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PRACTICAL PEARL

Stone Silent

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A 73-year-old right-handed female with an extensive cardiovascular history developed acute apraxia of speech and non-fluent expressive aphasia without limb or facial weakness at the end of her cardiac catheterization and stent placement procedure. The patient did have a large atherosclerotic burden, as evidenced by her advanced coronary artery disease. There was no evidence of cardiac dysrhythmia or resting wall motion abnormalities.

Immediate CT scan of the head demonstrated a rounded 3-mm focus of hyperdensity near the Sylvian point, at the junction of the M2–M3 vascular distribution (Fig. 1a and b). Based on the appearance on the bone window, this density was suspicious for a small calcified embolus. Ensuing MR imaging, performed about 5 h after the initial CT, did not show any evidence of acute infarction, based on diffusion-weighted imaging (DWI) sequences (Fig. 1d). However, the calcified embolus was again visualized on GRE sequences (Fig. 1c). Mechanical retrieval of the embolus was deemed inappropriate secondary to significant risk of vessel perforation. Intra-arterial thrombolysis was deemed ineffective because of the nature of the embolized material.

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Patient was simply placed on intravenous heparin, adjusted to an activated partial thromboplastin time (aPTT) of 40–60 s, and concurrent Clopidrogel therapy (as per institutional post-coronary stent placement guidelines). Within 12 h, patient's symptoms had progressed to include right-sided facial and upper limb weakness. Repeated CT imaging of the head, at the 24-h time-point, demonstrated a 2.2 × 1.4 cm area of low attenuation in the periventricular white matter of the left cerebral hemisphere (Fig. 2a and b). The rounded focus of hyperdensity near the Sylvian point was still present (figure not shown).

Neurological complications of cardiac catheterization have been previously reviewed [1]. Registries of coronary interventions have documented a stroke incidence as high as 0.3%. Possible sources of cardiac catheterization-related cerebral embolism include air introduced during the preparatory phase, a thrombus formed on the catheter or guide wires, dislodged atheromatous material from the aortic arch, and displaced apical thrombus during left ventriculography. Several case reports have suggested the effectiveness of intra-arterial thrombolysis for cerebral strokes during coronary angiography [2]. Unfortunately, therapeutic options in cases of distal-branch calcium fragment embolization remain limited.

We believe that our patient's clinical course correlated with a progressive focal cerebral hypoperfusion, eventually leading to infarction. At the 5-h time-point, irreversible neuronal injury had not yet occurred, as demonstrated by the unremarkable DWI images.

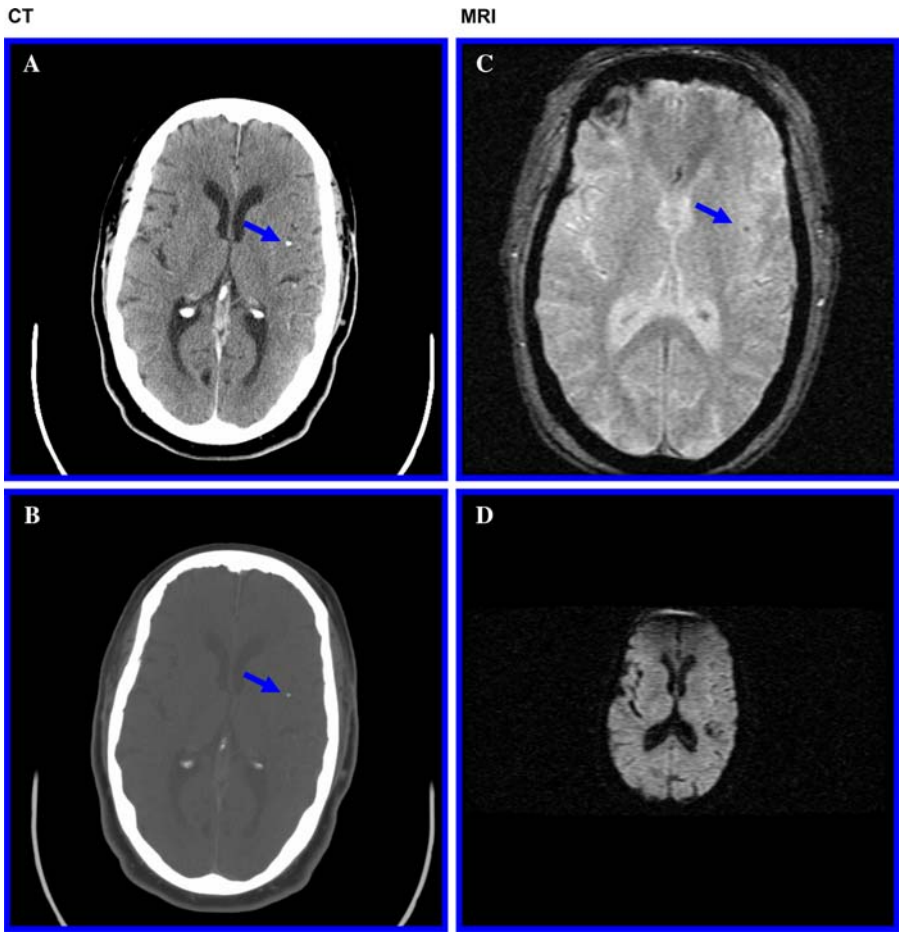

Fig. 1 Post-coronary catheterization CT image of the head demonstrates a rounded 3-mm focus of hyperdensity (arrow) near the Sylvian point, at or near the junction of the M2–M3 vascular distribution (Soft-tissue, **a**; Bone, **b**). MR imaging was performed about 5 h after the initial CT. The gradient echo sequence shows a dark punctate focus corresponding to the embolized material (**c**, arrow). The DWI sequences (**d**) are not suggestive of an ischemic stroke

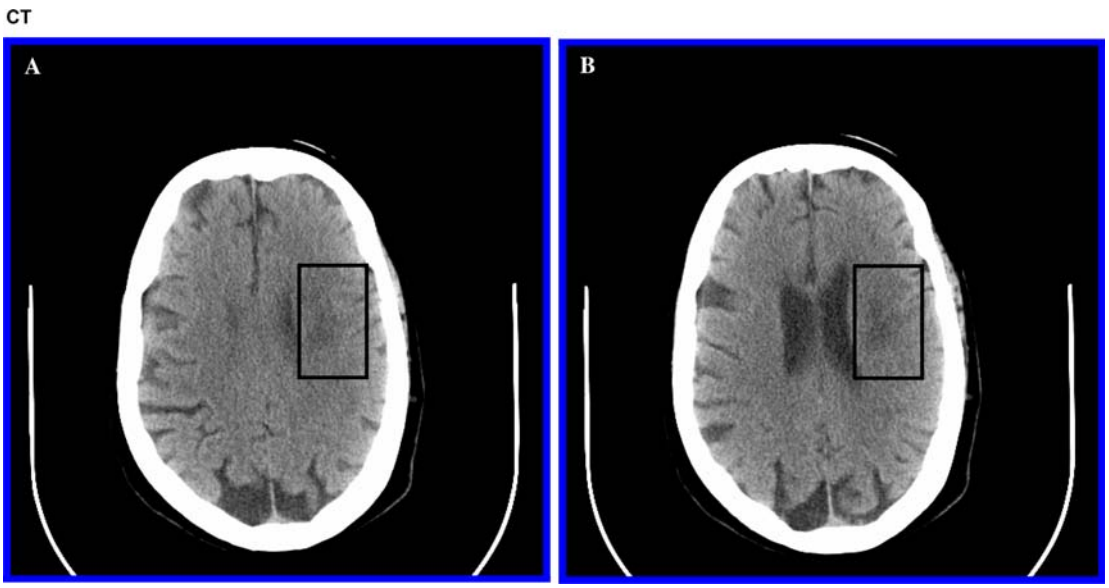

Fig. 2 Follow-up CT image of the head, at the 24-h time-point, demonstrates a new 2.2 × 1.4 cm area of low attenuation in the left periventricular white matter (boxed area)

References

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