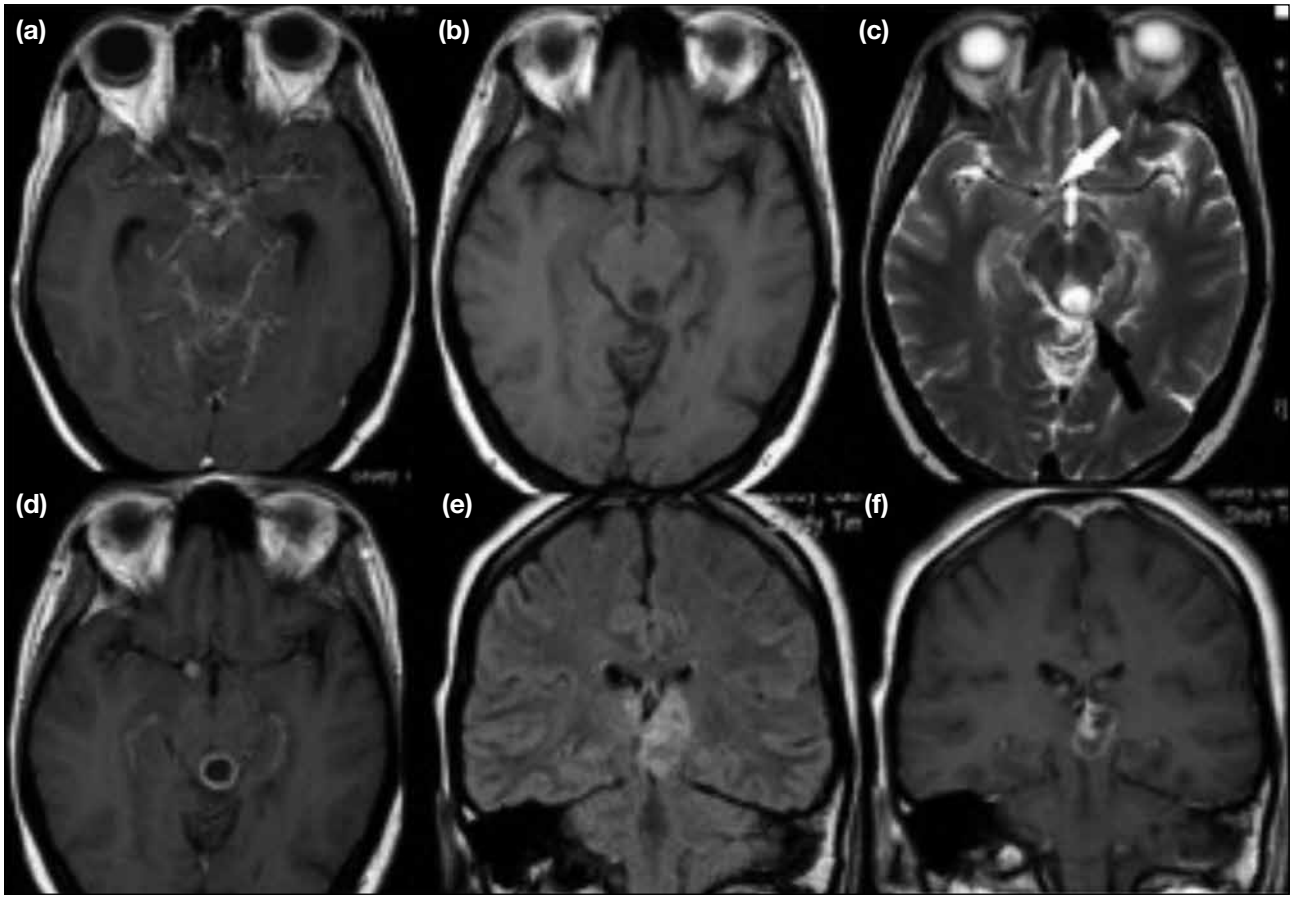

Figure 3. Case of tuberculous meningitis and tuberculoma. (a) Initial post-contrast sequence showing diffuse leptomeningeal enhancement around the basal cistern, suggestive of tuberculous meningitis. (b, c) Follow-up magnetic resonance imaging showing lesions at the suprasellar and left quadrigeminal cistern, suggestive of tuberculoma. The lesion at the right suprasellar cistern (white arrow) is T2 isointense. The lesion at the left quadrigeminal cistern (black arrow) is T2 hyperintense, suggestive of internal caseating content. (d) Ring enhancement is noted at the tuberculoma on post-contrast magnetic resonance imaging. (e) Failed suppression of signal of internal content of the tuberculoma on fluid-attenuated inversion recovery sequence. (f) Sometimes ring enhancement of tuberculoma can appear heterogeneous.

All lesions ultimately become calcified with no more adjacent oedema and the disease enters a nodular calcified stage. MRI features of neurocysticercosis are essentially thin-walled ring-enhancing lesions. The lesions can be distributed at the subarachnoid space, brain parenchyma especially grey-white junction, and the ventricles of the brain. At the early stage, enhancing nodules may be seen within the ring-enhancing lesions. However, the most typical appearance of neurocysticercosis is multiple calcified nodules with no significant oedema in the final nodular calcified stage. Hypointensity is seen on T2-weighted sequence due to underlying calcification. Differing from dystrophic calcification of brain, persistent ring enhancement can be noted despite calcification of the lesion (Figure 6).[12]

NEOPLASTIC CAUSES

Glioblastoma

Glioblastoma is a high-grade astrocytoma and is the most common primary brain tumour in adults. It can be classified as primary or secondary (arising from a low-grade astrocytoma). Most primary glioblastomas are isocitrate dehydrogenase wild-type whereas secondary glioblastoma is more likely to have isocitrate dehydrogenase–mutant status.[11] Up to 90% of glioblastomas are primary and more commonly seen in elderly patients. The tumour arises in cerebral white matter and has a high tendency to spread across the corpus callosum. Similar to most brain tumours, it is T1 hypointense and T2 hyperintense, but the signals are much more heterogeneous and the outline of the lesion is more irregular. These signal changes may alter in the presence of superimposed haemorrhage or central necrosis. Susceptibility artefact may occur due to haemorrhage and is often irregular. Focal restricted diffusion may be seen within the lesion, although the signal may not be as homogenous as that with pyogenic abscess. Irregular ring enhancement is a feature of

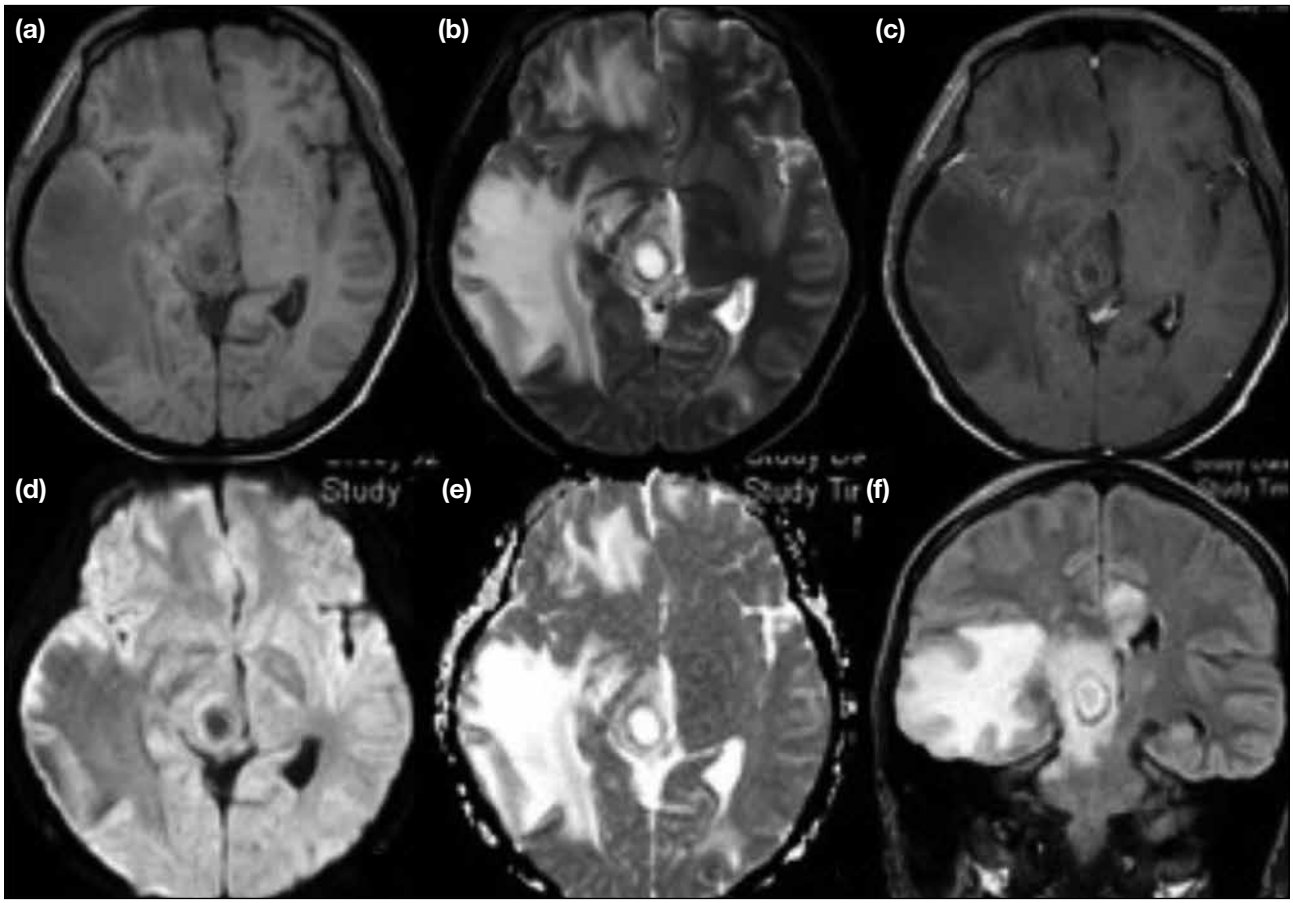

Figure 4. Case of microbiological proven *Penicillium marneffei* cerebral abscesses. (a, b) Multiple abscesses are noted at right frontal and parietal white matter. One of the abscesses being T1 hypointense and T2 hyperintense is noted at the right thalamus. Involvement of deep grey matter is not common in pyogenic abscess but is seen in fungal cerebral abscess. (c) Ring enhancement pattern of fungal abscess is noted. (d, e) Diffusion-weighted imaging sequence and apparent diffusion coefficient map showing restricted diffusion of abscess wall at the right thalamus. No restricted diffusion of the abscess cavity is seen. This feature suggests the underlying aetiology is more likely to be fungal than pyogenic. (f) Hyperintensity of abscess cavity is seen on fluid-attenuated inversion recovery sequence.

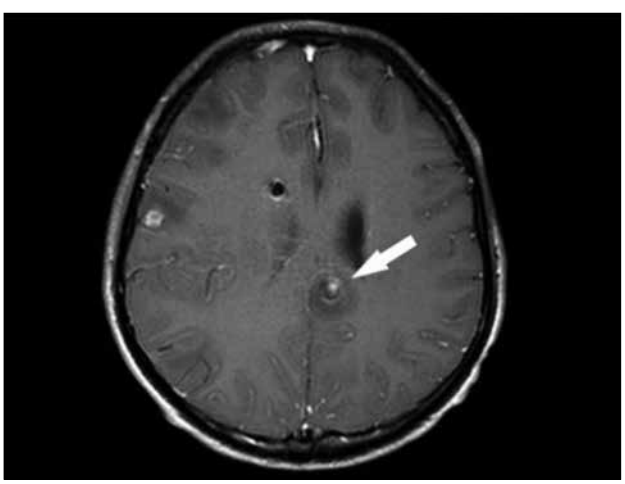

Figure 5. Case of cerebral toxoplasmosis with one of the lesions at the left parasagittal region demonstrating an "eccentric target sign" (arrow). Within the outer enhancing ring, an eccentrically located enhancing focus is seen.

glioblastoma, often associated with a thick enhancing rim (Figure 7). Prognosis of the disease is generally very poor due to its fast growth and aggressive behaviour.

Brain Metastasis

Brain metastasis is a more common intracranial malignancy than primary malignant brain tumour. Common tumours that metastasise to the brain include those of lung cancer, malignant melanoma, renal cell carcinoma, breast cancer, and colorectal carcinoma.[13] Brain metastasis can be solitary or multiple. Most metastatic lesions are T1 hypointense and T2 hyperintense except for malignant melanoma, in which the intrinsic melanin pigment will cause a reduction in T1 relaxation time with a consequent T1 hyperintense appearance.[14] When the tumour is complicated with haemorrhage, T1 hyperintensity of the lesion will be

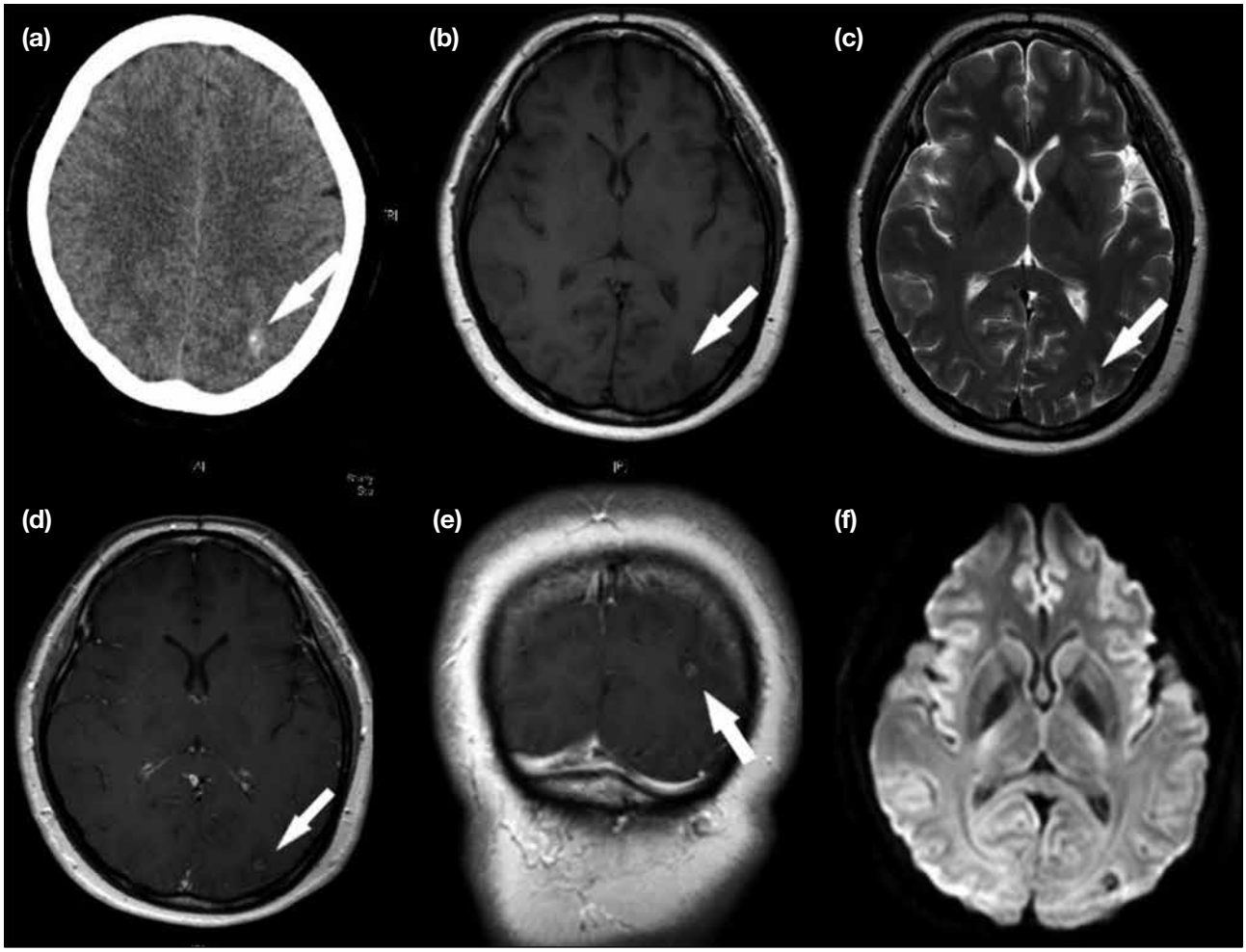

Figure 6. Case of neurocysticercosis at left parietal lobe. (a) A small calcified lesion without significant adjacent oedema is seen at the left parietal lobe grey-white junction, in keeping with known nodular calcified stage of neurocysticercosis. (b, c) The lesion appears T1 hypointense. The lesion shows peripheral signal drop-out on T2-weighted sequences, in keeping with underlying calcification. Minimal internal T2 hyperintensity is still noted. (d, e) Persistent thin wall ring enhancement is seen at the lesion despite calcification. (f) No restricted diffusion is seen at the lesion.

noted. Enhancement can be uniform or ring-shaped and the wall of ring-enhancing lesions is often thick and irregular. Central necrosis may be seen within the tumour, and no restricted diffusion can be seen within the necrotic region (Figure 8).

Differentiation between metastases and primary glioblastoma may be difficult because the latter may also present with multiple enhancing foci. For metastases, they tend to involve the grey-white junction and rarely spread along the corpus callosum unlike those of glioblastoma. Moreover, if multiple enhancing tumours are not connected by a single patch of T2/FLAIR abnormality, they are more likely due to metastases.[15] If the same characteristic is seen in glioblastoma, it is termed multicentric glioblastoma and considered a rare entity since it means there are multiple synchronous glioblastoma within the brain.[15] MR spectroscopy may be useful to distinguish the two diseases by investigation of the region of T2 hyperintensity around the ring of enhancement. In metastases, the region of adjacent T2 hyperintensity often represents vasogenic oedema and there will not be any increased choline-to-creatinine ratio. In glioblastoma, the T2 hyperintensity might represent non-enhancing tumour infiltration and will be evidenced by increased choline-to-creatinine ratio at those regions (Figures 9 and 10).[16]

Primary Central Nervous System Lymphoma

Primary CNS lymphoma means there is no systemic lymphomatous involvement when the disease is diagnosed. Otherwise, it is just classified as secondary intracranial involvement of lymphoma. The presentation again depends on the location and size of the lesions.

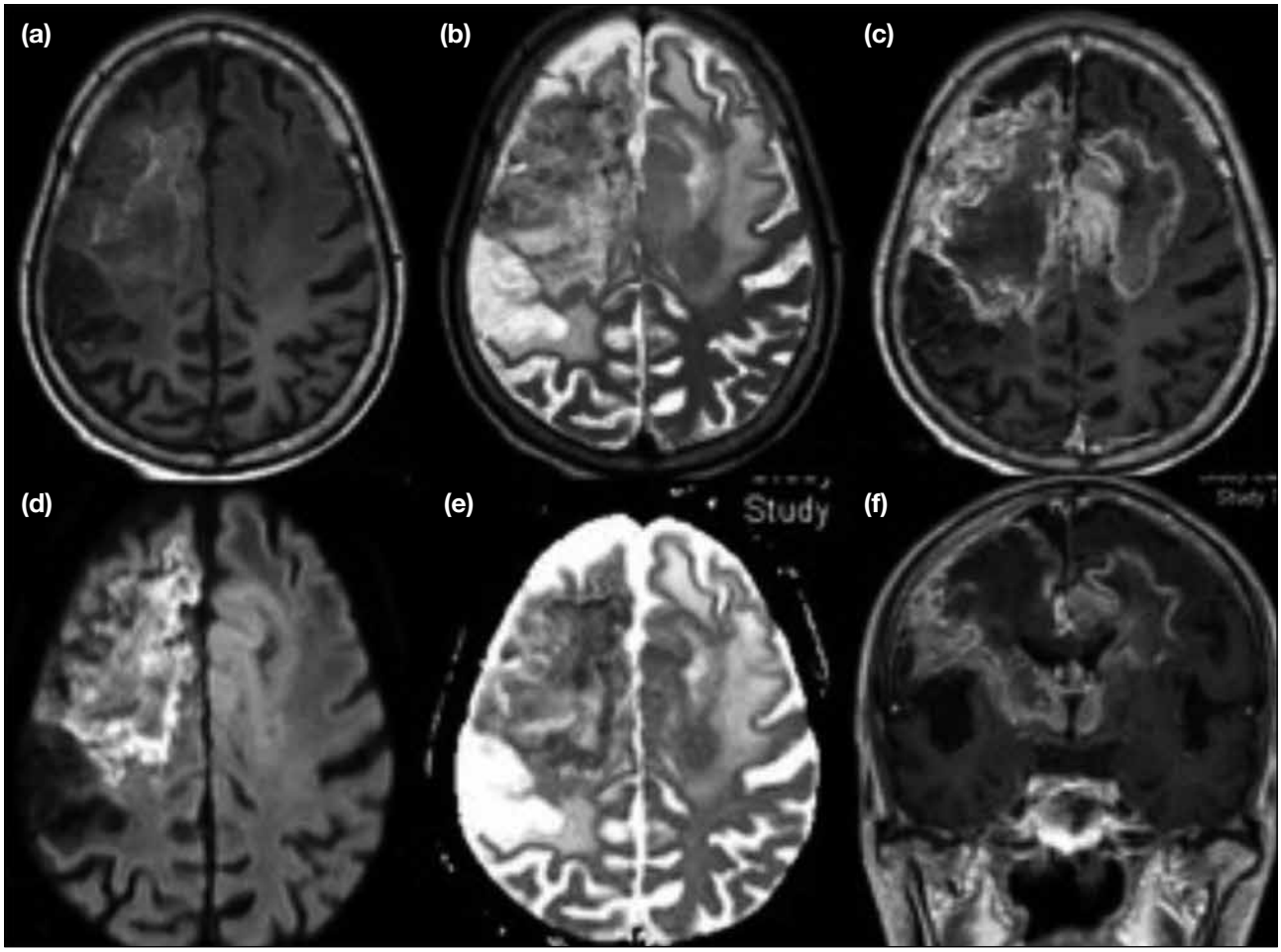

Figure 7. Case of glioblastoma. (a, b) Heterogeneous irregular mass lesion is seen at the bilateral frontal region, with predominantly T1 hypointensity and T2 hyperintensity. Superimposed T1 hyperintensity likely represents foci of haemorrhage. Evidence of haemosiderin deposition is noted within the tumour mass. (c) Irregular thick-walled peripheral rim enhancement is seen. (d, e) Evidence of restricted diffusion at the solid component at the peripheral aspect, whereas the central necrotic region shows no restricted diffusion. The appearance of restricted diffusion is heterogeneous. (f) Post-contrast sequence showing predilection of the tumour in white matter and the tumour spreading across the corpus callosum.

One special characteristic of primary CNS lymphoma is a predilection for supratentorial white matter.[17] Similar to glioblastoma, it has a tendency to spread across the corpus callosum. The disease can be seen in immunocompetent and immunocompromised patients although the appearance may be slightly different depending on the patient's immune status. On computed tomography, it appears as a homogenous hyperdense lesion with diffuse contrast enhancement. On MRI, primary CNS lymphoma is typically T1 hypointense and T2 hypointense/isointense with restricted diffusion. The T2 hypointensity of primary CNS lymphoma makes it a special characteristic as most intracranial masses are T2 hyperintense. In immunocompetent patients, primary CNS lymphoma usually demonstrates homogenous enhancement with diffuse restricted diffusion (Figure 11). In immunocompromised patients, the enhancement pattern is more heterogeneous and the lesion will more likely demonstrate ring enhancement.[17] Central necrosis of tumour tends to occur in immunocompromised patients, so focal T2 hyperintensity may be evident within the lesions. A characteristic of MR spectroscopy in primary CNS lymphoma is markedly elevated choline-to-creatinine ratio.[18]

POST-RADIATION CAUSE

Pseudoprogression and Cerebral Radiation Necrosis

Brain tumours such as glioblastoma and brain metastases often require radiation therapy. This often imposes diagnostic challenges as post-radiation changes to the tumour may simulate the appearance of residual or recurrent tumour. Pseudoprogression and cerebral radiation necrosis are both sequelae of radiation

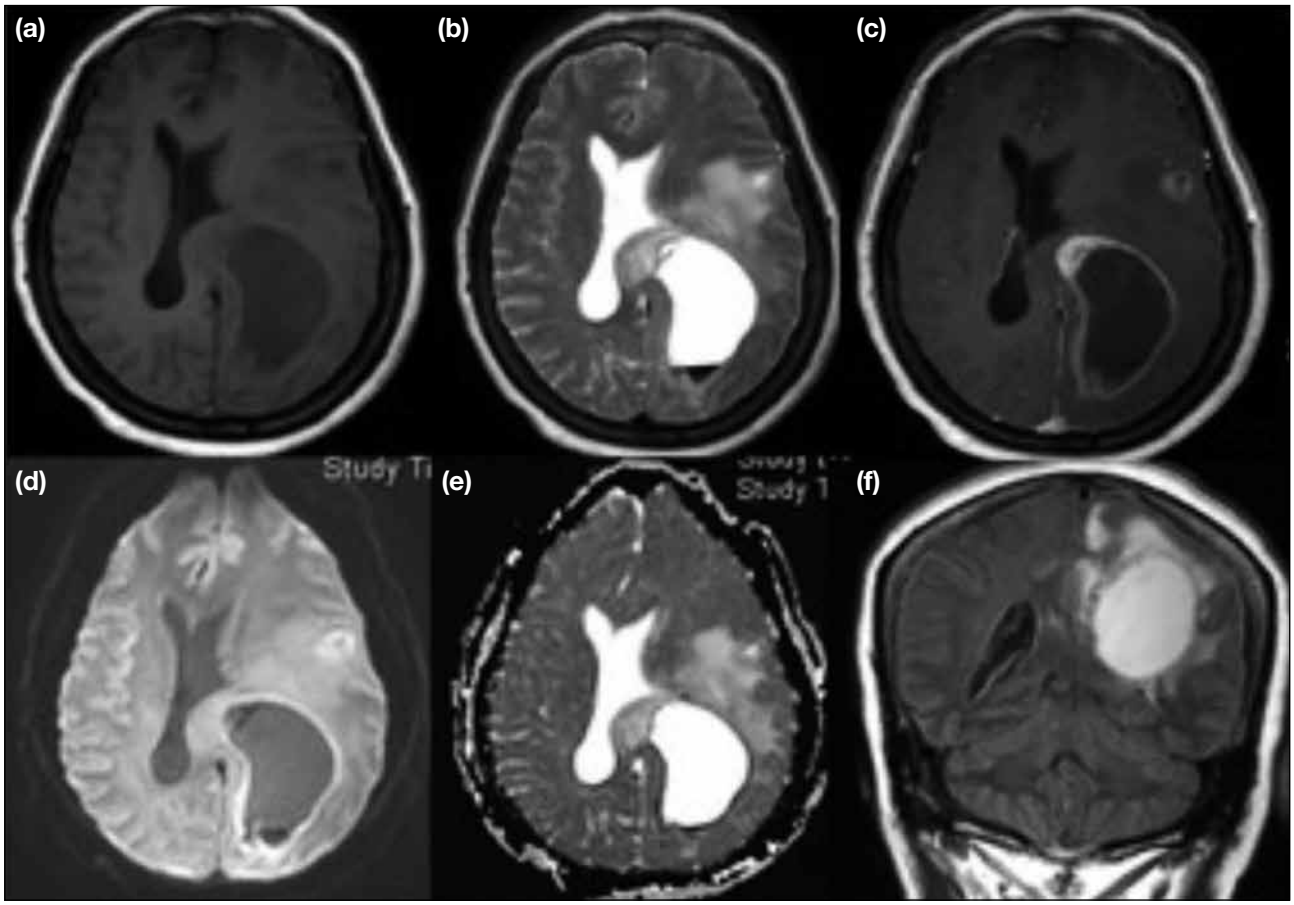

Figure 8. Case of cystic cerebral metastases from small cell lung cancer. (a, b) Cystic tumour masses are seen at left frontal and parietal lobes, with the predominantly large cystic lesion at the left parietal lobe causing significant mass effect. The perilesional T2 hyperintensity is due to vasogenic oedema that can be confirmed by magnetic resonance spectroscopy, compared with that of glioblastoma due to tumour infiltration. (c) Irregular peripheral ring enhancement with focal thickened wall suggestive of malignant lesion. (d, e) Restricted diffusion at peripheral solid wall is noted. (f) Internal cystic component cannot be suppressed on fluid-attenuated inversion recovery sequence, signifying non- cerebrospinal fluid content.

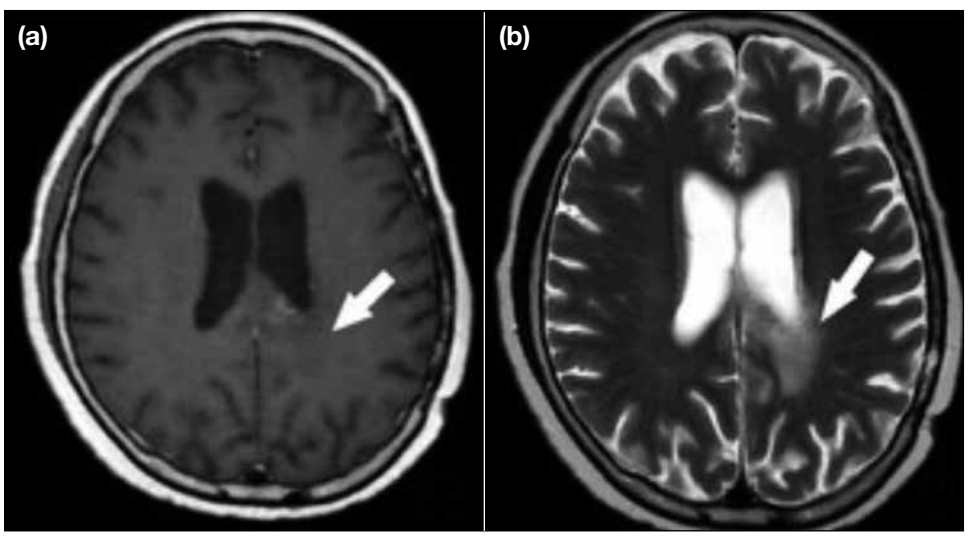

Figure 9. Case of glioblastoma. (a) Mild ring enhancement at the left periventricular region. (a, b) T1 hypointensity and T2 hyperintensity (arrows) at the left parietal region adjacent to the enhancing lesion. Magnetic resonance spectroscopy is useful to further characterise this region.

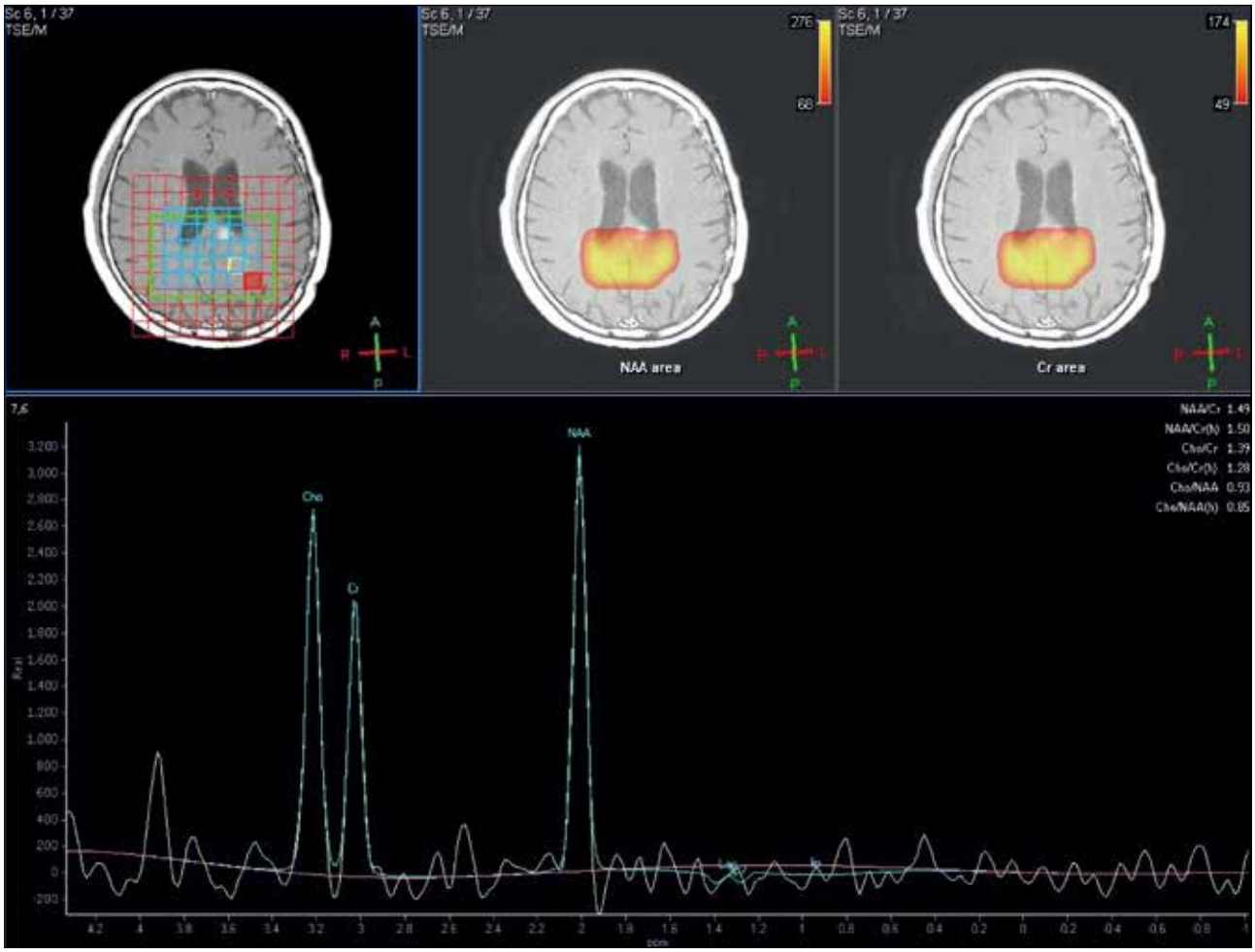

Figure 10. Case of glioblastoma (same case as Figure 9). Magnetic resonance spectroscopy showing elevated choline peak at the T2 hyperintense region at the left parietal lobe, with increased choline/creatine and choline/N-acetylaspartate ratios. Features are suggestive of tumour infiltration rather than perilesional oedema.

therapy of the brain. Pseudoprogression usually occurs in the first 3 months following completion of brain radiation but can occur up to 6 months post-treatment. It appears as an enlarging irregular ring-enhancing lesion mimicking disease progression whereas it actually represents a change related to underlying cell death. The border of the lesion with pseudoprogression might have a "Swiss cheese" or "soap bubble" appearance.[19] A few advanced MR sequences may contribute to diagnosis of pseudoprogression. On MR perfusion scan, pseudoprogression will show reduced cerebral blood volume whereas tumour usually has increased cerebral blood volume (Figure 12). MR spectroscopy of pseudoprogression often shows reduced metabolites and increased lactate peak (Figure 13).[20] Stability or shrinkage of the lesion through interval follow-up imaging can also confirm that the lesion is related to post-radiation changes. Cerebral radiation necrosis refers to a more long-term effect of radiation therapy, usually beyond 6 months to years after treatment. The mass effect of the lesion will be lost, unlike pseudoprogression. On the other hand, the enhancing features, and findings on MR spectroscopy and MR perfusion are similar to those of pseudoprogression.

DEMYELINATING CAUSE

Multiple Sclerosis

One of the most common demyelinating diseases is multiple sclerosis. The disease has several presentation patterns. Involvement can be at the cerebrum, cerebellum, brain stem, cranial nerves especially optic nerves, and spinal cord. In general, multiple sclerosis is predominantly seen in women, with a female-to-male ratio up to 2:1.[21] MRI is the important imaging modality in the radiological diagnosis of multiple sclerosis. Diseased regions typically show foci of T1 hypointensity and T2/FLAIR hyperintensity, with a predilection for the callososeptal interface. They can slowly progress

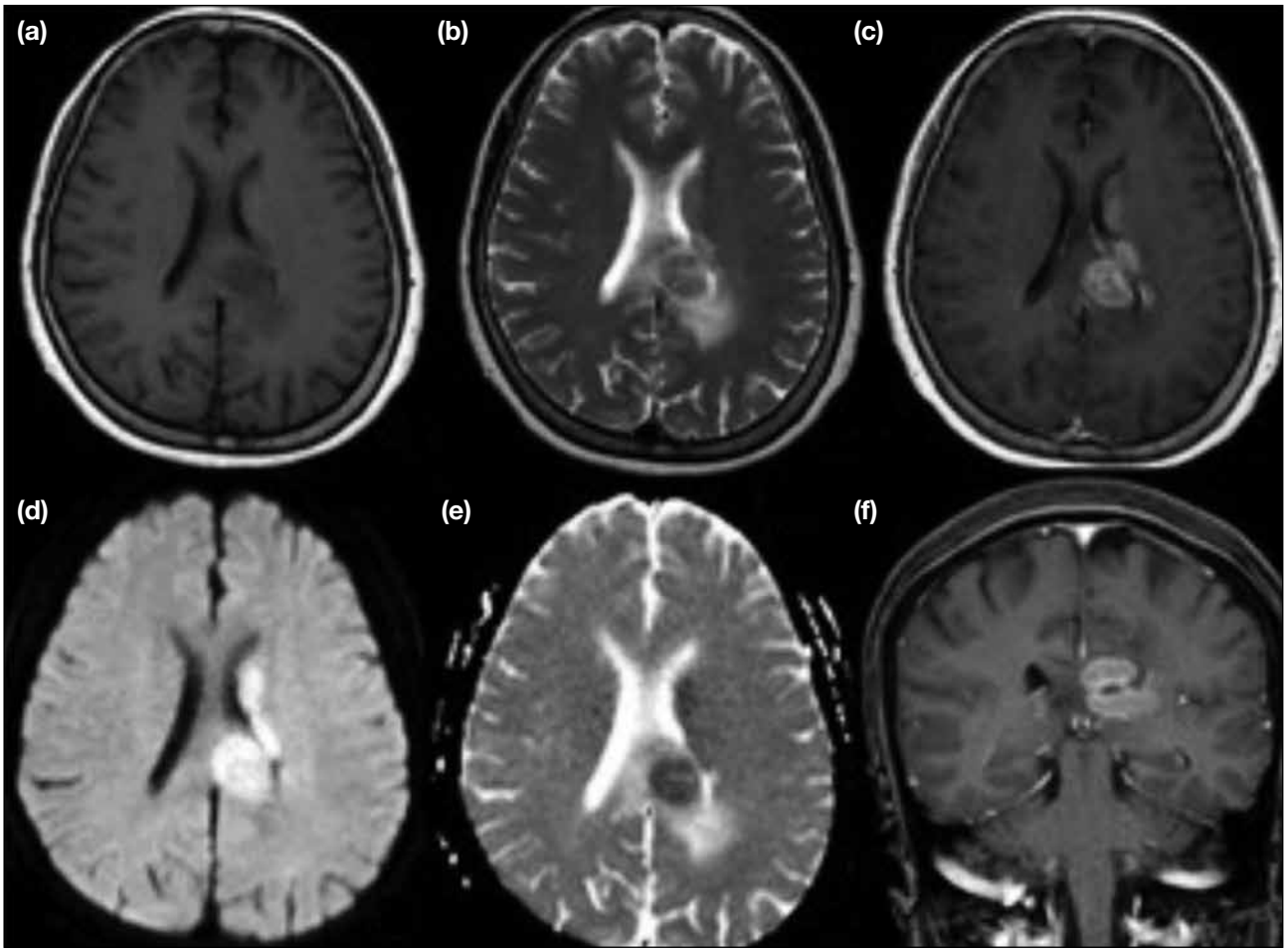

Figure 11. Case of primary central nervous system lymphoma in an immunocompetent patient. (a, b) T1 and T2 hypointense lesion at the corpus callosum with mild perilesional oedema with T2 hypointensity characteristic of lymphoma. (c) Homogenous enhancement is usually seen in immunocompetent patients, compared with ring-shaped irregular enhancement in immunocompromised patients. (d, e) Primary central nervous system lymphoma typically demonstrates restricted diffusion. (f) Similar to glioblastoma, it tends to involve and cross the corpus callosum.

with longitudinal extension perpendicular to the lateral ventricles, giving the typical "Dawson's fingers" appearance. Post-contrast and DWI sequences are useful to detect active lesions/plaque. Sometimes these active lesions may be quite large and mimic a mass lesion. Active demyelinating lesions can be distinguished from other malignant causes by the special ring-enhancing pattern seen in the demyelinating disease known as "open ring enhancement",[22] which means there is incomplete ring enhancement (Figure 14). The enhancing edge represents an active demyelinating process in those regions. This sign is rather specific for demyelination as the underlying cause.[23]

VASCULAR CAUSE

Haematoma

Hypertensive intracranial haemorrhage is the most common cause of intracranial haemorrhage. It typically affects the basal ganglia, thalami, cerebellum, and pons. When the haematoma enters a subacute phase or early chronic phase, thin peripheral enhancement around the haematoma is often evident and may mimic tumour mass lesion. Imaging characteristics of subacute haematoma include T1 and T2 hyperintensity of the lesion. Also, the peripheral ring enhancement should be thin. Perilesional vasogenic oedema is usually not very significant. A haemosiderin rim that is complete is often seen on T2-weighted image (Figure 15) and blooming artefact is noted on DWI sequence. Interval follow-up scan can exclude malignant lesions that will progress in size. If MR perfusion is available, the cerebral blood flow will decrease in case of subacute haematoma.[24]

CONCLUSION

Cerebral ring-enhancing lesions are common features in neuroradiological imaging. Although no imaging

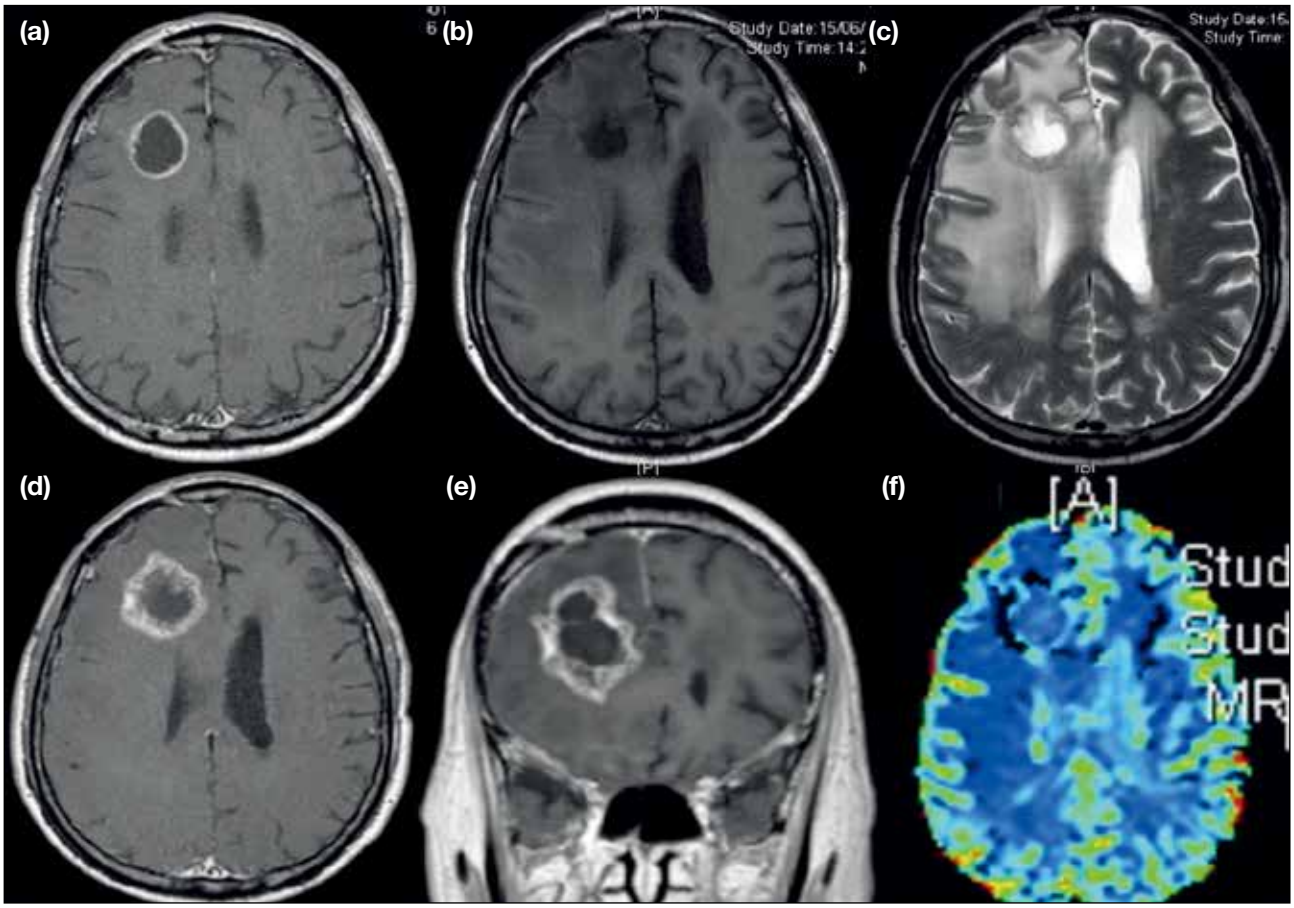

Figure 12. Case of pseudoprogression. (a) Ring-enhancing metastatic adenocarcinoma of lung before radiation therapy at the right frontal lobe. (b, c) T1 hypointense T2 hyperintense right frontal lesion with increased size and extent of perilesional oedema is seen 3 months after radiation therapy. (d, e) Increased size of ring-enhancing lesion 3 months after radiation therapy. Notice the "Swiss cheese" or "soap bubble" appearance at the wall of the enhancing lesion. (f) Magnetic resonance perfusion scan showing no increased cerebral blood volume at the right frontal lesion, suggestive of pseudoprogression rather than residual/recurrent tumour. It is confirmed by subsequent follow-up imaging that shows stability of the lesion.

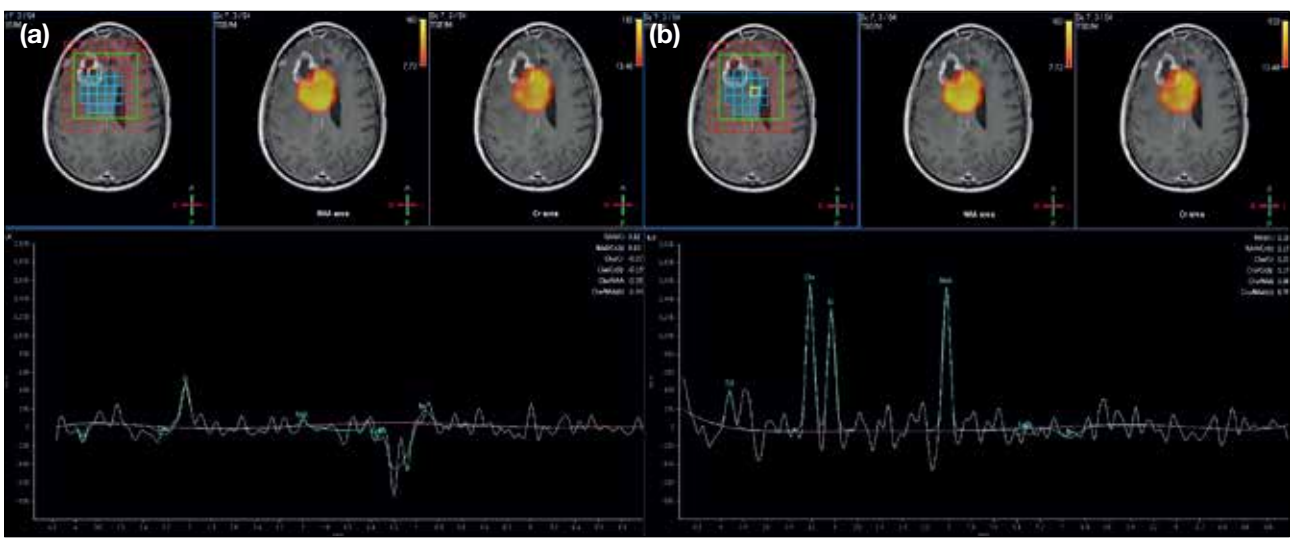

Figure 13. Case of pseudoprogression (same case as Figure 12). (a) The trace appears flattened with reduced peaks of metabolites including choline and N-acetylaspartate. Notice the inverted lactate peak on intermediate echo time (144 ms). (b) Another voxel showing normal brain parenchyma with taller choline, creatinine, and N-acetylaspartate peaks.

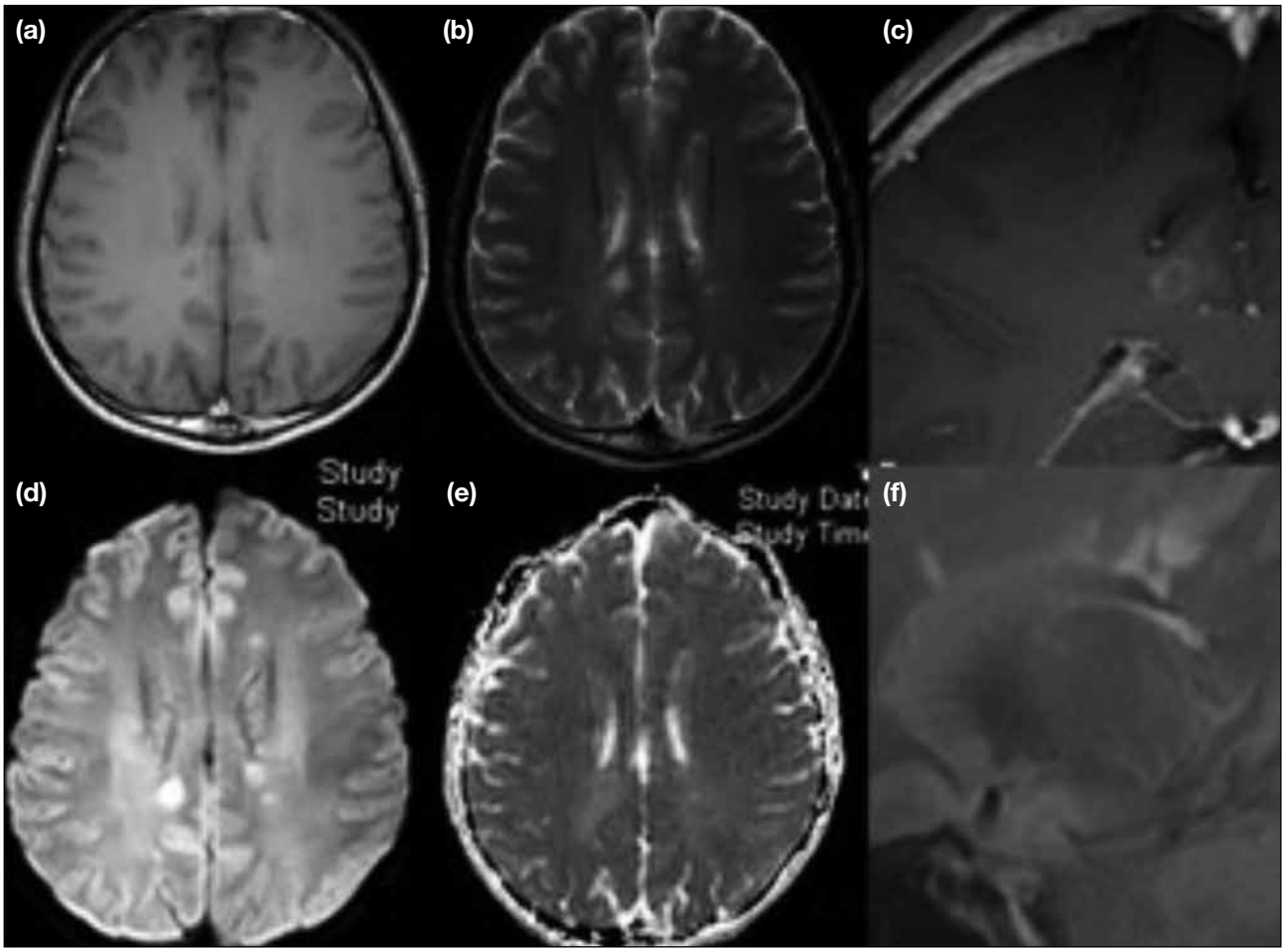

Figure 14. Case of multiple sclerosis. (a, b) Diseased region is usually T1 hypointense and T2 hyperintense, the most common site being the callososeptal interface. (c) Typical active demyelinating disease enhancement pattern–open ring enhancement. The enhancing edge represents an active demyelinating process. (d, e) Active plaque will demonstrate restricted diffusion. (f) Sagittal fluid-attenuated inversion recovery sequence is best to evaluate the longitudinal plaque perpendicular to the lateral ventricle known as "Dawson's fingers".

features are pathognomonic for certain disease entities, a diagnosis can be reached or the differential diagnoses narrowed through careful evaluation of a patient's clinical history, blood test results, certain radiological features, and interval follow-up.

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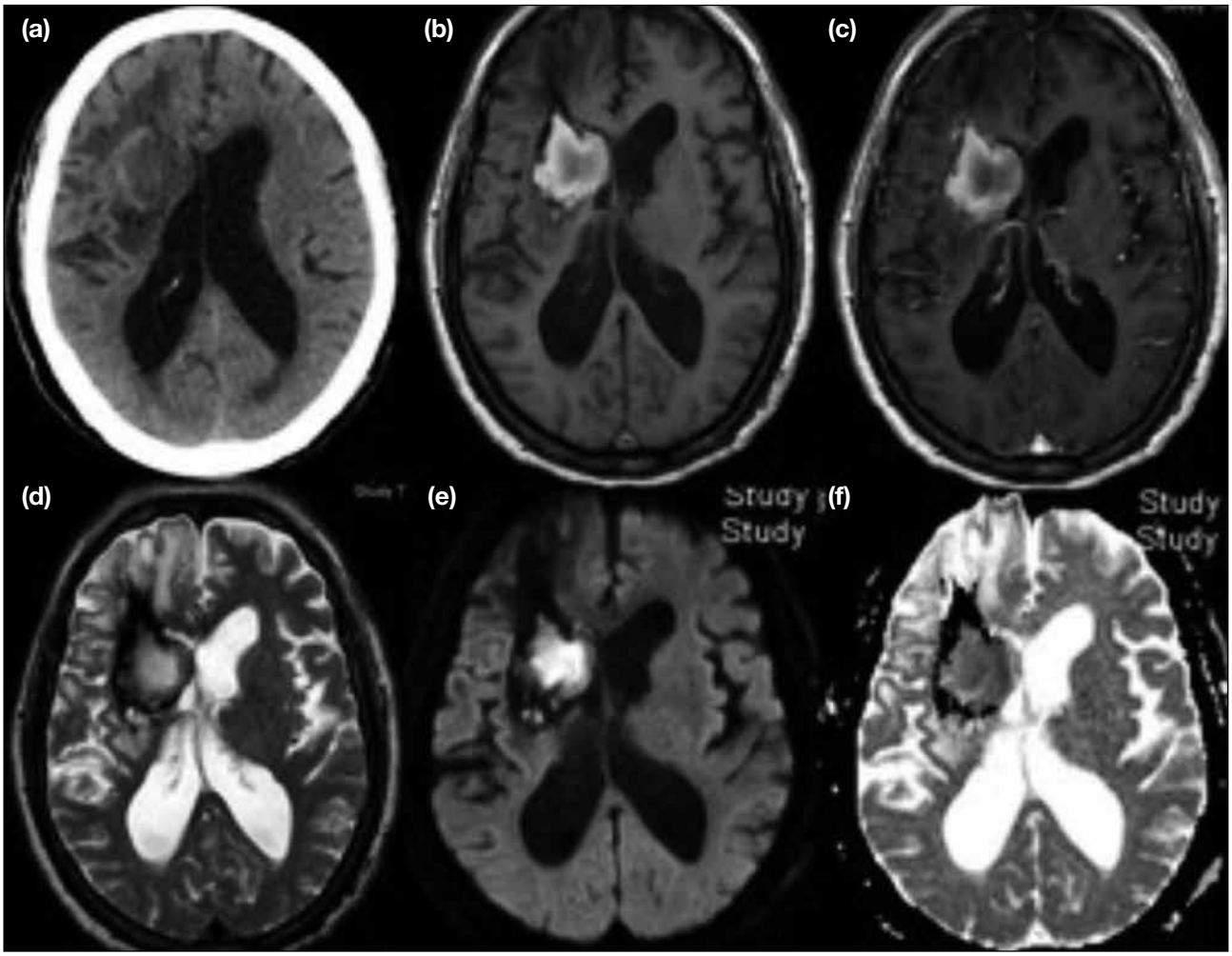

Figure 15. Case of subacute haematoma. (a) Non-contrast computed tomography brain showing hypodense lesion with peripheral hyperdensity at the right frontal lobe, mimicking tumour mass. (b, c, d) On magnetic resonance imaging, the lesion is T1 and T2 hyperintense. A complete haemosiderin rim is seen on T2-weighted image. A mild degree of thin ring enhancement is seen on the post-contrast image, and can persist for a few months even after the lesion becomes T1 and T2 hypointense. Overall features are suggestive of subacute haematoma. (e, f) Diffusion-weighted imaging and apparent diffusion coefficient map showing that restricted diffusion can present within subacute haematoma.

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