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CASE REPORT

CT and MRI features of the "Fogging Effect": A case report and literature review

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Abstract

The "fogging effect" is a phenomenon observed in Computed Tomography (CT) or Magnetic resonance imaging (MRI) imaging that results in a transient normalization of parenchymal abnormalities, such as changes in density or signal, in the context of ischemic strokes. This effect is typically seen in 10% to 54% of cases during the subacute phase of cerebral infarction and is caused by the migration of lipid-rich macrophages, leukocytes, and the extravasation of red blood cells into the infarcted area. These changes can cause an apparent improvement in imaging findings, leading to an incorrect interpretation of the patient's condition. If this phenomenon is not recognized, it can result in diagnostic errors, potentially causing unnecessary treatment discontinuation or altered clinical management. In this report, we present the case of a 60-year-old patient with a subacute ischemic stroke, where the "fogging effect" was evident on both CT and MRI scans, emphasizing the importance of awareness in preventing misdiagnosis and ensuring appropriate treatment.

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Introduction

The progression of an ischemic stroke on imaging during the subacute phase can exhibit a variety of changes that reflect the underlying pathophysiological processes of tissue damage and repair. One notable phenomenon observed during this phase is the gradual disappearance of density or signal abnormalities within the infarcted region, a process commonly referred to as the "fogging effect", that typically occurs in the second and third weeks [1,2].

Although the "fogging effect" has been well documented in the context of conventional Computed Tomography (CT) and Magnetic resonance imaging (MRI) imaging, its occurrence on more specialized imaging sequences, such as magnetic susceptibility-weighted imaging (SWI), is rarely discussed in the literature.

Case Report

A 60-year-old male patient with a history of chronic smoking and hypertension, under medical treatment, presented with sudden-onset right hemiplegia 10 days ago, following a non-ST elevation myocardial infarction.

A brain CT scan performed on day 1 revealed a region of subtil hypodensity localized to the superficial territory of the left middle cerebral artery, without hemorrhagic infarction.

The patient was initiated on dual antiplatelet therapy, and the clinical course was marked by worsening symptoms.

A follow-up brain CT scan performed on day 10 showed a left fronto-parietal subcortical hypodensity (indicative of edema) and an isodense appearance of the cortical ribbon; this appearance was consistent with the "fogging effect" (Fig. 1).

An MRI performed on this patient revealed a subcortical area with hyperintensity on T2/FLAIR, with areas of diffusion restriction, along with thickening of the cortical ribbon with isosignal on both T1 and T2/FLAIR sequences. The SWI sequence showed a diffuse loss of cortical signal in the infarcted area (Fig. 2).

This appearance on the SWI sequence is rarely reported in the literature; it corresponds to the

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migration of lipid-rich macrophages and the extravasation of red blood cells containing deoxyhemoglobin and methemoglobin, which causes a paramagnetic effect.

Discussion

Ischemic stroke is one of the leading causes of morbidity and mortality worldwide, second only to myocardial infarction in terms of cardiovascular disease burden. It results from cerebral injury due to the abrupt interruption of blood flow in a vascular territory, leading to ischemia and neuronal necrosis. [1]

Hypoperfusion may result from an *in-situ* thrombus, embolic migration, or reduced blood flow (due to conditions such as heart failure, dissection, or stenosis from vasculitis), all of which cause a reduction in cerebral blood flow, leading to impaired cellular exchanges and cytotoxic edema. Clinically, it manifests as motor or sensory deficits, aphasia, or altered consciousness, with a sudden onset [1].

Ischemic stroke progresses through four stages, corresponding to varying degrees of injury, with their characteristics on CT and MRI described in the table below (Table 1).

The progression of cerebral infarction (in the absence of early reperfusion) leads to the formation of infarcted tissue, which is manifested in imaging by a progressive exaggeration of hypodensity on CT, the appearance of hyperintensity on FLAIR and T2 sequences, with a decrease in diffusion-weighted imaging hyperintensity and pseudonormalization of Apparent diffusion coefficient (ADC) values around 10–14 days post-infarct.

During the subacute phase, between the second and third week, there is migration of lipid-rich macrophages and leukocytes, capillary proliferation, extravasation of red blood cells, and a reduction in the edematous component [2].

These processes are reflected on CT by an increase in density, which unmasks the infarcted area and can be a source of confusion. This phenomenon corresponds to the "fogging effect," which occurs in 10% to 54% of cases and may lead to confusion due to the radiological pseudonormalization of the cerebral tissue [1–4].

This pseudonormalization of density is transient, and hypodensity reappears on follow-up CT scans [5].

The administration of contrast agents helps refine the diagnosis by showing gyriform enhancement due to the disruption of the blood-brain barrier, thus delineating the affected territory. In cases where perfusion CT is performed, a "luxury perfusion" pattern is noted, reflecting an adaptive increase in blood flow, which better highlights the infarcted area [5].

On MRI, the fogging effect is characterized by either the disappearance of signal abnormalities on T2/FLAIR and diffusion sequences or by blurring and attenuation of T2/FLAIR hyperintensity [1,2].

The "fogging effect" may be visible on SWI as signal voids caused by the presence of deoxyhemoglobin and methemoglobin within extravasated red blood cells [1]. These signal voids should be interpreted as hemorrhagic transformation if they present as punctate lesions exceeding 5 mm in size, or as microbleeds if smaller [6].

Conclusion

The "fogging effect" is a well-recognized phenomenon in the subacute phase of ischemic stroke imaging, where the infarcted area appears isodense or isointense with surrounding tissue, which can

Table 1. CT and MRI appearance of the evolutionary stages of ischemic stroke [7].

Evolution		CT	MRI			
			Diffusion	ADC	FLAIR	T1
Hyperacute <6 hours		Isodensity	Hyper intensity	Low	Iso intensity	Iso intensity
Acute 6–48 hours		Moderate hypodensity	Marked hyper intensity	Low	Hyper intensity	Iso/hypo intensity
Subacute	Early 2–10 days	Hypodensity	Hyper/iso intensity	Low	Hyper intensity	Hypo intensity
	Late 10–21 days		Hyper/iso intensity	High	Hyper intensity	Hypo intensity

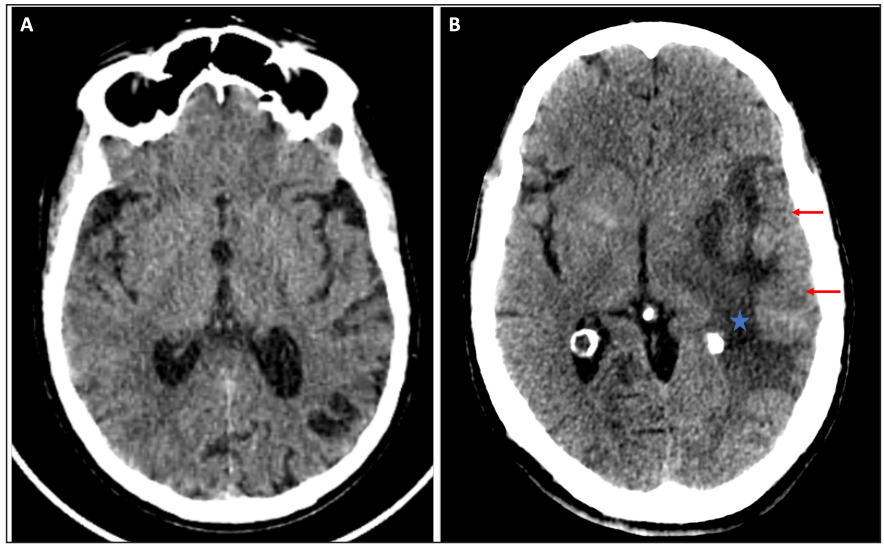


Figure 1. Axial section of the brain CT scan was performed on day 1 (A) and day 10 (B). Note the iso density of the infarcted left parietal cortex (red arrows) and the subcortical edematous region (asterisk).

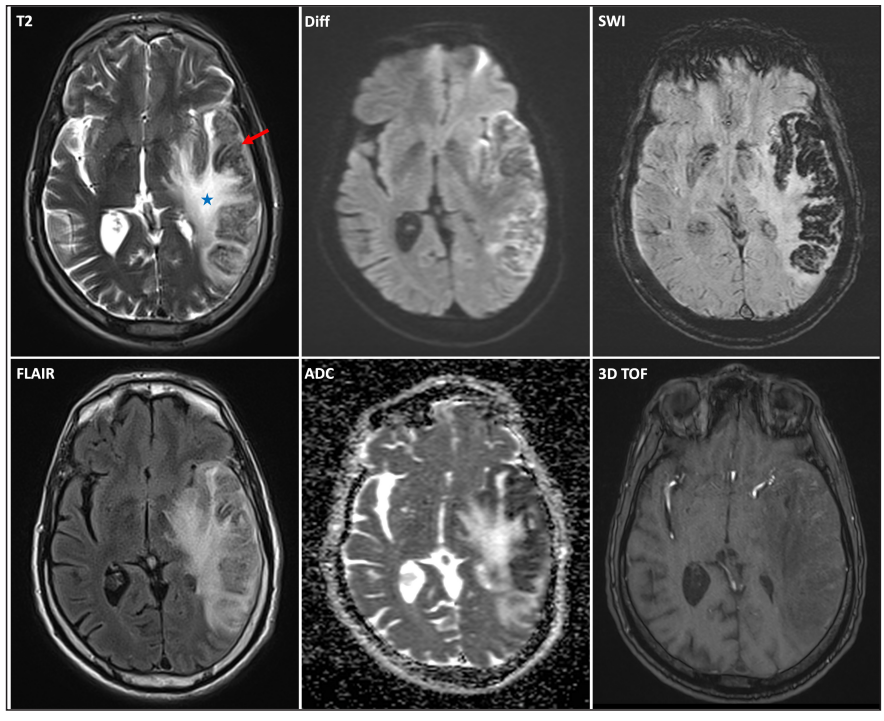


Figure 2. Axial sections of the brain MRI were performed on day 10. Normalization of the signal in the left parietal cortex, with iso intensity on T1, T2/FLAIR, and diffusion sequences, along with low ADC (red arrows), and the subcortical edematous region (asterisk). The SWI sequence demonstrates a cortical signal void, corresponding to the paramagnetic effect of blood components. The absence of T1 hyperintensity does not suggest parenchymal hemorrhagic changes.

sometimes be misinterpreted as improvement of the ischemic injury.

Familiarity with the "fogging effect" phenomenon helps prevent diagnostic errors, such as mistaking it for improvement or resolution of ischemia, which could lead to inappropriate discontinuation of treatment or secondary prevention strategies.

Its appearance on MRI magnetic susceptibility-weighted sequences is rarely described in the

literature, characterized by a paramagnetic effect in the form of signal voids within the infarcted area.

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