A 37-year-old man with a history of hypertension presented to the emergency department with acute left-sided weakness and slurred speech within 160 minutes from symptom onset. On examination, he was afebrile, with a blood pressure of 147/75 mm Hg and a heart rate of 88 bpm. Heart auscultation revealed a regular rate and rhythm without any murmurs, and peripheral arterial pulses were symmetrical. Neurological examination disclosed severe left-sided hemiparesis, dysarthria, left-sided hemineglect, left hemisensory loss, and right-gaze deviation. His National Institutes of Health Stroke Scale score was 18 points. Emergent chest x-ray and brain computed tomography were normal. A 12-lead ECG showed sinus rhythm (92 beats) and T-wave inversions in the inferolateral leads. The patient complained of no chest pain, and the blood creatine kinase and troponin levels were within normal range.

The patient was diagnosed with acute ischemic stroke (AIS) and was considered a potential candidate for intravenous thrombolysis. He underwent emergent neurovascular assessment at the bedside (with the use of portable ultrasound equipment and fast-track insonation protocol) as part of our standard stroke treatment protocol. The ultrasound evaluation was performed concurrently with the clinical evaluation before the decision was made to treat the patient with intravenous thrombolysis. Carotid duplex ultrasonography disclosed the presence of an intimal flap and thrombotic material in the right common carotid artery (Figure, panels A through C, and Movie I in the online-only Data Supplement). Left common carotid artery evaluation showed a double-lumen appearance with thrombotic material and residual stenosis (Figure, panels D through F). On the basis of ultrasound findings, an aortic arch dissection (AAD) extend-
According to both common carotid arteries was suspected, and emergent chest computed tomography/angiography was performed, confirming the diagnosis of type A AAD (Figure, panels G and H). Consequently, administration of intravenous tissue plasminogen activator was withheld, and the patient was transferred to the cardiothoracic intensive care unit, where an emergent surgical repair was planned.

Intravenous thrombolysis is the only approved treatment for AIS within 4.5 hours from symptom onset. However, because of the narrow time window, the underlying stroke pathogenesis may not be investigated, and therefore careful selection of appropriate candidates may not be performed. AAD may present with predominant neurological symptoms of acute cerebral ischemia without the typical appearance of chest pain, hypotension, and absent peripheral pulses. Patients with AAD mimicking acute myocardial infarction who have been inadvertently treated with intravenous tissue plasminogen activator have suffered dire complications, including extension of dissection into the pericardium, leading to cardiac tamponade and death. Moreover, the high risk of aortic arch or ascending aorta rupture constitutes AAD as an absolute contraindication for thrombolysis in patients with myocardial infarction. The experience with thrombolytic treatment in patients with AIS caused by AAD is limited, and underscores the dismal outcome of these patients when they are accidentally selected for tissue plasminogen activator infusion (one patient died as a result of cardiac tamponade and intrapleural hemorrhage, and the other one developed a fatal symptomatic intracranial hemorrhage).

The present case highlights the importance of concurrent, noninvasive vascular evaluation in identifying the appropriate AIS candidates for intravenous thrombolysis. Despite the fact that vessel imaging is not required for thrombolytic therapy, ultraearly ultrasound evaluation at the bedside (simultaneously with the clinical evaluation) of AIS patients was instrumental in the timely diagnosis of AAD and prevention of the inadvertent administration of intravenous tissue plasminogen activator in both our patient and a similar case evaluated in a tertiary-care, high-volume stroke center. As the number of AIS patients receiving thrombolytic therapy increases in exponential fashion, AAD manifesting with symptoms of acute cerebral ischemia presents a unique diagnostic challenge to stroke physicians that may be aptly managed with the use of emergent neurosonology evaluation.

Disclosures

None.

References

Aortic Arch Dissection Causing Acute Cerebral Ischemia: An Uncommon Contraindication for Intravenous Thrombolysis

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_Circulation_. 2011;124:657-658
doi: 10.1161/CIRCULATIONAHA.111.033308

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/124/5/657

Data Supplement (unedited) at:
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