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CASE REPORT

Stanford Type A Aortic Dissection Masquerading as Acute Ischemic Stroke: A Case Report

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Abstract: Aortic dissection (AD), a life-threatening cardiovascular emergency, is characterized by the separation of the aorta's inner and middle layers due to a tear in the intima. It is classified as Stanford type A or B based on the tear's location and extent. Symptoms vary but commonly include severe pain in the chest, back, or abdomen, along with atypical presentations such as shock, heart failure, or syncope. End-organ ischemia, including stroke and limb necrosis, may occur. Timely diagnosis and intervention are crucial for survival. Here, we report a 31-year-old male patient who presented with acute neurological symptoms, initially suspected of having a stroke, but was ultimately diagnosed with Stanford type A AD upon computed tomography (CT) angiography. This case underscores the importance of considering AD in the differential diagnosis of patients with neurological symptoms for accurate and prompt management.

Keywords: Aortic Dissection; Ischemic Stroke; Emergency Medicine; Computed Tomography Angiography; Case Reports

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1. Introduction

Aortic dissection (AD), a catastrophic event involving the tearing of the intima of the aorta, leading to the separation of the aortic wall layers, is a cardiovascular emergency that demands immediate medical attention (1). This condition, which can be classified as Stanford type A or type B depending on the involvement of the ascending or descending aorta, respectively, is associated with high mortality rates if not treated promptly. While the incidence of AD is estimated at approximately 4.8 cases per 100,000 individuals per year (2), it is crucial to note that the condition is often underdiagnosed or misdiagnosed, particularly in the early stages when symptoms may be non-specific or attributed to other pathologies (3).

Clinical manifestations of AD can be diverse, ranging from the classic symptoms of severe chest or back pain and pulse deficit to more atypical presentations such as syncope, heart failure, or even stroke. The heterogeneity of symptoms presents a diagnostic challenge, frequently resulting in treatment delays. Moreover, the potential for end-organ ischemia, such as stroke and limb necrosis, further underscores the importance of early recognition and intervention.

Here, we report a patient who presented with acute neurological symptoms, prompting an initial evaluation for stroke. However, the subsequent clinical course and imaging findings led to the diagnosis of Stanford type A AD. This case highlights the necessity of maintaining a broad differential diagnosis when assessing patients with neurological symptoms, emphasizing the life-saving potential of timely and accurate diagnosis.

2. Case presentation

A 31-year-old male patient, presented to our emergency department with acute onset of slurred speech and left-sided limb weakness lasting for over one hour. The patient reported no clear precipitating factors for the onset of the symptoms, which included difficulty speaking, leftward deviation of the tongue, and weakness in the left limbs, without loss of consciousness, dizziness, headache, nausea, vomiting, chest tightness, chest pain, back pain, abdominal pain, or diarrhea. The patient had no significant past medical history. Given the neurological deficits, the patient was promptly admitted to the emergency rescue room, and a Code Stroke was activated for further management.

On physical examination, the patient was found to have significantly elevated blood pressure, with the blood pressure in the left arm (195/134 mmHg) higher than that in the right arm (162/116 mmHg). No appreciable cardiac murmurs were auscultated. Muscle strength was rated at grade 4 on the left side, in contrast to normal strength observed on the right

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side. The patient's National Institutes of Health Stroke Score (NIHSS) was calculated to be 4. The patient's symptoms and signs indicated an initial assessment of a stroke.

Emergency non-contrast head computed tomography (CT) scan revealed normal morphology of both cerebral hemispheres and the brainstem. There was a patch of slightly hypodense material adjacent to the right lateral ventricle (Figure 1A). CT angiography (CTA) with 3D reconstruction revealed the absence of the right internal carotid artery within the intracranial space, and the left internal carotid artery and other intracranial arteries demonstrated a normal course with no evidence of significant stenosis or dilation (Figure 1B). CT perfusion (CTP) demonstrated reduced cerebral blood flow (CBF) and cerebral blood volume (CBV), prolonged time to drain (TTD), and extended time to peak (Tmax) in the right frontal and insular lobes (Figure 1C).

Laboratory studies revealed leukocytosis and marked elevations in brain natriuretic peptide (BNP, 648.60 pmol/L), creatinine (228.2 μmol/L), and blood urea nitrogen (8.8 mmol/L). Arterial blood gas analysis demonstrated mild respiratory alkalosis. Furthermore, there was a substantial elevation in D-dimer levels (8.17 mg/L), and urinary analysis revealed the presence of proteinuria (2+).

Given the patient's clinical presentation, imaging findings, and laboratory results, the preliminary diagnosis was acute ischemic stroke (AIS). To comprehensively assess the patient's condition and evaluate potential underlying causes, a thoracoabdominal CT scan was also performed. However, the thoracoabdominal CT scan revealed a widening of the thoracoabdominal aorta with heterogeneous intraluminal density (Figure 2A), suggesting the possibility of AD. This finding led our medical team to suspect an underlying AD that might be affecting the patient's clinical status. Although we were preparing to transfer the patient to the interventional suite for stroke treatment, a thorough assessment prompted us to prioritize a thoracoabdominal aorta CTA. This was done to precisely identify the extent and nature of the aortic abnormality before proceeding with other interventions. The CTA images, which included cross-sectional and three-dimensional reconstructions, revealed a bifurcation pattern from the root of the ascending aorta to the left common iliac artery and the right external iliac artery, with the involvement of the three branches of the aortic arch. There was a partial occlusion of the brachiocephalic trunk extending to the right common carotid artery. The false lumen of the thoracic aorta was notably wider, with a transverse diameter of approximately 59 mm at its widest point, and exhibited wall-attached annular and crescent-shaped low-density filling defects (Figure 2B).

The final diagnosis was Stanford type A AD. In response, the patient received rigorous management of blood pressure and heart rate, followed by expedited referral to the cardiothoracic surgery team for operative intervention. The surgical procedure consisted of aortic root replacement with concomitant stent grafting, utilizing the elephant trunk technique. The patient's postoperative course was favorable.

3. Discussion

In this case report, we present a patient who activated the Code Stroke due to suspected acute ischemic stroke (AIS), presenting with symptoms including slurred speech and left-sided limb weakness. The cranial CTA and CTP revealed an occlusion of the right internal carotid artery and concomitant hypoperfusion of the right cerebral hemisphere. However, these findings were later confirmed to be secondary to a Stanford type A AD, as evidenced by thoracoabdominal CTA, which had initially mimicked the clinical presentation of an ischemic stroke. A recent review has underscored the diagnostic difficulties associated with AD, uncovering an alarming misdiagnosis rate of 33.8% across 12 studies, encompassing a total of 1,663 patients. The predominant clinical presentations were chest pain (67.5%), back pain (24.8%), and syncope (6.8%) (4). Additionally, another study has emphasized the particular challenge in diagnosing pain-free dissections, which often leads to delayed identification. This challenge is particularly pronounced in patients under consideration for thrombolytic therapy in the setting of acute stroke, necessitating rigorous and timely diagnostic evaluations (5). This is reflected in a case of Type A AD that presented with cerebral infarction and neurological deficits, such as limb weakness and dysarthria. Despite negative head CT and initial thrombolytic therapy, the patient developed seizures, prompting discontinuation of thrombolysis. Neck CTA confirmed Type A AD, leading to surgical intervention with an unknown long-term prognosis (6). Another similar case involved a patient with syncope, abdominal pain, right gaze preference, and left-sided weakness, leading to "Code Stroke". CTA revealed a Stanford type A AD affecting the subclavian and right carotid arteries, resulting in multifocal cerebral and spinal cord infarcts. Despite intensive care, no neurological recovery was observed after 3 weeks (7), Our case's strength lies in the swift and precise diagnosis of Stanford type A AD using thoracoabdominal CTA, crucial for immediate intervention and preventing misdiagnosis-related disasters. However, our report is limited by the absence of long-term outcome data. Its utility would be improved by including the patient's postoperative recovery, potential complications, and the long-term efficacy of surgery to assess treatment impact on quality of life and prognosis. Moving forward, we will pay greater attention to these aspects in our work.

AIS constitutes a medical emergency marked by high morbidity and mortality rates, as well as a significant risk of recurrence and complications. The most effective treatments for AIS, such as intravenous thrombolysis and endovascular thrombectomy, are time-dependent, underscoring the critical nature of rapid intervention (8, 9). The principle of "Time is Brain" emphasizes the importance of immediate treatment within a specific time window to minimize brain damage and improve patient outcomes (10). In contrast, AD is a less com-

mon but life-threatening condition that requires immediate medical attention (11). The Stanford classification system categorizes AD based on the location of the proximal tear (12).

Type A involves any part of the aorta proximal to the origin of the right brachiocephalic trunk, and is associated with high mortality rates, with up to 23.7% mortality within 48 hours if solely on medical treatment (13). Surgical intervention is considered the gold standard for managing this type (14). Type B, originating distal to the left subclavian artery, is managed with either endovascular repair or medical treatment involving the control of blood pressure, heart rate, and pain.15 The presence of stroke-like symptoms in patients with Stanford type A AD can be attributed to the anatomical and physiological consequences of the condition. Type A AD, which involves the ascending aorta, may lead to embolic events that affect the cerebral circulation. These events may occur due to the dislodgment of thrombi or atherosclerotic plaques, which can travel to the carotid or vertebral arteries, resulting in stroke. Furthermore, the compromised integrity of the aortic wall in type A AD can lead to aortic insufficiency, which can decrease systemic blood pressure and subsequently reduce cerebral perfusion, potentially resulting in ischemic stroke.

It has been reported that approximately 6% of patients with type A AD present with stroke at hospital admission (16). While type B AD, involving the descending aorta distal to the left subclavian artery, typically does not result in stroke-like symptoms. The clinical presentation of type A AD can mimic the symptoms of AIS, such as altered consciousness or motor impairment of limbs, without the classic complaints of chest or back pain. This similarity in symptoms can lead to misdiagnosis and inappropriate treatment, like thrombolytic therapy, which may result in catastrophic complications, including aortic rupture.

In differentiating between AD and AIS, several laboratory and clinical findings have been identified that can help distinguish between these two conditions (17). Firstly, a significantly elevated D-dimer level, with a threshold greater than 6.9 g/mL, raises suspicion for the presence of AD (18). Secondly, a bilateral systolic blood pressure difference exceeding 20 mmHg suggests the presence of AD (19). Besides, carotid artery ultrasound may reveal an intimal flap or the absence of clear blood flow, which indicates the presence of AD(20). Additionally, enlargement of the mediastinum on chest radiography is a significant sign of AD (21). It is also important to note that left hemiparesis is more commonly present in AD, due to the more common involvement of the right carotid artery in dissection cases (22).

4. Conclusions

This case underscores the critical role of considering aortic dissection in patients presenting with neurological symptoms, particularly in the absence of classic stroke risk factors. Timely and accurate diagnosis, followed by prompt intervention, is essential to prevent life-threatening complications and optimize patient outcomes.

5. Declarations

5.1. Acknowledgments

None.

5.2. Ethics approval

This case report was approved by the Jinling Hospital Ethics Committee (ethics code: 2021DZSKT-YBB-014).

5.3.

5.3. Informed consent

Written informed consent was obtained for publication of this case report.

5.4. Author contribution statement

All authors contributed substantially to the writing and revision of this manuscript and approved this final version.

- Data curation: Bo Zhang, Ji Xie.
- Formal analysis: Liyu Lu.
- Supervision: Shinan Nie
- Writing – original draft: Chao Liu.
- Writing – review & editing: Jiangrong Ma.

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5.6. Data availability statement

Data will be made available on reasonable request.

5.7. Competing interest

The authors declare that they have no competing interests.

5.8. Using artificial intelligence chatbots

None.

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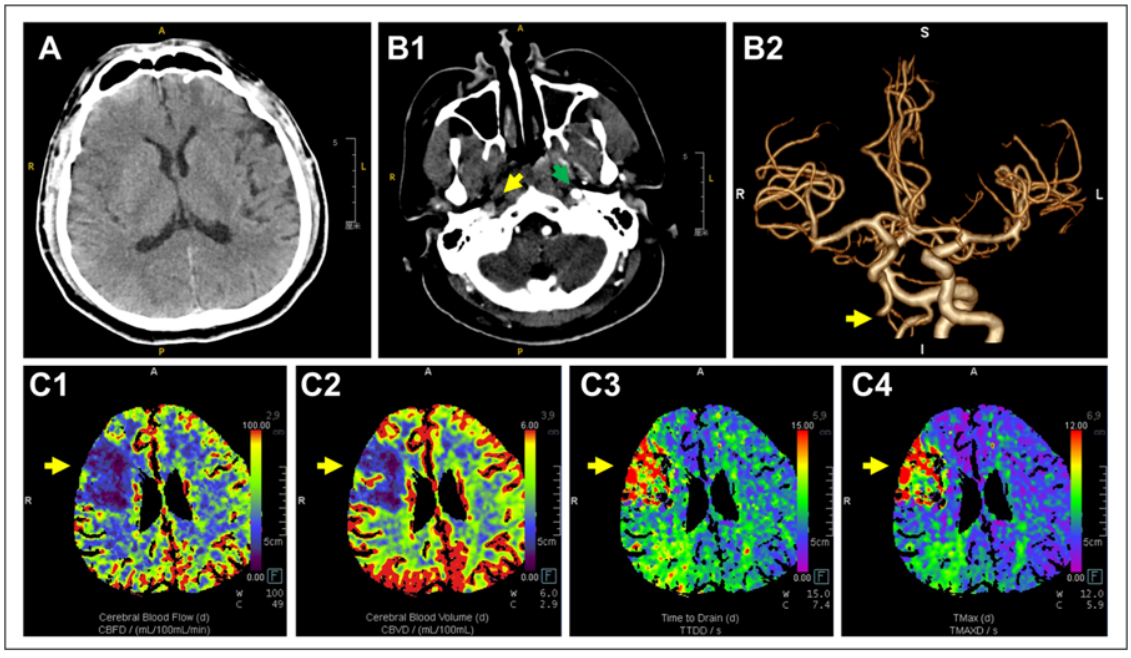

Figure 1: Imaging examination results obtained following the patient's admission to the stroke alert protocol. A. The non-contrast head CT scan revealed unremarkable morphology of both cerebral hemispheres and the brainstem. Additionally, there was a focal area of mild hypodensity adjacent to the right lateral ventricle. B1. The cross-sectional image of the cranial CTA showed non-visualization of the right internal carotid artery. B2. The 3D reconstruction of the cranial CTA suggested occlusion of the right internal carotid artery. C1-C4. The CTP demonstrated reduced cerebral blood flow (C1) and cerebral blood volume (C2), prolonged time to drain (C3), and extended time to peak (C4) in the right frontal and insular lobes.

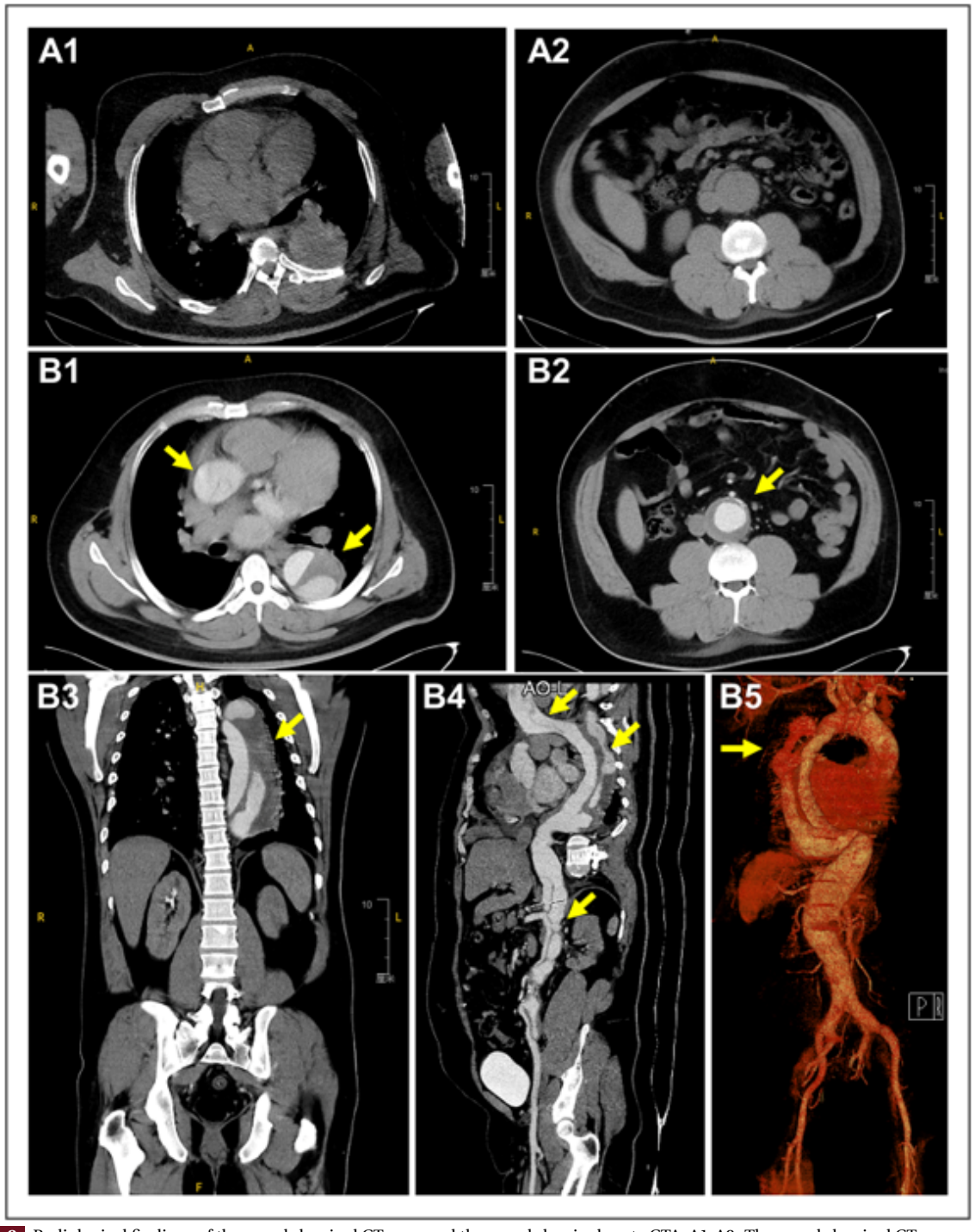

Figure 2: Radiological findings of thoracoabdominal CT scan and thoracoabdominal aorta CTA. A1-A2. Thoracoabdominal CT scan revealed a dilated thoracoabdominal aorta with heterogeneous intraluminal density, suggestive of aortic dissection. B1-B5. Thoracoabdominal CTA cross-sectional images (B1-B4) and the corresponding 3D reconstruction (B5) revealed the presence of a Stanford type A aortic dissection, characterized by the separation of the aortic wall layers.