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Rapid dystrophic calcification mimicking hemorrhagic transformation after postoperative cerebral infarction: A case report

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Keywords

Cerebral Infarction; Computed Tomography; Dystrophic Calcification; Ischemic Stroke; Magnetic Resonance Imaging

Abstract

Background: Dystrophic calcification is a rare complication of cerebral infarction. This study presents the case of a 67-year-old man with a postoperative stroke who developed dystrophic calcification.

Case Report: A 67-year-old man with atrial fibrillation (AF) underwent surgery for a femoral fracture. On postoperative day 2, he developed acute right arm paralysis, and diffusion-weighted imaging (DWI) showed acute infarction in the left posterior cerebral artery (PCA) territory. Apixaban was initiated. On postoperative day 12, follow-up computed tomography (CT) showed focal hyperdensity prompting apixaban discontinuation. However, serial imaging revealed progressive hyperattenuation with

cortical ribbon distribution, absence of mass effect, and clinical stability.

Conclusion: This case highlights the diagnostic challenge and importance of multimodal imaging in differentiating dystrophic calcification from hemorrhagic transformation, particularly when managing anticoagulated patients. Recognition of temporal evolution, attenuation values, and anatomical distribution—alongside the absence of clinical deterioration—is essential for making appropriate therapeutic decisions.

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Introduction

Intracranial calcification on computed tomography (CT) encompasses physiological

(pineal gland, choroid plexus), neoplastic (oligodendroglioma, meningioma, craniopharyngioma), infectious (cytomegalovirus, toxoplasmosis, neurocysticercosis, tuberculosis), vascular (atherosclerosis, aneurysms, chronic hematomas), metabolic (hypoparathyroidism, Fahr disease), and genetic (Sturge-Weber syndrome, tuberous sclerosis) causes.¹ Dystrophic calcification is a rare complication of cerebral infarction.² To date, the literature describing the detailed multimodal imaging appearance of dystrophic calcifications in cerebral infarcts, particularly magnetic resonance imaging (MRI) findings, is limited. This case report presents a well-documented example of rapid dystrophic calcification developing after postoperative ischemic stroke, emphasizing the diagnostic importance of integrating imaging parameters with the clinical context to differentiate this benign entity from hemorrhagic transformation.

Case Report

A 67-year-old man with a history of hypertension (HTN), diabetes, and persistent atrial fibrillation (AF)—who had stopped taking an anticoagulant several years prior—underwent surgery for a left femur fracture. Aspirin was initiated on postoperative day 1. Two days after surgery, he developed paralysis in his right arm upon awakening. On examination, he had sensory aphasia, right homonymous hemianopsia, facial palsy, and mild weakness in his right arm. A brain diffusion-weighted MRI (DW-MRI) conducted 2.5 hours after symptom onset revealed an acute cerebral infarction in the left posterior cerebral artery (PCA) territory (Figure 1), while gradient recalled echo (GRE) images demonstrated no associated hypodensity within the infarcted area. However, GRE performed 5 hours after stroke showed hypointensity in the left medial temporal lobe (Figure 1).

Susceptibility-weighted imaging (SWI) was not performed. Subsequently, the patient was initiated on dual antiplatelet therapy. A CT scan of the brain obtained three days after stroke showed no evidence of hemorrhagic transformation (Figure 2), and apixaban was initiated four days later because of a large infarct secondary to AF. The antiplatelet agent was discontinued. A follow-up CT scan performed 10 days post-stroke revealed a small hyperdensity within the cerebral infarction (Figure 2), although the patient showed progressive improvement in his arm weakness and aphasia.

Four days later, apixaban was discontinued, as the clinical team suspected hemorrhagic transformation. Hyperattenuation on CT and hypointensity on GRE became increasingly extensive (Figures 1 and 2).

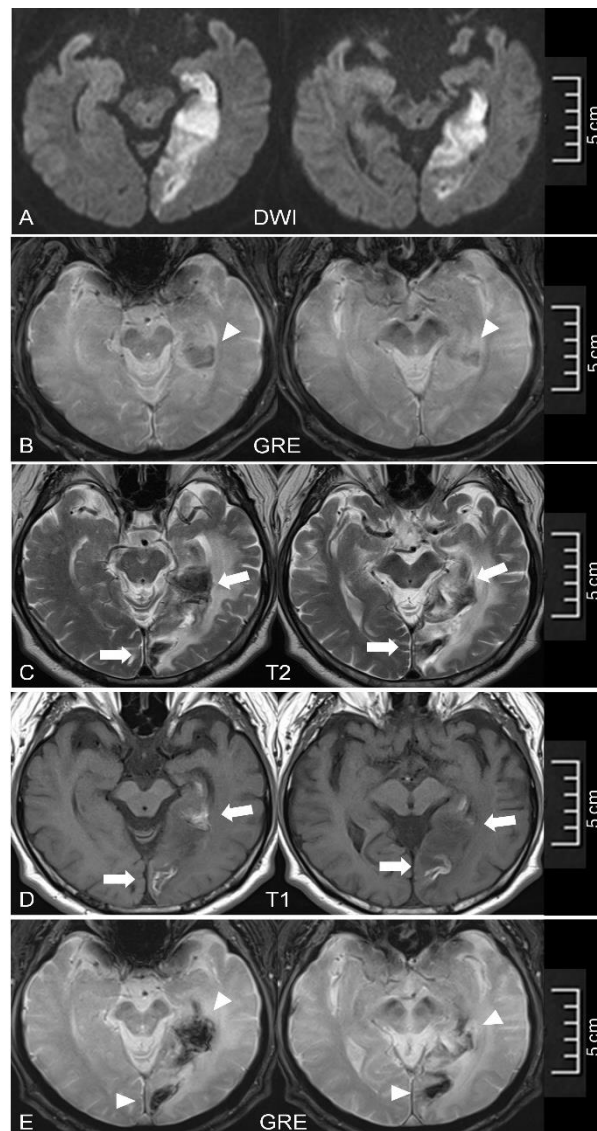


Figure 1. A) Axial diffusion-weighted magnetic resonance imaging (DW-MRI) (7200/98) performed 2.5 hours after symptom onset showing an acute infarction in the left posterior cerebral artery (PCA) territory; B) Axial gradient recalled echo (GRE) (822/26) acquired 5 hours after stroke onset showing hypointensity (arrowheads) in the left medial temporal lobe; C, D) Axial T2-weighted (5370/98) and T1-weighted (618/12) images obtained 6 weeks post-stroke demonstrating heterogeneous signal (arrows) in the left temporo-occipital area; E) Axial GRE (891/26) showing low signal intensity (arrowheads) in the same area. Magnetic resonance imaging (MRI) was performed using a 3-Tesla scanner.

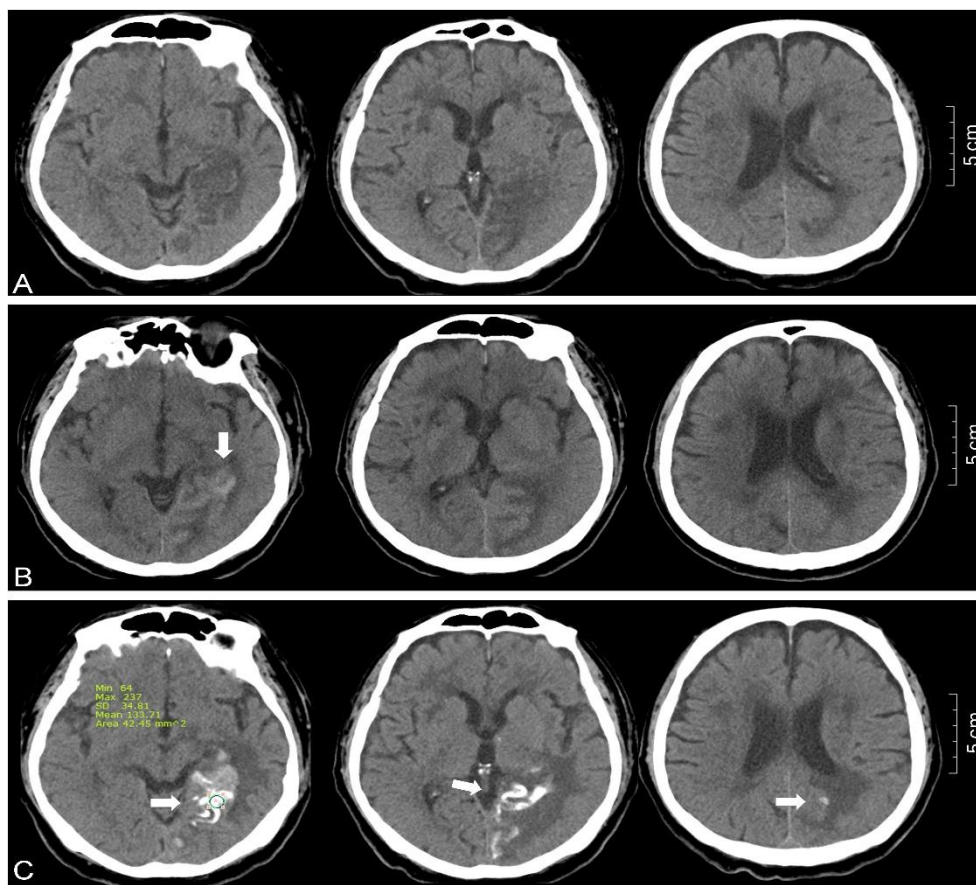


Figure 2. A) Brain axial computed tomography (CT) scans obtained 3 days after stroke onset showing hypodensity in the left posterior cerebral artery (PCA) territory; B) Axial CT scans performed 10 days post-stroke demonstrating hyperdensity of the cortical ribbon (arrow) in the infarcted region; C) Axial CT scans obtained 40 days post-stroke showing striking hyperdensity (arrows) of the cortical ribbon. Hounsfield Unit (HU) measurements were obtained using a circular region-of-interest (ROI) analysis (diameter: 7.4-mm) placed in the hyperdense area (green circle). CT was performed using a Discovery CT750 HD scanner with the following parameters: tube voltage, 120 kVp; reconstruction kernel, soft; and slice thickness, 5 mm.

Hounsfield unit (HU) measurements were obtained using region-of-interest (ROI) analysis with a 7.4-mm diameter circular ROI placed in the cortical ribbon region of the infarct 40 days post-stroke (INFINITT PACS M6, INFINITT Healthcare Co., Seoul, Korea) (Figure 2). The attenuation of the lesion on CT was 134 ± 35 HU (Figure 2). Serum calcium, phosphate, creatinine, and 1,25-dihydroxyvitamin D₃ [1,25-(OH)₂D₃] concentrations were within the normal range. Parathyroid hormone concentration was slightly high but became normalized 2 weeks later. Considering the attenuation values of the lesions and the absence of worsening symptoms, these findings were interpreted as dystrophic calcification, and apixaban was restarted 47 days post-stroke. The patient's Mini-Mental State Examination (MMSE)

score was 22. A 20-month follow-up CT scan showed persistent hyperdensity of the cortical ribbon, with an MMSE score of 21. He exhibited persistent right homonymous hemianopsia; these cognitive and visual deficits were attributed to the sequelae of his previous stroke.

Discussion

Dystrophic calcification as a complication of cerebral infarction occurs 10 days to 13 years after stroke onset.² The ages of the affected patients ranged from 5 months to 71 years.² Notably, dystrophic calcification tends to occur in patients with postoperative stroke.^{2,3} Approximately one-third of reported cases were associated with such surgical complications.² The exact pathophysiology of this condition remains

unknown. Postmortem examination of a case of dystrophic calcification revealed the presence of calcium salts dispersed throughout the infarcted tissue, with higher concentrations in the grey matter at the margins of the infarct and in the perivascular areas.⁴ However, calcium salts were not found in the vessel walls.⁴ Surgical stress and anesthesia disrupt blood-brain barrier (BBB) integrity through multiple mechanisms, including matrix metalloproteinase 9 (MMP-9) activation, Caveolin-1 upregulation, high mobility group box-1 (HMGB1) release, and systemic inflammation, leading to increased BBB permeability that may facilitate calcium deposition in postoperative stroke patients.⁵

To date, few reports have described the MRI appearance of dystrophic calcifications in cerebral infarcts.² In our patient, the GRE obtained 5 hours after stroke onset showed low signal intensity in the area that became hyperdense on the 10-day follow-up CT. When dystrophic calcification occurs in patients using anticoagulants, it is important to differentiate it from hemorrhagic transformation. Acute intracerebral hemorrhages (ICHs) usually demonstrate attenuation values ranging from 50 to 100 HU, whereas calcifications demonstrate attenuation values of > 100 HU.⁶ In the present patient, the measured attenuation

value far exceeded the typical range for acute hemorrhage, while the progressive temporal evolution—rather than the expected density decrease of a resolving hematoma—strongly favored dystrophic calcification.^{3,7} The cortical ribbon anatomical distribution conforming to infarct margins, absence of mass effect or perilesional edema on serial imaging, and clinical stability without neurological deterioration further distinguished this entity from hemorrhage.^{4,8}

Conclusion

This case underscores the diagnostic challenge and significance of multimodal brain imaging in differentiating dystrophic calcification from hemorrhagic transformation, especially when treating anticoagulated patients. Recognizing temporal evolution, attenuation values, and anatomical distribution—alongside the absence of clinical deterioration—is essential for making appropriate therapeutic decisions.

Conflict of Interests

The authors declare no conflict of interest in this study.

Acknowledgments

None.

References

1. Duncan IC, Sher BJ. Dystrophic calcification in an old cerebral infarction. *S Afr J Radiol* 2003; 7(2): 48-9.
2. Bharath J, Reddy VN, Nagarajan K, Adithan S, Pillai VM. Calcifications in Cerebral Infarcts: Report of Two Cases. *J Stroke Med* 2023; 6(1): 64-8.
3. Ju R, Shi J. Rapid calcification after large area cerebral infarction. *Panminerva Med* 2023; 65(4): 539-40.
4. Parisi J, Place C, Nag S. Calcification in a recent cerebral infarct—radiologic and pathologic correlation. *Can J Neurol Sci* 1988; 15(2): 152-5.
5. Qiu Y, Mo C, Xu S, Chen L, Ye W, Kang Y, et al. Research progress on perioperative blood-brain barrier damage and its potential mechanism. *Front Cell Dev Biol* 2023; 11: 1174043.
6. Morales H. Pitfalls in the Imaging Interpretation of Intracranial Hemorrhage. *Semin Ultrasound CT MR* 2018; 39(5): 457-68.
7. Sener RN. Gyral calcifications detected on the 45th day after cerebral infarction. *Pediatr Radiol* 1993; 23(7): 570-1.
8. Wityk RJ, Lapeyrolerie D, Stein BD. Rapid brain calcification after ischemic stroke. *Ann Intern Med* 1993; 119(6): 490-1.