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Case Report

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Aortic dissection with cerebral infarction

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Abstract

Aortic dissection is a fatal cardiovascular health problem. Chest and back pain are among the common complaints of the patients, and they may also apply with atypical clinics. It is very difficult to diagnose with examination and anamnesis, especially in patients who present with poor consciousness and stroke symptoms. In this study, we wanted to present a 62-year-old female patient who had syncope at home and was unconscious for about 1 hour, has stroke symptoms, and aortic dissection was detected in her examinations. When no hemorrhage was detected in non-contrast brain CT, neck and brain contrast-enhanced CT angiography imaging was performed. Aortic dissection flap extending to the right carotid communis was detected in the imaging. Clinicians should pay attention to detailed examination and be alert for further examinations in order not to harm the patient in terms of underlying causes, especially in unconscious patients who have a stroke clinic and cannot express their complaints in a healthy way.

Keywords: stanford type A, stroke, syncope, emergency

1. Introduction

Aortic dissection (AoD) is a fatal aortic disease and prompt diagnosis is essential for prompt surgical intervention and good patient outcomes. Mortality of Stanford type A AoD is greater than 50% if early surgical intervention is not performed. Especially in Stanford type A AoD, the ascending aorta is affected and sometimes accompanies acute ischemic stroke. Patients with ischemic stroke as a complication of Stanford type A dissection; often do not complain of chest or back pain, possibly due to impaired consciousness, amnesia, or aphasia. It has a fatal course following inappropriate intravenous rt-PA therapy, and delayed proper surgical treatment also increases mortality (1). The latest guidelines indicate AoD as a contraindication to the use of intravenous thrombolytics for acute cerebral infarction (CI). A case of AoD with a rare CI occurring at our emergency department has been analyzed and summarized. This case will serve as a reference for the management of similar patients.

2. Case Report

A 62-year-old female patient was admitted to the emergency service by ambulance after fainting at home and unconscious for about 1 hour. The patient's Glasgow Coma Score was 5 and other vital parameters were normal, and direct and indirect light reflexes could not be detected in the neurological examination. Left Babinski reflex was positive, right upper and left lower extremity distal pulses could not be palpated. There were known diagnoses of hypertension, diabetes mellitus and atrial fibrillation. Rapid serial intubation was performed to maintain cardiopulmonary stability. Brain CT imaging was performed to detect central events.

Blood tests showed C-reactive protein 3.6 mg/L, D-dimer 19.6 mg/L, white blood cells 9.69 × 103/uL, red blood cells 3.98 × 106/uL, and hemoglobin 3.98 g/L. When no hemorrhage was detected in non-contrast brain CT (Fig. 1a) neck and brain contrast-enhanced CT angiography imaging was performed. It was observed that there was no blood supply in the right cerebral hemisphere and decreased blood supply in the left cerebral hemisphere (Fig.1b). AoD flap extending to the right carotid communis was detected in the imaging (Fig. 2ab). The ascending aorta of the patient, whose echocardiography was performed, was measured 54 mm and a dissection flap was seen. The patient was taken to intensive care follow-up for further examination and treatment. He died after 15 hours of intensive care follow-up.

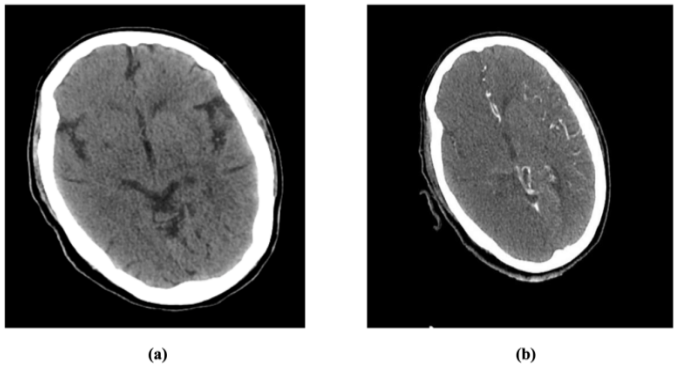

Fig. 1. Non-contrast brain CT (a), Lack of contrast material passage in the right cerebral hemisphere (b)

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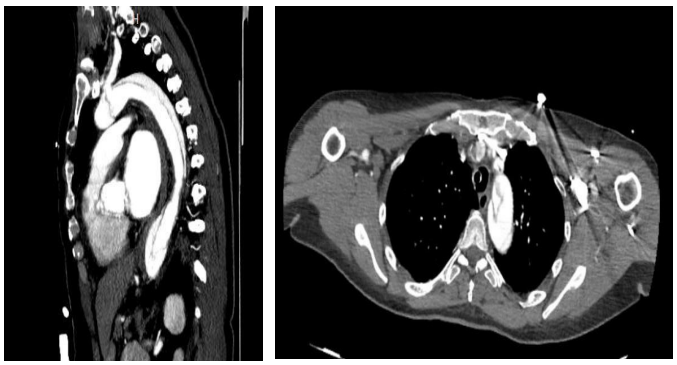

Fig. 2. Stanford type A dissection (a), dissection flap starting from the brachiocephalic artery and extending to the right common carotid artery (b)

3. Discussion

The most frequent causes of AoD are hypertension, atherosclerosis, trauma, Marfan syndrome, arteritis, and pregnancy. This patient had there was a history of only hypertension. Clinical symptoms are intimately related to pathophysiological development. The most classic clinical symptom in AoD is chest or back pain. However, only about half of the patients with CI have such a complaint (chest or back pain), likely due to the presence of amnesia or cortical symptoms such as aphasia and/or impaired consciousness (1,2). Some auxiliary examinations can also provide remarkable clinical information. If symptoms such as unexplained hypotension, shortness of breath, chest discomfort, asymmetry of blood pressure between the arms, loss of consciousness, and cold limbs are present, aortic CT angiography and echocardiography should be added to determine if AoD is present. The guidelines have suggested intravenous thrombolysis as the top level of therapy for CI within the time window. If a patient has AoD, intravenous thrombolysis can have catastrophic results. For example, new embolic events caused by the disintegration of emboli in the dissection, pericardial effusion, tamponade due to aortic rupture, and the delay of life-saving surgery. Published reports on aortic steering warnings in intravenous thrombolytic therapy emphasize the importance of keeping in mind the potential existence of AoD as a cause of ischemic stroke (3). Depending on the extent of involvement of AoD, it can cause various organ disorders and clinical symptoms. Approximately 6% of patients experience a transient ischemic attack (TIA) due to accompanying ischemic stroke or spread to the cervicocerebral arteries or embolism from the dissection site (4, 5). A study of 1637 consecutive patients with suspected stroke found a total of 5 patients with ischemic stroke as a complication of type A aortic dissection (1). AoD patients need to preserve low blood pressure to prevent further tearing. In contrast, low blood pressure will decrease stroke hemisphere perfusion and then grow the CI (6). The presence of ischemic stroke has been accepted as an indicator of poor prognosis (7,8). In addition, it has been reported that with early diagnosis of AoD and appropriate surgical treatment, accompanying neurological symptoms are not associated with increased mortality (2,9,10). Patients may present with a combination of various neurological manifestations such as hemiplegia, impaired consciousness, syncope, convulsions, amnesia, spinal cord ischemia, and peripheral neuropathy (11). Clinicians should pay attention to detailed examination and be alert for further examinations in order not to harm the patient in terms of underlying causes, especially in unconscious patients who have a stroke clinic and cannot express their complaints in a healthy way.

Today, due to intensive care occupancy, the first treatment of most ischemic stroke patients is performed in the emergency room conditions. In particular, patients who are candidates for fibrinolytic therapy and present with stroke symptoms should be investigated for etiology. Although it is not common in stroke patients, as in our case, the presence of underlying AoD changes the course of treatment. Otherwise, the fibrinolytic therapy given can be fatal.

Conflict of interest

None to declare

Acknowledgments

None to declare

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