Revisiting Magnetic Resonance Imaging Gadolinium Contrast Enhancement in Subacute Cerebral Infarction:

Two Cases Leading to Misdiagnosis

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Brain magnetic resonance imaging (MRI) is essential in the process of dating and staging cerebral ischemic lesions.[1–4] Classical findings include restriction in diffusion-weighted sequences. Characteristic patterns are also found in other sequences, such as contrast-enhanced T1-weighted.[3] Usually 3 types of contrast enhancement are described: intravascular, meningeal, and parenchymal.[3] While intravascular enhancement is occasionally seen in the acute phase when there is vessel occlusion, parenchymal enhancement usually occurs during the subacute phase.[4] Interpretation of imaging findings may prove challenging when there is an unclear history or atypical symptoms.

Case Report 1

An 80-year-old woman presented new-onset bilateral horizontal diplopia and gait instability. She had a past medical history of hypertension and diabetes mellitus type 2. Timing of symptom onset was unclear. Brain computed tomography (CT) revealed a left thalamic round lesion. MRI (first week of admission) showed no evidence of restriction on diffusion-weighted sequences but showed contrast enhancement on contrast-enhanced T1-weighted sequence (Figure 1). Considering the possibility of brain metastasis, the patient was referred to the oncology department for a full diagnostic workup and was started on dexamethasone (15 mg/d). Six months later, the patient presented acute confusion and left homonymous hemianopsia. MRI showed evidence of an acute ischemic stroke in the right posterior cerebral artery territory. The previous thalamic lesion had evolved to a small lacuna. After detection of atrial fibrillation, the patient was started on anticoagulation therapy.

Case Report 2

A 60-year-old man was admitted to the hospital due to brief episodes of involuntary movements of his left foot. His medical history revealed several cardiovascular risk factors (smoker, hypertension, diabetes mellitus type 2) and HIV infection (CD4 cell count > 250 cell/mm^3). Brain CT was unremarkable. The patient was started on levetiracetam (2 g/d) following a presumptive diagnosis of focal motor seizures. MRI (3 weeks after symptom onset) revealed a cortical round lesion in the right precentral gyrus (15 mm in diameter) without vasogenic edema—hyperintense on T2, fluid-attenuated inversion recovery, and post-gadolinium without restricted diffusion (Figure 1). Due to possible opportunistic infection, a lumbar puncture was performed, and the patient was started on empirical antibiotics and antiviral and antifungal medication. Laboratory results were all negative. Electroencephalogram revealed right frontal epileptogenic activity that remitted with antiepileptic therapy. A follow-up brain MRI 3 months later revealed that the lesion progressed to an area of encephalomalacia without contrast enhancement. Vascular studies revealed right internal carotid artery occlusion that had been detected in the first MRI. The patient successfully underwent carotid endarterectomy.

Discussion

Although both patients presented cardiovascular risk factors, presentation and neuroimaging findings were less typical of acute stroke. In case 1, there was uncertainty about the onset of the symptoms. In case 2, paroxysmal episodes with history of HIV suggested a possible secondary infection. Imaging findings consisted of lesions without restricted diffusion or a clearly defined vascular territory with the presence of contrast enhancement. Contrast-enhancement patterns in brain ischemic lesions may result from vessel occlusion, flow impairment, disturbance of the blood-brain barrier, altered local hemodynamics, and luxury perfusion.[2–4] While arterial and meningeal enhancement appear early during the first week after stroke, parenchymal enhancement usually begins in the second week, around the time when collateral flow is established and immature capillaries of granulation tissue allow contrast accumulation in the area.[2,3] After 8–12 weeks, contrast enhancement is undetectable, and other causes should be considered.[2] During the subacute phase, enhancement occurs when diffusion-weighted imaging may be normal due to pseudonormalization of the diffusion coefficient.[4] Infarct enhancement can be heterogeneous, sometimes showing bizarre parenchymal patterns, further complicating the diagnosis.[4] Parenchymal enhancement may lead to misdiagnosis with future deleterious outcomes, delaying an adequate approach to the underlying cerebrovascular disease. There is a lack of knowledge about the normal presence of contrast enhancement in ischemic lesions. Moreover, contrast is not part of typical MRI stroke protocols. Our report highlights the normal presence of contrast enhancement in the subacute phase of ischemic stroke that can be misleading without the knowledge of different contrast-enhancement patterns in these lesions.

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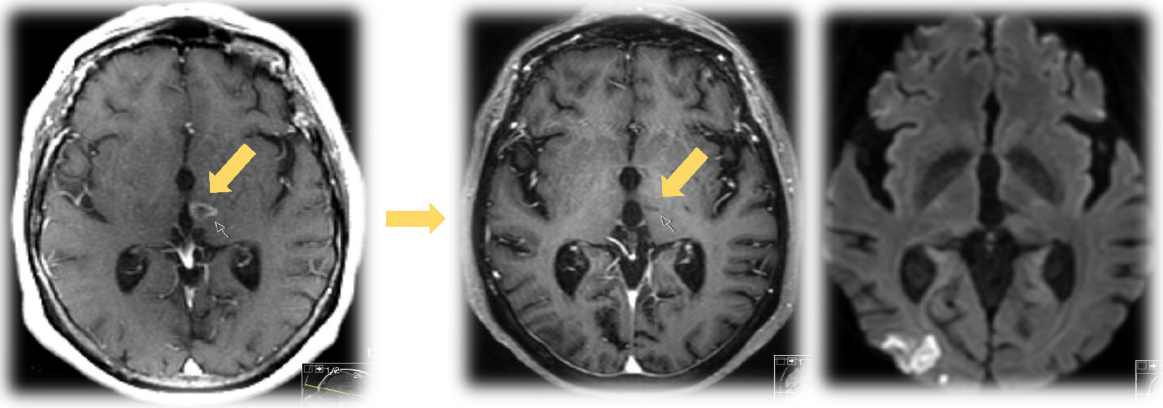
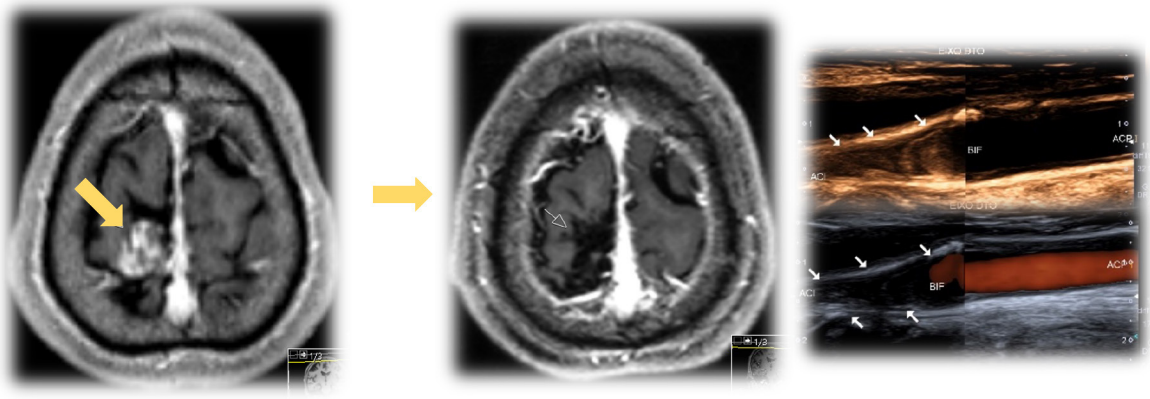
Figure 1. Neuroimaging Findings

Case 1: Initial brain MRI showed a round lesion on the left thalamus, with ring enhancement on post-gadolinium sequence (A1). This lesion progressed to a small lacuna on MRI performed 6 months later (A2) that also showed an acute right occipital ischemic stroke on diffusion-weighted sequence (A3).



Case 2: Circumscribed cortical lesion (15 mm in diameter) on the right precentral gyrus, with nodular enhancement on post-gadolinium sequence (B1). Repeated brain MRI 3 months later showed that the lesion progressed to an area of encephalomalacia without further contrast enhancement (B2). Carotid Doppler ultrasound with right internal carotid artery occlusion (B3).



Abbreviation: MRI = magnetic resonance imaging.

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