

Acephalgic Migraine Presenting as Episodic Fatigue and Nausea: A Case Report

Daniel G. Oliveira, MD, MSc

Acephalgic migraine (AM), or migraine aura without headache, is a subtype of migraine, characterized by transient neurological symptoms without the accompanying headache phase. Its diagnosis remains challenging due to varied clinical presentation and the tendency to misattribute symptoms to other neurological, psychiatric, or systemic disorders. This case report describes a female patient, age 38 years, presenting with episodic, incapacitating fatigue, nausea, and cognitive dysfunction, initially leading to an extensive but inconclusive diagnostic workup. Partial symptom relief with ondansetron, but not with metoclopramide, suggested involvement of migraine-associated pathways. A detailed history revealed a family history of migraine and specific triggers, including dietary and hormonal factors. A therapeutic trial with triptans led to dramatic symptom resolution, supporting an AM diagnosis. Retrospectively, the patient was able to identify additional aura symptoms, reinforcing the diagnosis. This case underscores the diagnostic challenge of AM and migraine aura variants. It also highlights the pivotal role of careful history-taking, patient–physician communication, and clinical reasoning in the evaluation of atypical symptom presentations. Clinician awareness of migraine spectrum disorders is essential to prevent misdiagnosis, reduce unnecessary testing, and improve patient outcomes. Further research is needed to refine diagnostic criteria and optimize management strategies.

Keywords: Acephalgic migraine; Migraine aura; Headache; Triptans; Diagnosis

Acephalgic migraine (AM), also known as migraine aura without headache or “silent migraine,” is a recognized subtype of migraine characterized by transient neurological symptoms occurring in the absence of headache.¹ Patients typically experience visual auras, such as transient visual loss or zigzag, often scintillating lines (known as fortification spectra), and other sensory disturbances (e.g., paresthesia), transient aphasia, or cognitive impairment, particularly in attention and processing speed.^{1–3} These episodes are usually self-limiting, with considerable variability in frequency and duration.

Despite being part of the migraine spectrum, AM remains underdiagnosed due to the absence of headache and its diverse clinical presentation, often mimicking neurological,

psychiatric, or systemic conditions.⁴ Studies suggest only about 4% of migraineurs experience aura without headache exclusively, although up to 38% may report such episodes at some point.^{1,3} As in other migraine with aura types, AM follows a biphasic age distribution, peaking between ages 20–39 years and 60–69 years, with a slight female predominance.⁴

The non-specific nature of AM and lack of diagnostic biomarkers frequently lead to misdiagnosis, excessive testing, and patient anxiety. Common differential diagnoses include epilepsy, transient ischemic attacks, functional neurological disorders, and psychiatric conditions.⁵ Nonetheless, early recognition is critical, as most patients respond well to standard migraine therapies.⁶ This case report illustrates the

Corresponding Author: Dr. Daniel G. Oliveira, UMIB, School of Medicine and Biomedical Sciences
University of Porto, Rua Jorge Viterbo Ferreira 228, 4050-313 Porto, Portugal
Email: daniel.gtro@gmail.com

Received: May 4, 2025
Revised: July 22, 2025
Accepted: August 14, 2025

Funding Disclosure: DGO is kindly supported by a grant from FCT - Fundação para a Ciência e Tecnologia and from ICBAS/BIAL in support of his doctoral studies. This funding is unrelated to the present work, and the funding bodies had no role in any aspect of the research or manuscript.

doi: 10.3121/cmr.2025.2030

diagnostic challenge posed by AM and demonstrates how a structured, context-based clinical approach—grounded in careful history-taking, open physician-patient communication, and symptom-directed treatment trials—can lead to timely recognition and effective management.

Case Presentation

A Caucasian woman, age 38 years, presented to a private internal medicine diagnostic clinic with a year-long history of episodic nausea, profound fatigue, and cognitive dysfunction. These episodes, which varied in frequency but occurred at least once a month, lasted between 24 and 72 hours. During each episode, she experienced nausea that prevented eating, an overwhelming and incapacitating sense of exhaustion, and a diminished ability to concentrate or perform cognitively demanding tasks, especially in her role as a practicing physician. The episodes followed a stereotypical pattern, with abrupt onset of nausea, which gradually built up to the other symptoms and resolved slowly. She reported increased irritability during these episodes, stating she had “little patience.” She also expressed frustration about their recurring nature and impact on her quality of life. She experienced no symptoms or impairment in the period between episodes.

Some of the episodes were accompanied by abdominal bloating and diarrheal transit with no blood or mucus, which had led to a trial of gluten-free diet for suspected celiac disease, without improvement. She reported a few episodes of vertigo in the two preceding years, which had been diagnosed as benign paroxysmal positional vertigo (BPPV) by an ear-nose-and-throat surgeon. She did not report headache, photophobia, or phonophobia. On some occasions, she also experienced a sensation of heaviness in her limbs, though she struggled to describe the exact nature of this phenomenon. The patient denied fever, flushing, hyperdiaphoresis, or vomiting. She had tried metoclopramide for nausea, but only ondansetron exerted any effect. During one episode, a previous physician prescribed a one-week course of prednisolone (20 mg/day). The patient vaguely associated this treatment with a quicker resolution of symptoms and interruption of new episodes

Table 1. Summary of previously available results and subsequent diagnostic workup

| Test | Reference Range | Result |
|-----------------------------------|-------------------------------|----------|
| Hematology | | |
| Hemoglobin | 11.2–15.7 g/dL | 13 |
| Hematocrit | 34.1–44.9% | 38 |
| Leukocytes | 4.0–10.5 ×10 ³ /μL | 4.54 |
| Platelets | 150–400 ×10 ³ /μL | 261 |
| ESR | <20 mm/h | 7 |
| Chemistry | | |
| C reactive protein | <0.3 mg/dL | <0.05 |
| NT-proBNP | <125 pg/mL | 138 |
| Creatine phosphokinase | 20–200 U/L | 134 |
| Creatinine | 0.6–1.3 mg/dL | 0.57 |
| Urea | 15–44 mg/dL | 17 |
| Sodium | 135–145 mmol/L | 144 |
| Potassium | 3.5–5.1 mmol/L | 4.5 |
| Calcium | 7.4–11.0 mg/dL | 9.91 |
| Albumin | 3.4–5.0 g/dL | 4.89 |
| Aspartate aminotransferase | 15–37 U/L | 17 |
| Alanine aminotransferase | 12–78 U/L | 9 |
| Total bilirubin | 0.2–1.0 mg/dL | 1 |
| Total alkaline phosphatase | 46–117 U/L | 55 |
| Gamma-glutamyl transferase | <55 U/L | 10 |
| Ferritin | 30–300 ng/mL | 55 |
| Fecal calprotectin | < 30 ug/g | 56 |
| Endocrinology and vitamins | | |
| Vitamin B12 | 200–900 pg/mL | 728 |
| Vitamin D, 25-hydroxy | 30–100 ng/mL | 37 |
| Thyroid-stimulating hormone | 0.5–5.0 μIU/mL | 1.7 |
| ACTH | 7.2–63.3 pg/mL | 29.2 |
| Cortisol | 3.7–19.4 μg/dL | 16 |
| Dehydroepiandrosterone sulfate | 35–430 ug/dL | 95 |
| Infection and Serology | | |
| HBV serology | Negative | Negative |
| HCV serology | Negative | Negative |
| HIV serology | Negative | Negative |
| Syphilis serology | Negative | Negative |
| Coxiella serology | Negative | Negative |
| Borrelia serology | Negative | Negative |
| Rickettsia serology | Negative | Negative |
| EBV serology | Negative | Negative |
| CMV serology | Negative | Negative |
| Tropheryma whipplei (fecal PCR) | Negative | Negative |
| Giardia lamblia fecal antigen | Negative | Negative |
| Fecal parasites | Negative | Negative |

Table 1. Summary of previously available results and subsequent diagnostic workup (continued)

| Test | Reference Range | Result |
|-------------------------------------|-----------------|----------|
| Immunology | | |
| Serum protein immunoelectrophoresis | Normal | Normal |
| IgG | 700-1,600 mg/dL | 1215 |
| IgM | 40-230 mg/dL | 153 |
| IgA | 70-400 mg/dL | 103 |
| IgE | < 378 UI/mL | 180 |
| C3 | 80-180 mg/dL | 108 |
| C4 | 10-40 mg/dL | 16 |
| Antinuclear antibody | <1/80 | <1/80 |
| Anti-Thyroid peroxidase IgG | <35 UI/mL | 165.5 |
| Anti-Transglutaminase IgA | <7 U/mL | <0.2 |
| Additional tests | | |
| Electrocardiogram | Normal | Normal |
| CT chest abdomen pelvis | Normal | Normal |
| CT head | Normal | Normal |
| MRI head | Normal | Normal |
| Upper gastrointestinal endoscopy | Normal | Normal |
| Lower gastrointestinal endoscopy | Normal | Normal |
| SIBO | Negative | Negative |

ACTH - Adrenocorticotropic Hormone; C3 - Complement component 3; C4 - Complement component 4; CMV - Cytomegalovirus; CT - Computerized tomography; DHEAS - Dehydroepiandrosterone sulfate; EBV - Epstein Barr virus; ESR - Erythrocyte Sedimentation Rate; IgA - Immunoglobulin A; IgE - Immunoglobulin E; IgG - Immunoglobulin G; IgM - Immunoglobulin M; MRI - Magnetic resonance imaging; NT-proBNP - N-terminal prohormone of brain natriuretic peptide; PCR - Polymerase Chain Reaction; SIBO - Syndrome of Intestinal Bacterial Overgrowth, exhaled air hydrogen and methane test

of severe nausea for a few weeks, although she maintained paroxysms of fatigue. Other previous treatments had included empirical doxycycline, with no effect.

The patient had been a vegetarian for the last 11 years. Past medical history was relevant for irritable bowel syndrome, autoimmune thyroiditis (with reportedly normal thyroxine levels on recent testing), and prior episodic bouts of depression. She had no history of migraine, epilepsy, or other autoimmune or metabolic disorders. A review of travel history revealed visits to several countries in Europe and Southern Asia, with ingestion of street food. She lived in an urban area with a stable partner and owned a dog, which had recently been treated for a parasitic infection. The household was similarly treated at the time.

Her family history included migraine with aura in her father, who also had irritable bowel syndrome, and irritable bowel syndrome in her sister. She denied tobacco and alcohol use, and had no known allergies.

On assessment, the patient appeared well, with stable vital signs. Physical examination was within normal limits, including unremarkable neurological, cardiopulmonary, and abdominal findings. The body mass index was 20.

The patient had previously sought evaluation from multiple specialists, including a general practitioner, an internist, a gastroenterologist, a neurologist, and an otolaryngologist, due to the persistence and impact of her symptoms. Despite extensive testing, including laboratory studies, imaging, and endoscopic evaluations, no significant abnormalities were identified, and no definitive diagnosis was established. A summary of prior investigations is provided in Table 1. She described growing frustration with the lack of answers, at times wondering whether her symptoms were truly indicative of an underlying medical problem or psychosomatic. This uncertainty contributed to a sense of helplessness and personal distress, further compounding the impact of her condition.

The diagnostic picture was summarized as recurrent, episodic, incapacitating fatigue with nausea. Workup was performed to look for or exclude a broad range of additional possible neurological, metabolic, and autoimmune differentials (Table 1).

Laboratory evaluations were unremarkable. Thyroid function remained normal despite the known autoimmune thyroiditis. Routine clinical biochemistry, including electrolytes, renal and liver function tests, glucose, and vitamin B12 and D levels, was within normal limits. Autoimmune testing, including antinuclear antibodies, anti-double stranded DNA, and antiphospholipid antibodies, was also unremarkable. An evaluation for gastrointestinal disorders, including serologic testing for celiac disease and upper endoscopy, revealed only mild gastritis, with no evidence of malabsorption or inflammatory pathology.

A clinical suspicion of a migraine spectrum disorder emerged based on the episodic pattern, stereotypical recurrence of attacks, and with normal interictal periods. The discussion of family history, including her father's migraine with aura, helped the patient notice a series of episodes with potential association with sleep loss, curry ingestion, and the immediate pre-menstrual period. There was no definite association with emotional stress, coffee or alcohol intake, or physical activity. The patient

Table 2. ICHD-3 Diagnostic Criteria for Migraine Aura Without Headache (Code 1.2.1.2)

| Diagnostic criteria | |
|--------------------------------|----------------------------------------------------------------------------------------------------------|
| A. Attack Frequency | At least two attacks fulfilling criteria B-D |
| B. Aura Symptoms | At least one of the following criteria (fully reversible) |
| 1. | Visual (e.g., scotomas, fortification spectra, zigzag lines) |
| 2. | Sensory (e.g., paresthesia, numbness) |
| 3. | Speech/language disturbances (e.g., transient aphasia) |
| 4. | Motor (e.g., hemiparesis) [Only in hemiplegic migraine] |
| 5. | Brainstem (e.g., vertigo, tinnitus, diplopia) [Only in migraine with brainstem aura] |
| 6. | Retinal symptoms (e.g., monocular visual disturbances) [Only in retinal migraine] |
| C. Aura Characteristics | At least three of six |
| 1. | At least one aura symptom spreads gradually over ≥ 5 minutes |
| 2. | Two or more aura symptoms occur in succession |
| 3. | Each individual aura symptom lasts 5-60 minutes |
| 4. | At least one aura symptom is unilateral |
| 5. | At least one aura symptom is positive (e.g., flickering lights, pins-and-needles rather than numbness) |
| 6. | The aura is followed by a headache within 60 minutes (not mandatory in acephalgic migraine) |
| D. Exclusion Criteria | None of the below |
| 1. | Not better explained by another disorder (e.g., transient ischemic attack, epilepsy, multiple sclerosis) |
| 2. | No headache accompanies or follows the aura within 60 minutes |

repeatedly denied headache, but referred to feeling her “head was heavier” during these attacks. Thus, AM with aura was considered a potential differential. The improvement of nausea with ondansetron and not with metoclopramide that had been noted in the first appointment was considered particularly relevant in this context.

Given this suspicion and following discussion with the patient, a therapeutic trial with a triptan and ondansetron was prescribed, and she was encouraged to record her symptoms prospectively. Taking the medication during the first hour of an episode resulted in marked improvement of symptoms within 3 hours, a stark contrast to the usual 3-day duration. Repeated use of triptans during subsequent episodes resulted in a consistent and rapid resolution of symptoms, strongly supporting the diagnosis of AM.

During the follow-up appointment at 3 months, the patient reported a newfound ability to recognize subtle aura symptoms that had previously gone unnoticed, including mild visual disturbances with “undulating lines”, a sensation of limb heaviness, and transient word-finding difficulties that occurred with some of the episodes, after the start of nausea. With the recognition of migraine as the underlying mechanism, and increased awareness and patient engagement, additional potential triggers were identified, including hormonal

fluctuations and dietary factors, allowing for targeted lifestyle modifications. These have mitigated, if not resolved, the frequency of episodes.

While she declined prophylactic medication due to concerns about side effects and pregnancy considerations, she continues to manage her episodes with abortive therapy using triptans. Given the established association between migraine with aura and increased cardiovascular risk,⁷⁻¹⁰ she was counseled on appropriate risk factor screening and management, which is also managed by her internist.

Discussion

Migraine is a highly heterogeneous neurological disorder, and its clinical manifestations extend well beyond the classic headache phenotype. Among its recognized variants, AM, or migraine aura without headache, remains underdiagnosed. As per current criteria, according to the International Classification of Headache Disorders, third edition (ICHD-3) (Table 2), AM is suggested by the presence of transient, fully reversible neurological symptoms (visual, sensory, or speech and language) typical of aura, often with a history of migraine or familial predisposition, in the absence of cerebrovascular or structural causes.² This case illustrates the diagnostic complexity of AM, particularly when non-traditional aura symptoms predominate, as nausea, cognitive dysfunction, and profound fatigue are often not immediately recognized as potentially migrainous in origin.

The absence of headache does not preclude cortical hyperexcitability or susceptibility to cortical spreading depression, which are at the base of the pathophysiology of migraine aura, reinforcing the need for clinician awareness of migraine variants that do not conform to the traditional headache-centered paradigm.^{5,11} The specific aura symptoms experienced by a patient depend on the cortical regions affected. Visual phenomena—whether illusions, hallucinations, or transient visual loss—remain the most common manifestations of migraine aura, even in headache-free variants.^{1,12} While the International Classification of Headache Disorders defines aura as consisting of reversible focal neurological symptoms, the patient's cognitive and executive dysfunction may reflect transient cortical dysfunction of the frontal and parietal cortex that remains underrecognized in current classifications.¹³

The episodic and stereotypical nature of the patient's symptoms necessitated consideration of a broad differential diagnosis. When AM is experienced by older adults, distinguishing the event from a transient ischemic attack (TIA) may be difficult; however, in the case of this young patient, TIA was considered inconsistent with the prolonged symptom duration and recurrent, identical episodes in the absence of vascular risk factors. The gradual buildup of symptoms, rather than sudden onset, further supported a migrainous origin, reflecting the underlying dynamics of cortical spreading depression.³ Neuroimmune and metabolic disorders, including multiple sclerosis and autonomic dysfunction, were also explored, but no laboratory, imaging, or autonomic findings supported these diagnoses. Seizures remained a consideration, but these were ultimately deemed less likely due to lack of personal or family history.

Two details in the patient's history offered critical insights into the mechanisms behind her symptoms. First, her selective response to antiemetics was notable: ondansetron—a selective 5-HT₃ receptor antagonist—provided relief from nausea, while metoclopramide—a dopamine antagonist with limited serotonergic activity—did not. This pattern aligns with the role of serotonin in migraine-associated nausea and the expected pharmacological profiles of these agents.^{14,15} Second, the patient reported a temporary reduction in the frequency of severe nausea episodes following a short course of corticosteroids. Although corticosteroids are not typically effective for acute migraine symptoms, they have been shown to reduce recurrence rates when used adjunctively.^{16,17} This could explain the partial and transient benefit observed, particularly as episodes of fatigue persisted.

In hindsight, the BPPV diagnosed by the otolaryngologist may be reinterpreted as another associated symptom of migraine, as recognized by the ICHD-3.² Epidemiologic studies suggest migraine increases the risk of developing BPPV by up to 2.5 times.¹⁸ Moreover, since migraine typically

presents at a younger age,^{6,8} compared with the later onset of BPPV in the general population,¹⁹ it is plausible the BPPV observed in this case is not an isolated vestibular disorder but rather a manifestation associated with the underlying migraine pathology.

The rapid and significant improvement of symptoms following a therapeutic trial with triptan provided further therapeutic evidence for the diagnosis. Triptans, which target serotonin 5-hydroxytryptamine 1B/1D receptors, are a mainstay of acute migraine treatment²⁰ and are generally not effective in other conditions that mimic migraine, such as vestibular disorders²¹ or functional gastrointestinal disorders.²² The patient's response, thus, suggested that despite the absence of headache, the underlying pathophysiology aligned with that of migraine with aura.

The recognition of AM has critical clinical and therapeutic implications. Although often regarded as a benign neurological disorder, migraine with aura—including its headache-free variants—has been associated with increased risks of stroke, thromboembolism, myocardial infarction, arrhythmias, and heart failure.⁷⁻¹⁰ This risk is present even in women under age 45, particularly those using hormonal contraceptives.²³ Long-term management should, therefore, include cardiovascular risk stratification and lifestyle counseling.^{6,23} In this case, the patient's response to triptans not only provided symptom relief but also reinforced the diagnostic hypothesis.⁶ Identifying individual triggers, such as hormonal fluctuations or dietary factors, allowed for targeted preventive strategies and improved self-management.⁶ It is still unclear if anti-calcitonin gene-related peptide (CGRP) monoclonal antibodies may be useful for resistant AM cases.²⁴⁻²⁸

Beyond diagnostic and management considerations, this case underscores the value of patient-physician communication and patient engagement in navigating clinical uncertainty. The patient's openness, symptom tracking, and collaboration in treatment decisions were instrumental in establishing a diagnosis. A strong therapeutic alliance fostered trust and awareness, ultimately enhancing clinical outcomes.

Conclusion

Acephalgic migraine (AM), or migraine aura without headache, presents a diagnostic challenge due to its rarity and the absence of the hallmark headache that typically defines migraine. In this case, suspicion of a migraine-related mechanism arose from the patient's selective response to treatment and a pattern of stereotypical, paroxysmal episodes. The consistent response to triptans provided both therapeutic relief and supported the clinical suspicion of a migrainous mechanism. This case illustrates the importance of a holistic, context-driven diagnostic approach, rooted in thorough history-taking, clinical reasoning, and the judicious use of therapeutic trials, particularly when evaluating patients with

unexplained, atypical, or complex symptoms. Greater awareness of AM can reduce diagnostic delays, limit unnecessary investigations, and improve patient outcomes.

References

1. Shah DR, Dilwali S, Friedman DI. Migraine Aura Without Headache [corrected]. *Curr Pain Headache Rep.* 2018;22(11):77. doi:10.1007/s11916-018-0725-1
2. Headache Classification Committee of the International Headache Society (IHS) The International Classification of Headache Disorders, 3rd edition. *Cephalalgia.* 2018;38(1):1-211. doi:10.1177/0333102417738202
3. Thomsen AV, Ashina H, Al-Khazali HM, et al. Clinical features of migraine with aura: a REFORM study. *J Headache Pain.* 2024;25(1):22. doi:10.1186/s10194-024-01718-1
4. Viana M, Khaliq F, Zecca C, et al. Poor patient awareness and frequent misdiagnosis of migraine: findings from a large transcontinental cohort. *Eur J Neurol.* 2020;27(3):536-541. doi:10.1111/ene.14098
5. Foroozan R, Cutrer FM. Transient Neurologic Dysfunction in Migraine. *Neurol Clin.* 2019;37(4):673-694. doi:10.1016/j.ncl.2019.06.002
6. Puledda F, Sacco S, Diener HC, et al. International Headache Society global practice recommendations for the acute pharmacological treatment of migraine. *Cephalalgia.* 2024;44(8):3331024241252666. doi:10.1177/03331024241252666
7. Kurth T, Rist PM, Ridker PM, Kotler G, Bubes V, Buring JE. Association of Migraine With Aura and Other Risk Factors With Incident Cardiovascular Disease in Women. *JAMA.* 2020;323(22):2281-2289. doi:10.1001/jama.2020.7172
8. Adelborg K, Szépligeti SK, Holland-Bill L, et al. Migraine and risk of cardiovascular diseases: Danish population based matched cohort study. *BMJ.* 2018;360:k96. doi:10.1136/bmj.k96
9. Mahmoud AN, Mentias A, Elgendy AY, et al. Migraine and the risk of cardiovascular and cerebrovascular events: a meta-analysis of 16 cohort studies including 1 152 407 subjects. *BMJ Open.* 2018;8(3):e020498. doi:10.1136/bmjopen-2017-020498
10. Ng CYH, Tan BYQ, Teo YN, et al. Myocardial infarction, stroke and cardiovascular mortality among migraine patients: a systematic review and meta-analysis. *J Neurol.* 2022;269(5):2346-2358. doi:10.1007/s00415-021-10930-x
11. Frimpong-Manson K, Ortiz YT, McMahon LR, Wilkerson JL. Advances in understanding migraine pathophysiology: a bench to bedside review of research insights and therapeutics. *Front Mol Neurosci.* 2024;17:1355281. doi:10.3389/fnmol.2024.1355281
12. Scutelnic A, Bracher J, Kreis LA, et al. Symptoms and patterns of symptom propagation in incipient ischemic stroke and migraine aura. *Front Hum Neurosci.* 2023;16:1077737. doi:10.3389/fnhum.2022.1077737
13. Petrusic I, Zidverc-Trajkovic J, Podgorac A, Sternic N. Underestimated phenomena: higher cortical dysfunctions during migraine aura. *Cephalalgia.* 2013;33(10):861-867. doi:10.1177/0333102413476373
14. Doğan NÖ, Pekdemir M, Yılmaz S, et al. Intravenous metoclopramide in the treatment of acute migraines: A randomized, placebo-controlled trial. *Acta Neurol Scand.* 2019;139(4):334-339. doi:10.1111/ane.13063
15. Villalón CM, VanDenBrink AM. The Role of 5-Hydroxytryptamine in the Pathophysiology of Migraine and its Relevance to the Design of Novel Treatments. *Mini Rev Med Chem.* 2017;17(11):928-938. doi:10.2174/1389557516666160728121050
16. Huang Y, Cai X, Song X, et al. Steroids for preventing recurrence of acute severe migraine headaches: a meta-analysis. *Eur J Neurol.* 2013;20(8):1184-1190. doi:10.1111/ene.12155
17. Woldeamanuel YW, Rapoport AM, Cowan RP. The place of corticosteroids in migraine attack management: A 65-year systematic review with pooled analysis and critical appraisal. *Cephalalgia.* 2015;35(11):996-1024. doi:10.1177/0333102414566200
18. Chu CH, Liu CJ, Lin LY, Chen TJ, Wang SJ. Migraine is associated with an increased risk for benign paroxysmal positional vertigo: a nationwide population-based study. *J Headache Pain.* 2015;16:62. doi:10.1186/s10194-015-0547-z
19. Bhattacharyya N, Baugh RF, Orvidas L, et al. Clinical practice guideline: benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg.* 2008;139(5 Suppl 4):S47-S81. doi:10.1016/j.otohns.2008.08.022
20. Ferrari MD, Roon KI, Lipton RB, Goadsby PJ. Oral triptans (serotonin 5-HT_{1B/1D} agonists) in acute migraine treatment: a meta-analysis of 53 trials. *Lancet.* 2001;358(9294):1668-1675. doi:10.1016/S0140-6736(01)06711-3
21. Webster KE, Dor A, Galbraith K, et al. Pharmacological interventions for acute attacks of vestibular migraine. *Cochrane Database Syst Rev.* 2023;4(4):CD015322. doi:10.1002/14651858.CD015322.pub2
22. De Ponti F, Tonini M. Irritable bowel syndrome: new agents targeting serotonin receptor subtypes. *Drugs.* 2001;61(3):317-332. doi:10.2165/00003495-200161030-00001
23. Bushnell C, McCullough LD, Awad IA, et al. Guidelines for the prevention of stroke in women: a statement for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke.* 2014;45(5):1545-1588. doi:10.1161/01.str.0000442009.06663.48

24. A Al-Khazali HM, Ashina H, Wiggers A, et al. Calcitonin gene-related peptide causes migraine aura. *J Headache Pain.* 2023;24(1):124. doi:10.1186/s10194-023-01656-4
25. Ashina M, Goadsby PJ, Dodick DW, et al. Assessment of Erenumab Safety and Efficacy in Patients With Migraine With and Without Aura: A Secondary Analysis of Randomized Clinical Trials. *JAMA Neurol.* 2022;79(2):159-168. doi:10.1001/jamaneurol.2021.4678
26. Shibata Y. Anti-Calcitonin Gene-Related Peptide Monoclonal Antibody Is Effective for Preventing Migraine Aura Without Headache. *Neurol Int.* 2024;16(6):1279-1284. doi:10.3390/neurolint16060097
27. Braca S, Miele A, Stornaiuolo A, Cretella G, De Simone R, Russo CV. Are anti-calcitonin gene-related peptide monoclonal antibodies effective in treating migraine aura? A pilot prospective observational cohort study. *Neurol Sci.* 2024;45(4):1655-1660. doi:10.1007/s10072-023-07241-6
28. Cresta E, Bellotti A, Rinaldi G, Corbelli I, Sarchielli P. Effect of anti-CGRP-targeted therapy on migraine aura: Results of an observational case series study. *CNS Neurosci Ther.* 2024;30(2):e14595. doi:10.1111/cns.14595

Author Affiliation

*Daniel G. Oliveira, MD, MSc**

**Instituto Médico de Estudos Imunológicos (IMEI), Porto; Autoimmune Diseases and Immunology Unit (UDAI), Internal Medicine Department, ULS Tâmega e Sousa, Penafiel; Unit for Multidisciplinary Research in Biomedicine (UMIB), School of Medicine and Biomedical Sciences (ICBAS), University of Porto (UIDB/00215/2020; UIDP/00215/2020), Porto; and ITR -Laboratory for Integrative and Translational Research in Population Health, (LA/P/0064/2020), Porto, Portugal*