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PICTORIAL ESSAY

Multimodality Imaging and Interventional Radiological Management of Neurological Complications of Infective Endocarditis

EH Chan, HM Kwok, NY Pan, LF Cheng, JKF Ma

Department of Diagnostic and Interventional Radiology, Kowloon West Cluster, Hong Kong SAR, China

INTRODUCTION

Infective endocarditis (IE) affects 1.7 to 6.2 individuals per 100,000 population per year and remains a life-threatening condition.[1] *Staphylococci* are the most frequent causative organisms.[1,2] Neurological complications are the most common and severe extracardiac complications of IE[3] and have been reported as the presenting symptom in up to 47% of cases.[4] These complications are caused by cerebral septic embolisation of endocardial vegetations. Patients with neurological complications have significantly higher mortality compared to those without (24% vs. 10%; $p < 0.03$).[3] Neuroimaging leads to the identification of valvular surgery indications in about 22% of patients with symptoms of neurological complications of IE, and in 19% of asymptomatic IE patients.[2] Up to 82% of patients have cerebral lesions on magnetic resonance imaging (MRI) performed within 7 days after admission.[1] MRI findings influence diagnostic classification and other clinical decisions in 28% of patients, including modification of medical or surgical treatment plans.[5] According to the 2015 European Society of Cardiology guidelines,[6] the presence of cerebral emboli in patients with left-sided valvular vegetations greater than 10 mm is an indication for urgent valve surgery to prevent further embolisms. Familiarity with the neurological imaging findings is essential for early diagnosis of this complication of IE, allowing a window for early and specific treatment, thereby reducing mortality. However, the wide spectrum of presentations on neuroimaging poses diagnostic challenges to radiologists, especially when cerebral septic embolism is the first presentation. This pictorial essay aims to review the spectrum of presentations and the use of multimodality imaging to increase awareness of the classic diagnostic imaging findings of cerebral septic emboli secondary to IE, and to highlight the role of interventional radiology in clinical management.

Diagnosis

The diagnosis of IE is made according to the Modified Duke criteria,[1] which include the presence of major arterial emboli, mycotic aneurysm, and intracranial haemorrhage as part of its minor criteria.

Correspondence: *Dr EH Chan, Department of Diagnostic and Interventional Radiology, Kowloon West Cluster, Hong Kong SAR, China*
Email: eh278@ha.org.hk

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Computed tomography (CT) is the first-line imaging study in patients with neurological symptoms as it is readily available. MRI, including susceptibility-weighted imaging (SWI) and diffusion-weighted imaging (DWI), is required to detect more subtle findings such as cerebral microbleeds and early infarcts. Further investigations with computed tomographic angiography (CTA) or magnetic resonance angiography (MRA) are useful for detecting mycotic aneurysms, while digital subtraction angiography (DSA) remains the gold standard and should be performed in clinically suspicious cases with negative CTA or MRA.[7] There is growing support for performing screening MRI in patients with suspected or confirmed IE, given the frequency of asymptomatic findings and its usefulness in decision-making. However, its cost-effectiveness and impact on mortality reduction remain to be seen.[8]

Imaging Spectrum

Neurological complications of IE may present as cerebral infarcts, micro- or macro-haemorrhage, abscess, and meningitis. The pooled frequency of individual findings on MRI is as follows: acute ischaemic lesions (61.9%), cerebral microbleeds (52.9%), macro-haemorrhages (24.7%), abscess or meningitis (9.5%), and intracranial mycotic aneurysm (6.2%).[8] Accurate identification of these lesions allows early diagnosis of IE complications and individualised management strategies.

Ischaemic Stroke

Ischaemic stroke is the most common neurological manifestation of IE. It can result from embolisation of endocardial vegetations, leading to occlusion of intracerebral arteries.[3] The incidence of cerebral ischaemia is correlated with left-sided endocarditis (especially involving the anterior mitral valve leaflet), larger endocardial vegetation size (>10 mm), mobile vegetations, and *Staphylococcus aureus* infection.[3]

Disseminated ischaemic lesions may result from multiple emboli occurring over a short period or fragmentation of an embolus in the heart or aorta. The presence of multiple cortical and subcortical cerebral infarcts of varying ages within different vascular territories (especially watershed areas) or bihemispheric involvement suggests the diagnosis of septic emboli[4] (Figure 1). Large emboli tend to cause cortical infarction in the middle cerebral artery (MCA) territory, while smaller emboli often lodge distally in terminal cortical branches of the anterior cerebral artery and MCA, resulting in small peripheral infarcts at the grey-white matter junction.[9] It is worth noting that isolated brainstem strokes are rarely caused by cardioembolism.

DWI is useful for assessing the temporal relationship of ischaemic lesions. Acute infarcts appear as hyperintense on DWI and hypointense on apparent diffusion coefficient

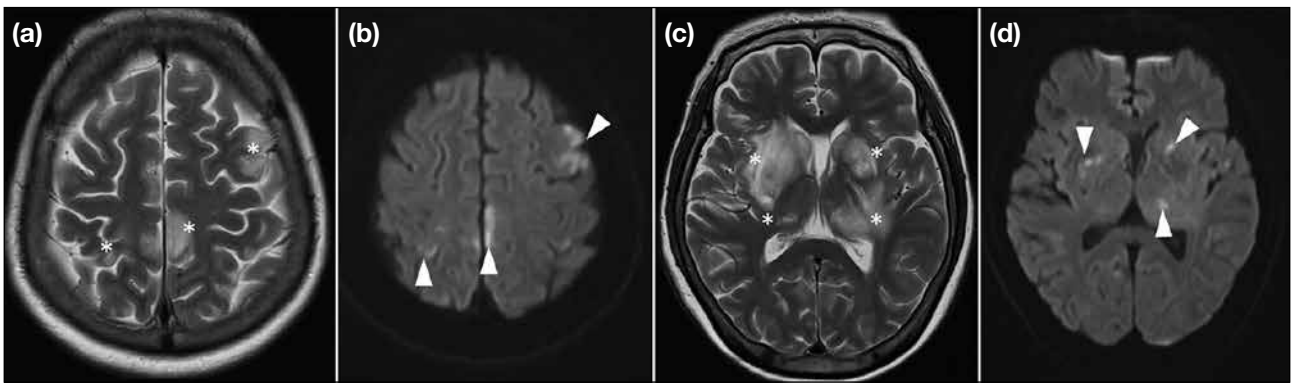

Figure 1. A 69-year-old woman presented with fever and confusion 3 days after dental surgery. (a) Axial T2-weighted magnetic resonance imaging (MRI) shows multiple foci of hyperintense signals involving the bilateral high frontal and parietal cortices (asterisks). (b) Axial diffusion-weighted imaging (DWI) with a high b-value revealed corresponding hyperintense signal (arrowheads) with low signal on apparent diffusion coefficient map (ADC) [not shown], suggestive of restricted diffusion. (c) Axial T2-weighted MRI shows hyperintense signals involving the bilateral basal ganglia, capsular regions, and thalami (asterisks). (d) Axial DWI with a high b-value shows patchy areas of hyperintense signal (arrowheads), with low signal on the ADC map (not shown), suggestive of restricted diffusion. These findings were suggestive of acute infarcts. The distribution pattern raised suspicion for an embolic shower, involving both deep perforating arteries and cortical branches. Consequently, an echocardiogram was performed and revealed a ventricular septal defect and tricuspid valve vegetation, consistent with a paradoxical embolism. The patient was managed conservatively with intravenous antibiotics and showed good neurological recovery.

mapping. Over time, the apparent diffusion coefficient signal increases and pseudonormalises in about 1 week, while the DWI signal decreases and pseudonormalises in about 2 weeks.[10]

Cerebral Abscesses and Meningitis

Cerebral abscesses and meningitis are uncommon neurological manifestations of IE, occurring in up to 9.5% of patients.[8]

Typically, multiple abscesses appear in the MCA territory at the grey-white matter junction, often with vasogenic oedema and associated mass effect or haemorrhage.[3] On CT, cerebral abscesses are usually hypodense with ring enhancement, but MRI is more sensitive. Classic MRI features include lesions that are hypointense on T1-weighted images and hyperintense on T2-weighted images, with ring enhancement and central restricted diffusion (Figure 2). A dual rim sign, two concentric rims surrounding the abscess cavity, where the outer rim is hypointense and the inner is relatively hyperintense, may be visible on SWI or T2-weighted imaging. Cerebral abscesses may also arise near mycotic aneurysms (Figures 3 and 4). The presence of leptomeningeal enhancement on MRI or CT can suggest concomitant meningitis.

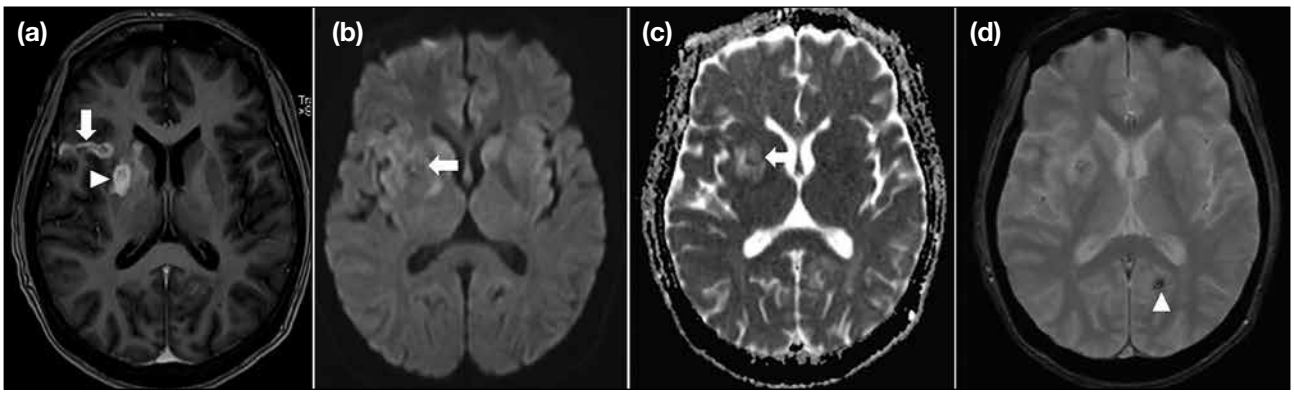

Figure 2. A 56-year-old woman presented with fever and a bilateral lower extremity rash. Physical examination revealed a pansystolic murmur with radiation to the left axilla and splinter haemorrhages. Echocardiography demonstrated mitral valve regurgitation and prolapse with vegetation. Blood culture yielded *Streptococcus sanguinis*. (a) Axial post-contrast T1-weighted image shows a small ring-enhancing lesion in the right basal ganglia (arrowhead). The lesion is hyperintense on T2-weighted image (not shown). There is associated focal leptomeningeal enhancement in the adjacent right frontal lobe cortex (arrow), suggestive of leptomeningitis. (b) Axial diffusion-weighted imaging with a high b-value demonstrates a focal central hyperintense signal (arrow) within the previous right basal ganglia ring-enhancing lesion. (c) The corresponding apparent diffusion coefficient map shows hypointense signal (arrow). These findings were indicative of restricted diffusion and therefore consistent with an abscess. (d) Axial gradient echo sequence reveals a focus of susceptibility artefact in the left occipital lobe (arrowhead), consistent with a microbleed. All findings resolved with conservative management with a 6-week course of broad-spectrum antibiotics. Mitral valve replacement was proposed but declined by the patient.

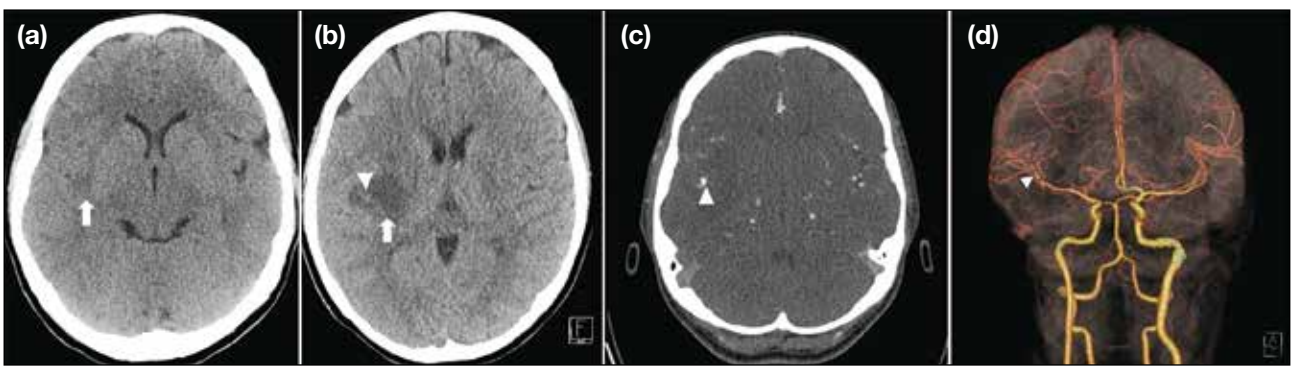

Figure 3. A 27-year-old woman presented with fever, left-sided weakness, and slurred speech. (a) Initial axial non-contrast computed tomography (CT) of the brain shows a small focus of hypodensity in the right temporoparietal region (arrow). (b) Follow-up axial non-contrast CT 1 day later demonstrates rapid enlargement of the hypodensity in the right temporoparietal region (arrow), with a small acute haemorrhagic focus (arrowhead). (c) Axial computed tomography angiography (CTA) shows a small contrast-enhancing focus at the proximal M2 segment of the right middle cerebral artery (arrowhead) within the infarct, suggestive of an aneurysm. (d) Volumetric rendering of the CTA shows the saccular morphology of the proximal right M2 aneurysm (arrowhead), consistent with a mycotic aneurysm.

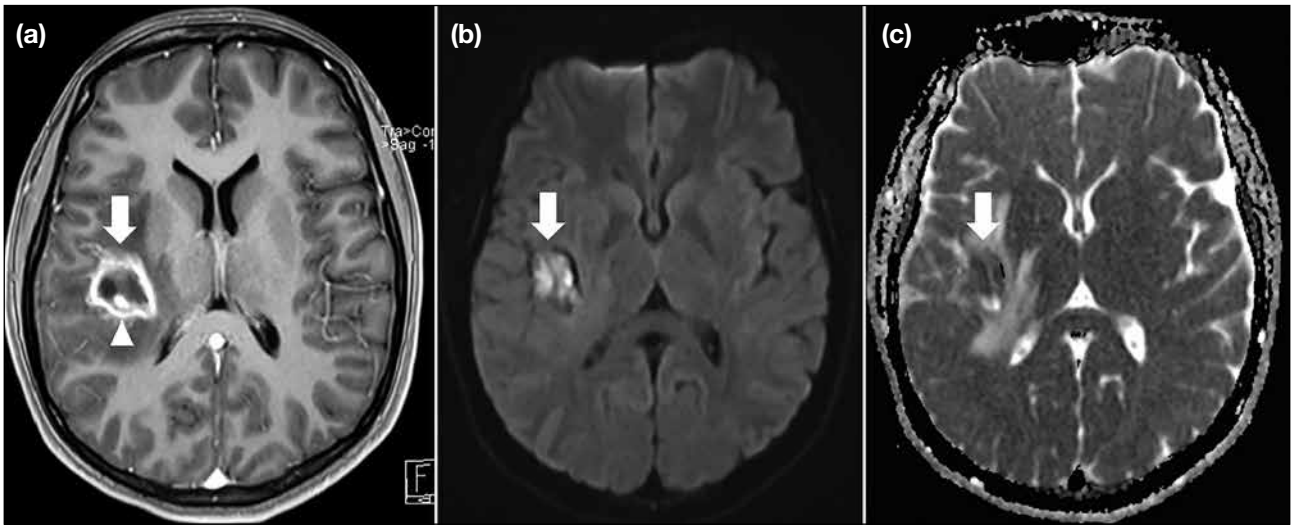

Figure 4. Same patient as in Figure 3. (a) Axial post-contrast T1-weighted magnetic resonance image shows a ring-enhancing lesion in the right temporoparietal region (arrow), with a central non-enhancing area and a peripheral enhancing focus (arrowhead). (b) Axial diffusion-weighted image with a high b-value demonstrates central hyperintense signal (arrow). (c) The apparent diffusion coefficient map shows corresponding hypointense signal (arrow). Overall findings are suggestive of a cerebral abscess with a mycotic aneurysm. Subsequent echocardiography revealed mitral valve regurgitation and prolapse with vegetation. Blood culture was negative. The patient underwent burr hole drainage of the abscess, and pus culture yielded *Staphylococcus aureus*. She was subsequently treated with a course of intravenous vancomycin.

Cerebral Haemorrhages

Macrohaemorrhage usually results from haemorrhagic transformation of ischaemic stroke, progression of microhaemorrhages, or rupture of mycotic aneurysms. Haemorrhagic transformation occurs more frequently in embolic strokes (51%-71%) than in non-embolic strokes (2%-21%)[11] and may present as petechial haemorrhage or large parenchymal haematomas.[9] Cerebral ischaemic lesions of varying ages across multiple vascular territories and different haemorrhagic patterns would raise suspicion for cardiac emboli. In the context of underlying IE, cerebral septic embolism is a likely diagnosis (Figure 5).

Septic emboli damage the endothelium and disrupt the blood-brain barrier, resulting in inflammatory vasculitis and small vessel rupture, often leading to cerebral microbleeds or even intracerebral haemorrhage. One study found that cerebral microbleeds in 57% of patients with IE.[4] These microbleeds appear as hypointense foci on T2* imaging or SWI MRI often in the cortex, and less frequently in subcortical white matter, basal ganglia, or posterior fossa.[4]

Mycotic Aneurysms

Cerebral septic emboli can trigger inflammation and weakening of vessel walls, forming mycotic aneurysms.[7] These aneurysms are found in about 6.2% of patients with IE and may shrink, enlarge, or develop de novo within 1 week to 3 months of starting antibiotics.[12] Mycotic aneurysms have a 2% to 10% risk of rupture regardless of their size and are associated with a high mortality rate of 80%.[7] About 22% of IE patients presenting with intracerebral haemorrhage have mycotic aneurysms which should be promptly identified.[13] CTA or MRA should be performed to confirm the diagnosis (Figures 5 and 6), followed by DSA for clear delineation of the number, size and location of the mycotic aneurysms and surgical or endovascular planning.

CTA and MRA have low sensitivity for small (<5 mm) or distal mycotic aneurysms.[1] Aneurysms near the skull base may be overlooked on CTA, while those in low-flow areas may be missed on time-of-flight MRA.[14] In cases with clinical suspicion of mycotic aneurysm but negative CTA or MRA, DSA should be performed.[1]

Features favouring mycotic aneurysms include multiplicity, saccular shape, distal location (such as MCA segments 2 to 4 or posterior cerebral artery), size or morphological changes on consecutive angiograms, presence of other intra- or extra-cranial mycotic aneurysms, adjacent arterial occlusion or stenosis, and cerebral infarction at the aneurysm site[13] (Figure 6).

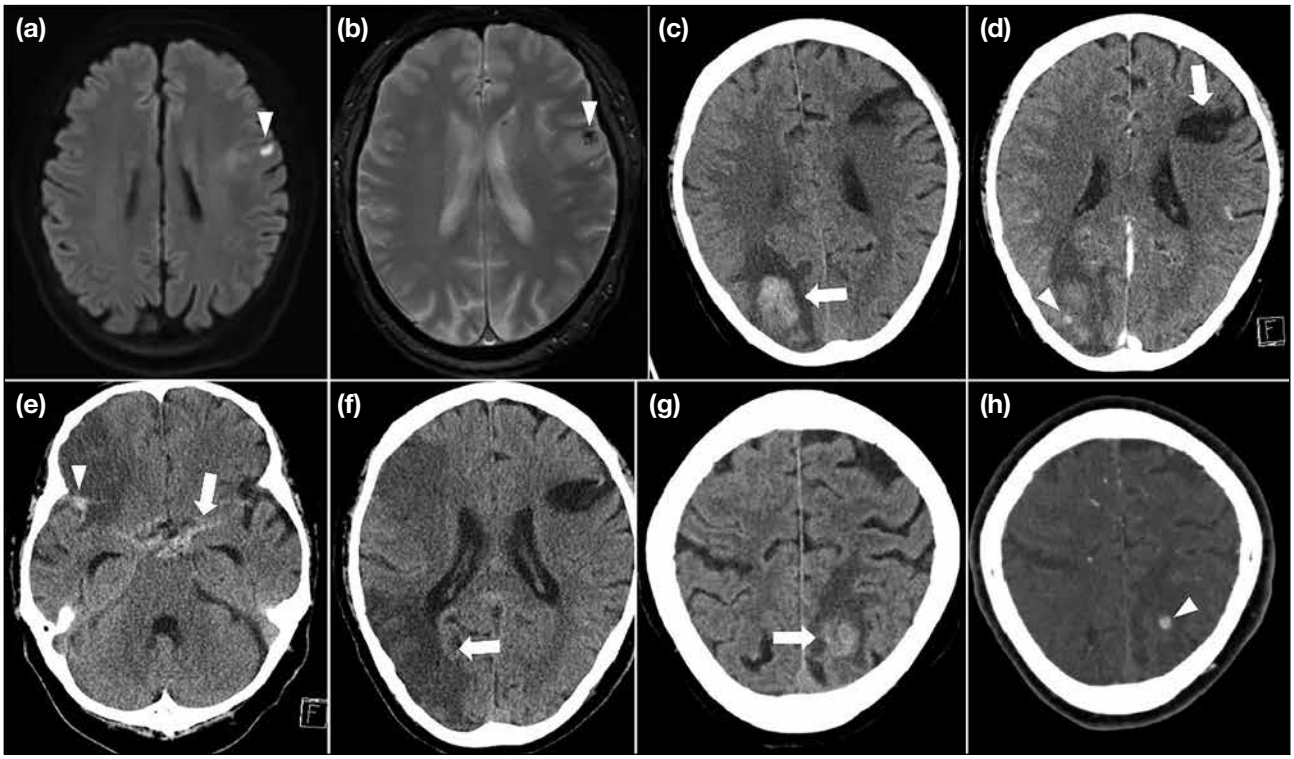

Figure 5. A 62-year-old man with known mitral valve regurgitation. (a) Axial diffusion-weighted imaging of the brain with a high b-value shows a focal hyperintense signal in the left frontal lobe (arrowhead), with corresponding hypointensity on the apparent diffusion coefficient map (not shown). (b) Associated susceptibility artefact is noted in the same region (arrowhead), suggestive of haemorrhagic transformation. (c) Axial unenhanced computed tomography (CT) of the brain 1 month later shows a new haemorrhagic infarction in the right occipital lobe with mild perilesional oedema (arrow). (d) Axial computed tomography angiography (CTA) of the brain shows a tiny contrast-enhancing focus within the haemorrhagic infarct, suggestive of an aneurysm (arrowhead), while the left frontal infarct shows signs of chronicity (arrow). (e) Axial non-contrast CT of the brain 1 day later shows a new right frontal lobe infarct, as well as new subarachnoid haemorrhage in the right frontal region (arrowhead) and suprasellar cistern (arrow). (f) Axial non-contrast CT of the brain 3 days later reveals a new right parieto-occipital lobe infarct (arrow). (g) Axial non-contrast CT of the brain 1 month later shows a new haemorrhagic infarct in the high left parietal lobe (arrow). (h) Axial CTA of the brain shows a tiny contrast-enhancing focus (arrowhead) within the haemorrhagic infarct suggestive of aneurysm. The presence of multiple haemorrhagic infarcts of different timing, some with associated aneurysms, is highly suggestive of cerebral septic emboli with mycotic aneurysms. Subsequent echocardiography revealed mild mitral and tricuspid regurgitation with vegetations on both mitral valve leaflets. Blood cultures yielded *Rothia dentocariosa*. A diagnosis of infective endocarditis was established and the patient was treated with intravenous antibiotics.

Management

Neurological complications from IE are life-threatening and require multidisciplinary management, involving neurosurgeons, radiologists, cardiologists, and microbiologists. Empirical intravenous antibiotic therapy is promptly administered and later tailored according to culture sensitivity results. Valvular replacement combined with antibiotics yield better outcomes than antibiotics alone in left-sided endocarditis.[15]

Radiologists play a key role in both diagnosis and guiding treatment by accurately reporting the type and severity of each lesion. Surgical drainage can be considered in cases of cerebral abscesses with significant mass effect. Antiplatelet drugs and anticoagulants are contraindicated in both ischaemic stroke and macrohaemorrhage caused by septic embolism due to the high risk of bleeding.[3] Cardiac surgery should be postponed for at least 4 weeks after a clinically significant intracranial haemorrhage or large ischaemic infarct.[3] Mycotic aneurysms should be excluded before open heart surgery for valvular replacement requiring anticoagulation to reduce bleeding risk.[15]

Interventional radiologists play an evolving role of in treating mycotic aneurysms in collaboration with neurosurgeons. Techniques include preoperative CTA or MRA with volumetric rendering, road-map technique for neuro-navigation, and cone beam CTA for postprocedural monitoring. Given the unpredictable nature of mycotic

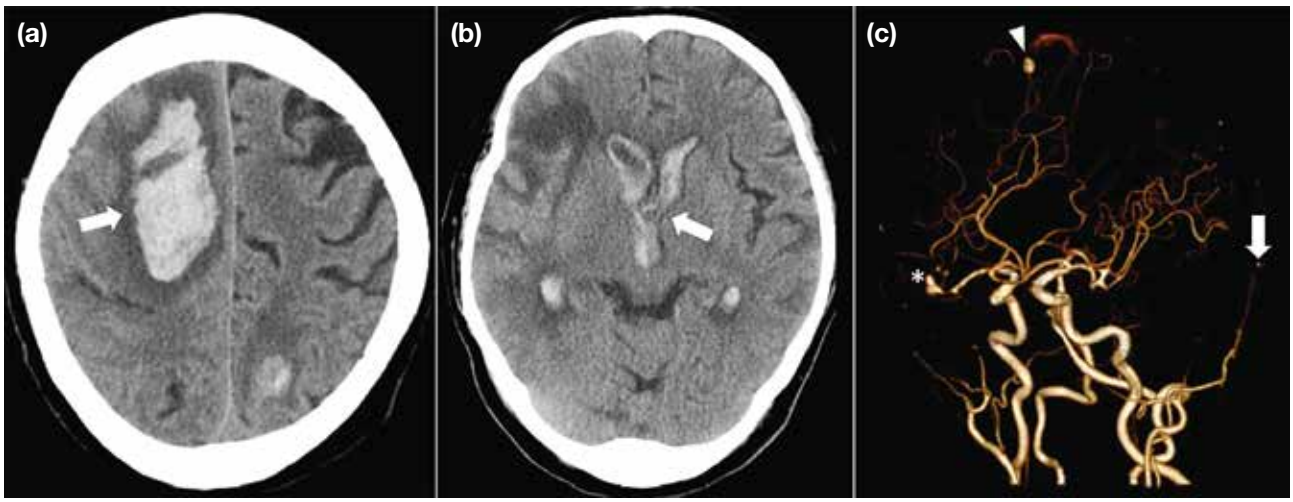

Figure 6. Same patient as in Figure 5. (a) Axial plain computed tomography of the brain 2 weeks after antibiotic therapy shows a new, large haemorrhagic infarct in the right frontal lobe with mass effect (arrow). (b) Axial unenhanced computed tomography at a lower level from the same study demonstrates a preexisting haematoma with intraventricular extension (arrow). (c) Volumetric rendering of computed tomography angiography reveals a saccular aneurysm arising from a distal branch of the right anterior cerebral artery (arrowhead), saccular aneurysms of the right middle cerebral artery (asterisk), and a left posterior cerebral artery aneurysm (arrow). The peripheral location and saccular morphology of these aneurysms are highly suggestive of mycotic origin.

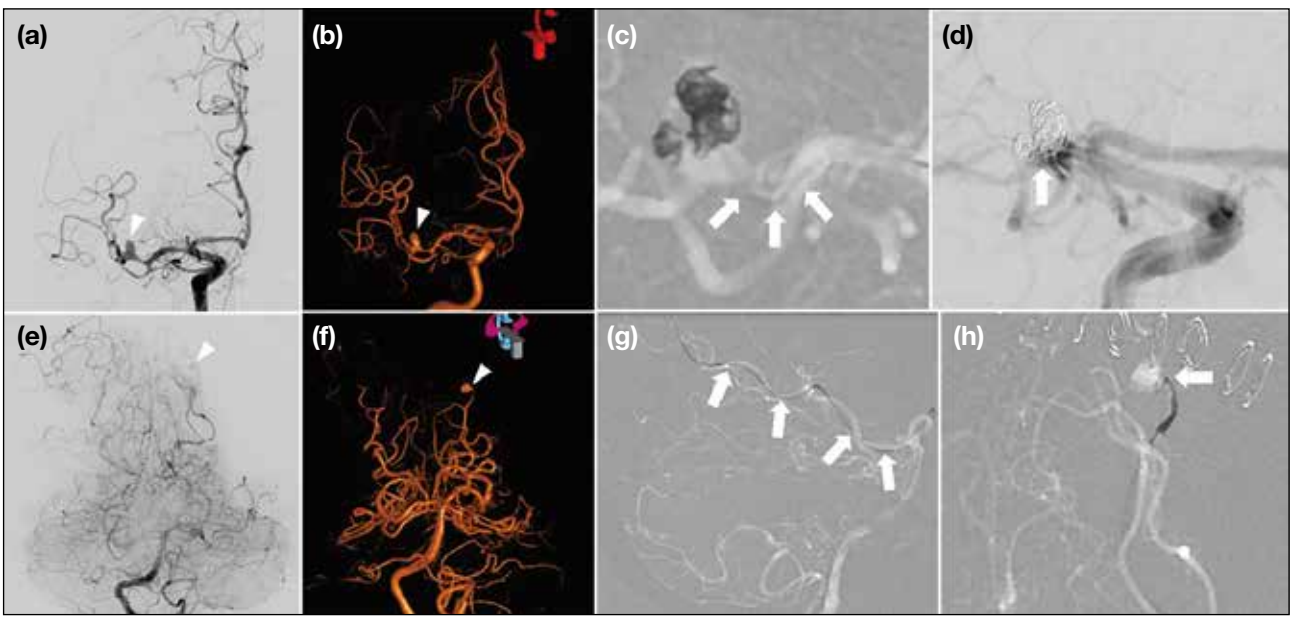

Figure 7. The same patient as Figures 5 and 6 underwent surgical clipping of the right distal anterior cerebral artery mycotic aneurysm. (a) Frontal view of digital subtraction angiography (DSA) of the right internal carotid artery shows a saccular distal right M1 aneurysm (arrowhead). (b) Volumetric rendering depicts the saccular aneurysm (arrowhead), allowing accurate preoperative measurement of its size, height, and neck. The arterial supply from the right middle cerebral artery (MCA) and its angulation in three-dimensional space are also visualised. (c) DSA of the right MCA using the roadmap technique enabled neuronavigation with the use of a guidewire to access the M1 aneurysm for precise coil embolisation (arrows). (d) Post-embolisation DSA of the right MCA shows successful occlusion of the mycotic aneurysm with preserved flow to the distal branches (arrow). (e) Frontal DSA of the right vertebral artery demonstrates a peripherally located P4 aneurysm (arrowhead). (f) Volumetric rendering depicts its saccular morphology with a narrow neck and clearly shows the arterial supply (arrowhead) from the left posterior cerebral artery (PCA), aiding in accurate preoperative planning. (g) DSA of the left PCA enabled direct neuronavigation using a guidewire (arrows) to the target aneurysm for embolisation. (h) DSA of the distal left PCA shows precise coil embolisation of the peripherally located mycotic aneurysm (arrow), performed through an indirect approach that resulted in parent artery occlusion. Despite the challenging locations of the mycotic aneurysms, DSA with volumetric rendering and the roadmap technique allowed successful neuronavigated embolisation.

aneurysms and the weak correlation between size and rupture risk, surgical or endovascular treatment should be considered for unruptured aneurysms that enlarge or do not regress on follow-up imaging.[7,14] Ruptured or symptomatic mycotic aneurysms also require surgical or endovascular intervention.[14] A surgical approach is indicated when an aneurysm exerts mass effect[14] or supplies an eloquent brain region.[1] However, clipping may be difficult due to a wide or absent aneurysmal neck and fragile vessels.[3]

An endovascular approach is indicated for those unfit for surgery due to cardiac disease.[3] It can be divided into direct or indirect approaches. An indirect approach with parent artery occlusion is the endovascular treatment of choice, especially for distally located aneurysms and circumferential vessel involvement. However, parent artery sacrifice is not possible at times and the direct approach may remain the only viable option. The direct approach using coils or liquid embolic agents allows precise control of the aneurysm while preserving distal flow from the parent artery. Endovascular coiling may be a safer option with higher occlusion and lower procedure-related complication rates[7] (Figure 7). Detachable coils allow precise deployment and better durability compared with liquid embolic agents. They are preferred in proximal aneurysms, while liquid embolic agents are more suitable for distal aneurysms not accessible by microcatheter. Intracranial flow diverters can be used to divert turbulent blood flow from the aneurysm and preserve laminar blood flow in the main vessel and its side branches. With reduced blood flow to the aneurysm and gradual vessel remodelling, this results in progressive aneurysmal sac thrombosis[16] (Figure 8). It is important to note that mycotic aneurysms may grow after simple coiling, while the parent artery may thrombose after flow diverter placement in the setting of infection.

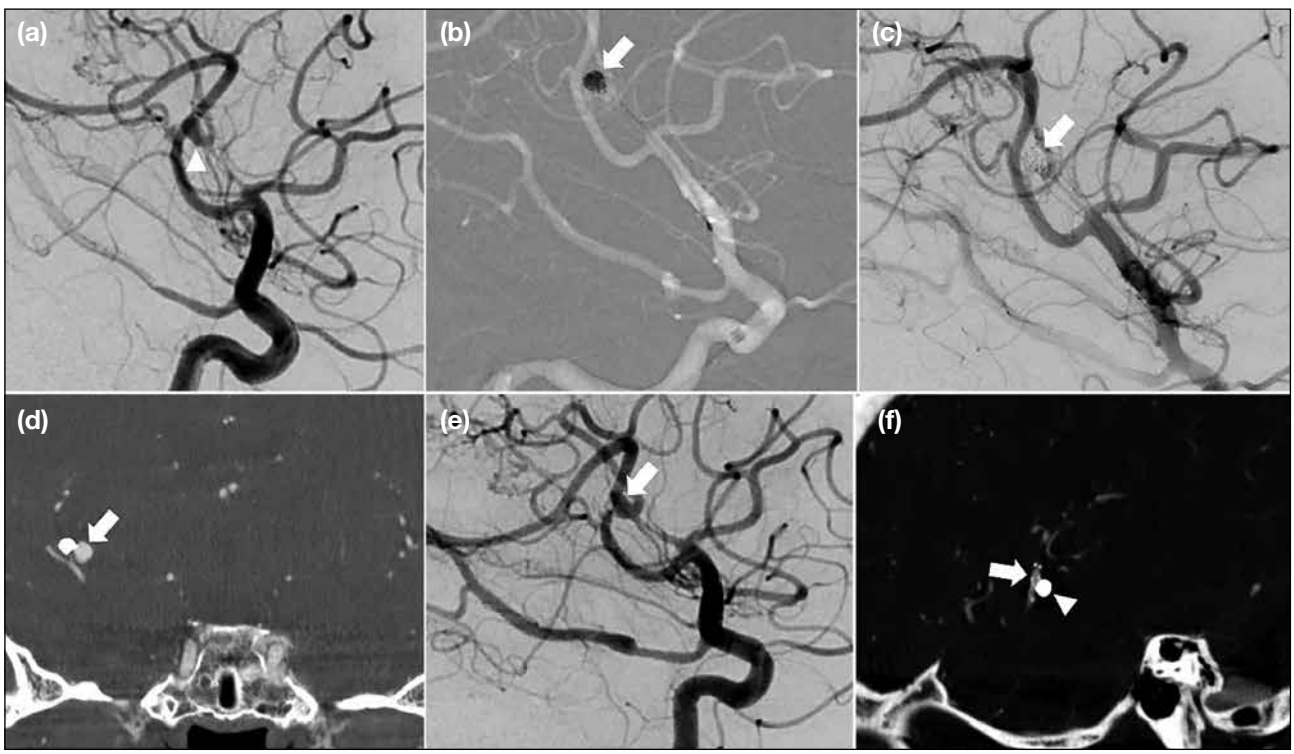

Figure 8. Same patient as Figures 3 and 4. Follow-up computed tomography angiography (CTA) 2 months later showed a persistent M2 mycotic aneurysm (not shown). (a) Oblique projection of digital subtraction angiography (DSA) of the right internal carotid artery (ICA) shows a lobulated M2 mycotic aneurysm arising from the middle cerebral artery (arrowhead). (b) DSA with the roadmap technique enabled neuronavigation for precise embolisation of the mycotic aneurysm (arrow). (c) Post-embolisation DSA of the right ICA shows the successfully embolised right M2 aneurysm (arrow), with preservation of distal flow. (d) Follow-up coronal cone beam CTA 2 months post-embolisation shows a new saccular aneurysm adjacent to the previously embolised aneurysm (arrow). (e) Oblique projection DSA of the right ICA confirms the presence of the new narrow-neck mycotic aneurysm arising from the medial wall of the M2 segment (arrow). (f) Coil embolisation of the second aneurysm was performed, and a flow diverter was deployed across the aneurysmal neck. Follow-up sagittal cone beam CTA 1.5 years later shows the flow diverter in situ (arrow), with successfully embolised aneurysm (arrowhead). The stent remains patent and the distal branches are preserved.

CONCLUSION

Neurological complications secondary to IE require prompt recognition of its typical presentations and imaging manifestations to facilitate early diagnosis of neurological complications and their subsequent treatment, including possible radiological intervention.

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