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【 CASE REPORT 】

Successful Delayed Aortic Surgery for a Patient with Ischemic Stroke Secondary to Aortic Dissection

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Abstract:
The diagnosis of aortic dissection (AD) is sometimes difficult within the limited time window of recombinant tissue plasminogen activator (tPA) for ischemic stroke (IS). A 60-year-old man developed sudden left hemiparesis due to IS. During tPA infusion, his blood pressure dropped and consciousness declined. After transfer to our hospital, carotid duplex ultrasonography led to a diagnosis of AD. Emergency surgery was postponed because of the risk of hemorrhagic transformation. The patient successfully underwent aortic surgery on day 5 and was discharged with a remarkable improvement in his symptoms. Delayed surgery may avoid hemorrhagic transformation in patients with AD-induced IS who have received tPA.

Key words: aortic dissection, hemorrhagic transformation, ischemic stroke, recombinant tissue plasminogen activator

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Introduction

Aortic dissection (AD) is a life-threatening emergency in which patients usually present with sudden severe chest and back pain sometimes accompanied by neurological symptoms such as ischemic stroke (IS). Recombinant tissue plasminogen activator (tPA) is approved for the treatment of acute IS within 4.5 hours of onset. The eligibility for tPA treatment according to the current guidelines in the US, Europe, Canada, and Australia (1-4) is based on the criteria adapted from National Institute of Neurological Disorders and Stroke (NINDS) rt-PA Stroke Study (5). In that study, patients who have arterial puncture at a noncompressible site are not indicated to receive this treatment. Previous reports strongly suggest that AD should be considered as a cause of IS before using tPA (6-11). According to the current Japanese guidelines, if a patient with IS is diagnosed with AD, tPA should not be administered (12). However, a precise diagnosis of AD is sometimes difficult to achieve within the narrow time window of tPA administration because the patients sometimes lack typical medical histories or findings. If tPA is misused in patients with AD-induced IS, emergency aortic surgery may be required (9-11). However, several reports have recommended delaying surgery until the patient's neurologic condition stabilizes because of concerns regarding hemorrhagic transformation (HT) with high-dose heparin for extracorporeal circulation (13-16). We herein report a case involving a patient with AD-induced IS who was treated with tPA and underwent delayed surgical treatment with a remarkable outcome.

Case Report

A 60-year-old man developed sudden left hemiparesis and visited a nearby hospital 60 minutes after onset. On neurologic examination, he was awake exhibited left hemiparesis and left facial palsy, and had a blood pressure (BP) of 128/64 mmHg, pulse of 66 bpm, body weight of 86 kg, and National Institute of Health Stroke Scale (NIHSS) score of

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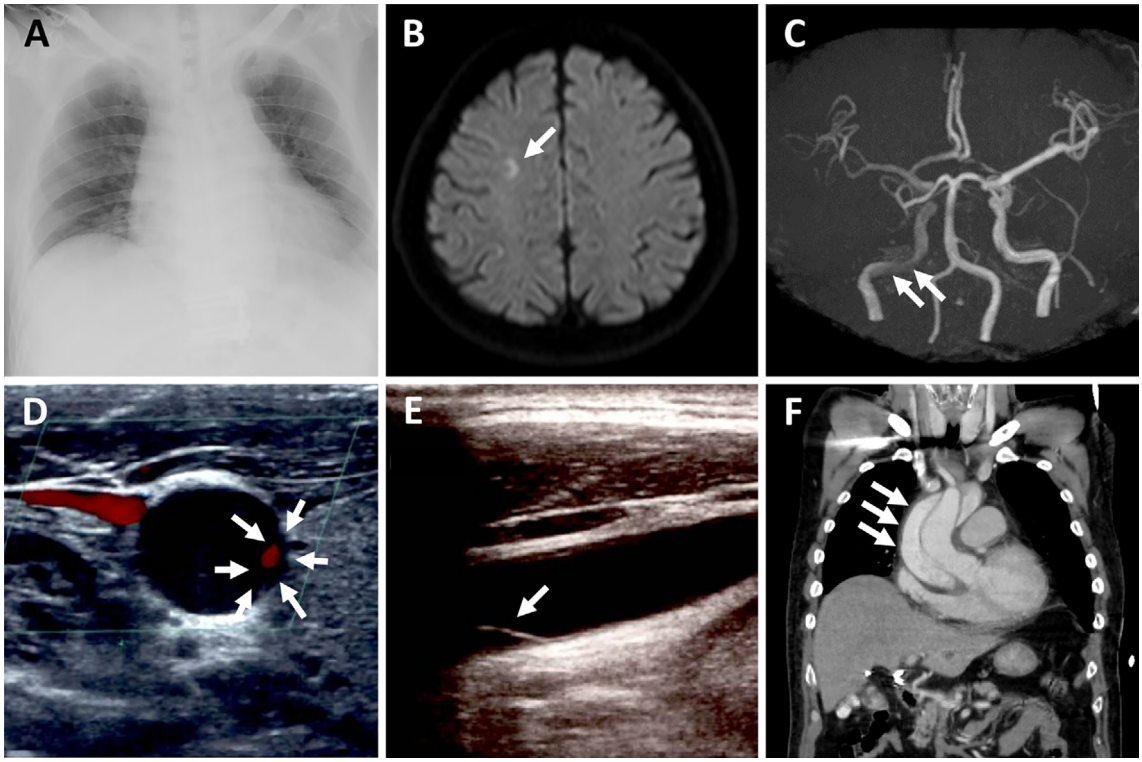

Figure. **(A) A supine chest X-ray (anteroposterior view) showed no abnormal findings, including no enlargement of the mediastinum. (B) Brain magnetic resonance imaging revealed a right frontoparietal ischemic lesion (arrow). (C) Brain magnetic resonance angiography demonstrated a low signal in the right internal carotid artery (arrows). (D) Carotid duplex ultrasonography demonstrated 95% occlusion of the right common carotid artery with a large false lumen and small true lumen (arrows), and a dissecting intima was present in the left internal carotid artery (arrow, E). (F) Chest computed tomography revealed a Stanford type A ascending aortic dissection extending to the aortic arch (arrows).**

2. A supine chest X-ray (Figure A) and electrocardiogram were normal, and general serum tests were unremarkable. Brain magnetic resonance imaging revealed a right watershed infarct located on the border of the anterior cerebral artery and middle cerebral artery territories (Figure B, arrow). Brain magnetic resonance angiography demonstrated a low signal in the right internal carotid artery (Figure C, arrows). Based on the diagnosis of IS, infusion of tPA was started at a dose of 0.6 mg/kg (total, 51.6 mg) 137 minutes after onset. During the tPA infusion, the patient's BP dropped to 72/53 mmHg, consciousness declined, and left hemiparesis progressed. Thus, the tPA was stopped after infusing 75% of the total dose (38.7 mg), and he was emergently transferred to our hospital.

On admission to our hospital, the patient's NIHSS score worsened to 26 with a Glasgow coma scale score of E2V2M5. His BP was 75/50 mmHg in his right arm but 140/80 mmHg in his left arm. Repeated interviews revealed slight right shoulder pain before his consciousness decline. He was temporally intubated and ventilated. Carotid duplex ultrasonography demonstrated 95% occlusion of the right common carotid artery with a large false lumen and small true lumen (Figure D, arrows), and a dissecting intima was present in the left internal carotid artery (Figure E, arrow). Chest computed tomography revealed a Stanford type A ascending AD extending to the aortic arch (Figure F, arrows). Emergency surgery was postponed due to concerns regarding the risk of HT induced by high-dose heparin for extracorporeal circulation. With conventional medical treatment, the patient was removed from the ventilator with a BP of 109/42 mmHg and Glasgow coma scale score of E4V5M6 on admission day 2. He underwent open surgery for reconstruction of the ascending aorta and proximal aortic arch on day 5. After the establishment of full cardiopulmonary bypass with 24,000 units of unfractionated heparin, aortic cross-clamping was achieved and deep hypothermic circulatory arrest was induced. The intimal and medial layers of the false lumen were sealed, and the proximal end of the ascending aorta and proximal aortic arch (between the innominate artery and left common carotid artery) were reconnected by a graft. His BP was maintained at 100-120/50-60 during the surgery. The surgical duration was 9 hours, and he recovered with clear consciousness. Follow-up brain computed tomography on day 10 showed no intracranial hemorrhage. On day 46, he was transferred to a rehabilitation hospital with a remarkable improvement in his NIHSS score from 26 to 1. At the 3-month follow-up, the patient was living at home and able to carry out all usual activities

despite slight muscle weakness in the left hand with a modified Rankin scale score of 1.

Discussion

The eligibility for tPA treatment at 0.9 mg/kg according to the current guidelines in the US, Europe, Canada, and Australia (1-4) is based on the selection criteria from NINDS rt-PA Stroke Study (5). In the study, patients who have arterial puncture at a noncompressible site are not indicated for this treatment. According to the current Japanese 2012 guidelines, a diagnosis of AD should be excluded before administering tPA at 0.6 mg/kg (12). The misuse of tPA to treat IS due to AD may have catastrophic consequences. There are several reports of IS patients receiving tPA with unrecognized AD who took a sudden turn for the worse after tPA, leading to death (6, 7, 17). However, the treatment of AD-induced IS is challenging in the era of tPA because the diagnosis of AD is sometimes difficult within the limited time window (up to 4.5 hours) of stroke onset. One-third of patients with AD who develop neurological symptoms do not report typical pain at onset (18), as was found in the present case at the nearby hospital. Because it is difficult to conduct all diagnostic tests for AD in every patient who is eligible for tPA, clinical suspicion and physical examination findings are important, especially in patients with a consciousness decline. In the present case, an examination of the bilateral BP at a local hospital may have led to a faster diagnosis, but repeated history-taking, physical examination, and careful conduction of carotid duplex ultrasonography led to a definitive diagnosis after transfer to our hospital.

Previous reports have described eight cases of tPA therapy for AD-induced IS. Three of these patients died without aortic surgery (6, 7, 17), and the remaining five survived after aortic surgery (8-11, 19). Of the five survivors, four underwent emergency surgery on the same day as IS onset (8-11), and one underwent surgery on day 3 because of complications of jugular vein thrombosis and arrhythmia (19). Surgery is the gold standard treatment for AD, but it may be associated with high morbidity and mortality in patients with AD-induced IS who have received tPA. Stroke is not a contraindication for emergent surgery to treat acute type A and B ADs, and the former should be done as soon as possible in all patients except those with profound or evolving neurological deficits (20-24). Because the possibility of HT is a concern when administering high-dose heparin for extracorporeal circulation, several reports have suggested delaying surgery until the patient's neurologic condition stabilizes (13-15). Fukuda et al. (16) reported good results in some patients with brain edema due to AD-induced IS by delayed surgery. In the present case, we delayed performing aortic surgery because of concerns over the risk of HT, and the patient successfully underwent reconstruction of the ascending aorta without any neurological complications.

In conclusion, the present case suggests that AD should be carefully considered as a cause of IS before administrating tPA. The misuse of tPA to treat IS patients due to AD can lead to a very poor outcome. We delayed the aortic surgery for 5 days until the risk of HT was reduced. The patient successfully underwent the aortic surgery and was discharged with a remarkable improvement in his neurologic manifestations. Delayed surgery could therefore be an alternative choice to avoid HT in patients with AD-induced IS who have received tPA.

The authors state that they have no Conflict of Interest (COI).

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