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Moyamoya Disease Masquerading as a Transient Ischemic Attack: The Importance of Early Vascular Imaging

Muhammad Ali Akhtar [1], Muhammad Saleem [1], Thair Dawood [1], Huda Marcus [1]

1. Internal Medicine, Hurley Medical Center/Michigan State University, Flint, USA

Corresponding author: Muhammad Ali Akhtar, doc.aliakhtar@gmail.com

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Abstract

Moyamoya disease is a rare progressive occlusive cerebrovascular disorder characterized by stenosis of intracranial arteries and compensatory collateral formation. Adult presentations most commonly include ischemic or hemorrhagic stroke, while transient ischemic attack (TIA) is less frequently reported. A 35-year-old woman presented with transient dizziness, dysarthria, left-sided weakness, and numbness lasting approximately one minute. Initial CT and MRI were unremarkable. CT angiography revealed near occlusion of the proximal left M1 segment. Diagnostic cerebral angiography demonstrated characteristic collateral vessels consistent with unilateral moyamoya disease (Suzuki stage II). Incidental nonfunctional pituitary enlargement was also identified. The patient was managed conservatively with antiplatelet therapy and outpatient follow-up. This case highlights the importance of early vascular imaging in patients presenting with transient neurological symptoms and normal MRI findings. Moyamoya disease should remain in the differential diagnosis for young adults with suspected TIA.

Categories: Neurology, Radiology, Medical Education
Keywords: angiography, cerebrovascular stenosis, moyamoya disease, transient ischemic attack, unilateral moyamoya

Introduction

Moyamoya disease is a rare progressive cerebral occlusive vasculopathy. It is characterized by progressive stenosis of the terminal internal carotid and proximal middle and anterior cerebral arteries, with the formation of collateral networks that produce a characteristic "puff of smoke" appearance on cerebral angiography [1]. It has a higher prevalence in East Asia, and the incidence is 0.1-0.5 per 100,000 in Western populations. It exhibits a bimodal age distribution, affecting children around the age of 10 and adults in their 30s and 40s [2].

Pediatric populations mostly present with ischemic events, and the disease in adults often presents as ischemic or hemorrhagic stroke; however, transient ischemic attacks (TIAs) have also been reported [3,4]. Diagnosis is primarily based on radiologic studies such as MRI/MRA or digital subtraction angiography. The unilateral variant of moyamoya is relatively uncommon and poorly understood. Approximately 10-15% of patients may initially present with unilateral disease, and progressive development of bilateral disease has been reported in some cases [5]. Early recognition of the disease is crucial for timely management.

We describe the case of a 35-year-old woman who presented with symptoms consistent with a TIA and was ultimately diagnosed with unilateral left-sided moyamoya disease. There was a concurrent nonfunctional pituitary adenoma identified on imaging, which could be an incidental finding. However, a rare association between moyamoya and functional and nonfunctional pituitary enlargement has been previously reported [6].

This case report emphasizes the diagnostic importance of early vascular imaging in adults presenting with TIAs, including consideration of moyamoya disease in the differential diagnosis of patients with TIA.

Case Presentation

A 35-year-old woman presented to the emergency department (ED) for evaluation of an episode of sudden-onset dizziness, numbness, right-sided weakness, and dysarthria. The episode lasted approximately one minute and was associated with a headache that lasted several hours; however, she was asymptomatic at the time of presentation. The patient's past medical history was significant for hypertension and a remote history of seizures, for which she was not taking any antiseizure medications.

Upon arrival at the ED, the patient was hypertensive, with a blood pressure of 153/96 mmHg, and otherwise had stable vital signs. She was alert and oriented, and no focal neurological deficits were appreciated on physical examination. The initial Glasgow Coma Scale (GCS) score was 15, and the National Institutes of

Health Stroke Scale (NIHSS) score was 0. A noncontrast computed tomography (CT) scan of the head was unremarkable. Neurology evaluated the patient, and she received a loading dose of aspirin. Computed tomography angiography (CTA) of the head and neck showed a focal area of critical stenosis and near occlusion involving the proximal M1 segment; however, no sizable aneurysm or arteriovenous (AV) malformation was seen (Figure *1*). MRI of the head was negative for acute intracranial hemorrhage or infarct. Neurology and interventional neurology evaluated the patient. The episode was treated as a TIA, and the patient was started on antiplatelet therapy.

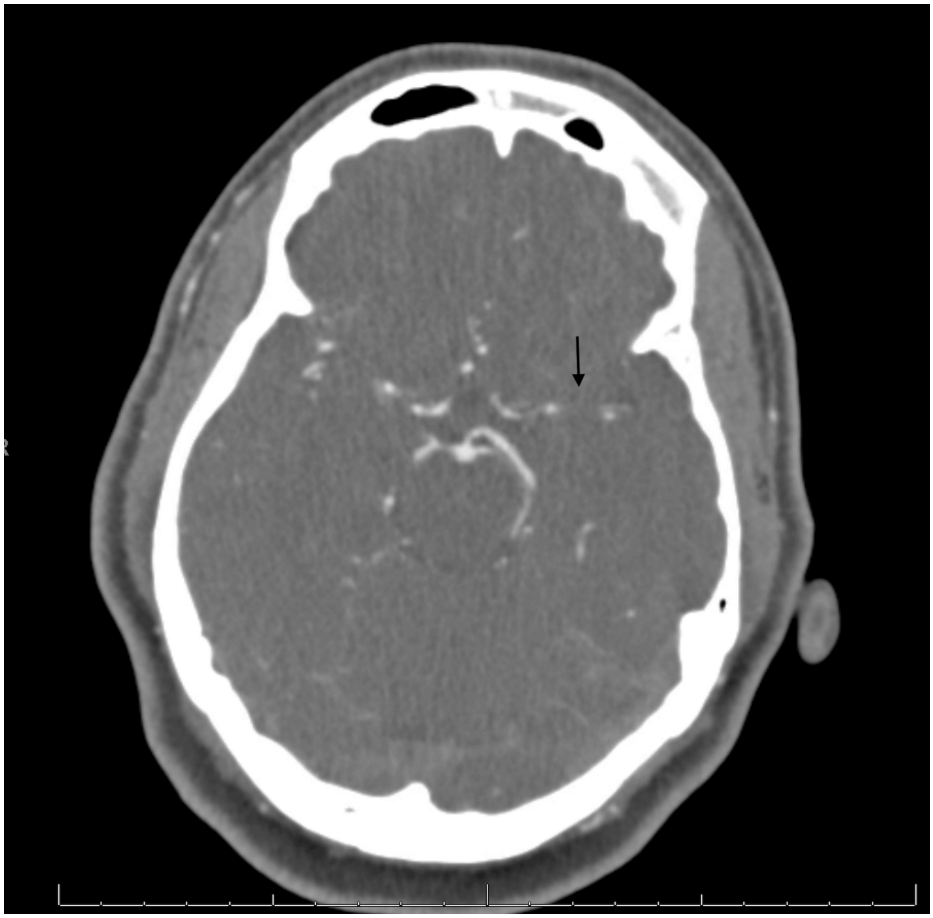


FIGURE 1: CTA head and neck showing focal area of critical stenosis/near occlusion seen involving the proximal M1 segment of left MCA

CTA: computed tomography angiography; MCA: middle cerebral artery.

Neurosurgery was consulted for further evaluation, and the patient underwent a diagnostic cerebral angiogram, which demonstrated characteristic narrowing of the left M1 segment with fine basal collateral vessels consistent with unilateral moyamoya disease, Suzuki stage II (Figure *2*). No intervention was performed, and conservative management was advised.

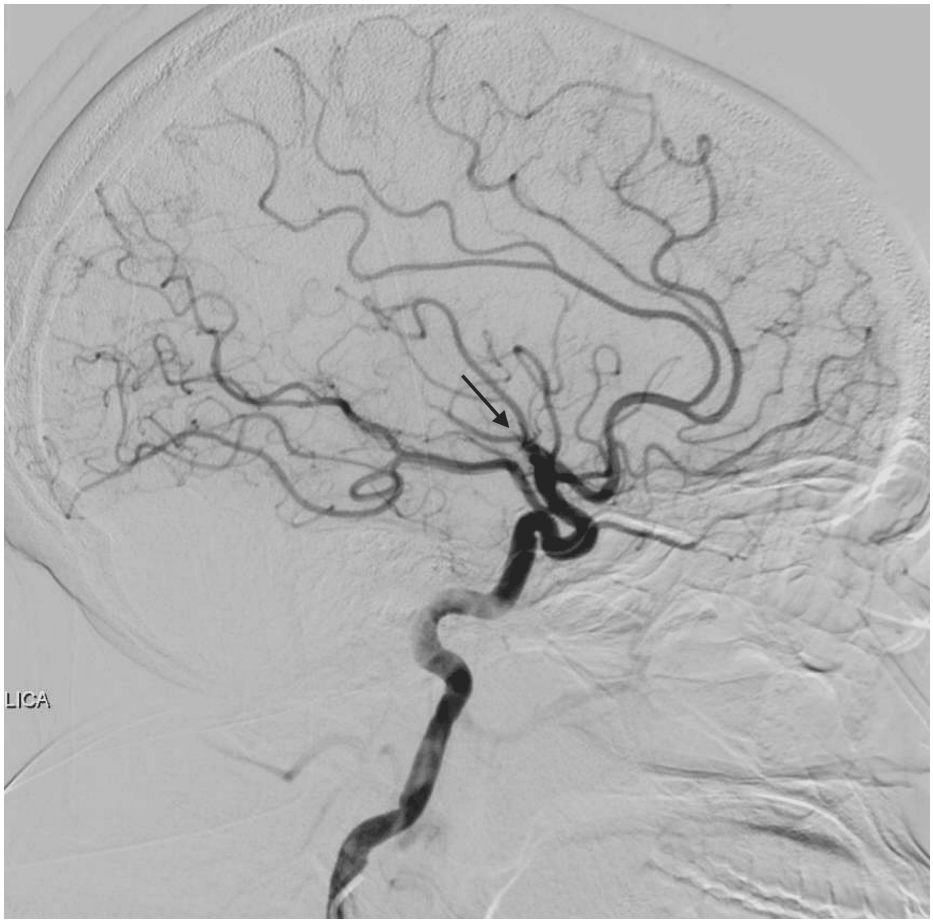


FIGURE 2: Diagnostic cerebral angiogram of the left internal carotid artery demonstrating vascular findings consistent with moyamoya angiopathy, including prominent lenticulostriate collateral vessels and preserved caliber of the distal middle cerebral artery branches

Hematology and endocrinology also evaluated the patient. Comprehensive hypercoagulable testing was performed, including factor V Leiden mutation, lupus anticoagulant, protein C and S, and antithrombin III, all of which were negative. An incidental sellar mass was noted on MRI, which was identified as a homogeneous pituitary enlargement measuring 1.3 × 1 cm with superior convexity, without compression of the optic chiasm or deviation of the infundibulum. Endocrine workup, which included prolactin, luteinizing hormone (LH), follicle-stimulating hormone (FSH), growth hormone (GH), insulin-like growth factor (IGF), thyroid-stimulating hormone (TSH), cortisol, and adrenocorticotropic hormone (ACTH), was negative for any evidence of pituitary hypersecretion.

The patient remained neurologically stable and was discharged on dual antiplatelet therapy. The patient was advised to follow up with endocrinology, neurosurgery, and neurology.

Discussion

Moyamoya angiopathy is a progressive steno-occlusive cerebrovascular disorder with variable clinical presentations, including TIAs, ischemic stroke, intracranial hemorrhage, and headache [3,4]. Although TIAs are a recognized manifestation, their transient nature and the absence of acute infarction on conventional brain MRI may complicate the diagnostic evaluation. The absence of acute ischemic changes on MRI does not exclude underlying cerebrovascular pathology, emphasizing the importance of clinical suspicion and appropriate vascular imaging.

In our patient, brain MRI did not demonstrate acute ischemic injury, whereas CT angiography (CTA) identified severe stenosis involving the proximal left M1 segment of the middle cerebral artery. Subsequent diagnostic cerebral angiography demonstrated associated lenticulostriate collateral vessels, raising concern for moyamoya angiopathy [1,2]. These findings illustrate the complementary roles of parenchymal imaging, noninvasive vascular imaging, and catheter angiography. However, intracranial arterial stenosis alone is insufficient to establish a diagnosis of primary moyamoya disease. The diagnosis requires characteristic

angiographic findings and careful exclusion of alternative etiologies, including intracranial atherosclerosis, inflammatory vasculopathy, arterial dissection, and other steno-occlusive disorders. Moyamoya syndrome refers to similar vascular abnormalities occurring in association with underlying conditions such as autoimmune disorders, hematological diseases, neurofibromatosis type 1, and prior cranial irradiation. In our patient, the hypercoagulable evaluation, including factor V Leiden mutation, lupus anticoagulant, protein C and S, and antithrombin III, was negative. However, these investigations do not comprehensively exclude all secondary causes of moyamoya angiopathy.

Unilateral moyamoya disease is a recognized but less commonly reported presentation [5]. Although its underlying pathophysiology remains incompletely understood, proposed mechanisms include genetic predisposition, asymmetric vascular involvement, and early-stage disease that may subsequently progress to bilateral involvement [2,5,7]. Progression from unilateral to bilateral disease has been documented, although the frequency and timing vary among reported cohorts [5,7]. These findings emphasize the importance of long-term clinical surveillance and follow-up vascular imaging. Repeat cerebral angiography was recommended for our patient to evaluate for disease progression; however, it had not been performed at the time of manuscript preparation.

Management of moyamoya angiopathy is individualized according to the clinical presentation, vascular anatomy, and cerebral hemodynamic status. Surgical revascularization is an important treatment option for selected symptomatic patients, particularly those with recurrent ischemic events or evidence of impaired cerebral perfusion. Antiplatelet therapy may be considered for ischemic presentations, although evidence supporting specific medical regimens remains limited [3,8]. In our patient, neurosurgical evaluation was performed, and no immediate intervention was recommended following diagnostic angiography. The patient remained neurologically stable and was discharged on dual antiplatelet therapy with recommendations for outpatient neurological and neurosurgical follow-up. Continued surveillance is important to assess for recurrent symptoms or progression of the underlying vascular disease.

An additional finding in our patient was incidental pituitary enlargement. Rare cases describing the coexistence of moyamoya disease and pituitary adenomas, including functional and nonfunctioning lesions, have been reported [6,9]. Although shared vascular or inflammatory mechanisms have been proposed, the available evidence is limited, and a causal association has not been established [9,10]. In our patient, MRI demonstrated homogeneous pituitary enlargement measuring approximately 1.3 × 1.0 cm without optic chiasm compression or infundibular deviation. Endocrine evaluation did not reveal evidence of pituitary hormone hypersecretion. Although a nonfunctioning pituitary adenoma was considered, the exact nature of the enlargement was not definitively established. Given the absence of associated hormonal or compressive manifestations, the finding was considered likely incidental. Further endocrinological follow-up was recommended for continued evaluation and monitoring.

Conclusions

In conclusion, our case highlights the importance of considering moyamoya disease in the differential diagnosis of adults presenting with transient neurological symptoms. It emphasizes the importance of performing vascular imaging, such as CTA and MRA, in patients with suspected cerebrovascular ischemia despite the absence of acute infarction on brain MRI.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Muhammad Ali Akhtar, Muhammad Saleem, Thair Dawood, Huda Marcus

Acquisition, analysis, or interpretation of data: Muhammad Ali Akhtar, Muhammad Saleem, Thair Dawood, Huda Marcus

Drafting of the manuscript: Muhammad Ali Akhtar, Muhammad Saleem, Thair Dawood, Huda Marcus

Critical review of the manuscript for important intellectual content: Muhammad Ali Akhtar, Muhammad Saleem, Thair Dawood, Huda Marcus

Supervision: Thair Dawood, Huda Marcus

Disclosures

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