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IMAGING HIGHLIGHTS

Significance and fate of hyperdensity on non-contrast CT immediately following intra-arterial thrombolysis

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A non-contrast CT (NCCT) scan is usually obtained after intra-arterial (IA) thrombolysis to assess for possible hemorrhagic complication. Hyperdensity on NCCT following IA thrombolysis can represent either hemorrhage or extravasated iodinated contrast material, and these can be difficult to distinguish.[1] This is illustrated by the following case report.

CASE REPORT

A 73-year-old woman with a history of hypertension and diabetes mellitus was admitted in a local hospital, unequipped to deliver thrombolysis, at about 9:30 AM, after experiencing sudden onset of dizziness and slurred speech at 6:00 AM. NCCT, obtained at 10:00 AM, was normal. Oral aspirin was initiated. At 1:00 PM, the patient developed a stuporous mental state and was eventually transferred to our stroke center at 4:00 pm with fluctuating level of consciousness. NCCT was repeated at 4:25 pm and showed a hyperdense basilar artery (Figure 1A). Diffusion weighted MR images (DWI), obtained at 6:00 PM, revealed multiple areas of restricted diffusion in bilateral paramedian thalami and left cerebellum (Figure 1B, C and D). During this imaging work-up, the Glasgow Coma Scale (GCS) of this patients fluctuated from 5 to 8, and she was intubated because of central respiratory dysfunction.

Because some studies show potential benefit of thrombolysis even up to 24 hours in patients with basilar artery occlusions[2], the emergency stroke team initiated intravenous rtPA thrombolysis, which started at 6:15 PM, after the patient's relatives were informed of the proposed procedure. As the patient did not show any clinical improvement within the next 30 minutes, endovascular treatment was decided. Angiography revealed a complete occlusion of the left vertebral artery, but only irregularity of basilar artery with partial occlusion of the right posterior cerebral artery (PCA) (Figure 2A). 10mg rtPA was administered through intra-arterial catheter at 8:00 pm. Fifteen minutes later, this patient started showing progressive clinical improvement and regained complete consciousness with a National Institutes of Health Stroke Scale (NIHSS) score going down to 4 in a few hours. Angiography

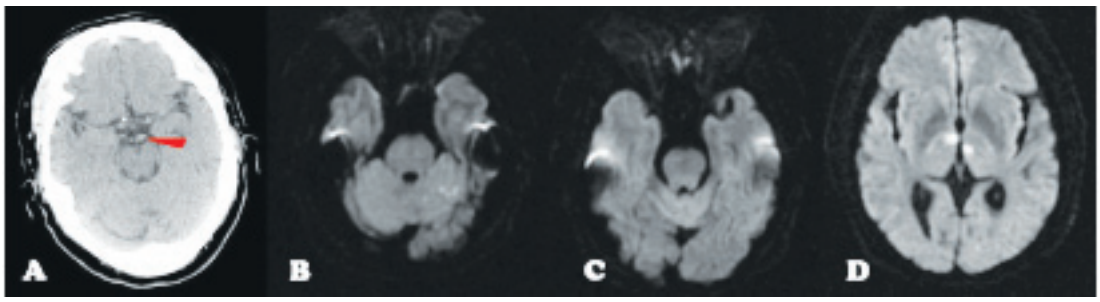

Figure 1.
A: Hyperdense basilar artery (red arrowhead)on NCCT obtained at 10 hours from symptom onset.
B, C and D: Multiple areas of diffusion restriction, or infarcts, in bilateral paramedian thalami and left cerebellum. Please note that no such lesion in seen in the right temporal lobe.

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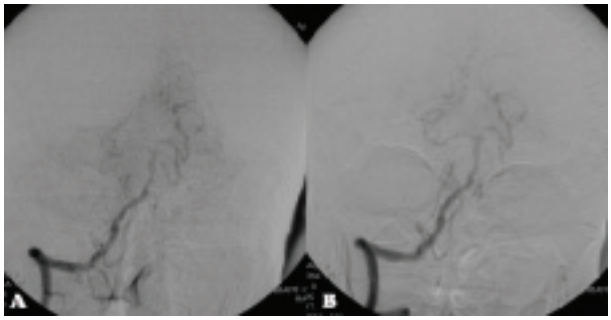

Figure 2.
A: Before IA thrombolysis, basilar artery was irregular and right PCA was partially occluded.
B: After IA thrombolysis, the basilar artery and right PCA are recanalized.

demonstrated improved opacification of the right PCA (Fig. 2B).

A NCCT scan obtained at 9:17 PM, immediately after the endovascular therapy, showed high attenuation in bilateral paramedian thalamic regions and in the right temporal lobe. The Hounsfield units measured in the hyperdense lesions ranged from 70 to 90 (Figure 3A, B and C).

On the next day, the patient's NIHSS was still 4. A follow-up NCCT was obtained at 4:00 PM, about 20 hours after completing the IA thrombolytic therapy. The NCCT demonstrated the disappearance of the hyperdensities previously observed, and the development of hypodensities, in the bilateral paramedian thalamic regions, as well as a severe hemorrhagic transformation in the right temporal lobe (Figure 4A, B). Mannitol was initiated to reduce edema and mass effect of hematoma, while oral aspirin and other antiplatelet were not administered due to the bleeding, until six months later. Nevertheless, the patient's clinical condition continued to improve. Fourteen days later, the patient was discharged with functional independency (NIHSS of 2 and modified Rankin score of 1), but still with mild short and long-term memory dysfunction.

DISCUSSION

NCCT is usually performed after IA thrombolytic therapy for acute ischemic stroke to investigate the possibility of Intracerebral hemorrhage (ICH). However, a NCCT hyperdense lesion in this setting could represent either a hemorrhage or the extravasation of iodinated contrast.[1,3]

Yoon *et al*.[3] distinguished two different types of non-hemohrragic hyperdensities: contrast enhancement and contrast extravasation. Both of them tend to develop in the basal ganglia. Contrast enhancement does not exert mass effect, disappears at 24 hours, and is not associated with intracranial hemorrhage. It results from a damaged blood-brain barrier (BBB) combined with a prolonged angiographic procedure. On the contrary, contrast extravasation shows a density > 90 HU, persists beyond 24 hours, and is caused by an injury to the basal lamina. Contrast extravasation (but not contrast enhancement) is associated with intracranial hemorrhage and

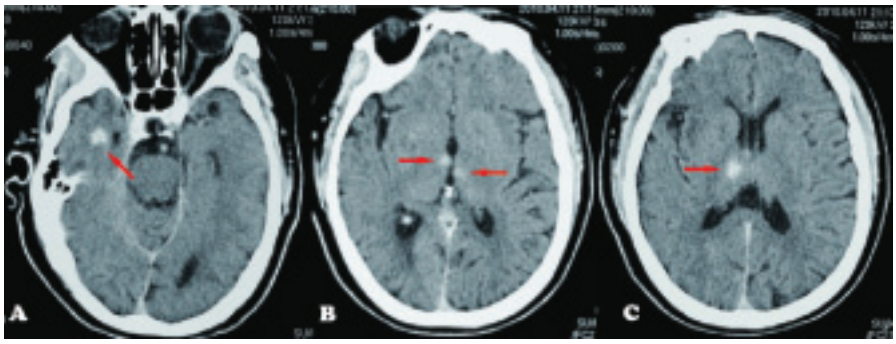

Figure 3.
NCCT performed immediately after IA thrombolysis shows high attenuations (red arrows) in the right temporal lobe **(A)** and in the bilateral paramedian thalami **(B and C)**.

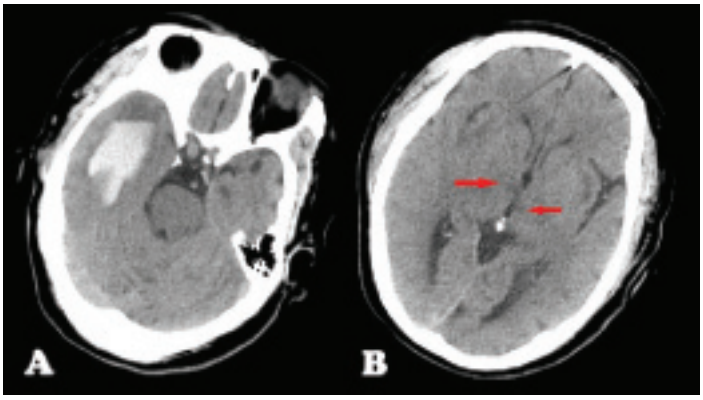

Figure 4.
20 hours after IA thrombolysis, a large hematoma developed in the right temporal lobe **(A)**. The hyperdensities in the bilateral thalami had resolved and were replaced by hypodensities **(B)** .

should therefore be considered as a negative prognostic sign.

In another study, Jang *et al*.[4] classified post-IA hyperdense lesions into four types according to their volume, shape, location and density. (1) cortical high density (HD): a hyperdense lesion that was limited to the cortices. Cortical HD lesions are caused by contrast staining of the already-infarcted cortex. (2) Soft HD: a hyperdense lesion with a maximum CT unit measurement < 80 HU and a small volume without contour bulging. (3) Metallic HD: a hyperdense lesion with a maximum CT unit measurement > 80 HU and a larger volume with bulging contour. It is nearly identical to the lesion described as 'contrast extravasation' above. Although all metallic HD lesion show some degree of hemorrhagic hemorraghic transformation, 70% of them did not resulted in a hematoma exerting mass effect. (4) Diffuse HD: a hyperdense area involving a large vascular territory. In Jang's study, all the patients with diffuse HD experienced significant hemorrhage.

In our patient, the hyperdense lesions in bilateral thalami should be labeled as "contrast enhancement" and "soft HD", according to the Yoon and Jang's classifications, respectively. These hyperdense NCCT lesion match the areas of restricted diffusion on the MRI obtained prior to IA thrombolysis (Figure 1D and Figure 3B). Disruption of BBB associated with acute infarction is the likely cause for these hyperdense lesions, which resolved within 24 hours, and acute hypodense infarct lesions can be detected by NCCT (Figure 4B, indicated by the arrows).

There could be two possibilities about hyperdense lesion developed in the right temporal lobe. Firstly, it can be hemorrhagic transformation after IA thrombolysis, which is hardly differentiated from extravasated contrast, if compared with these enhancements in bilateral thalami. Secondly, it can be labeled as "contrast enhancement" and "cortical HD", due to a new infarction. Although there was no acute infarct lesion on the initial DWI before IA thrombolysis, two hours elapsed between the baseline MRI and the IA treatment, and new areas of infarct may have occurred. If so, based on the Yoon and Jang's classifications, this is an atypical location with hemorrhagic transformation, as usually basal ganglia are affected. But a large hematoma developed at this site on the 20-hour follow-up NCCT (Fig 4A).

In conclusion, it is difficult not only to differentiate between hemorrhage and extravasated contrast on NCCT obtained shortly after IA therapy, but it is also difficult to prognosticate the risk of hemorrhage associated with extravasated contrast.[5] MRI, more specifically susceptibility-weighted and gradient Echo images, can be useful in this setting as it allows to differentiate between blood and extravsated contrast.

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