

CLUSTER HEADACHE MIMIC: CT-NEGATIVE SUBARACHNOID HEMORRHAGE FROM MCA ANEURYSM

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ABSTRACT:

Introduction: Primary cluster headache is a debilitating trigeminal autonomic cephalalgia. While diagnostic criteria are well-established, secondary structural lesions can closely mimic this phenotype, posing a significant diagnostic challenge in emergency settings.

Case Presentation: A 51-year-old woman presented with sudden-onset, severe, unilateral right temporal headache clinically mimicking a primary cluster headache. Initial emergency non-contrast computed tomography (NCCT) demonstrated no acute intracranial abnormalities. Due to persistent symptoms, a subsequent brain MRI was performed. Multi-sequence MRI revealed a small saccular aneurysm arising from the M1 segment of the right middle cerebral artery (MCA). Accompanying right temporal sulcal hyperintensities on FLAIR and subtle hyperintensity on T1-weighted imaging confirmed a localized, late subacute subarachnoid hemorrhage (SAH).

Discussion: This case illustrates a critical clinical pitfall driven by time-dependent radiologic attenuation, where transitional subacute blood becomes isodense on NCCT. The structural mimicry could be explained by mechanical irritation or a minor leak from the MCA aneurysm stimulating the closely adjacent ophthalmic branch (V1) of the trigeminal nerve, triggering the trigeminovascular pain pathway.

Conclusion: Multi-sequence MRI is indispensable for ruling out occult, life-threatening secondary vascular pathologies when strong clinical suspicion persists despite negative initial CT findings.

Keywords: Cluster Headache, Subarachnoid Hemorrhage, Aneurysm, MRI, CT scan

INTRODUCTION

Cluster headache is a debilitating primary disorder categorized within the trigeminal autonomic cephalalgias, characterized by strictly unilateral, excruciating, or stabbing pain in the orbital, supraorbital, or temporal regions [1,2]. Pain typically peaks within 15 minutes and is accompanied by ipsilateral cranial autonomic symptoms, such as lacrimation and ptosis, with attacks lasting 15 to 180 minutes and occurring up to 8 times daily [3]. Although the ICHD-3 outlines the criteria for primary cluster headache, various destructive secondary lesions can mimic this phenotype [1,4]. These mimics encompass diverse intracranial pathologies, including neoplasms, dissections, and vascular malformations [4]. Among these, intracranial aneurysms and non-traumatic subarachnoid hemorrhages are life-threatening conditions that necessitate urgent identification to prevent catastrophic rebleeding and neurological morbidity [5].

Even after the acute Subarachnoid Hemorrhage (SAH) phase, many survivors experience persistent headaches that severely impact their quality of life (QoL) [6]. Because secondary headaches can closely mimic primary disorders, misdiagnosis remains a critical clinical pitfall [7]. We present a rare case of an initially underdiagnosed subarachnoid hemorrhage (SAH) mimicking a primary cluster headache. Recent evidence shows that vascular etiologies account for 37.7% of structural pathologies in cluster-like headaches (CLH), leading to frequent diagnostic delays [8-12]. However, intracranial aneurysms are surprisingly rare, with only six cases reported in systematic reviews [12]. These few cases usually involve intact, unruptured aneurysms near parasellar or cavernous regions pressing on local nerves. An actual aneurysmal leak or SAH from an M1 MCA segment presenting with cluster-like symptoms is a rare clinical occurrence.

CASE PRESENTATION

A 51-year-old woman with no significant past medical history presented to the emergency department with a sudden-onset, severe, unilateral headache clinically resembling a cluster headache. The patient reported spontaneous, intense pain localized strictly to the right temporal and retro-orbital regions. The pain was abrupt and persistent, lasting over 30 minutes and causing restlessness at home. The patient also presented with ipsilateral cranial autonomic features included forehead and facial sweating, retro-orbital soreness, and conjunctival injection, alongside nausea and vomiting. However, rhinorrhea, eyelid edema, miosis, and ptosis were not seen. No loss of consciousness, visual aura, or reduced visual acuity was reported. Thus, the presentation clinically mimicked a cluster headache, though it did not fully meet ICHD-3 diagnostic criteria due to an insufficient history of attack frequency and periodicity. Neurological examination was inconclusive. non-contrast brain CT (NCCT) was done immediately planned after ED arrival. The initial NCCT scan produced no acute intracranial or extracranial abnormalities.

Due to persistent severe symptoms and clinical concern for secondary headache, further evaluation with a brain MRI was performed. MRI revealed a small saccular aneurysm arising from the right middle cerebral artery (MCA) at the M1 segment. An adjacent hyperintense signal within the right temporal subarachnoid space was noted, consistent with a localized late subacute subarachnoid hemorrhage. The findings suggested a small ruptured aneurysm that was not detected on the initial CT examination. Regrettably, the patient passed away prior to undergoing digital subtraction angiography (DSA).

DISCUSSION

The clinical presentation of this patient highlights a diagnostic dilemma where a

secondary vascular pathology mimicked a benign primary headache disorder. The anatomical proximity of the middle cerebral artery (MCA) and the surrounding subarachnoid space to the ophthalmic branch (V1) of the trigeminal nerve is one mechanism where structural vascular anomaly could trigger the same neurovascular pain pathway [8]. In cases of non-traumatic intracranial or subarachnoid hemorrhage, pain typically arises from mechanical nociception caused by the physical stretching or displacement of pain-sensitive tissues, such as the meninges and dura, paired with increased intracranial pressure [8]. In our patient, local structural tension, focal mass effect, or a minor leak from a right middle cerebral artery (MCA) M1 segment aneurysm likely irritated these perivascular trigeminal afferents, triggering an acute, severe temporal headache that closely mimicked a primary cluster headache attack to the patient.

This case is an example of the critical clinical pitfall of relying solely on emergency neuroimaging when the clinical timeline is transitional or subacute. Non-contrast computed tomography (NCCT) remains the gold standard in acute settings due to its near-perfect sensitivity if performed within 6 hours of symptom onset, and 86% to 97% sensitivity within 6 to 72 hours [5,7]. In this patient, the initial NCCT demonstrated no acute abnormalities (Figure 1), a classic false-negative result directly attributable to this time-dependent radiologic attenuation. When emergency NCCT yields negative results but strong clinical suspicion of a secondary etiology persists, magnetic resonance imaging (MRI) sequences become crucial for evaluating a missed diagnosis [5]. The patient's MRI panel shows the radiologic image of a transitional subarachnoid hemorrhage. On standard T1-weighted imaging (T1WI), acute subarachnoid blood typically mimics normal CSF and appears dark. However, our patient's scan demonstrated a subtle

right temporal hyperintensity. This subtle T1-bright signal provides evidence of early hemoglobin degradation, driven primarily by methemoglobin's intrinsic T1-shortening effect [9]. Fluid-Attenuated Inversion Recovery (FLAIR) sequences circumvent this limitation, exhibiting good sensitivity across both the acute and subacute phases [9,10,11]. By suppressing the signal from normal, moving CSF, FLAIR highlights hyperintense signals within the sulci. This was seen in our patient's right temporal subarachnoid space. This failure of FLAIR suppression of CSF was initially caused by the high protein content of the fluid mixture shifting the null point of suppression and was further sustained by the evolving subacute methemoglobin signal in the CSF [7,9]. Matching these parenchymal changes with three-dimensional time-of-flight magnetic resonance angiography (MRA) enables further identification of vascular anomalies, such as our patient's right middle cerebral artery (MCA) M1 segment aneurysm. magnetic resonance angiography (MRA) serves as a valuable non-invasive tool for identifying underlying vascular anomalies. However, MRA cannot replace conventional digital subtraction angiography (DSA), which remains the gold standard modality essential for precise anatomical characterization, collateral flow assessment, and definitive neurointerventional or surgical planning. In our case, although emergency DSA and subsequent endovascular coiling or surgical clipping were planned following the MRA findings, no definitive invasive management could be done as the patient experienced a rapid clinical decline and passed away prior to intervention [11].

CONCLUSION

Cluster-like headache may rarely represent aneurysmal subarachnoid hemorrhage. When CT findings are negative but clinical suspicion persists, MRI plays an important role in detecting occult hemorrhage and

identifying the underlying vascular lesion. In these scenarios, advanced multi-sequence MRI is indispensable for detecting occult hemorrhage and identifying the underlying vascular anomaly. Missing an evolving subacute SAH carries a catastrophic prognosis, exposing the patient to an imminent risk of aneurysmal re-bleeding, vasospasm, and permanent neurological morbidity [5]. Therefore, clinicians must maintain a low threshold for ordering multi-modal MR imaging in patients presenting with new-onset or atypical cluster phenotypes to definitively rule out dangerous structural mimics before diagnosing a benign primary headache disorder.

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FIGURE LEGENDS:

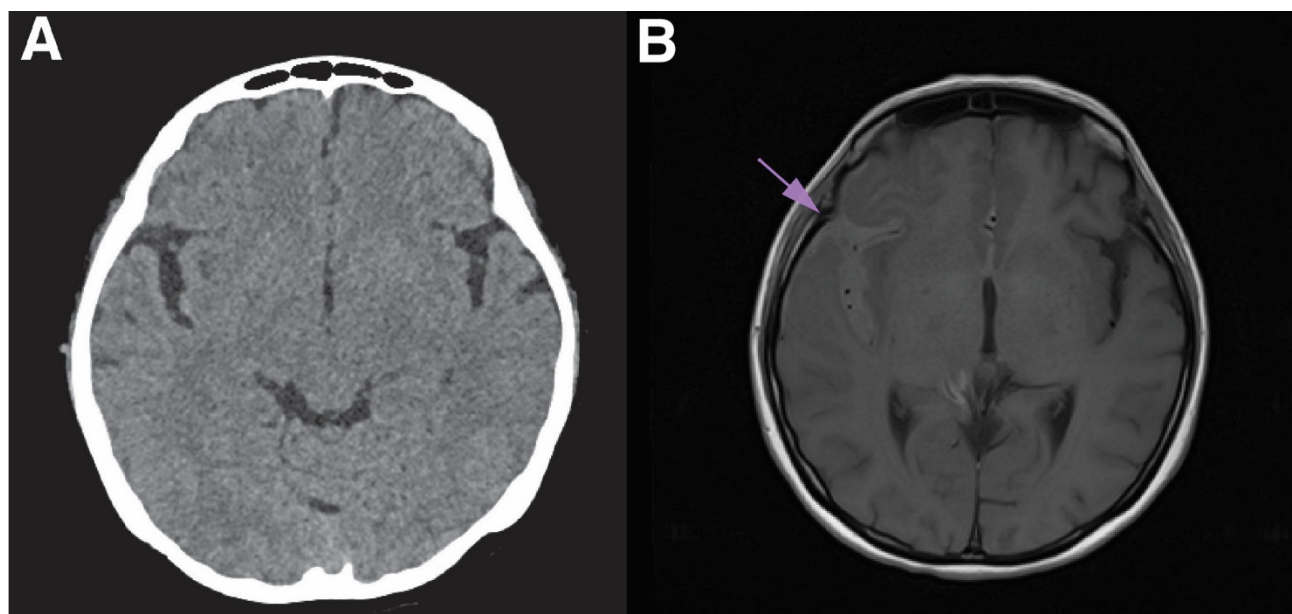


Figure 1. (A) Axial non-contrast brain CT (NCCT) shows no evidence of acute intracranial hemorrhage or gross focal lesions. (B) Axial T1-weighted MRI (T1WI) reveals subtle hyperintensity within the right temporal subarachnoid space (purple arrow).

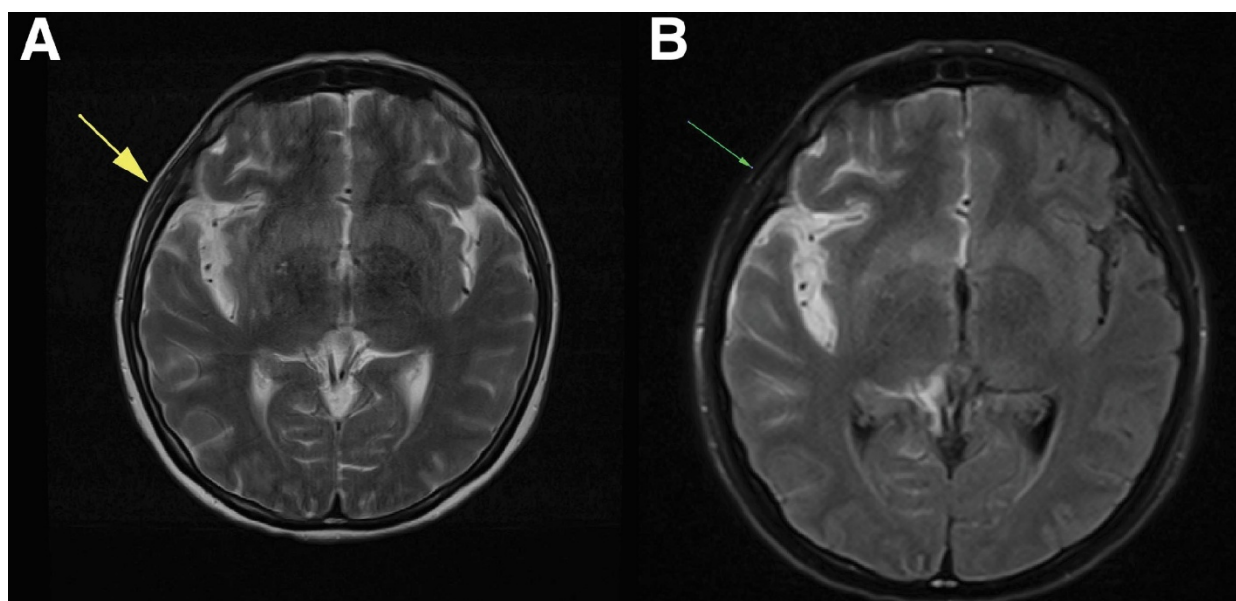


Figure 2: (A) Axial T2-weighted MRI (T2WI) shows a corresponding focal signal abnormality along the right sylvian/temporal region (yellow arrow). (B) Axial Fluid-Attenuated Inversion Recovery (FLAIR) image highlights sulcal hyperintensity in the right temporal lobe, consistent with subarachnoid hemorrhage (SAH) (green arrow).