CASE REPORT

Misdiagnosis of a Dissecting Aneurysm as a Cerebral Aneurysm: A Preoperative Case Report

Xiaohong Guo, MBBS; Yi Wu, MM; Junkang Fang, MBBS

ABSTRACT

Background • Intracranial Aneurysm (IA) is characterized by abnormal dilation of intracranial arterial walls, a tumor-like protrusion, often occurring in the anterior communicating artery. Intracranial Dissecting Aneurysm (IDA) refers to hemodynamic changes within intracranial arteries, leading to ruptures between blood vessel walls, disrupting normal arterial blood flow within the arterial lumen. IDA is relatively uncommon in the anterior circulation. To date, there have been no reported cases of dissecting aneurysms misdiagnosed as cerebral aneurysms before surgical intervention. This case report presents a patient's detailed clinical diagnosis, treatment, and imaging data.

Case Presentation • A 56-year-old female patient experienced post-work headaches. Cranial Computed Tomography (CT), Magnetic Resonance Imaging (MRI), and Digital Subtraction Angiography (DSA) examinations revealed a small hemorrhage following infarction and aneurysms in the initial part of the right A2 segment. Initially, the patient was diagnosed with a ruptured cerebral aneurysm, accompanied by hemorrhage and cerebral vasospasm (cerebral infarction in the right frontal lobe). Subsequently, cerebral aneurysm clipping was performed. During surgery, it was observed that the aneurysm originated from the ipsilateral A2 starting site and displayed dissecting-like changes extending towards the distal end. The final diagnosis confirmed an aneurysm evolving from intracranial artery dissection. Artificial meninges were employed to encase and clip the aneurysm. Post-surgery, the patient was transferred to a superior hospital for A3 bypass. Follow-up assessments indicated a successful recovery.

Conclusion • Cerebral aneurysms typically involve larger arteries with rare possibilities of stenosis. Moreover, the cerebral artery is relatively small, making it challenging for Brain Computed Tomography Angiography (CTA) to distinguish true and false lumen within blood vessels. The diagnosis of dissecting aneurysms is difficult and often susceptible to clinical misdiagnosis. (*Altern Ther Health Med.* 2023;29(8):663-667).

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BACKGROUND

Intracranial Aneurysm (IA) is characterized by a tumor-like protrusion resulting from abnormal dilation of the intracranial arterial wall. It stands as one of the most prevalent cerebrovascular diseases. Epidemiological studies indicate that the prevalence of IA in China is approximately 10%, which continues to escalate annually, surpassing the rate observed in Western countries.[1]

The Anterior Communicating Artery Aneurysm (AcoAA) ranks among the most prevalent aneurysms in the anterior circulation, comprising approximately 30%-37% of all intracranial aneurysms.[2] Notably, AcoAA exhibits a higher susceptibility to rupture, with Subarachnoid Hemorrhage (SAH) stemming from AcoAA rupture accounting for roughly 40% of non-traumatic subarachnoid hemorrhage cases.[3] Morphologically, anterior communicating aneurysms (ACA) can be categorized into cystic aneurysms, fusiform aneurysms, and dissecting aneurysms.

Due to the relatively short length of the communicating artery, aneurysms frequently develop at the bifurcation of the anterior cerebral artery and the anterior communicating artery, making cystic aneurysms the most prevalent among them. While AcoAA and dissecting aneurysms share certain common characteristics, the distinction between cystic aneurysms and dissecting aneurysms can be readily made based on their morphology.[1-3] To date, there have been no reported instances of dissecting aneurysms being misdiagnosed as cerebral aneurysms before surgical intervention.

Intracranial Dissecting Aneurysm (IDA) refers to a condition in which congenital factors lead to hemodynamic changes within the intracranial artery, including endothelial injury. This can result in ruptures occurring between the walls of blood vessels, causing the accumulation of blood and the formation of hematomas. These events often lead to luminal stenosis, occlusion, and the bulging of both inward and outward blood vessel walls, ultimately disrupting normal blood flow within the arterial lumen.[4]

IDA clinical presentations commonly include SAH and ischemic stroke. It is noted that approximately 50%-60% of IDA patients experience SAH as their primary symptom, while an additional 30%-78% of IDA patients exhibit symptoms related to ischemic stroke.[5] IDA predominantly manifest within the posterior circulation of the intracranial artery, comprising approximately 76%-93% of all IDA cases.[5,6] IDA occurrences in the anterior circulation are notably rare, with dissecting aneurysms in the ACA being particularly infrequent and sparsely documented [7, 8]. Despite its relatively low incidence, IDA poses a significant threat, as it is associated with elevated mortality and disability rates. IDA primarily affects individuals at a young age, with an average onset occurring around 30 years and predominantly afflicts males.

Therefore, we presented a comprehensive case report highlighting the diagnostic challenges, clinical manifestations, and treatment considerations of IDA occurring in the anterior communicating artery, contributing to a better understanding of this rare cerebrovascular condition. This study received approval from the Ethics Committee of Dongyang People's Hospital and informed written consent was obtained with the consent of the patient and her relatives.

Presented here is a case involving a 56-year-old female patient who experienced a sudden headache following work and was initially misdiagnosed with a cerebral aneurysm before undergoing surgery. It became evident upon surgical exploration that the aneurysm had its origin at the ipsilateral A2 starting site and exhibited characteristics resembling a dissecting aneurysm extending towards the distal end. Subsequently, the patient received a diagnosis of IDA.

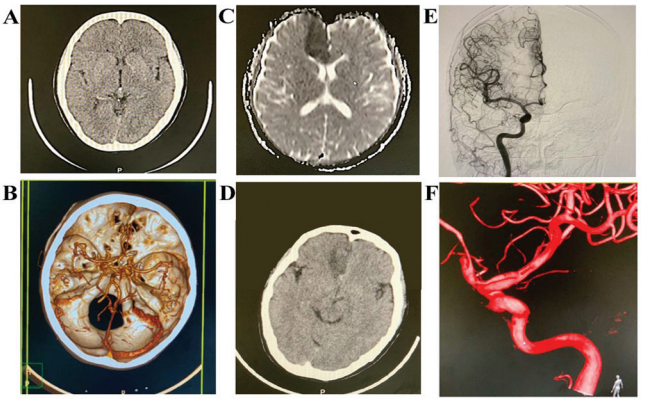
CASE PRESENTATION

A 56-year-old female patient presented to the emergency department with a complaint of persisting headache for 2 days. She had no family history of hypertension, diabetes, or heart disease and no history of smoking or alcohol consumption. The patient experienced a sudden headache at her workplace two days before admission. Initially, the pain was localized in the occipital area, characterized by a persistent throbbing sensation of mild intensity that the patient could tolerate. Following the resolution of occipital pain, she subsequently reported a frontal headache of similar intensity and nature without any accompanying discomfort.

Emergency Examination Findings

The results of the emergency examination revealed the following findings: White blood cell count (WBC): 21.35 × 10^9/L; Percentage of neutrophils (NEUR): 0.875; Hemoglobin (Hb): 192 g/L. Brain CT scans indicated a suspected subarachnoid hemorrhage, as depicted in Figure 1A-B. Further evaluation through brain and neck enhanced CTA revealed no apparent abnormalities.

Figure 1. Craniocerebral Imaging Findings



Note: CT scan of the brain suggested a suspected subarachnoid hemorrhage (A), and Brain CTA examination showed no apparent abnormality (B) at the onset of the case. (C): Four days after the onset of the disease, a brain MRI showed a new cerebral infarction in the frontal lobe and bilateral lacunar cerebral infarction. (D): Two weeks after the onset of the disease, a brain CT examination indicated that cerebral infarction had not progressed. (E and F): DSA examination revealed the formation of an anterior communicating aneurysm with a size of 5.7 × 1.9 × 2.8mm at the beginning part of right A2, accompanied by the formation of a 3.6mm ascus.

Admission and Initial Assessment

Subsequently, the patient was admitted to the Neurology Department of our hospital. Upon admission, a physical examination revealed normal tendon reflexes in the limbs (++), with no abnormalities detected during other neurological assessments. Routine symptomatic support treatment was administered.

Sudden Neurological Episode

On the fourth day of admission, the patient experienced sudden adverse movement in her left limbs. She could not walk unassisted, felt dizzy, and vomited a coffee-like substance twice. The patient self-reported getting out of bed and moving independently between 11:30 PM and 12 AM.

Physical Examination Findings

Physical examination revealed the following findings: Muscle strength in the left upper limb was graded as 4; Muscle strength in the left lower limb was graded as 3-; Muscle strength in the right limb was graded as 5; The left Babinski sign was positive; Other examinations showed no abnormalities.

Neuroimaging and Diagnosis

A Craniocerebral CT examination was conducted, which showed no evidence of bleeding. Subsequently, a Craniocerebral MRI revealed recent cerebral infarction in the frontal lobe and lacunar cerebral infarction in bilateral

frontal lobes (see Figure 1C for CT and MRI images). As a result, aspirin was added to the treatment regimen for antiplatelet therapy.

Lumbar Puncture Examination

A lumbar puncture examination was conducted on the fifth day following admission, yielding three tubes of uniform hemorrhagic cerebrospinal fluid with a cerebrospinal fluid pressure of 75 mmH_2O. The cerebrospinal fluid analysis revealed the following results: color: light red; nucleated cell count: 5/μl; cerebrospinal fluid direct smear: RBC: >30, WBC: 0-1/HP; and predominant normal erythrocyte morphology.

Diagnostic Implications

These findings strongly suggested subarachnoid hemorrhage, potentially indicating an aneurysm rupture. Consequently, aspirin treatment was discontinued, and conventional conservative care was initiated.

Follow-up Imaging and Diagnosis Confirmation

A brain CT scan (Figure 1D) was reviewed two weeks later, and the interventional department conducted a DSA examination (Figure 1E-F). These assessments revealed the presence of an anterior communicating aneurysm measuring 5.7 × 1.9 × 2.8 mm, accompanied by the formation of a 3.6 mm sacculus. The patient was found to have a small amount of bleeding following infarction and an aneurysm located at the beginning of the right A2 segment. These findings established the diagnosis of cerebral aneurysm rupture with concomitant cerebral vasospasm (resulting in cerebral infarction in the right frontal lobe). Consequently, the patient was transferred to the neurosurgery department for aneurysm clipping.

Intraoperative Discovery

During the surgical procedure, it was observed that the aneurysm originated from the ipsilateral starting site of A2 and exhibited a dissect-like change extending to the distal end. The presence of an aneurysm with dissection evolution was confirmed, particularly through indocyanine green fluorescence angiography. Due to the complexity of the case, simple clipping was deemed challenging. Artificial meninges were employed to encapsulate and secure the aneurysm, followed by clipping (Figure 2A-B). Post-surgery, the patient was transferred to the ICU for further care.

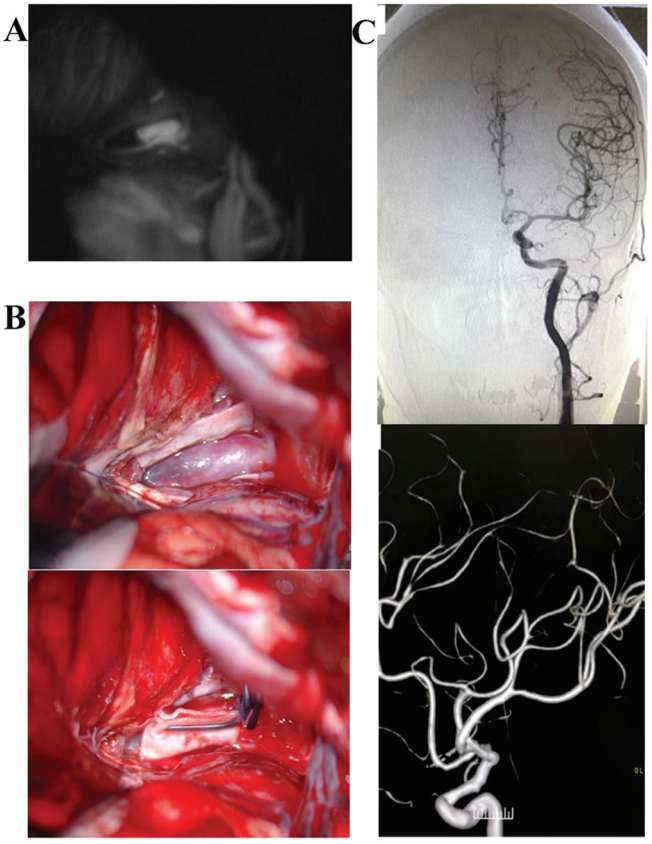
Post-Surgery CT Review and Hematoma Formation

On the second day following surgery, a CT review indicated the presence of hematoma formation in the surgical site, accompanied by an elevated level of intracranial hemorrhage. Remarkably, the patient maintained normal consciousness, prompting her family to request conservative treatment.

Emergent Surgical Intervention

During the afternoon, the patient exhibited a decline in mental clarity and unequal pupil size. Subsequent re-examination via CT scan revealed a linear, left displacement in the brain. Consequently, an emergency procedure was initiated to address the right intracranial hematoma, necessitating bone flap removal and dural repair. Following this operation, the patient's condition stabilized, leading to her transfer to a more specialized facility for an A3 bypass procedure (see Figure 2C).

Figure 2. The Patient Underwent Aneurysm Clipping Surgery



Note: (A): During the operation, it was found to be an A2 aneurysm with dissection evolution, which was confirmed by indocyanine green fluorescein angiography. (B): Artificial meninges were used to wrap the aneurysm and clamp the aneurysm. (C): A3 bypass was performed in a superior hospital.

Successful Recovery and Current Status

The patient's recovery post-surgery has been remarkable. Presently, she maintains full consciousness and exhibits normal limb function. Notably, there are no signs of hemiplegia, intellectual or psychological impairment, and her daily life remains unaffected.

DISCUSSION

The pathogenesis of IDA remains enigmatic, potentially linked to the absence of exogenous and endogenous factors contributing to injury and repair mechanisms. Existing literature suggests that IDA's pathogenesis primarily stems from the unique histological characteristics of the

cerebrovascular system. Compared to extracranial arteries, intracranial arteries lack an external elastic layer, possess a thicker internal elastic layer, exhibit fewer elastic fibers within the intermediate membrane, and feature a thinner outer membrane.[9]

According to Mizutani's classification of dissection,[10] it can be assumed that distinct pathological mechanisms underlie the occurrence of SAH, ischemic symptoms, or symptoms related to the space-occupying effects induced by dissection. When SAH arises due to a ruptured intracranial dissection aneurysm, it manifests as a "bursting" headache, often accompanied by symptoms such as vomiting. In cases of vertebrobasilar dissection, SAH has an incidence rate ranging from 61% to 69%, with aneurysmal dilation being the most prevalent presentation.[9,11,12] Additionally, 20% to 65% of intracranial carotid artery dissections result in SAH, while 12% to 75% of middle cerebral artery dissections lead to SAH.[9,13,14]

The symptoms of cerebral infarction resulting from intracranial dissection closely resemble those induced by other causes. They typically manifest as non-specific symptoms, including dizziness, vertigo, headache, and neck pain.[15] However, anterior circulation dissections tend to present with ischemic symptoms more frequently. Li et al.[16] reported that out of 11 patients with anterior circulation dissecting aneurysms, 8 cases (72.7%) presented with cerebral ischemic symptoms. Notably, spontaneous IDA accounted for 64.3% of cases in studies concerning anterior cerebral artery infarction.[17]

In the presented case, the patient exhibited prodromal symptoms consistent with a "bursting" headache, along with CT examination-suggested subarachnoid hemorrhage. Furthermore, cerebral infarction occurred at atypical sites in the later stages of the condition. Early lumbar puncture examination is recommended to aid in clinical assessment to elucidate the nature of the cerebrospinal fluid.

In recent years, there has been significant progress in imaging technology. MRI, Magnetic Resonance Angiography (MRA), CT, Computed Tomography Angiography (CTA), and Digital Subtraction Angiography (DSA) have gained prominence in diagnosing IDA. MRI/MRA and CT/CTA are the primary modalities employed for preliminary screening and subsequent reexamination of IDA. These techniques not only facilitate the detection of both extravascular and intracavitary abnormalities but also offer the advantage of being less invasive compared to DSA.[18] While MRI can effectively identify intramural hematomas in 32% of patients diagnosed with DSA, it is important to note that DSA remains the gold standard for diagnosing IDA. This preference for DSA persists due to its higher diagnostic accuracy despite its lower sensitivity.[19]

The radiological characteristics of IDA can vary depending on clinical presentation, making the establishment of a uniform radiological standard challenging. However, certain characteristic features serve as diagnostic indicators. These features encompass the presence of double lumen signs (indicating pseudolumen, intramural hematoma, or intimal flap), a string of pearl signs (manifesting as alternating lumen widening and narrowing), morphological alterations in affected segments evident in continuous imaging, and the retention of contrast agent within these affected segments.[18,20]

Other indicators associated with IDA encompass irregular stenosis (recognized as the thin line sign), abrupt narrowing or occlusion, localized fusiform dilation, and aneurysms occurring at a distance from arterial bifurcations.[18,21] However, the double cavity sign holds particular significance as the primary indicator of IDA.[15,18] In the case under consideration, the cerebral aneurysm identified via DSA was conjoined with the responsible vessel, specifically the initial segment of A2. Moreover, the distal portion of this fusion (distal A2) exhibited narrowing. Notably, it is essential to consider the possibility of intracranial arterial dissection in cases involving infarction or hemorrhage at corresponding positions on CT scans.

Cystic aneurysms typically originate at arterial bifurcations. Their occurrence is contingent on several factors, including the degradation or breakdown of the internal elastic layer within the affected vessels, the absence of a medial membrane, or the presence of an abnormal fibrous structure in the medial membrane at the intracranial artery bifurcation. These factors collectively weaken the local vascular wall, facilitating the development of tumor-like protrusions over extended periods of blood flow impact. These aneurysms typically present as spherical formations with a nodular neck, resembling the appearance of a berry.[21,22]

IDA result from the intimal tear within the arterial wall and the subsequent abrupt rupture of the internal elastic layer.[20] Blood within the vessel's lumen enters this tear, leading to the formation of an intramural hematoma. This hematoma progressively expands along the blood vessel's long axis. On MRI, it may appear oval in shape. However, it is important to distinguish that the tumor-like bulge created by IDA lacks an obvious nodular neck. On the other hand, DSA can reveal abnormal dilation and curvature of the lesion's lumen, which is differentiated from cystic aneurysms in terms of morphology.

In the presented case, the dissecting aneurysm manifested within the A2 segment, with DSA examination indicating the formation of an anterior communicating aneurysm accompanied by ascus development. Upon surgical exploration, dissecting changes were observed within the A2 segment, ultimately confirming the diagnosis of a dissecting aneurysm. It is worth noting that the anterior communicating artery is characterized by its complex anatomy, developmental variations, and deep location. Consequently, the occurrence of dissecting aneurysms in the anterior communicating artery is exceedingly rare, rendering their diagnosis significantly challenging.

Cerebral aneurysms typically involve larger arteries, and the occurrence of stenosis is relatively rare in these cases. Conversely, cerebral arteries are comparably smaller, rendering it challenging for brain CTA to differentiate between the true and false lumens of blood vessels. Moreover, the diagnosis of dissecting aneurysms poses inherent difficulties, making them susceptible to misdiagnosis in

clinical practice. Therefore, the early-stage diagnosis of intracranial dissection aneurysms can be quite challenging based solely on clinical symptoms. It becomes critical to promptly conduct CT/CTA and MRI/MRA examinations to address this issue. Additionally, DSA examinations should also be considered for a comprehensive evaluation.

CONCLUSION

In conclusion, this case report underscores the rarity and diagnostic challenges associated with IDA, particularly when they occur in the anterior communicating artery. The intricate relationship between clinical symptoms and imaging findings necessitates a multidimensional diagnostic approach, incorporating CT/CTA, MRI/MRA, and DSA examinations. This case highlights the critical need for timely and accurate diagnosis to inform appropriate therapeutic strategies. As the medical community continues to unravel the complexities of IDA, further research and clinical experience will undoubtedly refine our understanding, leading to enhanced diagnostic precision and improved treatment outcomes for patients facing this enigmatic cerebrovascular condition.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

This study was approved by the Ethics Committee of Dongyang People's Hospital. Informed written consent was issued with the consent of the patient and her relatives.

CONSENT FOR PUBLICATION

Not applicable.

AVAILABILITY OF DATA AND MATERIAL

All data generated or analyzed during this study are included in this. Further enquiries can be directed to the corresponding author.

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The authors have not received any funding support.

CONFLICTS OF INTEREST

The authors declare no conflict of interest in this study.

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ABBREVIATIONS

CT, Computed Tomography; MRI, Magnetic Resonance Imaging; DSA, Digital Subtraction Angiography; CTA, Computed Tomography Angiography; IA, Intracranial Aneurysm; AcoAA, Anterior Communicating Artery Aneurysm; SAH, Subarachnoid Hemorrhage; IDA, Intracranial Dissecting Aneurysm; ACA, Anterior Cerebral Artery; WBC, White Blood Cell Count; NEUR, Neutrophils; Hb, Hemoglobin; MRA, Magnetic Resonance Angiography.

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