ภาวะหลอดเลือดแดงใหญ่ฉีกขาดที่แสดงอาการคล้ายโรคหลอดเลือดสมองตีบเฉียบพลัน : ข้อผิดพลาดในการวินิจฉัยและผลลัพธ์จากการให้ยาละลายลิ่มเลือดทางหลอดเลือดดำ

Aortic Dissection Presenting as Acute Ischemic Stroke: Diagnostic Pitfalls and Outcomes Following Intravenous Thrombolytic

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ความเป็นมา: ภาวะผนังหลอดเลือดแดงใหญ่ปริแตกเซาะ (aortic dissection) ชนิด Stanford type A อาจนำมาด้วยอาการแสดงของโรคหลอดเลือดสมองตีบ (acute ischemic stroke:AIS) ซึ่งนำไปสู่การให้ยาละลายลิ่มเลือด (rtPA) โดยไม่ทราบการวินิจฉัยที่แท้จริงและเพิ่มความเสี่ยงต่อการเสียชีวิต

วัตถุประสงค์: นำเสนอรายงานผู้ป่วยและทบทวนวรรณกรรมเพื่อสะท้อนความสำคัญของการวินิจฉัย aortic dissection ที่แฝงมากับอาการ AIS และผลกระทบของการให้ rtPA

วิธีการ: ศึกษาย้อนหลังผู้ป่วย 4 รายระหว่างปี พ.ศ. 2562-2567 ที่มีอาการ AIS และได้รับการวินิจฉัยภายหลังว่าเป็น Stanford type A Aortic Dissection วิเคราะห์อาการ การให้ rtPA การรักษา และผลลัพธ์ พร้อมทบทวนวรรณกรรมกรณีคล้ายคลึงกันจากรายงานต่างประเทศ

ผลการศึกษา:ในผู้ป่วย 4 ราย พบว่า 3 รายไม่ได้รับ rtPA โดย 2 รายผ่าตัดฉุกเฉินและรอดชีวิต ส่วนอีก 1 รายปฏิเสธการผ่าตัด ได้รับการดูแลแบบประคับประคอง ผู้ป่วยเพียง 1 รายที่ได้รับ rtPA เสียชีวิตในโรงพยาบาล

สรุป: ในผู้ป่วยที่มีอาการ AIS ควรระวังอาการนำของ aortic dissection เช่น เจ็บหน้าอก ความดันเลือดต่างกันระหว่างแขน หรือ mediastinal widening และควรพิจารณาส่งตรวจ CTA ก่อนพิจารณาให้ rtPA

คำสำคัญ: Aortic Dissection, Acute Ischemic Stroke, rtPA, การวินิจฉัย, การผ่าตัดฉุกเฉิน

Abstract

Background: Stanford type A aortic dissection may initially present with symptoms mimicking acute ischemic stroke (AIS), which can lead to the inadvertent administration of intravenous thrombolysis (rtPA) without an accurate diagnosis, significantly increasing the risk of mortality.

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Objective: To present a case series and literature review emphasizing the importance of recognizing aortic dissection presenting as AIS and the consequences of administering rtPA in such cases.

Methods: A retrospective review was conducted on four patients between 2019 and 2024 who initially presented with AIS symptoms and were subsequently diagnosed with Stanford type A aortic dissection. Clinical presentation, rtPA administration, treatment approaches, and outcomes were analyzed. Similar cases from international literature were also reviewed.

Results: Of the four patients, three did not receive rtPA; two underwent emergency surgery and survived, while one declined surgery and received palliative care. The one patient who received rtPA died in the hospital.

Conclusion: Aortic dissection should be considered in AIS patients with suggestive symptoms such as chest pain, inter-arm blood pressure difference, or mediastinal widening. Computed tomography angiography (CTA) should be performed before administering rtPA to prevent fatal complications.

Keywords: Aortic dissection, Acute ischemic stroke, rtPA, Diagnosis, Emergency surgery

Introduction

Aortic dissection is a rare but life-threatening medical emergency with a high mortality rate if not diagnosed and treated promptly. Its incidence is estimated at approximately 2.6–3.5 cases per 100,000 persons per year.[1] In contrast, acute ischemic stroke (AIS) is a common emergency condition that is currently treatable with high efficacy, particularly when intravenous thrombolytic agents are administered within 3 to 4.5 hours after symptom onset. Early thrombolytic therapy has been shown to significantly improve patient outcomes.[2,3] However, thrombolytic in AIS patients carries a risk of hemorrhagic complications, especially when an undiagnosed condition such as aortic dissection is present. Identifying patients with occult aortic dissection during the critical window for rtPA administration is therefore urgent and poses a major diagnostic challenge for treating physicians.

Pathophysiology[4-7]

Aortic dissection results from a tear in the inner most layer of the aortic wall (intima), allowing blood to enter the medial layer and split the aortic wall into two channels: the true lumen and the false lumen. This separation may obstruct blood flow to critical organs and lead to severe complications, such as acute myocardial infarction, renal failure, or stroke.

Clinically, aortic dissection is categorized using two widely accepted classification systems. One of the most commonly used is the Stanford classification, which is based on the location of the dissection:

1. **Stanford Classification**
   - **Type A** involves a dissection in the ascending aorta or aortic arch, and may extend to the descending aorta or beyond. It is the more common and more dangerous form, requiring urgent surgical intervention. Without treatment, the mortality rate increases by approximately 1% per hour during the initial phase, reaching 50% within 3 days and up to 80% within two weeks.
   - **Type B** refers to dissections confined to the descending aorta. In uncomplicated cases—i.e., without signs of end-organ ischemia or aneurysmal expansion medical management with blood pressure control is typically sufficient.

However, in the presence of complications, surgical repair or endovascular intervention may be indicated.

2. **DeBakey Classification:** This system categorizes aortic dissection based on the site of origin and the direction of propagation within the aorta. It is divided into three primary types:

- **Type I**: The dissection originates in the ascending aorta and extends beyond the aortic arch into the descending aorta. This is the most common and most severe form, requiring emergency surgical intervention due to its extensive involvement.
- **Type II**: The dissection is confined to the ascending aorta and does not extend to other segments. Despite its localized nature, surgical treatment is still indicated because of the high risk of life-threatening complications.
- **Type III**: The dissection originates in the descending aorta and is further subclassified into:
- **Type IIIa**: The dissection is limited to the descending thoracic aorta.
- **Type IIIb**: The dissection extends beyond the thoracic segment into the abdominal aorta and may involve distal arterial branches.

Table 1 Stanford and DeBakey classifications of aortic dissection.[8]

STANFORD	TYPE A		TYPE B	
Site of dissection	Involves the ascending aorta (may extend to the aortic arch and descending aorta)		Originates in the descending aorta (distal to the aortic arch) arch)	
DE BAKEY	TYPE I	TYPE II	TYPE IIIa	TYPE IIIb
Site of dissection	Originates in the ascending aorta and extends to aortic arch and descending aorta.	Confined to the ascending aorta only.	Originates in the descending aorta and extends only within the thoracic aorta.	Originates in the descending aorta and extends into the abdominal aorta.
NORMAL				
Treatment	Usually requires emergency surgical intervention due to high risk of complications.		Initially managed with medical therapy focusing on strict blood pressure control. In cases of complications (e.g., acute ischemia or rupture), endovascular repair (TEVAR) may be considered.	

Risk Factors for Aortic Dissection[9-14]

1. **Hypertension** – The most common risk factor, present in > 70 % of patients. Sustained elevated blood pressure increases intraluminal stress, accelerates medial degeneration, and facilitates intimal tearing.

2. **Connective tissue disorders** – Inherited defects of aortic elastin/collagen, such as *Marfan syndrome, Loeys-Dietz syndrome,* and *Ehlers-Danlos syndrome type IV*, are strongly associated with early-onset dissection.

3. **Aortic aneurysm** – Dilatation, particularly of the ascending aorta, markedly raises wall tension; diameters ≥4.5–5.0 cm confer a high rupture/dissection risk.

4. **Iatrogenic causes** – Cardiac surgery, catheter-based aortic manipulation, or valve replacement can produce mechanical injury to a vulnerable aortic wall.

5. **Age and sex** – Incidence rises sharply after 60 years and is roughly twice as high in men as in women.

6. **Cigarette smoking** – Chronic inflammation and oxidative stress accelerate medial degeneration, predisposing to dissection.

7. **Pregnancy** – Haemodynamic and hormonal changes, especially in the third trimester, increase aortic wall distensibility; the risk is heightened in women with underlying connective tissue disease.

8. **Family history** – First-degree relatives of patients with thoracic aortic aneurysm or prior dissection have a significantly elevated genetic risk.

9. **Sympathomimetic drug use** – Acute blood pressure surges from cocaine or amphetamines precipitate dissection in otherwise low-risk young adults.

Signs and Symptoms

The most common presenting symptom of aortic dissection is sudden, severe chest pain described as tearing or stabbing in nature. The pain is typically abrupt in onset and reaches maximal intensity immediately, especially in Stanford type A dissection. In contrast, Stanford type B dissections more often present with back pain, which may radiate downward toward the abdomen following the course of the descending thoracic and abdominal aorta. This characteristic pain has a sensitivity of 82.9% and specificity of 70.7% for the diagnosis of aortic dissection.[15]

Chest pain is frequently accompanied by signs of hemodynamic instability, and multiorgan symptoms may occur due to impaired blood flow or direct extension of the dissection into vital arteries:

- **Cardiovascular system:** Acute myocardial infarction, congestive heart failure, and acute limb ischemia may result from coronary artery involvement or systemic malperfusion.[16]
- **Respiratory system:** Acute lung injury and acute respiratory distress syndrome (ARDS) may arise secondary to systemic inflammation or volume overload.[17]
- **Neurologic system:** Acute ischemic myelopathy and acute ischemic stroke occur in 6–32% of patients with aortic dissection.[18,19] The two primary mechanisms are:

    1. **Flow obstruction** from dissection flap at or near the aortic root involving the carotid arteries, especially in Stanford type A.
    2. **Artery-to-artery embolism** from thrombus formation within the false lumen, leading to cerebral infarction.[19,20]

Diagnosis of Aortic Dissection

Clinical history and physical examination remain crucial in the initial assessment of patients, particularly when aortic dissection is suspected. Key diagnostic clues include characteristic chest pain, a systolic blood pressure differential of more than 20 mmHg between arms, pulse deficits,[21] and symptoms affecting other organ systems, as previously described. Chest radiography may reveal mediastinal widening, which is a classic finding suggestive of dissection. However, up to 20% of patients may have a normal mediastinum on chest X-ray.[22]

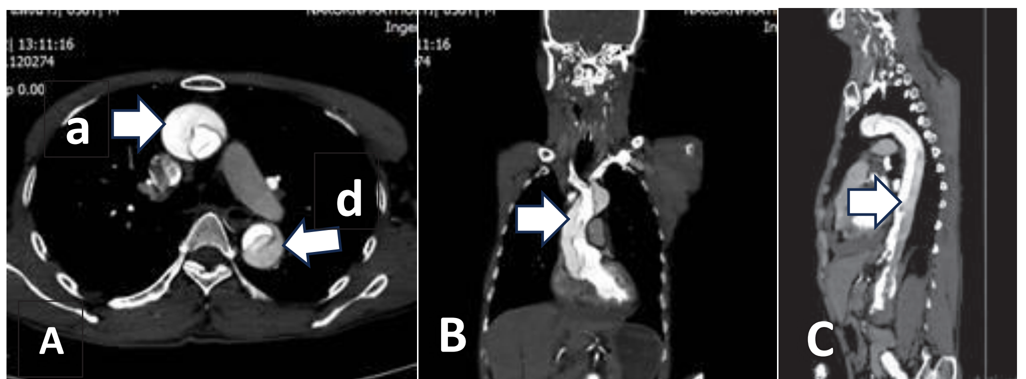
The current gold standard for diagnosis is computed tomography angiography (CTA), which provides a rapid and highly accurate assessment, with a reported sensitivity of approximately 95% and specificity of 98%.[23]

In our patient cohort, individuals initially presented with acute ischemic stroke (AIS) and were evaluated by an emergency physician between 2019 and 2024 under the Stroke Fast Track protocol. The case was managed through a coordinated approach involving a neurologist and the hospital's Stroke Fast Track team to assess eligibility for intravenous thrombolysis (rtPA) within the prescribed therapeutic window, in accordance with contemporary clinical practice guidelines. Subsequently, the patient was further diagnosed with aortic dissection and was referred to the cardiothoracic surgery team for comprehensive evaluation and definitive management planning.

Table 2 Clinical characteristics of patients with aortic dissection presenting as acute ischemic stroke

Case	Sex	Age	Risk Factors	Stroke Presentation	NIHSS	Chest Pain	BP Asymmetry	CXR finding: widening mediastinum	Aortic dissection Stanford/ DeBakey type	rtPA	Outcome
1	M	66	HTN	Left hemiplegia	>10	No	Yes	No	A, type I	No	Denied surgery --> Palliative care
2	M	57	HTN	Right hemiparesis	>10	Yes	No	Yes	A, type I	No	Ascending and hemiarch graft replacement surgery --> Improve
3	M	58	DM, HTN, DLP	Left hemiplegia	>10	Yes	No	No	A, type I	No	Ascending and hemiarch graft replacement surgery --> Improve
4	M	75	HTN	Left hemiplegia	>10	No (back pain post-IVT)	No	No	A, type I	Yes	Dead

Radiographic Image of Case 3



The transverse image (A) shows the dissection flap (arrow) extending through the ascending (a) and descending (d) aorta. The flaps are shown more clearly on the coronal (B)and sagittal (C) images.

Based on a literature review of nine published case reports between 2000 and 2022, patients with aortic dissection initially presented with clinical manifestations suggestive of acute ischemic stroke and subsequently received intravenous thrombolytic therapy (rtPA). Details of these cases are summarized in Table 3.

Table 3 Summary of published case reports of aortic dissection presenting as AIS

Ref.	Cases	Dead in hospital	Thoracic surgery after rtPA	Clinical stroke
Fessier(2000)[23]	1	Yes		
Chua(2005)[24]	1	-	Emergency	Improve
Uchio(2005)[25]	1	-	Emergency	Not improve
Moti(2007)[26]	1	-	Emergency	Improve
Iguchi(2010)[27]	2	Yes (2/2)		
Nari C(2016)[28]	1	Yes		
Young(2017)[29]	1	-	Emergency	Not improve
He ZY(2022)[30]	1	-	Emergency	Not improve
Ankur(2023)[31]	2	-	Emergency	Improve
		Yes (1/2)		

In cases where Stanford type A aortic dissection presents with clinical features of acute ischemic stroke (AIS) during the therapeutic “golden period” for intravenous thrombolytic therapy (rtPA), clinicians face a critical and potentially life-threatening challenge in both diagnosis and treatment decision-making within a limited timeframe.

The administration of rtPA under these circumstances requires careful evaluation, as a missed diagnosis of aortic dissection can result in

catastrophic complications. A review of risk factors should be undertaken, including advanced age,[12] hypertension,[13] and characteristic chest pain described as tearing or stabbing in nature, along with evidence of abnormal circulatory status.[16] Additional diagnostic clues such as widened mediastinum on chest radiograph may prompt further investigation. In such cases, computed tomography angiography (CTA) should be performed to confirm the diagnosis of aortic dissection prior to thrombolytic administration.

A synthesis of published case reports indicates that the administration of intravenous alteplase (rtPA) is associated with a heightened risk of aortic dissection-related complications, frequently necessitating emergent thoracic surgery. [24- 26,29-31] Several deaths have also been documented[23,27,28,31] Reported neurological presentations in patients ultimately found to have an underlying aortic dissection include aphasia[28], depressed level of consciousness[29], and convulsions. [31] Notably, many cases lacked the classical prodromal features of aortic dissection, complicating recognition prior to thrombolytic treatment. [23-31]

Across nine published case reports, a total of 11 patients received intravenous alteplase. [23-31] In-hospital mortality occurred in 5 of 11 cases (45 %). Survival to discharge was closely linked to the performance of emergent surgical repair. [24- 26,29-31] Neurological outcomes were reported in six patients; only three showed sustained clinical improvement on follow-up (50 %).

During emergent surgical intervention in several reported cases, blood component therapy—including cryoprecipitate and platelet transfusion—was administered to optimize hemostasis prior to surgery. [24- 26,29-31] In the report by Ankur et al.,[31] epsilon-aminocaproic acid, an antifibrinolytic agent, was also used to counteract the effects of rtPA. However, preparation and administration of these agents may contribute to delays in surgical intervention.

These findings highlight the complex balance between thrombolysis and surgical readiness in patients with aortic dissection presenting as acute ischemic stroke. Although the overall survival rate following emergency surgery in this subgroup was approximately 55%, only 50% of survivors demonstrated favorable neurological recovery. This underscores the clinical challenge of managing such cases and the importance of early recognition to prevent inappropriate thrombolytic therapy.

It is well established that, following a diagnosis of acute Stanford type A aortic dissection, the cumulative mortality rate increases by approximately 1–2% per hour without surgical intervention. If managed with medical therapy alone, the mortality rate may reach up to 20% within the first 24 hours and as high as 50% within one month.[32]

This aligns with our Case 1, in which the patient, after being diagnosed with Stanford type A aortic dissection, declined surgical intervention. As a result, the treatment plan was shifted to palliative care, focusing on symptom control and comfort measures.

Several reports have documented cases in which acute ischemic stroke (AIS) coexists with aortic dissection, a condition that may be suspected based on a patient's history of chest pain and subsequently confirmed through computed tomography angiography (CTA). [34- 36] In such cases, physicians often choose to withhold intravenous thrombolysis (rtPA) due to the high risk of life-threatening complications, and instead proceed with urgent

referral to cardiothoracic surgery for definitive management planning.

This clinical approach is consistent with Cases 2 and 3 in our series, in which patients were diagnosed with Stanford type A aortic dissection, promptly underwent emergency surgical repair, and survived. Following postoperative recovery and neurological rehabilitation, both patients showed notable improvement in neurological function.

Pitfalls in Patient Management

Based on the current case series and literature review, several common pitfalls have been identified in the management of aortic dissection presenting as acute ischemic stroke (AIS). These challenges often lead to delayed diagnosis and poorer clinical outcomes, particularly when initial clinical features are subtle or non-specific. Notable pitfalls include:

- Absence of **characteristic tearing chest pain**, which is typically associated with aortic dissection
- Lack of **inter-arm blood pressure discrepancy**, a classic but inconsistently present sign
- Absence of **mediastinal widening** on chest radiography
- **Misinterpretation or oversight** of subtle findings on imaging studies
- **Premature administration of rtPA** under stroke fast-track protocols, without sufficient screening for alternative life-threatening conditions such as aortic dissection

These pitfalls emphasize the importance of maintaining a high index of suspicion for aortic dissection in AIS patients, particularly in the presence of any atypical signs or inconclusive imaging. Careful clinical judgment and early use of CTA may improve diagnostic accuracy and outcomes in these high-risk scenarios.

System-Level Recommendations

1. **Develop an integrated screening protocol for aortic dissection within the acute stroke fast-track system.** Implementation of a structured screening pathway is essential to prevent inadvertent administration of rtPA in patients with undiagnosed aortic dissection. Such a protocol would help reduce the risk of catastrophic complications, improve early diagnosis, and increase the likelihood of survival and favorable neurological recovery.

2. **Consider immediate computed tomography angiography (CTA) in patients with any suggestive clinical features or red flags.** Early CTA should be incorporated into the diagnostic workflow for stroke patients exhibiting signs suspicious for aortic dissection. This approach ensures timely and accurate diagnosis prior to thrombolytic therapy, allowing appropriate surgical referral and management.

Conclusion

Thorough history-taking and physical examination play a critical role in evaluating patients with acute ischemic stroke (AIS) when Stanford type A aortic dissection is suspected. Key clinical clues, including chest pain, inter-arm blood pressure discrepancy, or absent peripheral pulses should prompt consideration of alternative diagnoses to avoid the potentially fatal consequences of inappropriate rtPA administration.

When suspicion arises, computed tomography angiography (CTA) should be performed without delay to confirm the diagnosis. Prompt consultation with cardiothoracic surgery for emergency operative management can significantly reduce mortality in these high-risk patients.

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