

VANISHING ACT: THE TRUTH BEHIND THE CT FOGGING PHENOMENON IN ISCHEMIC STROKE - A CASE REPORT

Tatjana DELEVA STOSHEVSKA¹, Avdi NAZIFI², Tatjana CEPREGANOVA CANGOVSKA⁶, Daniela RISTIKJ STOMNAROSKA¹, Dimitar VELJANOVSKI¹, Bojan STOSHEVSKI³, Sofija NIKOLOSKA⁴, Marko NIKOLOSKI⁵, Driton SELMANI²

1 CGH "8-mi Septemvri" , Skopje, Republic of North Macedonia*

*2*Faculty of Medical Sciences, University of Tetovo, Republic of North Macedonia*

3 PHI UC for Pulmonology and Allergology , Skopje, Republic of North Macedonia*

4 PHI UC for Ear, Nose and Throat, Skopje, Republic of North Macedonia*

5 PHI UC for Surgical Diseases "Sv. Naum Ohridski", Skopje, Republic of North Macedonia*

6 Faculty of Medical Sciences, University of Goce Delchev, Shtip, Republic of North Macedonia

Abstract

The fogging phenomenon is a well-recognized yet frequently underappreciated imaging pitfall in neuroimaging, particularly during the subacute phase of cerebral infarction. It is characterized by the transient normalization of previously hypodense ischemic brain tissue on computed tomography (CT), which may lead to misinterpretation as lesion resolution.

We present a case of subacute ischemic stroke in an 89-year-old patient, demonstrating the fogging effect on CT imaging, accompanied by a review of its pathophysiology, temporal evolution, and clinical implications. Recognition of this phenomenon is essential for neurologists and radiologists to prevent diagnostic errors and ensure appropriate patient management.

Keywords: ischemic stroke; fogging phenomenon; computed tomography (CT).

Introduction

Cranial CT remains a first-line imaging modality in the evaluation of acute and subacute stroke due to its accessibility and speed. However, its sensitivity varies depending on the timing of imaging. One notable limitation is the fogging phenomenon, first described in the 1970s, in which infarcted brain tissue transiently appears isodense relative to normal parenchyma.

Non-contrast CT is widely used in the evaluation of acute stroke; however, its sensitivity varies with time and anatomical location. Posterior fossa infarctions remain challenging due to technical limitations. The fogging phenomenon, first described in ischemic stroke imaging, represents a transient phase in which infarcted tissue becomes isodense, potentially obscuring diagnosis.

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", which typically occurs in the second and third weeks [1,2].

This phenomenon may falsely suggest lesion resolution or lead to underestimation of infarct size. Recognizing this temporal imaging pattern is essential, especially when clinical findings remain discordant with imaging.

Case Report

An 89-year-old patient was admitted due to generalized weakness, gait instability, and left-sided limb weakness, with symptoms developing approximately 10 days prior to the initial neurological evaluation. Because of suspected cerebrovascular insult, an urgent brain computed tomography (CT) scan was performed.

The initial CT demonstrated advanced global cortical atrophic changes without evidence of an expansive lesion. In the left cerebellar hemisphere, involving the anterior lobe and part of the

superior cerebellar peduncle, a well-demarcated hypodense area with cortico-subcortical distribution and mild surrounding edema was observed, without significant effect on the ventricular system. The findings were consistent with a late subacute/early chronic ischemic infarction.

Past medical history was notable for arterial hypertension treated with regular therapy. Admission ECG revealed atrial fibrillation, after which the patient was evaluated by an internist and admitted to the neurology department.

Neurological examination showed preserved consciousness and orientation without speech disturbances. Cranial nerve examination was unremarkable. Coordination testing was normal on the right side, while dysmetria was noted on the left. Mild left-sided weakness was present, characterized by drifting and slow descent of the left extremities during positional testing. Reflexes were symmetric, and plantar responses were flexor bilaterally.

Follow-up brain CT demonstrated increased density and less distinct demarcation of the previously described left cerebellar post-ischemic lesion, prompting a recommendation for brain magnetic resonance imaging (MRI). Although MRI was scheduled, the examination could not be completed due to technical reasons. A subsequent follow-up CT demonstrated near-complete resolution of the previously observed hypodensity in the left cerebellar hemisphere, a finding suggestive of the "fogging phenomenon." No new focal lesions were identified in the remaining brain parenchyma.

As part of the etiological workup, cardiology evaluation confirmed atrial fibrillation. In addition, Doppler ultrasonography of the carotid and vertebral arteries demonstrated atherosclerotic but hemodynamically insignificant changes. During hospitalization, the patient was treated as having a cardioembolic ischemic stroke with antiedematous, anticoagulant, antihypertensive, statins, and symptomatic therapy. Regular monitoring of vital signs was performed.

The patient was managed as a cardioembolic ischemic stroke. During hospitalization, the general condition deteriorated due to cardiogenic decompensation with subsequent cardiac arrest which ultimately resulted in a fatal outcome.

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, whose CT characteristics are described in the table below (Table 1).

Temporal Evolution of CT Findings in Ischemic Stroke

Phase	CT Appearance
Hyperacute (0–6 h)	Often normal, especially in posterior fossa
Acute (6–72 h)	Hypodensity becomes more apparent
Subacute (7–21 days)	Fogging phenomenon (isodense phase)
Chronic (>3 weeks)	Hypodensity with encephalomalacia and atrophy

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 reduction of the edematous component [1]. These processes are reflected on CT by an increase in density, which may obscure 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 [2,3]. The fogging phenomenon has been reported in 20–50% of infarctions, most commonly between days 10–20. It is associated with reperfusion and larger infarcts but may occur in posterior circulation strokes as well. This pseudonormalization of density is transient, and hypodensity reappears on follow-up CT scans [4]. 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]. 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 [4]. The fogging phenomenon is an important diagnostic pitfall in both supratentorial and cerebellar infarctions. In elderly patients and posterior circulation strokes, awareness is crucial. 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 [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 and may sometimes be misinterpreted as improvement of the ischemic injury. The fogging phenomenon is an important diagnostic pitfall in both supratentorial and cerebellar infarctions. In elderly patients and posterior circulation strokes, awareness is crucial. 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.

The fogging phenomenon has been reported in 20–50% of infarctions, most commonly between days 10–20. It is associated with reperfusion and larger infarcts but may occur in posterior circulation strokes as well.

References

- [1]. Park GS, Jang HG, Park JS, Lee JM, Koh EJ, Choi HY. Computed tomography/diffusion weighted magnetic resonance imaging fogging effect in the subacute stage of ischemic cerebral infarct: a report of three cases. *Nerve* 2021; 7(2):89–91.
- [2]. O'Brien P, Sellar RJ, Wardlaw JM. Fogging on T2-weighted MR after acute ischaemic stroke: how often might this occur and what are the implications? *Neuroradiology* 2004 Aug; 46(8):635–41

- [3]. Ricarte IF, Pedroso JL, Carvalho FA, Abrahão A, Valiente RA, Alves MM, et al. The essential can be invisible to the eyes: the “fogging effect” phenomenon in the subacute stage of ischemic stroke. *J Stroke Cerebrovasc Dis* 2013 Nov; 22(8):e628–9
- [4]. Hayden MR. Cerebral microbleeds associate with brain endothelial cell activation-dysfunction and blood–brain barrier dysfunction/disruption with increased risk of hemorrhagic and ischemic stroke. *Biomedicines* 2024; 12(7):1463
- [5]. Braileanu M, Weinberg BD, Hu R, Hoch MJ. Appearance of cerebral infarct fogging on CT perfusion. *Radiol Case Rep* 2019 May 14; 14(7):889–93; doi:10.1016/j.radcr.2019.04.014
- [6]. Moulin T, Tatu L, Vuillier F, et al. Role of imaging in posterior circulation stroke. *Stroke*. 2016;47(2):e45–8.