Hidden Telltale Signs in Hyperacute Ischemic Stroke Caused by Aortic Dissection

A Case Report and Post Hoc Analysis

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Introduction: Rapid identification of hidden telltale signs in hyperacute ischemic stroke caused by aortic dissection (AD) is challenging, mainly owing to the narrow time window for bridging therapy.

Case Report: A 63-year-old man was referred for sudden right-side weakness accompanied by a decreased level of consciousness for almost 1 hour and 37 minutes. He had a history of hypertension. His skin was clammy, and on physical examination, there was involuntary chest thumping in the left upper limb. Hyperacute cerebral infarction was considered after no bleeding was observed on emergency head computed tomography, and intravenous thrombolysis with alteplase was administered immediately after. The patient was then taken to the catheter room, ready for endovascular thrombectomy. Stanford type A AD was found by cerebral angiography before endovascular thrombectomy. The infusion of alteplase was stopped immediately during cerebral angiography, but the patient’s blood pressure, heart rate, and blood oxygen were still declining progressively, and the degree of consciousness disturbance deepened. The patient died after the combined but failed rescue attempts of multiple departments.

Conclusion: Hyperacute ischemic stroke caused by AD often hides some telltale signs. Clinicians should master basic clinical skills to exclude AD by looking for these telltale signs hidden in hyperacute ischemic stroke to avoid the fatal consequences of intravenous thrombolysis and/or cerebral angiography within the narrow window of time.

Key Words: aortic dissection, hyperacute ischemic stroke, intravenous thrombolysis, endovascular thrombectomy, cerebral angiography

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At present, the timely opening of occluded blood vessels is an internationally recognized method for the treatment of hyperacute ischemic stroke, including intravenous thrombolysis (IVT) and endovascular thrombectomy (EVT). IVT combined with subsequent EVT is known as bridging therapy. The major complication of IVT is hemorrhage, which can be fatal in patients with acute ischemic stroke (AIS) secondary to aortic dissection (AD). Current guidelines specifically state that AIS secondary to AD is a contraindication for IVT.1,2 However, it has been reported that IVT with alteplase is safe and beneficial for some of these patients.3–5 Cerebral angiography is necessary before performing EVT but is contraindicated for Stanford type A AD, even if it is not specified in the current guidelines. In China, not all hospitals can conduct AD screening for hyperacute ischemic stroke in a timely manner. IVT and cerebral angiography within the time window may lead to medical accidents and disputes. Effectively regulating such medical behavior is a problem we are facing. We are reporting a case with hyperacute ischemic stroke caused by AD on whom IVT and cerebral angiography were performed, and we also performed a post hoc analysis.

CASE PRESENTATION

A 63-year-old man was admitted to the emergency department of our hospital with sudden right-side weakness accompanied by a decreased level of consciousness for almost 1 hour and 37 minutes. At the time of onset, his companion found him sweating and presenting consciousness disturbance of almost 1 hour and 37 minutes. At that time, he was sent to the catheter room to prepare for bridging therapy [door to puncture time (DPT), 56 min]. Aortic arch angiography found narrowed endoluminal bands on the brachiocephalic trunk, the left common carotid artery (CCA), and the left subclavian artery (SCA) (Fig. 1A). Although the left vertebral artery was well developed seen from Figure 1A, after careful observation, a large rupture was found in the false lumen at the beginning of the left SCA, which ruptured into the pleural cavity (Fig. 1B). On the left internal carotid artery, a narrowed endoluminal band was found at the initial part with slow blood flow and only arterial phase imaging (Fig. 2). Abdominal angiography found that the right renal artery and superior mesenteric artery originated from the true lumen, but the lower abdominal aorta was occlusive due to dissection, which resulted in occlusion of the left renal artery and bilateral iliac arteries (including common iliac, external iliac, and internal iliac arteries) without imaging (Fig. 3). Stanford type A AD was finally confirmed, and the infusion of alteplase was stopped immediately.
during cerebral angiography. However, the patient’s blood pressure, heart rate, and blood oxygen were still found to be decreasing progressively, and the degree of consciousness disturbance deepened. The patient died after the combined but failed rescue attempts of multiple departments.

**DISCUSSION AND POST HOC ANALYSIS**

The incidence of AD is 2.53/100,000 in the general population, and it has a mortality rate of >50%.6–10 AD is classified as Stanford type A if the ascending aorta is involved and type B if not.11 Hypertension and atherosclerosis are the main causes of AD.12 The main pathological manifestations of AD are intimal flaps, cystic medial necrosis, and disruption to the tunica media.12 If AD involves the cerebral artery and results in mechanical obstruction or occlusion of the vessel, AIS may be the first manifestation.11 About 0.5% to 16% of Stanford type A AD patients show AIS as the primary presentation, and the most important clinical presentation of such patients is decreased consciousness (about 75%), which is higher than chest pain and low systolic BP.6–9

We performed a post hoc analysis of this case and found that AD should have been highly suspected upon admission. The classification of AD in our case was Stanford type A due to the involvement of the ascending aorta (in fact, the descending aorta was also involved), in accordance with the Stanford classification system.11 In terms of medical history and clinical manifestations, the patient developed the disease during activities. Although the patient could not complain of chest pain due to consciousness disturbance, he presented sweating at the onset of the disease, and the left upper limb exhibited the chest beating movement. We also found some valuable clues through physical examination: a lower body temperature, clammy skin, and asymmetry of branchial systolic BP (although <20 mm Hg). There was also the omission of checking aortic imaging, such as CT or CT angiography (CTA) or bedside duplex ultrasonography, on admission, but cerebral angiography was directly performed after IVT. The probable cause of death in this case was cardiac tamponade caused by the hematoma of the AD extending into the pericardium and rupture and hemorrhage of the left SCA dissection. IVT and cerebral angiography may have contributed to dissection extension and further rupture by interfering with hematoma formation in the intimal layer.

Most patients with AIS secondary to Stanford type A AD present with left-limb hemiplegia because the right supra-aortic vessels (including brachiocephalic trunk and right CCA) are closer to the ascending aorta or the origin of the dissection, so their intima can easily be torn under pressure from the hydraulic stress directly from the heart.7,9 Why did this case present with right-limb hemiplegia rather than left? First, the AD in this case

**FIGURE 1.** Aortic arch angiography. (A) Aortic dissection originates from the ascending aorta and involves the brachiocephalic trunk (blue arrow), the left common carotid artery (yellow arrow), and the left subclavian artery (white arrow). Aortic arch angiography. (B) Although the left vertebral artery was well developed seen from (A), after careful observation, a large rupture was found in the false lumen at the beginning of the left subclavian artery, which ruptured into the pleural cavity (red arrows), possibly causing massive hematocoele in the left pleural cavity.

**FIGURE 2.** Left carotid angiography: aortic dissection involved the left common carotid artery to the beginning of the left internal carotid artery (red arrow), with slow blood flow and only arterial phase imaging, suggesting significant hypoperfusion.
or obscured by unconsciousness or dysphasia. Although IVT might be effective in thromboembolic mechanism and there have been many successful reports. IVT is still contraindicated for AIS secondary to AD. The hydraulic stress during cerebral angiography, especially aortic arch angiography, may aggravate the extent of the intimal tear. Although there are no guidelines on whether cerebral angiography before EVT is contraindicated for AIS secondary to AD, we believe that it is also contraindicated for such patients. Therefore, in the era of strict control of DNT and DPT, rapid identification of AIS secondary to AD is particularly important.

Although auxiliary examinations, such as chest x-ray, aortic CTA, or bedside duplex ultrasonography, were not performed, the medical history, clinical manifestations, and physical examination could strongly support a diagnosis of AD, as mentioned above. These auxiliary examinations for screening AD also have their own disadvantages and cannot be completely relied on. Only some patients with AD were found to have mediastinal widening on chest x-ray, and it was not recommended because of the obvious delay in DNT. Bed-side duplex ultrasonography is indeed the fastest and simplest screening tool for AD, but it depends heavily on the experience of the diagnosing physician. As in our case, with Stanford type A AD, cerebral arteries sometimes could not be reconstructed by CTA due to cerebral artery hypoperfusion, thus, CTA seems to be less sensitive in such patients. Therefore, consistent with previous findings, we believe that pre-thrombolytic vascular imaging examination is not necessary in nonhighly suspected cases. During the current period of COVID-19 prevention and control, chest CT (which takes <10 seconds) can detect partial AD while excluding lung lesions. Following the occurrence of this case, we have routinely performed head CT in addition to chest CT in all emergency stroke patients, and we have also screened out a few cases of AD. Maybe it is worth recommending. Stanford type A AD is clearly not appropriate for cerebral angiography because high pressure injection during angiography can aggravate the tear of the dissection. Imaging studies should be performed on admission only for cases strongly suspected of AD before bridging therapy.

Some telltale signs are often hidden in hyperacute ischemic stroke caused by AD. Rapid identification of hyperacute ischemic stroke caused by AD could avoid IVT and cerebral angiography in the era of strict control of DNT and DPT. Repeated history-taking and physical examination rather than auxiliary examinations were especially important for the early recognition and management of AD, especially in hospitals with inadequate auxiliary inspection equipment. AD is highly suspected if there is a history of hypertension plus the following telltale signs: chest/back pain, convulsion, mild dyspnea, decreased blood pressure that cannot be improved with volume and dopa, reduced peripheral pulse and/or diastolic murmur, wet and cold skin, asymmetry of branchial systolic BP (not necessarily >20 mm Hg), carotidbruits, consciousness disturbance, or syncope caused by cerebral hypoperfusion. Rapid identification of telltale signs hidden in hyperacute ischemic stroke caused by AD contributes to discovering the vast majority of such cases.

BP-elevating with volume and dopa in such patients is controversial. Low pressure may lead to cerebral hypoperfusion, and prolonged cerebral hypoperfusion may lead to expansion of the infarct. However, patients with AD need to maintain a low pressure level to avoid further laceration of AD. As in our report, the patient was unconscious due to hypoxic brain ischemia caused by hypoperfusion, but BP-elevating actually made the patient worse on the surgical table in the catheter room. Surgery is the gold standard treatment and can reduce the mortality of AD, so rapid identification and referral is particularly important for hospitals that do not have a thoracic surgery department like ours.

Our case report highlights the importance of mastering basic clinical skills in the rapid identification of telltale signs in hyperacute ischemic strokes caused by AD. We also strongly recommend that future guidelines include cerebral angiography as a contraindication for Stanford type A AD along with IVT.
REFERENCES
1. Toyoda K, Koga M, Iguchi Y, et al. Guidelines for intravenous thrombolysis (recombinant tissue-type plasminogen activator): the third edition. March 2019: a guideline from the Japan Stroke Society. Neurrol Med Chir. 2019;59:449–491.
2. Powers W, Rabinstein A, Ackerson T, et al. Guidelines for the early management of patients with acute ischemic stroke: 2018 update to the 2013 guidelines for the early management of acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke. 2019;50:e344–e418.
3. Cao L, Huang X, Zui F. Successful intravenous thrombolysis for acute ischemic stroke caused by aortic dissection with severe hypofibrinogenemia: a case report and literature review. Int J Neurosci. 2021:1–7.
4. Hong K, Park S, Whang S, et al. Intravenous recombinant tissue plasminogen activator thrombolysis in a patient with acute ischemic stroke secondary to aortic dissection. J Clin Neurol (Seoul, Korea). 2009;5:49–52.
5. Malek E, Nasreddine W, Makki A. Successful thrombolysis in the setting of Marfan syndrome and chronic aortic dissection. Neurologist. 2019;24:183–184.
6. Gaul C, Dietrich W, Friedlich I, et al. Neurological symptoms in type A aortic dissections. Stroke. 2007;38:292–297.
7. Guglielmi V, Groeneveld N, Posthuma L, et al. Aortic dissection masquerading as a code stroke: a single-centre cohort study. Eur Stroke J. 2020;5:56–62.
8. Ohara T, Koga M, Tokuda N, et al. Rapid identification of type A aortic dissection as a cause of acute ischemic stroke. J Stroke Cerebrovasc Dis. 2016;25:1901–1906.
9. Lee S, Kim J, Na C, et al. Eleven years of experience with the neurologic complications in Korean patients with acute aortic dissection: a retrospective study. BMC Neurol. 2013;13:46.
10. Melvinsdottir I, Lind S, Agnarsson B, et al. The incidence and mortality of acute thoracic aortic dissection: results from a whole nation study. Eur J Cardio-thorac Surg. 2016;50:1111–1117.
11. Fukui T. Management of acute aortic dissection and thoracic aortic rupture. J Intensive Care. 2018;6:15.