



The imaging features of cerebral septic infarction in two patients with infective endocarditis

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Abstract

Background Neurologic complications are frequently seen in infective endocarditis (IE) and were identified in about 70% of patients with IE. However, the imaging features of the cerebral septic infarction were less investigated.

Purpose To demonstrate the imaging features of the cerebral septic infarction of IE.

Material and methods Two patients were clinically diagnosed as IE according to the modified Duke criterion. We studied their imaging profiles and reviewed the literature of the imaging features of neurologic complications of IE.

Results The critical features are multiple ischemic and hemorrhagic lesions, most of which locate at the cortical-medullary junction. The septic infarctions are irregular patchy in shape and have characteristic imaging features indicating complications of IE.

Conclusion Magnetic resonance imaging (MRI) with different sequences can detect the features and provide clinical evidence to physicians to make the correct diagnoses and then the treatment plans.

Keywords Infarction · Abscess · Magnetic resonance imaging · Infective endocarditis

Introduction

Infective endocarditis is defined as the infection of endocardial surface of the heart. Severe neurologic complications are frequently seen and were found in about 70% of patients with infective endocarditis (IE) [1–3]. Among these complications, cerebral ischemic and hemorrhagic lesions are mostly commonly seen and the incidence of them in patients with IE are 50.0–87.2 and 25.6–69.0%, respectively. Meningitis, abscess, and aneurysm are less common; the incidence is 1–20, 1–12.8, and 2–8%, respectively [1, 2, 4–8]. Patients with potential neurologic complications of IE should not be treated with anticoagulant therapy [4]. Alternatively, MRI can detect these complications accurately and help the physicians make the correct diagnosis

and treatment plan. There has been a rapid growth in the field of imaging features of neurologic implications, for instance, Bakshi et al. [9] have described cranial MRI findings in 12 IE patients, and first defined “septic infarction.” Encouraged by these findings and focusing on the imaging features of septic infarction, we herein investigated a series of imaging findings from two IE patients with neurologic symptoms.

Material and methods

Two patients were clinically diagnosed as IE according to the modified Duke criterion using major and minor criteria [3, 4]. Clinical evidence included positive blood cultures, endocardial vegetations, and valvular insufficiency detected by echocardiography. We studied their imaging profiles and reviewed the literature of the imaging features of neurologic complications of IE.

Results

Patient 1: A 39-year-old female presented with a 1-month history of fever up to 39 °C when admitted. Echocardiogram

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showed small vegetations on the mitral valve, mitral prolapse, and regurgitation. Blood cultures showed positive *Streptococcus gordonii*. The patient was treated with antibiotics and arranged mitral valve replacement operation. The day before the operation, she developed slurred speech, right lower extremity weakness, and transient unconsciousness. A brain CT (computed tomography) scan was performed and revealed patchy hemorrhage along the left middle main artery (MCA). On the same day, a digital subtraction angiography (DSA) was therefore taken and revealed that the left MCA trunk was obliterated. The patient received embolectomy, and the neurologic symptoms were alleviated. She was treated with antibiotics, antiplatelet, and anticoagulant. The neurologic symptoms deteriorated on the 4th day, and a brain CT showed irregular patchy hypodensities in the left temporal lobe and basal ganglia. Multiple hemorrhages were also observed in those areas as well as the left posterior temporal lobe, parietal lobe and anterior frontal lobe, and right temporal lobe, which are located at the gray-white matter junctions (Fig. 1a). The antiplatelet and anticoagulant therapies were discontinued. She underwent MRI examination on the 5th day after the emergence of symptoms of the nervous system. The lesion in the left temporal lobe and basal ganglia demonstrated hypointensity on the T1-weighted image (T1WI), hyperintensity on the T2-weighted image (T2WI), fluid-attenuated inversion recovery (FLAIR), and diffusion-weighted image (DWI) (Fig. 1b, c). There are many other punctate lesions with the same signal intensity disseminated in the left hemispheres. Apparent diffusion coefficient (ADC) showed restricted diffusion. All these findings were consistent with acute infarction. Susceptibility-weighted image (SWI) showed hemorrhagic transformation of the infarction in the left temporal lobe and basal ganglia, and more small hemorrhagic lesions disseminated in hemispheres than CT (Fig. 1d). Thrombolytic therapy other than anticoagulant was started, and antibiotic therapy was continued. MRI performed on the 12th day after neurological onset (Fig. 1e–g) showed infarction in the left temporal lobe, and basal ganglia developed an occupying effect with a clearer edge (Fig. 1e). DWI showed that the hyperintense areas became smaller and localized (Fig. 1f). Post-contrast showed patchy and ring-like enhancements around the hyperintense areas on DWI (Fig. 1g), which were consistent with abscesses. Antibiotic was changed to ceftriaxone sodium because it was sensitive, and treatment of cerebral infarction was continued. The patient's condition improved gradually, and MRI (performed on the 33th day after neurological onset) showed that the abscesses decreased but the edema around it were enlarged (Fig. 1h–j).

Patient 2: A 64-year-old male with a long history of heart valve disease and mitral valve prolapse was admitted after having 2 weeks of fever up to 39 °C and 2 days of slurred speech. MRI showed irregular patchy hypointensity on T1WI, hyperintensity on T2WI, FLAIR, and DWI in the cortical and

subcortical areas of the left frontal lobe (Fig. 2a, b), and punctate lesions with the same signal intensity disseminated in the gray-white matter junctions in hemispheres. ADC showed restricted diffusion. These findings are consistent with acute infarction. Hemorrhagic transformation was detected by T2*-weighted image (T2*WI) (Fig. 2c). The patient's condition was improved after treating with antibiotic, antiplatelet, lipid-modulating, and anticoagulant therapy. On the 15th day, his slurred speech worsened and delirium occurred. *Staphylococcus aureus* was identified in the blood cultures. Pre-contrast MRI showed that the lesion in the left frontal lobe developed an occupying effect with a clearer edge (Fig. 2d), and DWI showed round hyperintensity surrounded by a slight hypointense ring which was hypointense on T1WI and T2WI (Fig. 2e). The ring enhanced significantly on post-contrast T1WI (Fig. 2f). All these findings indicated abscess's formation. Anticoagulant therapy was stopped, and antibiotic therapy of higher level was continued. The patient's neurologic condition improved gradually, his MRI performed on the 31st day showed that the abscess got smaller, the wall got regular, and the edema around it was enlarged (Fig. 2g–i).

Discussion

Neurologic complications of IE such as infarction, hemorrhage, and abscess arise from embolization of valvular vegetation, i.e., septic embolization [2, 10], and sometimes may be the first symptom of IE [10]. Usually, the same patient can present one or more complications [4]. The most critical features are multiple ischemic and hemorrhagic lesions which are located at the cortical-medullary junction [2, 9], and about 40–65% of the lesions disseminated in the area of the cerebral middle artery [11, 12]. Our two patients demonstrated these characteristic features: multiple focal infarctions of various sizes with hemorrhagic transformations, hemorrhages of different time, and macro-abscesses. All of the small lesions are located at the cortical-medullary junction, macro-abscesses are located at both cortical and subcortical areas, and most lesions involve the cerebral middle artery distribution. Other complications such as meningitis, abscess, and aneurysm are not frequently seen, but the abscess is seen in both our two patients.

Geisenberger et al. [13] described the pathological process of the neurological complications of IE. The cardiac valvular vegetations are composed of fibrins, platelets, and bacteria. The mobile vegetations, which are synonymous with septic embolisms, are transported to cerebral arteries causing embolization and focal neurologic deficits. Then the inflammation occurs forming abscess; the necrosis of the arterial wall causes the formation of infectious aneurysm. If the vessel ruptures, there will be hemorrhages in the parenchyma, subarachnoid, ventricle, and subdura.

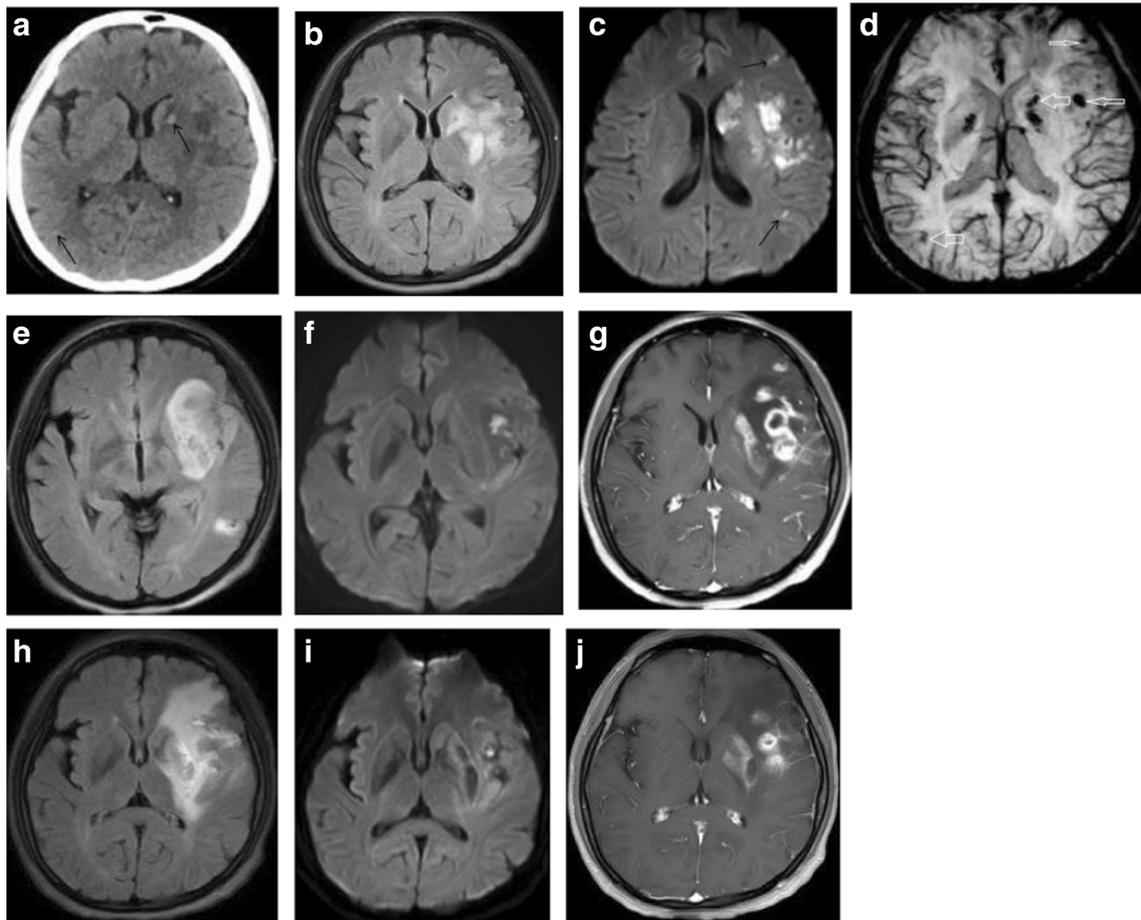


Fig. 1 Patient 1. **a** Brain CT performed 4 days later after onset of slurred speech and right lower extremity weakness shows irregular patchy hypodensities in the left temporal lobe and basal ganglia, and multiple small hemorrhages in the above-mentioned areas and gray-white matter junctions of the right parietal lobe (arrows). **b–d** MRI performed on the 5th day. The lesions in the left temporal lobe and basal ganglia are hyperintense on FLAIR (**b**) and DWI (**c**), and DWI shows many other punctate lesions (arrows). ADC image shows the diffusion is restricted (not included). **d** SWI shows multiple hypointensities disseminated in the above-mentioned areas (arrows; part of them not included in the picture) indicating hemorrhagic transformation of the infarction and hemorrhages of different time. The patient was treated with antibiotic and thrombolytic

therapy other than anticoagulant. **e–g** MRI performed on the 12th day after neurological onset. **e** FLAIR shows that infarction in the left temporal lobe and basal ganglia developed an occupying effect with a clearer edge. **f** DWI shows that hyperintensity is localized and surrounded by irregular hypointense rings. **g** Post-contrast T1WI shows that the rings enhance significantly and other patchy enhanced areas. All these are consistent with abscesses' formation. Antibiotic was changed to ceftriaxone sodium because it was sensitive, and treatment of cerebral infarction was continued. The patient's condition improved gradually. **h–j** MRI performed on the 33th day. **h** FLAIR, **i** DWI, and **j** post-contrast T1WI show that the abscesses become smaller, and the edema around enlarges

The imaging features of our two patients explained the pathological process of macro-abscesses of IE accurately. Initially, they demonstrated acute infarctions. About 2 weeks later, the lesions developed an occupying effect with clearer edges, and DWI showed irregular round hyperintensities with a hypointense wall consistent with abscesses. The abscesses were surrounded by edema, and their walls were enhanced significantly on post-contrast MRI. Our findings accord with “septic infarction” first defined by Bakshi et al., which was described as a unique circumstance of IE. We regard them as macro-abscesses of special time. After another 2 week's treatment, the abscesses decreased, but the edema around them was enlarged. No research for this phenomenon has been reported. We speculate that the reason for the phenomenon is the

increase in cerebral perfusion because septic embolism becomes smaller after treatment. More mechanistic studies should be further performed.

As a complication of IE, the cerebral septic infarctions before macro-abscesses' formation are slightly different from typical infarctions. First, they are hemorrhagic infarctions, easily detected by T2*WI or SWI. Second, they are irregular patchy in shape but not triangular or wedge-shaped. Finally, they are accompanied by small infarctions and hemorrhages of different times disseminated in the gray-white matter junctions of the hemisphere. These features are very important because they can expedite the diagnosis of IE and prompt immediate, proper treatment.

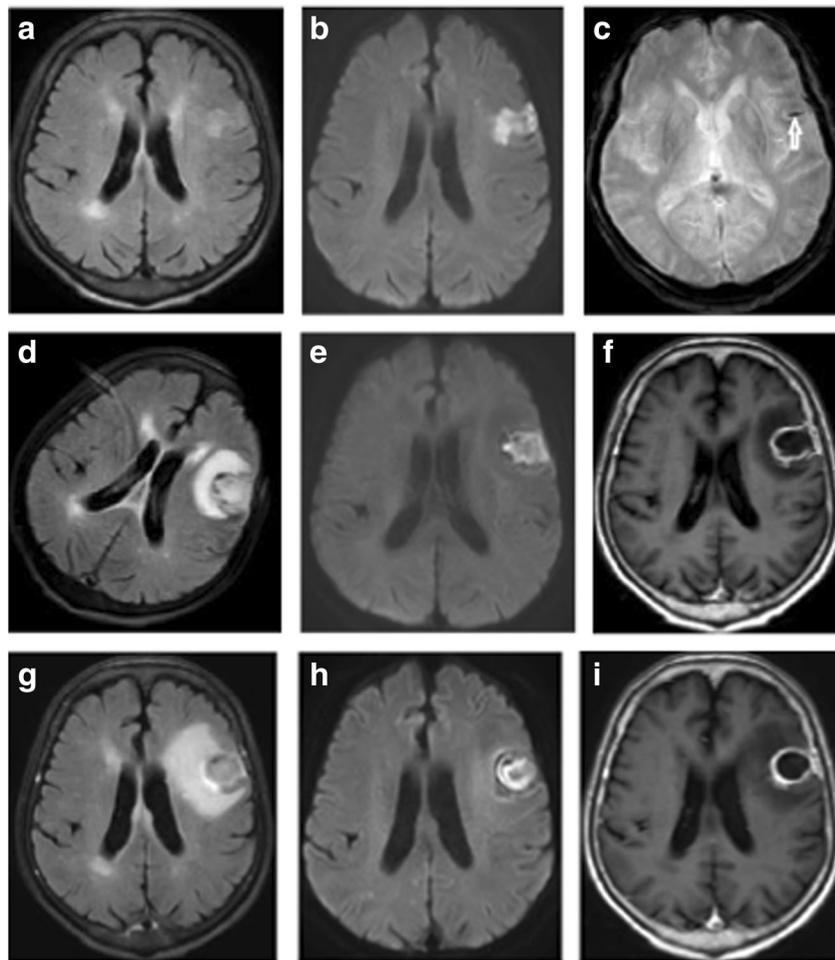


Fig. 2 Patient 2. **a–c** MRI performed 2 days later after onset of slurred speech. **a** FLAIR shows irregular patchy hyperintensity in the left frontal lobe which is hyperintense on DWI (**b**). ADC image shows that diffusion is restricted. All these are consistent with acute infarction. **c** T2*WI shows hemorrhagic transformation in the lesion of the left frontal lobe (arrow). The patient's condition was improved after treatment with antibiotic, antiplatelet, lipid-modulating, and anticoagulant therapy. On the 15th day, his slurred speech worsened and delirium occurred. *Staphylococcus aureus* was identified in the blood cultures. **d–f** MRI

performed on the 15th day. **d** FLAIR shows that the lesion developed an occupying effect with a clearer edge. **e** DWI shows irregular round hyperintensity surrounded by a hypointense ring. **f** Post-contrast T1WI shows ring-like enhancement. All these are consistent with abscess's formation. Anticoagulant therapy was stopped, and antibiotic therapy of higher level was continued. The patient's neurologic condition improved gradually. **g–i** Performed 31 days later. **g** FLAIR, **h** DWI, and **i** post-contrast T1WI show that the abscess has a more regular and smaller wall, and the edema around it enlarges

MRI should be performed for all the patients with IE because it can detect silent ischemic or hemorrhagic brain lesions sensitively and then modify the diagnosis and therapeutic plans [6]. MRI sequences should include DWI and T2*WI in addition to T1WI, T2WI, and FLAIR [4, 14]. Besides, CT is the best method to find hemorrhages, but T2*WI and SWI can find more lesions of different times. Our study indicates that post-contrast T1WI plays an important role in detecting septic infarction because it can display the wall of the abscess.

In conclusion, we reported two patients with IE and their neurologic complications focusing on the imaging features of the cerebral septic infarction. The lesions are multiple and locate at the cortical-medullary junction; the septic infarctions have characteristic imaging features indicating complications

of IE. MRI with different sequences can detect the features and provide evidence to clinical physicians to make the correct diagnoses and then the treatment plans.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval This article does not contain any studies with human participants performed by any of the authors.

Informed consent Informed consent was obtained from all individual participants included in the study.

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