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Figure. (A) Noncontrast CT scan of the head shows a triangular hypodensity in the territory of the lateral posterior inferior cerebellar artery compatible with a subacute infarct. (B) Follow-up noncontrast CT scan performed 11 days after the onset of the symptoms. The previously easily recognizable infarct is now not obvious. (C) CT scan performed 4 months later reveals an area of encephalomalacia in the same territory as the initial hypodensity.

The fogging effect

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A 64-year-old man developed acute vertigo, nausea, and vomiting. He was taken to a hospital, where he was diagnosed with acute labyrinthitis and discharged home. One day later he returned with worsening symptoms. CT of the head revealed hypodensity in the territory of the lateral branch of the left posterior inferior cerebellar artery (L-PICA) compatible with a subacute infarct (figure, A). The patient was found to have a large patent foramen ovale and was discharged home on oral warfarin. Ten days later, he returned to the emergency room complaining of facial numbness. A repeat noncontrast CT scan of the head showed that the previously obvious L-PICA infarct was now quite subtle (figure, B). A follow-up CT scan performed 4 months later revealed an area of encephalomalacia in the L-PICA territory, matching the territory involved in the first examination and compatible with a chronic L-PICA infarct (figure, C).

Normal findings on CT scan of patients with known radiologic evidence of cerebral infarcts can be a source of great perplexity that can lead to unnecessary neuroimaging. This phenomenon in which initially hypodense ischemic areas transiently become isodense to normal brain has been termed the “fogging effect.” It usually occurs in the second and third weeks after a stroke and is believed to be due to influx of lipid-laden macrophages, proliferation of capillaries, and decrease in bulk water in the infarcted area.^{1,2} Administration of IV contrast invariably demonstrates the otherwise unrecognizable infarct.² The fogging effect has been described with CT imaging and with T1-weighted MRI and both techniques may have diagnostic pitfalls if imaging studies without contrast are performed in the subacute phase of a stroke.^{1,2}

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