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Acute Cerebral Infarction Caused by Aortic Dissection: Caution in the Thrombolytic Era

To the Editor:

The National Institute of Neurological Disorders and Stroke (NINDS) Acute Ischemic Stroke Trial¹ has brought treatment of acute ischemic stroke into the thrombolytic era.² The inclusion and exclusion criteria were established to provide guidelines for the safe treatment of ischemic stroke within the first 3 hours.

Case: A 72-year-old woman was noted to drive off the road at 9:45 PM, with her car traveling at approximately 10 miles per hour. When they arrived minutes later, emergency medical services noted that the woman had a left facial droop and left hemiparesis. She arrived at the emergency department 20 minutes later. On initial evaluation, her temperature was 36.1°C, blood pressure 130/80 mm Hg, and pulse 54 and regular. She alerted to voice and could follow simple commands intermittently. She had minimal verbal output, except for perseveration of the word "hello." Cranial nerve examination was significant for a left upper motor neuron facial droop and right gaze preference. She had a left hemiplegia with reduced reflexes on the left and a Babinski sign. Heart auscultation revealed a regular rate and rhythm with no murmurs. Lungs were clear to auscultation bilaterally. Carotid pulses were diminished but present bilaterally, and no bruits were heard. Radial pulses were also present but diminished bilaterally (right greater than left). Femoral and distal pedal pulses were normal. There were no external signs of trauma.

Laboratory studies, including complete blood count, electrolytes, glucose, prothrombin time, and partial thromboplastin time, were within normal limits. An electrocardiogram revealed sinus rhythm at a rate of 52. A head CT without contrast revealed subtle sulcal effacement frontally on the right but no hypodensity or hemorrhage. Intravenous tissue plasminogen activator was considered at this point; however, a chest x-ray revealed a widened superior mediastinum (Figure 1), and intravenous tPA was not given. A CT scan of the chest with and without contrast revealed dilatation of the ascending aorta, with dissection beginning at the lateral aortic root (Figure 2). Only a thin crescent of contrast was seen in the brachiocephalic trunk, with opacification of the right subclavian artery. There was no flow in the left or right common carotid artery.

During evaluation in the emergency department, the patient's heart rate and blood pressure dropped, requiring atropine and fluid resuscitation. She was not considered a surgical candidate for aortic repair secondary to profound neurological deficits. She was admitted for medical management and died the next day.

The inadvertent administration of thrombolytics to patients with aortic dissection has been a concern for cardiologists since the advent of the thrombolytic therapy for acute myocardial infarction. Patients with aortic dissection may present with symptoms or signs consistent with an acute myocardial infarction. When these patients are given intravenous tPA, the majority have poor outcomes.³

The aggressiveness in screening for aortic dissection in patients meeting thrombolytic therapy criteria for myocardial infarction has been debated.⁴ The incidence of myocardial infarction far exceeds that of aortic dissection, and most feel it would be a disservice to delay thrombolytic therapy for a large number of patients who could benefit in order to protect the minority of those with aortic dissection.



Figure 1. Chest x-ray demonstrates a widened superior mediastinum.

A comprehensive review of the literature with a Medline database from 1966 to the present reveals no cases of tPA given intravenously to a person with acute ischemic stroke resulting from aortic dissection. It is likely that similar poor outcomes would occur in ischemic stroke if tPA were administered intravenously to such patients. Not only might intravenous tPA contribute to early death by worsening hemothorax or hemopericardium, but it also may delay potential surgical procedures and interfere with hemostasis. Therefore, it is important to clinically recognize this possibility. Because aortic dissection is a rare cause of ischemic stroke, the aggressiveness of screening for this entity is also questioned. Chest CT scanning and transesophageal echocardiography, while very sensitive for aortic dissection, are time consuming and expensive. Up to 20% of chest x-rays may be negative in patients with aortic dissection⁵; however, this is a relatively simple, quick study that is a routine part of a stroke mechanism evaluation.

An emergent chest x-ray should be considered as part of acute ischemic stroke protocols and should be completed in ischemic stroke patients before administration of intravenous tPA or other thrombolytic therapy. In the unusual setting in which chest x-ray would significantly delay emergent use of thrombolysis, it should be completed when history (chest or back pain or significant trauma immediately before the cerebral infarction) or physical examination (hypotension, reduced peripheral pulses, aortic regurgitation murmur) findings are suggestive of aortic dissection or when communication is impaired.

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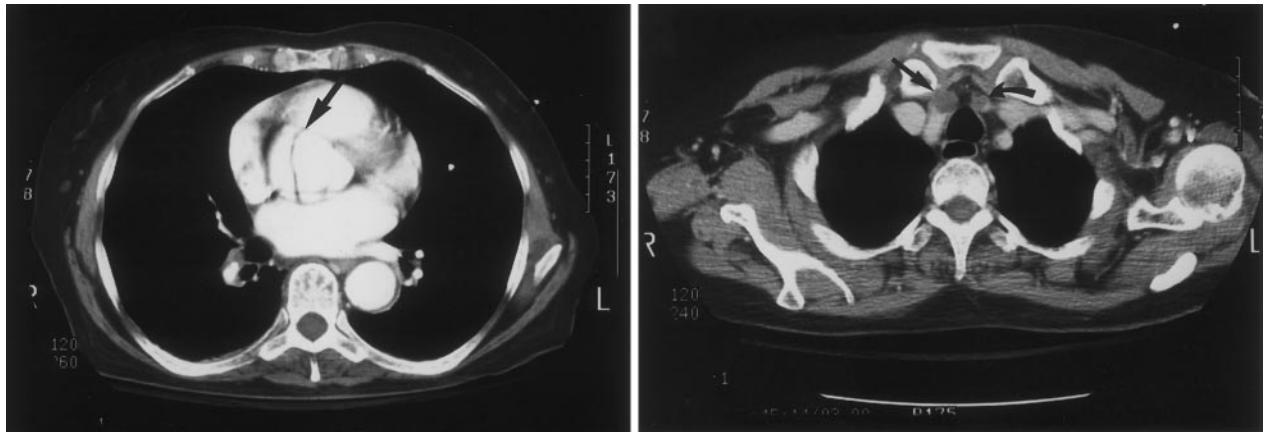


Figure 2. Chest CT with contrast. Left, Scan reveals dilatation of the ascending aorta with dissection beginning at the lateral aortic root (arrow). Right, There is no apparent flow in the right common carotid artery (arrow) or left common carotid artery (curved arrow).

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Diffusion-Weighted MRI in Severe Leukoaraiosis

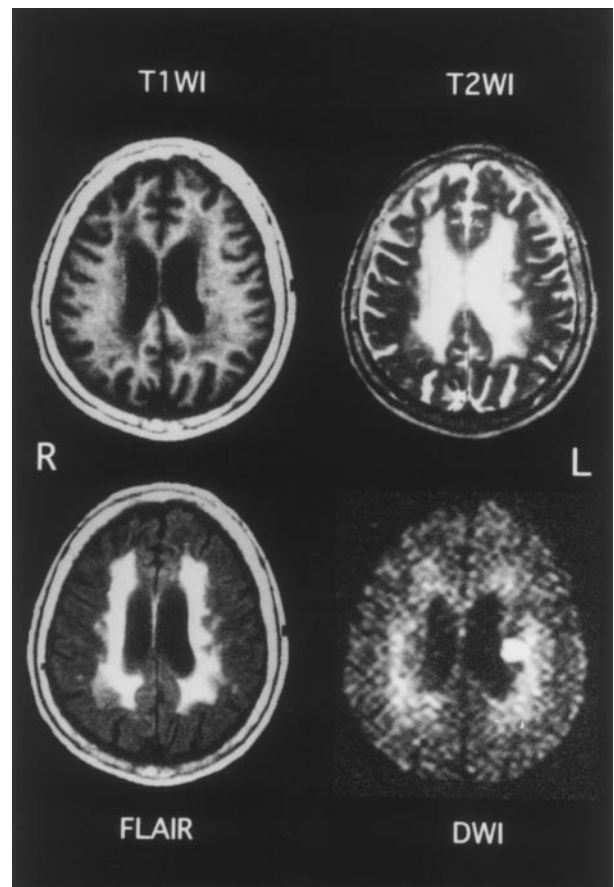
To the Editor:

Recent articles^{1,2} have reported that diffusion-weighted MRI (DWI) is useful in both detecting very early ischemic lesions and identifying the responsible lesion in multiple subcortical lesions. Singer and colleagues¹ described the very high accuracy of DWI for their 39 subjects with acute subcortical infarction. They reported that the sensitivity of DWI for acute subcortical infarction was 94.9% and the specificity was 94.1%. But they did not refer to its relation to the periventricular hyperintensity area (PVH).

We studied 18 cases (10 men and 8 women; mean age, 72 ± 7.9 years) with serial acute cerebral infarction. They were consisted of 3 cases with cortical infarction and 15 cases with subcortical infarctions, including infratentorial regions. MRI was performed with a 1.5-T MR scanner with hardware for echo-planar imaging (Gyrosan ACS-NT, Philips). In all cases, the images were obtained during the same imaging session and at the same slice locations; 6-mm-thick sections without gap and 240-cm field of view were used for all scans. T1-weighted spin-echo (SE) used a 256×256 matrix, TR=360 ms, TE=14 ms, number of excitations=1, and acquisition time=2:16 (min:sec). T2-weighted fast-SE used an echo train length of 11, TR=2000, TE=140, number of excitations=1, and acquisition time=4:03. Fluid-attenuated inversion recovery (FLAIR) imaging was obtained with a fast-SE method with an echo train length of 19, TR=5500, TE=140, number of excitations=1, and acquisition time=3:40. DWI, used in Multishot, SE/echo-planar image sequence, was performed with diffusion sensitivity $b=850 \text{ s/mm}^2$, TR=857, TE=20, number of excitations=1, and acquisition time=1:36. Image analysis was performed by 3 examiners.

The time intervals of imaging relative to onset of ictus ranged from 2 hours to 10 days (mean, 71 hours). Of the 18 patients, 5 underwent imaging <24 hours after onset, 7 from 24 to 72 hours after onset, and 6 at up to 240 hours after onset. Of 5 patients who could be examined within 24 hours, the lesion could be identified in

only 1 with T1WI, in 2 with T2WI, and in 3 with FLAIR. In contrast to these results, DWI revealed all responsible lesions. In patients with multiple subcortical lesions, in 6 of 7 cases the responsible lesion could not be correlated to neurological findings through routine MRI procedures. DWI revealed the responsible lesions in all



MRI of 72-year-old woman admitted to our hospital because of right hemiparesis. MRI was performed 7 days after onset. T1-weighted imaging revealed multiple low-intensity areas around the ventricle, and both T2-weighted imaging and FLAIR showed an area of severe periventricular hyperintensity with suspected multiple high-intensity lesions. DWI showed a high-intensity area that coincided with clinical features on the left corona radiata.

cases. Furthermore, in all 5 cases of severe PVH (so-called Binswanger type), which could not be identified through routine MRI methods, the responsible lesions could be identified by DWI (Figure).

White matter changes, including PVH, are often described as leukoaraiosis, which is defined as bilateral and either patchy or diffuse areas of hypodensity on CT or hyperdensity on T2-weighted MRI. The cause of leukoaraiosis is not fully understood. Clinically, severe leukoaraiosis is reported to relate to cognitive impairment, gait disturbances, and mood disorders.^{3,4} A pathological study of Binswanger's disease revealed that these PVH included lacunar infarct, incomplete infarction, état criblé, perivascular degeneration, and gliosis.⁵ But its clinical course and pathophysiology are unclear, especially in subjects with multiple subcortical lesions and severe PVH.⁴ DWI may provide the information for formation and extension of PVH and elucidate the mechanism of white matter changes to Binswanger's disease in vivo.

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