American Journal of Case Reports

e-ISSN 1941-5923
© Am J Case Rep, 2026; 27: e954918
DOI: 10.12659/AJCR.954918

Received: 2026.07.22
Accepted: 2026.10.07
Available online: 2026.10.28
Published: 2026.XX.XX

Side-Locked Migraine With Cranial Autonomic Symptoms in the Setting of a Duplicated Middle Cerebral Artery and Contralateral Cerebral Venous Sinus Hypoplasia: A Case Report

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Financial support: This study was supported by the Yunnan Provincial Science and Technology Department (Grant No. 202203AC100007-2)
Conflict of interest: None declared

Patient: Female, 30-year-old
Final Diagnosis: Migraine
Symptoms: Ipsilateral lacrimation • conjunctival injection • nasal congestion • nausea • nocturnal awakening • phonophobia • photophobia • rhinorrhea • side-locked left temporal-periorbital pulsating headache • vomiting
Clinical Procedure: —
Specialty: Neurology
Objective: Congenital defects / diseases
Background: Duplicated middle cerebral artery (DMCA) is an uncommon congenital arterial variant. Its relationship to primary headache is uncertain, and cranial autonomic symptoms in migraine may complicate its differentiation from trigeminal autonomic cephalalgias.
Case Report: A 30-year-old woman had a 15-year history of strictly left temporal-periorbital pulsating headaches, typically occurring approximately once monthly and lasting 2 to 72 hours, accompanied by nausea, vomiting, photophobia, and phonophobia. After sleep disruption, the headaches progressed to a daily nocturnal pattern lasting 3 weeks. The duration of individual nocturnal attacks during this recent phase was not reliably documented. The attacks awakened her from sleep and were accompanied by ipsilateral lacrimation, conjunctival injection, nasal congestion, and rhinorrhea. Magnetic resonance angiography revealed a left Teal type A DMCA, and magnetic resonance venography revealed right transverse-sigmoid sinus hypoplasia with left-dominant venous drainage. No parenchymal lesion, aneurysm, arterial stenosis, hemorrhage, epileptiform abnormality, or cardioembolic source was identified. The clinical diagnosis was migraine with a recent daily exacerbation and prominent cranial autonomic symptoms. Treatment with flunarizine, low-dose amitriptyline, and gabapentin, as well as lifestyle modifications, was followed by substantial improvement. At 3 months, headache frequency had decreased to fewer than 1 attack per month, and the Headache Impact Test-6 score had improved from 58 to 46.
Conclusions: Migraine with prominent cranial autonomic symptoms can mimic trigeminal autonomic cephalalgia. Coexisting arterial and venous variants may be incidental. This single case does not establish causality or the general diagnostic utility of detailed vascular imaging.

Keywords: Migraine Disorders • Duplicated Middle Cerebral Artery • Migraine • Cranial Autonomic Symptoms • Venous Sinus Hypoplasia • Case Report
Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/954918

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Introduction

Duplicated middle cerebral artery (DMCA) is an uncommon developmental variant in which an additional arterial trunk arises from the internal carotid artery and courses toward the Sylvian fissure. Teal et al distinguished DMCA from accessory middle cerebral artery (MCA): the former arises from the internal carotid artery, whereas the latter arises from the anterior cerebral artery [1]. Subsequent classifications further characterized type A DMCA as arising from the terminal internal carotid artery and running parallel to the main MCA trunk [2,3]. Most DMCAs are detected incidentally on vascular imaging; current evidence does not establish isolated DMCA as a direct cause of migraine, cranial autonomic symptoms, or any specific primary headache syndrome [2,3]. Transverse-sigmoid sinus hypoplasia may also represent an anatomical venous variant on magnetic resonance venography (MRV). Its relationship to the headache phenotype may be uncertain when there is no venous sinus thrombosis, papilledema, or other evidence of increased intracranial pressure [4]. Migraine is diagnosed clinically according to the International Classification of Headache Disorders, 3rd edition [5]. Some intracranial vascular variants may modulate migraine susceptibility or phenotype, but the strength and direction of these associations remain uncertain [6]. Cranial autonomic symptoms may occur in migraine and mimic trigeminal autonomic cephalalgias, particularly in patients with unilateral or high-burden attacks [7-9]. Here, we describe a patient with a long-standing side-locked migraine-like headache, a recent daily nocturnal exacerbation, and ipsilateral cranial autonomic symptoms, in whom magnetic resonance angiography (MRA) and MRV revealed an ipsilateral DMCA and contralateral venous sinus hypoplasia. This case is reported in accordance with the CARE framework [10].

Case Report

A 30-year-old woman presented with a 15-year history of recurrent left-sided pulsating headaches and a 3-week history of recent worsening with nocturnal awakening. Since adolescence, the attacks had been centered in the left temporal and periorbital regions; typically occurred approximately once monthly; lasted 2 to 72 hours; and were associated with nausea, vomiting, photophobia, and phonophobia. She reported no visual aura, focal sensory symptoms, limb weakness, or speech disturbances. Emotional stress could precipitate attacks. She had previously been diagnosed with migraine at other hospitals and had intermittently used unspecified analgesics. Three

Table 1. Timeline of clinical course, diagnostic assessment, therapeutic interventions, and follow-up outcomes.

Time point	Clinical events	Diagnostic assessment	Intervention	Outcome
Adolescence (~ 15 years before admission) onward	Recurrent left temporal-periorbital pulsating headaches, occurring approximately once monthly, lasting 2-72 hours, and associated with nausea, vomiting, photophobia, and phonophobia; no aura or focal neurological symptoms	Migraine diagnosed at other hospitals	Intermittent use of unspecified analgesics	Recurrent episodic attacks
3 years before admission	Transient increase in headache frequency	Not specified	Symptomatic treatment at other hospitals	Attack frequency subsequently decreased
3 weeks before admission	Sleep disruption during a holiday period, followed by daily nocturnal attacks. Pain awakened the patient 1-2 hours after sleep onset, usually between 1: 00 AM and 3: 00 AM. The duration of individual nocturnal attacks during this 3-week period was not reliably documented. VAS score, 9/10; ipsilateral lacrimation, conjunctival injection, nasal congestion, and rhinorrhea	Clinical evaluation at another hospital	Rizatriptan 5 mg as needed and gabapentin 0.1 g three times daily for 1 week	No clear improvement in attack frequency or severity

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Table 1 continued. Timeline of clinical course, diagnostic assessment, therapeutic interventions, and follow-up outcomes.

Time point	Clinical events	Diagnostic assessment	Intervention	Outcome
At admission	Persistent daily nocturnal side-locked headaches with prominent ipsilateral cranial autonomic symptoms; baseline HIT-6 score, 58	Head CT, video EEG, ECG, transthoracic echocardiography, and brain MRI revealed no alternative structural, epileptiform, or cardioembolic cause. MRA revealed a left duplicated middle cerebral artery; MRV revealed right transverse-sigmoid sinus hypoplasia with left-dominant venous drainage. No aneurysm or arterial stenosis was detected. Clinical diagnosis: migraine with a recent daily exacerbation and prominent cranial autonomic symptoms; trigeminal autonomic cephalalgia was considered less likely	Migraine-oriented preventive therapy planned	No alternative secondary structural cause identified
After treatment initiation	Preventive treatment and lifestyle modifications initiated after diagnostic evaluation	—	Flunarizine 5 mg nightly, amitriptyline 6.25 mg nightly, and gabapentin 0.1 g three times daily; sleep regularization, smoking cessation advice, alcohol restriction, and counseling regarding vascular findings	—
1-week follow-up	Nocturnal awakenings resolved	Clinical follow-up	Continued preventive treatment and lifestyle modifications	Daytime headache frequency decreased to fewer than 1 attack per week; pain intensity decreased to a VAS score of 3/10
3-month follow-up	Clinically stable	Follow-up complete blood count and liver and renal function test results were normal	Continued follow-up and lifestyle modifications	Fewer than 1 attack per month; no rescue medication required; no serious adverse reactions; HIT-6 score improved from 58 to 46

Abbreviations: CT, computed tomography; ECG, electrocardiography; EEG, electroencephalography; HIT-6, Headache Impact Test-6; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging; MRV, magnetic resonance venography; VAS, visual analog scale.

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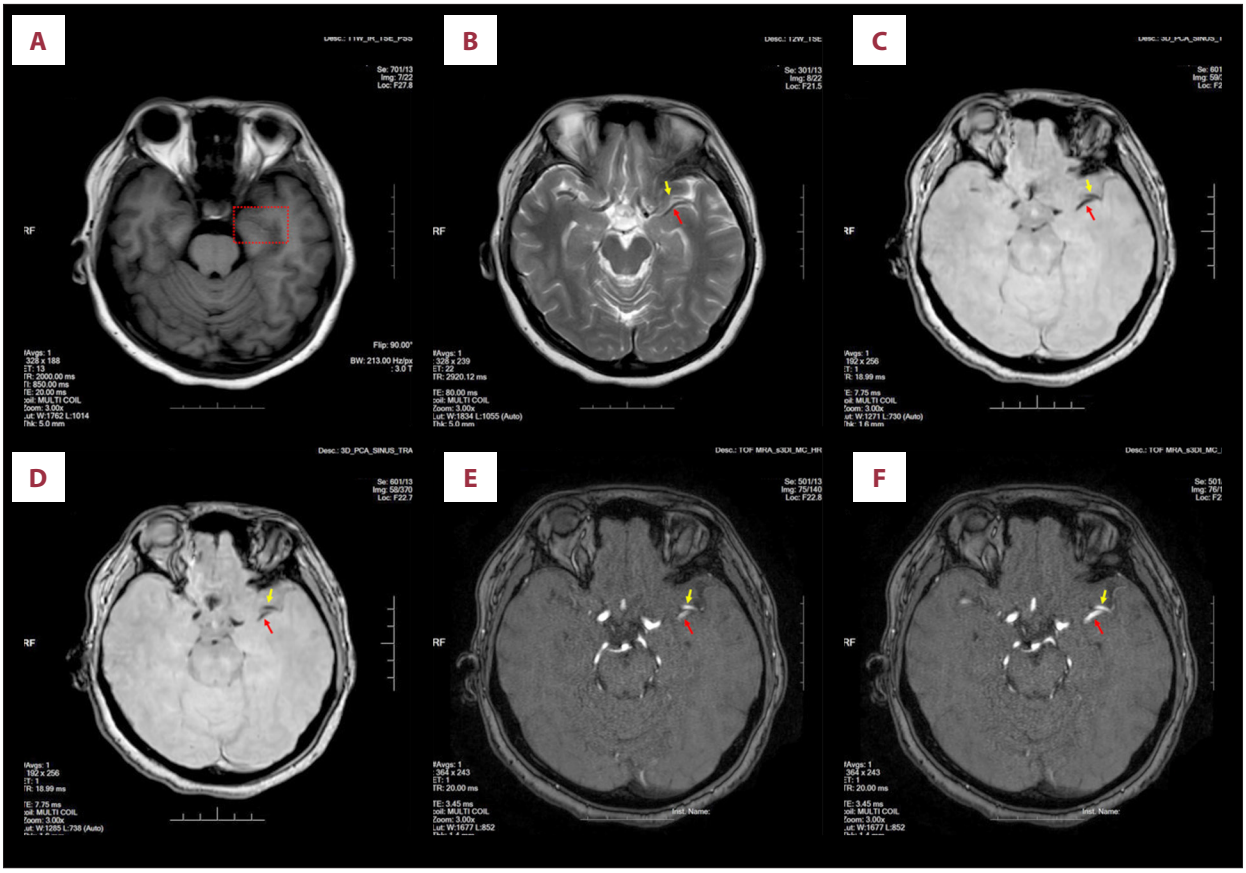


Figure 1. Brain MRI and 3-dimensional time-of-flight MRA demonstrating the left duplicated MCA. (**A**) Axial T1-weighted MRI shows no focal parenchymal lesion in the left temporal-periorbital region. (**B**) Axial T2-weighted MRI shows vascular flow voids in the left Sylvian region without adjacent structural abnormalities. (**C, D**) Axial angiographic source images further demonstrate 2 left MCA trunks coursing toward the Sylvian fissure. (**E, F**) Axial 3-dimensional time-of-flight MRA source images demonstrate the duplicated left MCA trunks arising near the terminal left internal carotid artery and running parallel to each other. Colored arrows indicate the duplicated arterial trunks. Panels **A-F** were rearranged for publication layout only; image content was not altered. Abbreviations: MCA, middle cerebral artery; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging.

years earlier, she had experienced a transient increase in attack frequency that subsequently resolved. The clinical course is summarized in **Table 1**.

Three weeks before the current presentation, after a holiday-related disruption of her sleep schedule, the headaches recurred and progressed to a daily nocturnal pattern. Attacks typically awakened her 1 to 2 hours after sleep onset, most often between 1: 00 AM and 3: 00 AM, with severe left temporal-orbital pain rated 9/10 on the visual analog scale. The patient could reliably recall the timing and daily frequency of these recent attacks, but the duration of individual nocturnal attacks during this 3-week period was not reliably documented. During severe attacks, she reported ipsilateral conjunctival injection, lacrimation, nasal congestion, and rhinorrhea. Acetaminophen was ineffective. A 1-week course of rizatriptan 5 mg as needed and gabapentin 0.1 g three times daily, prescribed at another hospital, did not clearly reduce attack frequency or severity. Her baseline Headache Impact Test-6 (HIT-6) score was 58.

She had no history of hypertension, diabetes mellitus, head trauma, stroke, epilepsy, or known intracranial disease. She reported poor sleep quality, difficulty initiating sleep, and frequent dreaming. She smoked 2 to 3 cigarettes daily for approximately 5 years and consumed alcohol approximately once weekly. A strong maternal family history of migraine was reported involving her mother and several maternal relatives. Vital signs were normal, and neurological examination was unremarkable. Funduscopic examination revealed no papilledema, and the Valsalva maneuver did not exacerbate the pain.

Head computed tomography revealed no hemorrhage or mass lesion. Video electroencephalography findings were normal.

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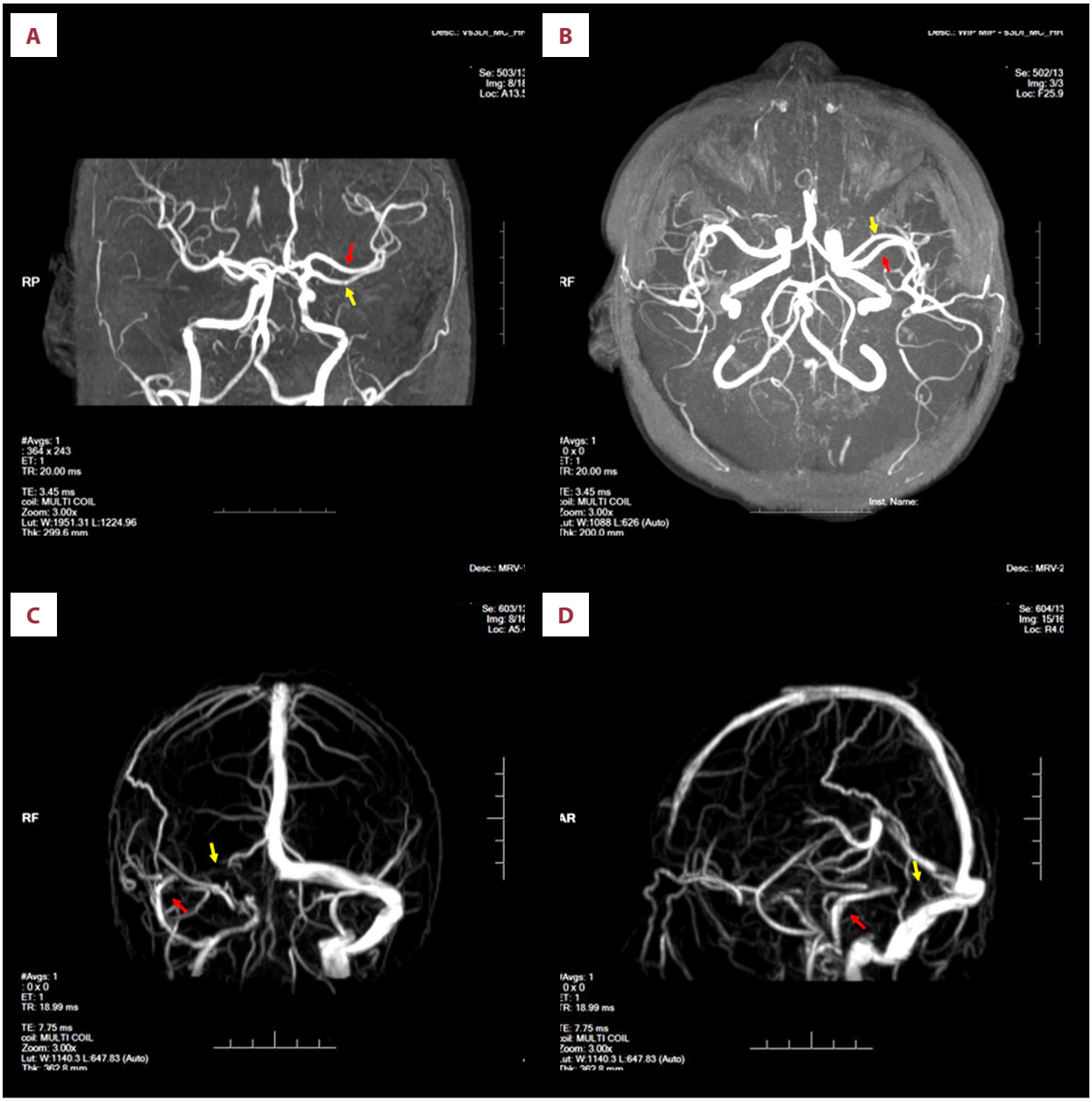


Figure 2. MRA and MRV demonstrating left-sided arterial duplication and hypoplasia of the right transverse and sigmoid sinuses with left-dominant venous drainage. (**A**) Coronal 3-dimensional time-of-flight MRA maximum-intensity projection shows the duplicated left MCA near the terminal internal carotid artery. (**B**) Axial 3-dimensional time-of-flight MRA maximum-intensity projection shows 2 parallel left MCA trunks. (**C**) Frontal MRV maximum-intensity projection shows attenuation of the right transverse-sigmoid sinus with relatively dominant left-sided venous drainage. (**D**) Lateral MRV maximum-intensity projection shows faint right sigmoid sinus/internal jugular venous outflow and dominant left transverse-sigmoid sinus drainage. Colored arrows indicate the corresponding arterial duplication and venous drainage asymmetry. Panels **A-D** were rearranged for publication layout only; image content was not altered. Abbreviations: MCA, middle cerebral artery; MRA, magnetic resonance angiography; MRV, magnetic resonance venography.

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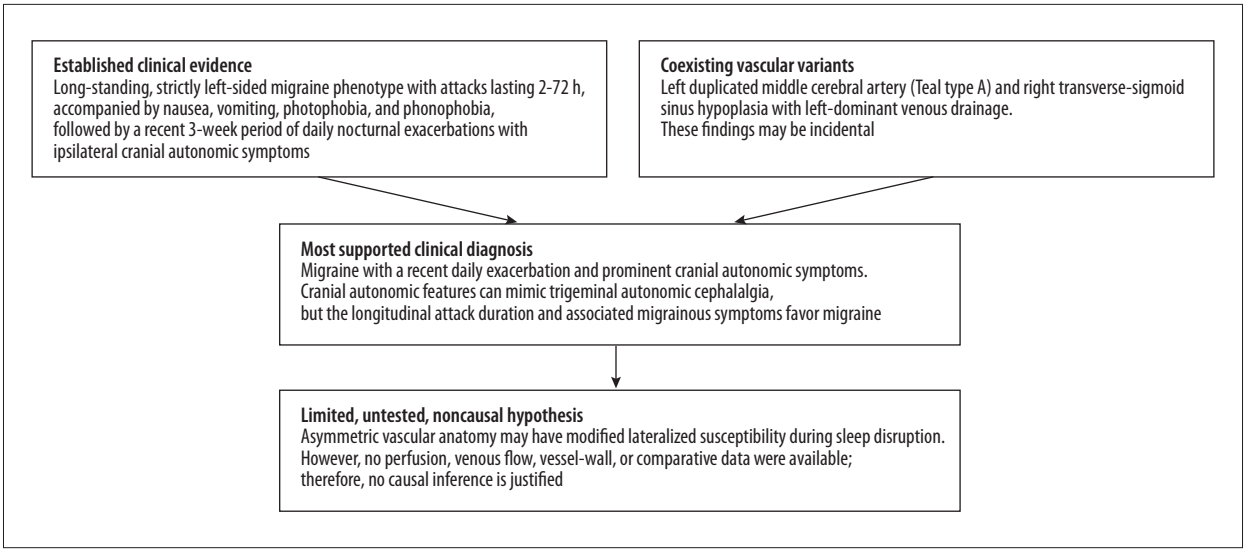


Figure 3. Clinical interpretation and limited neurovascular hypothesis. Clinical findings most strongly support migraine with a recent daily exacerbation and prominent cranial autonomic symptoms, whereas trigeminal autonomic cephalalgia is considered less likely. The duplicated middle cerebral artery and transverse-sigmoid sinus hypoplasia are presented as coexisting vascular variants that may be incidental. The vascular hypothesis remains untested, and no causal inference is justified.

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Electrocardiography and transthoracic echocardiography results were normal, without evidence of a cardioembolic source or patent foramen ovale. Brain magnetic resonance imaging revealed no parenchymal abnormalities, ischemic lesions, demyelinating changes, or intracranial masses.

Three-dimensional time-of-flight MRA revealed a left DMCA arising from the terminal internal carotid artery, with 2 similarly sized trunks running parallel toward the Sylvian fissure, consistent with Teal type A morphology [2,3] (**Figure 1**). MRV demonstrated hypoplasia of the right transverse and sigmoid sinuses, relatively faint visualization of the right internal jugular venous outflow, and left-dominant venous drainage (**Figure 2**). No intracranial aneurysm or arterial stenosis was detected.

Given the long-standing migraine phenotype, the transition to daily nocturnal attacks over 3 weeks after sleep disruption, the prominent ipsilateral cranial autonomic symptoms, and the absence of an alternative structural cause, the clinical diagnosis was migraine with a recent daily exacerbation and prominent cranial autonomic symptoms rather than a trigeminal autonomic cephalalgia [5,7-9].

Because the overall clinical course favored migraine and no secondary structural causes were identified, migraine-oriented preventive treatment was initiated. The patient received flunarizine 5 mg nightly, amitriptyline 6.25 mg nightly, and gabapentin 0.1 g three times daily. Nonpharmacological management included sleep regularization, advice on smoking cessation, alcohol restriction, avoidance of sleep deprivation, and counseling to reduce anxiety related to the incidental vascular findings. The patient was advised to maintain a headache diary and seek reassessment if headache laterality changed, neurological deficits developed, or attack severity abruptly worsened.

After 1 week of preventive therapy and lifestyle modifications, nocturnal awakenings had resolved. Daytime headache frequency decreased to fewer than 1 attack per week, and pain intensity decreased to 3/10 on the visual analog scale. The patient reported good adherence to the prescribed regimen.

At 3 months, she remained clinically stable and reported fewer than 1 headache attack per month without the need for rescue medication. No serious adverse drug reactions were reported. Follow-up complete blood count and liver and renal function test results were normal. The HIT-6 score improved from 58 at baseline to 46 at 3 months.

Discussion

This case is clinically notable because a patient with a long-standing side-locked migraine phenotype experienced a recent daily nocturnal exacerbation with cranial autonomic symptoms in the setting of an ipsilateral arterial variant and contralateral venous sinus hypoplasia. The central diagnostic lesson is that prominent cranial autonomic symptoms may accompany migraine and mimic trigeminal autonomic cephalalgia. The imaging findings are secondary observations and should be interpreted cautiously because DMCA is generally regarded as an incidental congenital variant, rather than an established cause of headache [2,3].

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The patient's longitudinal history favored migraine: earlier attacks lasted 2 to 72 hours and were accompanied by nausea, vomiting, photophobia, and phonophobia. The subsequent emergence of conjunctival injection, lacrimation, nasal congestion, and rhinorrhea did not, by itself, warrant reclassification as a trigeminal autonomic cephalalgia. Cranial autonomic symptoms are recognized features of migraine, often occur ipsilateral to pain, and are particularly common in patients with severe or unilateral attacks [7-9]. Thus, the broader temporal and symptom profile, rather than the autonomic symptoms alone, supported the diagnosis of migraine [5,7-9].

Prior reports and reviews of DMCA have primarily emphasized its embryological origin; anatomical classification; prevalence on angiographic imaging; and associations with aneurysms, subarachnoid hemorrhage, or ischemic events rather than primary headache phenotypes. Chang and Kim reviewed the classification and clinical implications of DMCA, highlighting aneurysms at the DMCA origin and cerebral infarction due to DMCA occlusion as clinically relevant associations [2]. Uchiyama also emphasized that MCA anomalies are primarily important for accurate neuroradiological diagnosis and safe surgical or endovascular intervention [11]. Reports of DMCA-associated aneurysms do not establish isolated DMCA as a cause of migraine-like headaches with cranial autonomic symptoms [12].

Therefore, the present case should not be interpreted as evidence that DMCA caused the headaches. Its incremental value is primarily descriptive: it documents the coexistence of a side-locked migraine phenotype, prominent ipsilateral cranial autonomic symptoms, ipsilateral DMCA, and contralateral venous sinus hypoplasia after several common secondary causes had been excluded.

Any vascular mechanism remains speculative. Although intracranial vascular variants have been discussed as potential modifiers of migraine susceptibility [6], our patient did not undergo perfusion imaging, vessel-wall imaging, transcranial Doppler monitoring, venous flow assessment, or computational flow analysis. Accordingly, there is no patient-specific evidence of hyperperfusion, altered shear stress, flow redistribution, venous outflow effects, or asymmetric trigeminovascular activation. Both vascular variants may be incidental. At most, this case generates an untested hypothesis that asymmetric vascular anatomy could have modified lateralized susceptibility during sleep disruption; this hypothesis should not be interpreted as evidence of causality (**Figure 3**).

The clinical improvement after a multimodal regimen should also be interpreted cautiously. Because flunarizine, amitriptyline, gabapentin, and lifestyle modifications were introduced simultaneously, the contribution of each component cannot be determined. The response does not validate the vascular hypothesis but supports the practical choice of a migraine-oriented management strategy after alternative secondary causes had been excluded.

This report has substantial limitations. It is a single observational case without perfusion imaging, vessel-wall imaging, computational fluid dynamics, formal autonomic testing, venous flow quantification, serial imaging, or a comparator group. Both vascular variants may have been incidental. Accordingly, the report provides a limited clinicoradiological observation and a practical diagnostic lesson, rather than evidence of a vascular mechanism or a basis for recommending detailed vascular imaging in general.

The patient reported that the recent nocturnal attacks caused pronounced anxiety because the pain repeatedly awakened her from sleep and was accompanied by eye redness, tearing, and nasal symptoms. After receiving an explanation of the imaging findings and initiating preventive treatment, she felt reassured that no aneurysm, tumor, stroke, or epileptiform abnormality had been identified. At follow-up, she reported substantial improvements in sleep quality and daily functioning; she expressed willingness to continue lifestyle modifications and follow-up monitoring.

Conclusions

This report has described a case of migraine with a recent daily nocturnal exacerbation and prominent ipsilateral cranial autonomic symptoms in a patient with left DMCA and contralateral venous sinus hypoplasia. The longitudinal clinical phenotype favored migraine over trigeminal autonomic cephalalgia. The vascular findings should be regarded as coexisting anatomical variants that may be incidental; this case does not establish causality or the general diagnostic utility of detailed vascular imaging. The principal clinical lesson is that cranial autonomic symptoms can accompany migraine and complicate the differential diagnosis of side-locked headache.

Ethics Approval

This study was approved by the Ethics Committee of The Second Affiliated Hospital of Kunming Medical University (Approval No. Examine-PJ-division-2025-158) and was conducted in accordance with the Declaration of Helsinki.

Patient Consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

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Availability of Data and Materials

All data generated or analyzed during this study are included in this manuscript.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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