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Beware of the DeBakey Type I Aortic Dissection Hidden by Ischemic Stroke: A Case Report

Chen SQ et al. Aortic Dissection presented as Ischemic Stroke

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Abstract

BACKGROUND

DeBakey Type I aortic dissection is one of the rare etiologies of ischemic stroke. It is

very important to identify arterial dissection before intravenous thrombolysis,

otherwise, fatal hemorrhage may occur.

CASE SUMMARY

Here, we report 2 painless DeBakey Type I aortic dissection cases with ischemic stroke

as the initial symptoms. Both cases had very sudden onset of conscious disturbance and

limbs weakness within minutes. Transient or persistent hypotension was found in both

cases. Thoracoabdominal Computed Tomography Angiography (CTA) was urgently

performed in an attempt to find out the underlying causes of hypotension, and aortic

dissection conforming to Type I according to the DeBakey classification was confirmed.

Both cases avoided intravenous thrombolysis because of timely diagnosis though they

eventually died of ruptured aortic dissection.

CONCLUSION

In conclusion, aortic dissection should always be excluded in ischemic patients with

unexplained hypotension or shock symptoms before intravenous thrombolytic therapy.

Key Words: Ischemic stroke; Aortic dissection; Diagnosis; Case report

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Core Tip: Aortic dissection should always be excluded in ischemic patients with

unexplained hypotension or shock symptoms before intravenous thrombolytic therapy.

These two painless DeBakey Type I aortic dissection cases with ischemic stroke as the

initial symptoms, avoided intravenous thrombolysis because of timely diagnosis,

thanks to the enough attention of the attending doctor paid to hypotension or insufficient limb perfusion in physical examination.

# 7 INTRODUCTION

Acute ischemic stroke (AIS) is the leading cause of death and disability in the world. It is defined as the sudden loss of blood flow to an area of the brain with the resulting loss of neurologic function. In order to save more penumbra tissue, vascular recanalization treatment such as intravenous thrombolysis or endovascular clot retrieval should be carried out as soon as possible, because the recanalization time is directly related to the outcome<sup>[1]</sup>. Although intravenous thrombolysis is widely used, carefully excluding patients with contraindications is critical. Aortic dissection as the absolute contraindication of thrombolytic therapy is an uncommon, life-threatening cause of ischemic stroke. Typical aortic dissection presents with tearing chest or back pain, but painless aortic dissection with only focal neurological deficits can be a challenge for emergency physicians, and inadvertently treating this situation with thrombolytic agents may threaten the patient's life.

Here, we describe 2 painless DeBakey Type I aortic dissection patients presenting as ischemic stroke who were eligible for thrombolytic treatment. We report these cases with the aim of emphasizing the importance of ruling out aortic dissection before fibrinolytic treatment in acute ischemic stroke.

# 9 CASE PRESENTATION

# Chief complaints

#### Case 1

A 73-year-old Chinese man was brought to the emergency room with a complaint of sudden loss of consciousness and right-limb weakness for 1 h.

#### Case 2

A 68-year-old male suddenly lost consciousness while standing up from squat position for 1.5 h.

# History of present illness

Case 1

Symptoms of right-limb weakness, altered consciousness, and incontinence suddenly occurred within ten seconds to minutes 1 h ago while he was cooking. No nausea or vomiting was observed during the clinical course.

Case 2

Symptoms occurred at the instant of postural change. After arriving at the emergency room, the patient became conscious and complained of neither chest or back pain, nor nausea or vomiting. He only presented with slight dysarthria and slow response.

# History of past illness

Case 1

The patient had intermittent gout for 3 years.

Case 2

21 years ago, the patient was diagnosed as "cerebral infarction" due to sudden left limb weakness. And he underwent decompressive craniectomy due to "cerebral hemorrhage" 19 years ago. He had "subarachnoid hemorrhage" 12 years ago and recovered after conservative treatment. Before this attack, the patient was independent in daily life and could go up and down stairs.

# 10 Personal and family history

Case 1

The patient denied any family history of similar medical history or genetic disease.

Case 2

He had a drinking history of more than 30 years and had given up drinking for more than 10 years. He smoked for more than 30 years and quit smoking 10 years ago. The patient denied any family history of similar medical history or genetic disease.

# Physical examination

#### Case 1

Physical examination found he had a pale face, wet and cold limbs, the vital signs were as follows: Body temperature, 36.8°C blood pressure, 98/58 mmHg (right arm), 101/60 mmHg (left arm); heart rate, 56 beats per min; respiratory rate, 22 breaths per min. There was no murmur in the heart valve auscultation area. Nervous system condition: blurred consciousness, passive posture, motor aphasia, grade II muscle strength of the right limb, normal muscle strength of the left limb, and positive Babinski sign of right lower limb.

#### Case 2

Low BP (84/68 mmHg) was detected at the emergency room, but was found to be normal at the neurology department. The vital signs at neurology department were as follows: Body temperature, 36.0°C blood pressure, 121/74 mmHg (right arm), 110/60 mmHg (left arm); heart rate, 89 beats per min; respiratory rate, 15 breaths per min. Furthermore, the patient had obvious cyanosis. The muscle strength of the four limbs had decreased slightly.

# Laboratory examinations

Case 1

Routine blood analyses were normal. Blood biochemistry indicates renal insufficiency (creatinine, 201  $\mu$ mol/L; urea nitrogen, 11 mmol/L). D-dimer was more than 16  $\mu$ g/mL.

Case 2

D-dimer was elevated (1.9  $\mu$ g/mL). Blood gas analysis indicates low blood oxygen (pressure of O2, 9.47 kPa). No abnormality was found in routine blood analyses.

# Imaging examinations

Case 1

Computed tomography (CT) of the brain did not find low density focus, which indicated that it was eligible for thrombolytic treatment. Doppler ultrasound of the carotid artery was applied in an attempt to determine the possible cause of the unexplained hypotension, and it found both right common carotid and right internal carotid artery dissection (Figure 1 A). Bedside chest X-ray indicated widening of the right mediastinum (Figure 1 B). Therefore, thoracic and abdominal CT-angiography (CTA) was urgently performed, which suggested aortic dissection conforming to Type I according to the DeBakey classification (Figure 1 C and D).

Case 2

The chest X-ray did not indicate mediastinum widening. A cranial CT scan showed multiple chronic brain infarctions (Figure 2 A) . Considering the high risk of bleeding in this patient, the family refused intravenous thrombolysis. In the light of the unexplained cyanosis and sweating limbs in this patient, a thoracoabdominal CT scan was accomplished in an attempt to determine the underlying causes. The CTA enabled a definite diagnosis of DeBakey Type I aortic dissection (Figure 2 B and C).

# **FINAL DIAGNOSIS**

Case 1

Combined with the patient's clinical history and imaging findings, the final diagnosis was DeBakey Type I aortic dissection complicated with cerebral infarction. Chronic renal insufficiency was the secondary diagnosis.

Case 2

Combined with the patient's clinical history and imaging findings, the final diagnosis was DeBakey Type I aortic dissection complicated with cerebral infarction. Chronic cerebral infarction was the secondary diagnosis.

### **TREATMENT**

Case 1

Since our hospital was unable to perform the surgery, we recommended the patient to transferred to another hospital. Carefully monitoring vital signs, controlling blood pressure and avoiding excessive exertion were implemented before referral.

#### Case 2

We carefully monitored the patient's vital signs, controlled blood pressure and gave appropriate sedation. We suggested the patient to transfer to other hospital for surgical treatment.

#### OUTCOME AND FOLLOW-UP

Case 1

On the second day of admission, the patient suddenly died due to rupture of the arterial dissection.

Case 2

The patient died 10 h later after admission when his family members were discussing whether to transfer him to a different hospital for surgical treatment.

# **DISCUSSION**

Acute aortic dissection (AAD) is characterized by the rapid development of an intimal flap. This is caused by blood flowing into the media and forcing the intima and the adventitia apart, which leads to life-threatening complications and death, particularly when the ascending aorta is involved<sup>[2]</sup>. According to the location of the dissection and or origin of the intimal tear and the extent of the dissection, AAD can be classified into Stanford Type A (or DeBakey Type I and Type II) and Stanford Type B (or DeBakey Type IIIa and Type IIIb). Stanford Type A aortic dissections involve the ascending aorta and usually require swift open surgical repair, whereas Stanford Type B dissections involve the descending but not the ascending aorta and are conventionally treated by endovascular repair and/or medical therapy<sup>[2]</sup>. AAD is an uncommon but life-threatening disease with a reported incidence of 3–5 cases per 100,000 people per year<sup>[3]</sup>, a figure which may rise to even as high as 35 cases per 100,000 people per year at

the age of 65-75 years old<sup>[4]</sup>. About 20% of the untreated patients with DeBakey Type I aortic dissection died on the spot, whilst a further 1-2% of the survivors died with every hour of delayed treatment<sup>[5]</sup>.

Stroke is one of the most serious complications of DeBakey Type I acute aortic dissection and the incidence of DeBakey Type I arterial dissection complicated with stroke is about 6% [6] to 10% [7]. Arterial dissection represents 5.7% of first-ever ischemic strokes of unusual cause in a clinical series [8]. Arterial dissection presented with stroke symptoms and without typical chest or back pain can lead to a delayed diagnosis of dissection and that can significantly increase the mortality. Furthermore, thrombolysis treatment can result in 3-5 folder higher probability of fatalities than non-thrombolysis treatment of patients [9]. Therefore, urgently identifying the presence of arterial dissection in stroke patients is very important.

The dissection can involve both of the carotid arteries while right hemisphere involvement is more common seen in previous reports<sup>[7]</sup>. The frequency of chest/back pain is much lower in AAD patients combined with stroke than those without stroke<sup>[7]</sup>. Altered consciousness, which may be either due to the sudden drop of blood pressure after arterial dissection or damage of the brain, can lead to delayed reporting of chest/back pain. SBP difference between the two upper limbs of greater than 20 mmHg is one of the characteristics of AAD, but in our case series, none of them had such manifestations. Furthermore, pulse deficits are present in about 20–30% of ADD cases which emphasizes the importance of serial physical examinations that can substantially contribute to the correct diagnosis. Transient or persistent low BP or shock symptoms should always be taken seriously in ischemic stroke patients because most ischemic stroke patients usually had higher BP<sup>[7]</sup>.

D-dimer is taken into an important predictive value in the diagnosis of AAD and D-dimer values less than  $0.5 \,\mu\text{g/mL}$  can be used to rule out AAD<sup>[10]</sup>. However, D-dimer elevation is also common in acute stroke, deep venous thrombosis and pulmonary embolism, particularly in cardioembolism<sup>[11]</sup>. So, D-dimer elevation has poor specificity

in the diagnosis of aortic dissection, but is helpful to exclude aortic dissection when Ddimer is negative.

The contrast-enhanced CT scan is a reliable method for the definite diagnosis of AAD. In addition to enhanced CT, other auxiliary examinations, such as carotid ultrasound and chest X-ray can also provide reference values for AAD diagnosis. Chest X-rays can be considered as part of the acute screening protocols in acute ischemic patients with special enlarged mediastinal shadow in AAD. A common or internal carotid artery dissection can easily be investigated by ultrasonography which can be regarded as a helpful, complementary tool for the current diagnostic workup<sup>[12]</sup>.

Arterial dissection is a life-threatening emergency which needs urgent surgery or endovascular intervention. Before that, keeping the SBP lower than <120 mmHg with intravenous  $\beta$ -blockers or vasodilators and careful management of any conditions that can increase thoraco-abdominal pressure to prevent the complication of aortic rupture is highly recommended.

# **CONCLUSION**

In conclusion, once neurologists encounter AIS patients showing a rapid peak of neurological symptoms, unexplained hypotension or shock symptoms, arterial dissection as a differential diagnosis should always be excluded before intravenous thrombolysis. Serial physical examinations, such as monitoring the SBP differential between the two arms and peripheral arterial pulsation can be a hint of arterial dissection. An abnormal carotid ultrasound finding and mediastinal widening on chest radiograph may also be helpful in identifying AAD. The elevation of D-dimer has no specificity value in diagnosing arterial dissection, but its negative value has high specificity in excluding AAD. AIS patients who are suspected of arterial dissection need timely enhanced CTA examination to confirmed the diagnosis. The close monitoring of vital signs, ensuring proper bed rest, and the treatment of coughing and constipation are very important to avoid arterial rupture.

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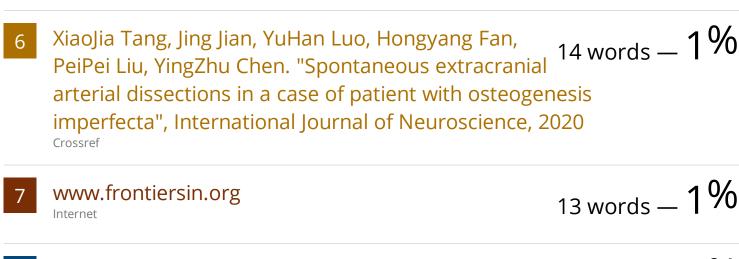
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