▪ Headache Medicine 2025, 16(4):209-223 ▪ p-ISSN 2178-7468, e-ISSN 2763-6178

Headache Medicine

▪ DOI: 10.48208/HeadacheMed.2025.35

Review

Migraine in pregnancy: an integrative review

Maria-Karina Vélez-Jiménez[1], Pablo Schubaroff[2], Maria Loreto Cid-Jeffs[3], Ana Mercedes Robles[4], Adriana Patricia Martínez-Mayorga[5,6], Ildefonso Rodriguez-Leyva[5,6], Daniel San-Juan[7,8], Javier Andrés Galnares-Olalde[1], Miguel Angel Morales-Morales[7], Kevin Espejo Campos[9]

[1]Migraine and Headache Clinic, Hospital Angeles Lomas, Mexico City, Mexico
[2]Dr. Bernardo A. Houssay Municipal Hospital, Buenos Aires, Argentina
[3]Pain Unit, Military Hospital of Santiago, Santiago, Chile
[4]Medical Center, Central University of the East, Santo Domingo, Dominican Republic
[5]Department of Neurology, Central Hospital "Dr. Ignacio Morones Prieto", San Luis Potosí, Mexico
[6]Faculty of Medicine, Autonomous University of San Luis Potosí, San Luis Potosí, Mexico
[7]Epilepsy Clinic, National Institute of Neurology and Neurosurgery Manuel Velasco Suárez, Mexico City, Mexico
[8]Epilepsy Clinic, Hospital Angeles Pedregal, Mexico City, Mexico
[9]Assistant neurologist, Lima, Peru

✉ María Karina Vélez-Jiménez
drakvj@aol.com

Edited by:
Marcelo Moraes Valença

Background
Migraine is the most prevalent primary headache disorder in pregnancy, with unique clinical implications requiring specialized management approaches.

Objective
To provide a comprehensive review of migraine pathophysiology, clinical course, diagnosis, and management during pregnancy, postpartum, and lactation.

Methods
Selective literature search of PubMed, Scopus, and Web of Science databases, complemented by AI-assisted tools (Elicit and Consensus) to ensure comprehensive coverage of emerging evidence, from 2003 to 2025.

Key Findings
Hormonal fluctuations drive migraine pathophysiology, with pregnancy's hormonal stability typically improving symptoms in 60-70% of women with migraine without aura during the second and third trimesters. However, migraine with aura shows less predictable improvement, and de novo attacks may occur. Maternal migraine, particularly with aura, is associated with hypertensive disorders, preterm birth, cesarean delivery, fetal growth restriction, and increased ischemic stroke risk. Psychiatric comorbidities, including depression and anxiety, are frequent and linked to poorer outcomes.

Management
Non-pharmacological interventions form the foundation of treatment. When pharmacological therapy is required, acetaminophen is first-line, NSAIDs may be used only in the second trimester, and sumatriptan is the safest triptan option. Preventive treatment should be reserved for severe, refractory cases, with magnesium, propranolol, and amitriptyline as evidence-based options.

Conclusion
Migraine during pregnancy and postpartum requires a multidisciplinary approach prioritizing safe pharmacological strategies, individualized monitoring, and lifestyle optimization to ensure optimal maternal and fetal outcomes.

Keywords:
Migraine
Pregnancy
Postpartum
Breastfeeding
Fetal complications

Received: October 7, 2025
Revised: October 19, 2025
Accepted: December 9, 2025

Introduction

Migraine is a common neurological disorder that profoundly impacts quality of life and imposes a significant burden on individuals having this disorder. It involves more than 12% of the general population, with a higher frequency in women (3:1 compare to men) (1,2). Although other primary headaches, such as tension-type headache, cluster headache, and trigeminal autonomic cephalalgias, also occur during pregnancy between 10-17% of pregnant women are estimated to experience migraines, making it the most common reason for consultation in clinical practice (accounting for up to 90% of visits due to headache) (3).

Pregnancy results in a distinct endocrine milieu with high, constant levels of placental estrogens and progestogens that suppress the cyclic hormone fluctuations of the menstrual cycle. This is associated with a reduction in migraine symptoms of up to 50% by the end of first trimester and further improvement over time during pregnancy (2). However, a small number of women who have had no history of migraine may develop an attack for the first time during the first trimester, with occurrence rates varying between 1.3% - 18% (1).

Therefore, the management of migraine in pregnancy presents a dual challenge: both the pain itself and the pharmacological interventions must be addressed without compromising maternal or fetal safety. Despite the clinical relevance of this issue, there are no established Latin American recommendations to support the comprehensive care of migraine during pregnancy (MdP), postpartum, and lactation. This represents a significant gap in care for a population with unique therapeutic needs and limited resources. Although this review explores hormonal, physiological, and clinical aspects of migraine in pregnancy, special emphasis is placed on evidence-based management strategies, as therapeutic decision-making during this period remains one of the main challenges for neurologists.

This review was conducted through a selective search of PubMed, Scopus, and Web of Science. Elicit and Consensus were used to complement the manual search, enhancing the retrieval of relevant literature and ensuring a comprehensive coverage of emerging evidence from 2003 to 2025. Eligible publications included original research, narrative reviews, and systematic reviews reporting clinical, physiological, or therapeutic aspects of migraine in pregnancy. This approach allows for a cohesive synthesis of current knowledge while highlighting areas where further research is needed.

Prevalence, risk factors, and clinical characteristics

Migraine is one of the most prevalent neurological disorders worldwide and shows a marked predominance in women of reproductive age. This sex difference is largely attributed to the modulatory effects of estrogens on serotonergic and trigemino-vascular pathways, which increase female susceptibility during the reproductive years (2). During pregnancy, the clinical course of migraine changes significantly. Migraine without aura (MwoA) typically improves in the majority of patients: about 60–70% report a reduction in frequency or even partial remission, especially in the second and third trimesters (4,5). In contrast, migraine with aura (MwA) follows a less favorable course, likely because cortical spreading depression (CSD) mechanisms involved in aura are less responsive to estrogen stabilization and even the phenotype of the migraine can vary, so MwA can start during pregnancy even if there is no history of migraine or if there is MwoA (2). The trimester-specific pattern has been consistently described. It has been reported that most begin in the first trimester, but between 70% and 80% decrease in the second and third trimesters, and only between 4% and 8% have worsened; 10% maintain the characteristics of the attacks in the first trimester. Importantly, if no improvement is observed after the first trimester, it is unlikely to occur later in pregnancy (6).

Another relevant phenomenon is the onset of de novo MdP. Although there is limited data, there are case reports in the third trimester that could begin without an underlying secondary condition, with the presence of an aura being more frequent than without an aura (7). Clinically, this is significant because the first occurrence of migraine-like headache during pregnancy should always prompt exclusion of secondary causes such as preeclampsia or cerebral venous thrombosis (CVT).

In terms of risk factors and triggers, the most frequently reported include psychosocial stress, sleep deprivation, fatigue, and dehydration, each affecting about 20–40% of pregnant women with migraine. Comorbid anxiety and depression are associated with worse outcomes and an increased risk of chronification (8).

When comparing episodic and chronic migraine, the available evidence is more limited. However, as in the reviews on migraine in women, overuse of medication is one of the biggest contributors to its chronicity (9). However, robust comparative studies are still lacking.

Overall, current evidence suggests that pregnancy is a period of clinical improvement in most women with episodic MwoA, while MwA or chronic migraine shows a less predictable course. Recognizing these patterns is essential for preconception counseling, anticipating clinical evolution, and planning safe management strategies during pregnancy.

Physiological mechanisms and hormonal influence of migraine in pregnancy

Sex hormones, particularly estrogens and progesterone, play a central role in migraine pathophysiology by modulating neuronal excitability, neurotransmitter systems, and vascular tone. Estrogens enhance serotonergic and glutamatergic activity while reducing GABAergic and noradrenergic tone, thereby promoting trigeminal hyperresponsiveness. In contrast, progesterone exerts neuroprotective effects by enhancing GABAergic activity, reducing neuroinflammation, and dampening trigeminovascular nociception. Estrogen also influences vascular tone through nitric oxide–mediated vasodilation, with effects varying according to receptor subtype, brain region, and hormonal milieu (10–13).

Estrogen demonstrates a dual role: its withdrawal is strongly linked to MwoA, whereas sustained high levels may trigger MwA through cortical hyperexcitability. Progesterone generally counteracts these effects and contributes to sleep regulation (12,13).

During pregnancy, 80–90% of women experience an improvement or remission of symptoms, particularly in the second and third trimesters, due to the stable levels of oestrogen and endogenous opioids present at this time. However, 4–8% report a worsening of symptoms, most often in the first trimester, and de novo migraine with aura (MwA) may emerge later, likely due to endothelial reactivity and altered oestrogen–progesterone ratios. As both pregnancy and migraine are associated with a hypercoagulable state, persistent or severe migraine symptoms require investigation for potential underlying causes, such as pre-eclampsia, stroke or CVT (1,14,15).

Vasoactive peptides further shape migraine mechanisms. Calcitonin gene–related peptide (CGRP), regulated by estrogen, mediates neuroinflammation and pain, and is the target of novel therapies, although safety in pregnancy remains uncertain. Oxytocin demonstrates antinociceptive effects and is a promising therapeutic candidate. Vasopressin and prolactin also influence trigeminal sensitivity and vascular responses, while testosterone may exert antinociceptive and neuroprotective effects, though evidence in women remains limited (10,11,13).

Across the reproductive lifespan, fluctuating sex hormones drive migraine variability: estrogen withdrawal precipitates MwoA, high estrogen states facilitate MwA, and progesterone provides a protective role. Pregnancy generally improves migraine symptoms, while perimenopause introduces irregular fluctuations, leading to heterogeneous outcomes. Additional contributors such as oxidative stress, mitochondrial dysfunction, and impaired glucose metabolism further modulate susceptibility (10,12,14).

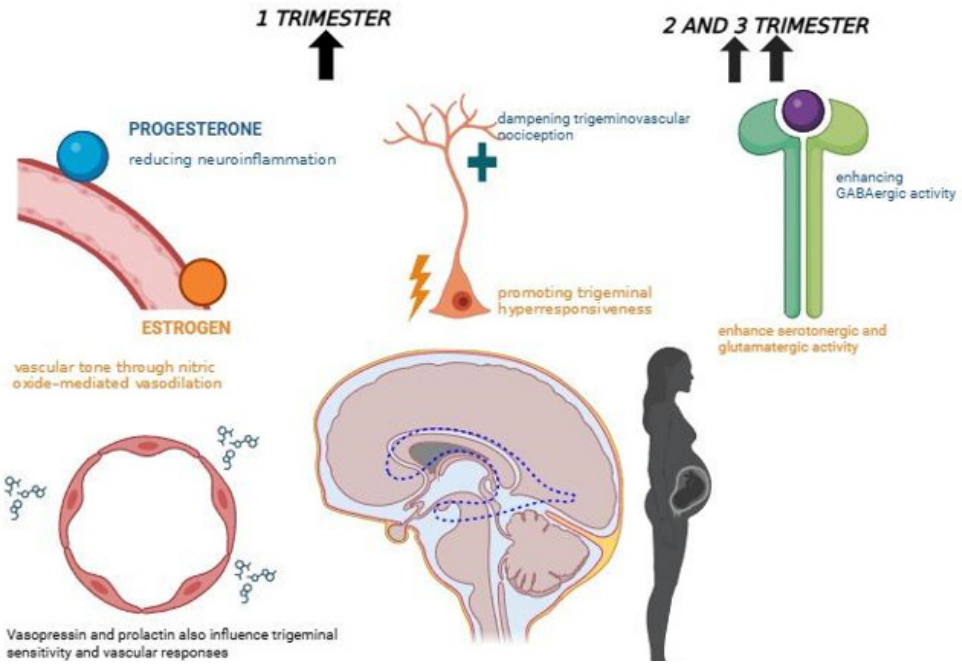

Figure 1. Hormonal and neurobiological mechanisms influencing migraine during pregnancy. In the first trimester, fluctuations in estrogen and progesterone affect vascular tone and neuroinflammation, promoting trigeminal hyper-responsiveness. In the second and third trimesters, enhanced GABAergic, serotonergic, and glutamatergic activity, together with dampened trigeminovascular nociception, contribute to symptom improvement. Vasopressin and prolactin also modulate trigeminal sensitivity and vascular responses.

Diagnostic criteria for migraine in pregnancy

Diagnosing MdP poses a particular clinical challenge due to limitations in both diagnostic methods and the use of medications during gestation. Despite these constraints, the criteria established by the International Classification of Headache Disorders (ICHD-3), while not specifically designed for pregnant women, are useful in differentiating between primary and secondary headaches through proper clinical assessment (Table 1) (16).

During pregnancy, MwoA tends to improve, whereas MwA may worsen or even appear for the first time, necessitating a careful differential diagnosis (7). However, several studies have shown that clinical, psychological, and psychophysical variables alone are insufficient to distinguish between women with and without migraine, highlighting the need for a comprehensive approach (17).

The diagnostic evaluation in pregnancy follows principles like those applied in non-pregnant women, beginning with a detailed medical history and physical examination aimed at identifying signs or symptoms that may suggest a secondary cause (18).

Table 1. Diagnostic criteria for migraine with and without aura according to the 2018 classification by the International Headache Society

Migraine without aura	Migraine with aura
At least 5 attacks fulfilling the following criteria: • Headache duration: 4–72 hours (and at least two of the following four characteristics): - Unilateral - Pulsating - Moderate to severe intensity - Aggravated by physical activity • During the headache, at least one of the following: - Nausea and vomiting - Photophobia and phonophobia	At least two attacks fulfilling the following criteria: • One or more of the following fully reversible symptoms: - visual, sensory, speech-language, motor, brainstem or retinal. At least three of the following six characteristics: • At least one aura symptom develops gradually over ≥5 minutes • Two or more aura symptoms occur in succession and last 5–60 minutes - At least one aura symptom is unilateral - At least one aura symptom is positive - The aura is followed by a headache within 60 minutes

Among the secondary headaches that may occur during pregnancy, the most relevant are those associated with prothrombotic states, such as stroke or CVT, and those linked to hypertensive disorders, including preeclampsia. Several clinical features should be considered red flags suggestive of a secondary origin. These include the onset of a new headache during pregnancy or the peripartum period, the appearance of a new aura, changes in the pattern of a preexisting headache, or the presence of concomitant hypertension (Table 2). Additional warning signs are a prolonged duration of attacks, a personal history of systemic arterial hypertension, absence of any prior history of headaches, and the presence of neurological abnormalities on examination (19).

Table 2. Red flags that should be assessed when evaluating headache in pregnant women

Clinical manifestations	Differential diagnosis	Diagnostic approach
Relapse thunderclap headaches	Subarachnoid hemorrhage	Brain CT or MRI and LP
	Reversible cerebral vasoconstriction syndrome (RCVS)	
	Primary cerebral vasculitis	
Progressive headache with papilledema, especially during the first trimester and the puerperium	CVT	Cerebral MRV ophthalmoscopy, Brain MRI, LP, or CT without contrast
	Idiopathic intracranial hypertension (IIH)	
Headache postural	Spontaneous intracranial hypotension	Brain MRI, PL
Progressive, refractory Headache, Visual symptoms, including scotoma	Gestational hypertension	Blood pressure, ophthalmoscopy, urine protein, Brain MRI/MRA/CT
	Preeclampsia reversible posterior encephalopathy (PRES)	
Seizures	Eclampsia	Blood pressure, ophthalmoscopy, urine protein levels, and Brain MRV MRI/MRA/CT
	CVT	
	PRES	
	RCVS	CT or diagnostic angiogram, MRI, LP
Fever / Meningismus	Meningitis	Brain MRI and LP
Severe, sudden retro-orbital headache, Campimetry deficit/ophthalmoplegia	IIH	Blood pressure, ophthalmoscopy, and urine proteins levels. Brain
	PRES	MRI/MRA/CT
	Preeclampsia/eclampsia	MRI
	CVT	
	Pituitary apoplexy	Ophthalmoscopy, MRI, MRV or LP

Abbreviations: MRI: Magnetic Resonance Imaging; MRA: Magnetic Resonance Angiography; CT: Computed Tomography; LP: Lumbar

Neuroimaging during pregnancy

When neuroimaging is warranted during pregnancy, magnetic resonance imaging (MRI) is preferred over computed tomography (CT) due to the absence of ionizing radiation and the reduced risk to fetal development. However, the use of iodinated contrast agents may transiently impair fetal thyroid function and should be considered with caution (20).

Despite being the modality of choice, MRI with gadolinium-based contrast agents is generally contraindicated during pregnancy unless necessary, given its potential adverse effects on the fetus. When vascular imaging is required, both brain Magnetic Resonance Angiography (MRA) and brain Magnetic Resonance Venography (MRV) can be performed safely without contrast by employing time-of-flight sequences.

Non-pharmacological migraine management in pregnancy

There is scientific evidence to support the effectiveness of non-pharmacological interventions in reducing the frequency and intensity of migraine attacks in women before and during pregnancy. The main methods are summarised in Table 3. These measures are safe in this context and contribute to overall well-being, establishing a solid foundation for adequate migraine control throughout pregnancy (14,21,22).

Medical devices, particularly non-invasive neuromodulation devices, represent an important treatment option for migraine management during pregnancy and breastfeeding when pharmaceutical therapies may pose safety concerns. Six FDA-cleared devices are currently available, including remote electrical neuromodulation (REN), noninvasive vagal nerve stimulation (nVNS), external trigeminal nerve stimulation (eTNS), single-pulse transcranial magnetic stimulation (sTMS), and external concurrent occipital and trigeminal neurostimulation (eCOT-NS) (23). These devices offer a safe, effective, and well-tolerated non-pharmacological alternative for pregnant patients (23,24).

Pharmacological acute management of migraine in pregnancy

The pharmacological management of migraine is challenging for physicians, as the teratogenic risk to the fetus must be considered when decisions are being made (see Table 4). The prescription of symptomatic treatment should be stratified according to the severity of the clinical picture and the associated risks. Figure 2 presents a proposed algorithm for decision-making.

Table 3. Non-pharmacological strategies for the initial management of migraine during pregnancy

Intervention	Benefits
Sleep hygiene	Routines of 7–9 hours of restful sleep, reducing screen time and ensuring a suitable environment decrease recurrence.
Nutrition	Identify triggers (caffeine, ultra-processed foods); avoid prolonged fasting; a balanced diet reduces attacks.
Hydration	Adequate fluid intake reduces the risk of seizures.
Stress management	Meditation, mindfulness, yoga, deep breathing, and CBT reduce the frequency of attacks.
Moderate exercise	Regular activity (walking, swimming, cycling) is safe and recommended; it improves circulation, weight, and stress management.
Complementary therapies	Biofeedback, acupuncture (with specialists), and CBT are effective as adjunctive strategies.

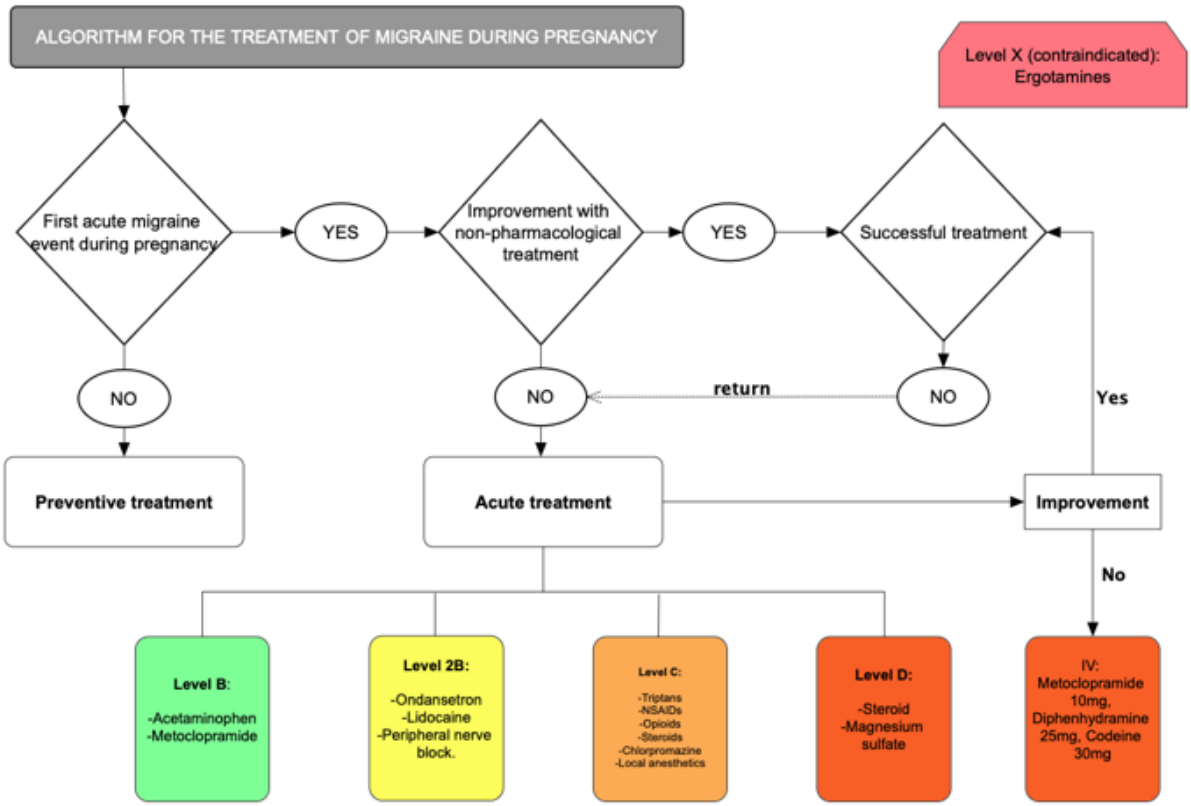

Figure 2. Algorithm for the treatment of migraine in pregnancy. Pharmacological interventions for migraine in pregnancy classified by safety: Level B – asthma, ADHD, extrapyramidal symptoms, QT alterations; Level 2B – cardiac defects, cleft palate, CNS depression (IV use); Level C – preterm birth, postpartum hemorrhage, uterine atony, spontaneous abortion, congenital malformations, ductus arteriosus closure, neonatal respiratory depression; Level D – orofacial malformations, growth disturbances, bone demyelination; Level X – contraindicated.

- Analgesics. Paracetamol is regarded as the preferred first-line therapy on account of its favourable safety profile throughout all trimesters of pregnancy and during lactation (25,26). It is widely accepted as the pharmaceutical intervention of choice for managing mild to moderate symptoms, and its safety has been established across all gestational trimesters.
- NSAIDs. May be considered second-line agents, with their use restricted to the second trimester. Their administration is contraindicated in the first trimester because of the risk of congenital malformations. Furthermore, its administration during the third trimester of pregnancy should be avoided due to the potential for premature closure of the ductus arteriosus (26).
- Triptans. A meta-analysis encompassing over 4,000 pregnant women exposed to triptans revealed no elevated risk of congenital malformations, miscarriage, or preterm birth in comparison to non-exposed women (26). Its use is considered compatible with breastfeeding (27), although its administration requires supervision. This group of drugs represents an effective and relatively safe option for moderate to severe attacks.
- CGRP-targeted Therapies. The use of gepants (ubrogepant, atogepant, rimegepant) and monoclonal antibodies (eptinezumab, fremanezumab, galcanezumab) lacks sufficient safety evidence in pregnancy and lactation. While rimegepant is FDA-approved for both acute and preventive treatment, animal studies suggest potential risks, including intrauterine growth restriction and hypertension. Current pharmacovigilance reports have not shown major maternal or fetal toxicity, but due to long half-lives and insufficient data, these agents are not recommended in pregnancy or lactation (28).
- Opioids. Opioids are not recommended given the risks of medication overuse headache, dependency,

neonatal abstinence syndrome, placental insufficiency, preterm birth, low birth weight, and neonatal respiratory depression. Limited, short-term use of weak opioids (e.g., codeine) may be considered only under strict medical supervision (18). Morphine and meperidine have not demonstrated human teratogenicity but should be avoided in the third trimester (29).

- Peripheral Nerve Blocks. Peripheral nerve blocks using local anesthetics (lidocaine, bupivacaine, ropivacaine) represent a safe, low-cost, and minimally invasive strategy for refractory migraine in pregnancy. Corticosteroid adjuvants may be considered, though local and systemic side effects (alopecia, cutaneous atrophy, hyperglycemia, Cushing's syndrome) have been reported. Evidence quality is rated at level C (30,31).

Other Pharmacological Approaches

- Lidocaine: Intranasal administration may be considered in refractory cases, but systemic routes are discouraged (3).
- Steroids: Systemic corticosteroids should be avoided due to risks of cleft palate and premature fetal lung maturation (3).
- Antipsychotics: Agents such as chlorpromazine or others are linked to adverse perinatal outcomes and are not recommended (3).
- Combination therapies: A randomized clinical trial compared IV metoclopramide + diphenhydramine vs. codeine in 70 pregnant women showed significant pain reduction at 30 minutes and complete resolution at 24 hours, without major adverse effects (3).

Table 4. Risk and safety levels of the main drugs used in the acute treatment of primary headaches during pregnancy

Drug	FDA classification	Risks and considerations
Acetaminophen	B (oral), C (IV)	The safest analgesic during pregnancy. However, some studies suggest a possible association with an increased risk of asthma or Attention-Deficit/Hyperactivity Disorder in offspring.
Metoclopramide	B	Useful as an antiemetic; it can induce extrapyramidal symptoms and prolong the QT interval.
NSAID´s	C (1st and 2nd trimester), D (≥ 30 weeks)	During the first trimester, spontaneous abortions and cardiac and digestive malformations have been reported. From week 30 onwards, the risk of premature closure of the ductus arteriosus and intracranial hemorrhages increases.
Acetylsalicylic Acid	D	Its late use in pregnancy can lead to neonatal pulmonary hypertension, intrauterine growth retardation, and intracranial bleeding in premature infants.
Triptans	C	Observational data indicate a potential risk of preterm delivery and postpartum hemorrhage; sumatriptan is the most studied.
Opioids (codeine, tramadol)	C	Potential neonatal respiratory depression and withdrawal syndrome in newborns.
Ergotamines	X	Induces uterine contractions, risk of miscarriage, and uteroplacental ischemia.
Caffeine	C	Moderate consumption appears to be safe but is dose-dependent; high doses may increase the risk of low birth weight.
Chlorpromazine and other dopamine antagonists	C	In the third trimester, it may cause extrapyramidal symptoms or withdrawal in the newborn.
Domperidone	X	Associated with QT interval prolongation; not recommended for use during pregnancy.
Ondansetron	B	It has been linked to heart defects, cleft palate, and QT prolongation.
Lidocaine	B	Well tolerated when administered locally or intranasally. Intravenous administration is contraindicated due to the risk of CNS depression.
Other local anesthetics	C	Bupivacaine may alter maternal cardiac conduction.
Prednisone, methylprednisolone	C (prednisone, methylprednisolone); D (dexamethasone)	Useful in selected cases; associated with fetal growth retardation and accelerated lung maturation.
Magnesium sulfate	D	Prolonged use is associated with bone demineralization; it may be used occasionally in specific contexts.
Lasmiditan	Not recommended	Unknown
Rimegepant	Not recommended	Unknown

Preventive and pharmacological prophylaxis for migraine in pregnancy

Preventive treatment for MdP should be reserved for patients with frequent or debilitating attacks that are refractory to non-pharmacological measures and acute treatments. Untreated severe migraine has been associated with adverse pregnancy outcomes such as preeclampsia, preterm birth, and low birth weight infants (24). Therefore, the decision to initiate prophylactic therapy must balance maternal quality of life with potential fetal risks, ideally using the lowest effective dose for the shortest possible duration (32). Decisions should always be made with the treating gynecologist and the patient about possible side effects (Table 5).

Several pharmacological agents have demonstrated relative safety in pregnancy and are considered first-line therapy:

- Magnesium: Oral magnesium (400–600 mg/day) is a well-tolerated supplement with evidence supporting its efficacy in migraine prevention. Oral magnesium appears to be safe in pregnancy, although high-dose intravenous use has been associated with fetal osteopenia in prolonged exposures (24).
- Beta-blockers, especially propranolol and metoprolol, are widely used and considered safe during pregnancy. Although some observational studies have linked beta-blockers to small for gestational age neonates and neonatal hypoglycemia or bradycardia, these effects appear dose-dependent and not uniformly replicated (26).
- Amitriptyline: This tricyclic antidepressant has a long history of use in migraine prophylaxis. At low doses, it is considered relatively safe in pregnancy. While withdrawal symptoms have been reported in neonates exposed late in gestation, no strong evidence suggests teratogenicity (26).
- OnabotulinumtoxinA (BoNT/A): Although BoNT/A is well established as an effective preventive therapy for chronic migraine, evidence regarding its safety in pregnancy remains limited. Data from the global pregnancy registry—including migraine and dystonia indications—show no increased risk of major congenital malformations or adverse pregnancy outcomes after therapeutic exposure (33). However, given the absence of controlled trials in pregnant migraine patients and the theoretical concerns regarding fetal toxicity derived from animal studies, its use should be considered only when migraine attacks are severe, disabling, and refractory to safer alternatives. The large molecular size of BoNT/A (~900 kDa) makes significant placental transfer unlikely, suggesting minimal fetal exposure (34). Still, treatment decisions must remain individualized and based on a careful risk–benefit assessment.

Table 5. Preventive pharmacological options for migraine during pregnancy

Preventive option	Mechanism of action	Typical adult preventive dose*	Effectiveness & safety in pregnancy (summary)
Propranolol (β-blocker)	Non-selective β-adrenergic blockade; dampens noradrenergic drive and trigeminovascular activation	80–160 mg/day (up to 320 mg/day used in some guidelines)	Strength of recommendation: moderate. Considered first-line if needed by ACOG/AHS with monitoring. Observational data show no increased major malformations; possible association with fetal growth restriction and neonatal bradycardia/hypoglycemia with late exposure—monitor fetal growth and neonate.
Amitriptyline (Tricyclic Antidepressant)	Inhibits 5-HT/NE reuptake; antinociceptive effects	Start 10–25 mg qHS; typical 50–100 mg qHS; up to 150 mg/day	Strength of recommendation: moderate. Acceptable with caution when benefits outweigh risks; widely used with no clear increase in major malformations. Possible neonatal adaptation symptoms if used near delivery. Recommended by ACOG/AHS as an option.
OnabotulinumtoxinA (BoNT-A) – chronic migraine	Blocks acetylcholine release at the neuromuscular junction; reduces peripheral/central sensitization	155 U IM across 31 sites q12 weeks (PREEMPT); up to 155–195 IU.	Strength of recommendation: low-moderate. ACOG allows case-by-case use in pregnancy. Large 29-year Allergan database and other series show major malformation rates ≈ background; minimal systemic absorption. Reasonable option in refractory chronic migraine after risk–benefit discussion.
CGRP monoclonal antibodies	Block the CGRP pathway (ligand or receptor)	Erenumab 70–140 mg SC monthly; Fremanezumab 225 mg monthly or 675 mg quarterly; Galcanezumab 240 mg load then 120 mg monthly; Eptinezumab 100–300 mg IV q12 weeks	Strength of recommendation: very low. Human data still limited. Pharmacovigilance shows no safety signal vs triptans so far, but due to long half-life and placental IgG transfer, most guidelines advise avoiding in pregnancy and stopping ~5–6 months before conception. Inadvertent early exposure has not shown a pattern of adverse outcomes.
Gepants (preventive) - Rimegepant, Atogepant	Small-molecule CGRP receptor antagonists	Orally Disintegrating Tablet Rimegepant 75 mg for prevention; Atogepant 10/30/60 mg once daily	Strength of recommendation: very low. Insufficient pregnancy data; generally, avoid; counsel on contraception and washout when planning conception.
Topiramate	Modulates voltage-gated Na+ channels, enhances GABA, blocks AMPA/kainate; weak CA-I	50–100 mg/day (e.g., 50 mg BID)	Strength of recommendation: contraindicated. Avoid in pregnancy: associated with oral clefts and lower birth weight; not recommended when trying to conceive.
Valproate	Increases GABA; multiple central mechanisms	500–1500 mg/day	Strength of recommendation: contraindicated. Contraindicated in pregnancy for major congenital malformations, neurodevelopmental injury/lower IQ. Avoid people who could become pregnant.
Candesartan / ACE inhibitors	Renin–angiotensin blockade	Candesartan 16–32 mg/day	Strength of recommendation: contraindicated. Contraindicated (esp. 2°–3° trimester): fetopathy (renal dysgenesis, oligohydramnios, skull hypoplasia, fetal/neonatal death). If on these at conception → switch/stop promptly and monitor.
Venlafaxine Serotonin-Norepinephrine Reuptake Inhibitor	Inhibits 5-HT/NE reuptake	75–150 mg/day (XR)	Strength of recommendation: low. Use with caution if comorbid depression/anxiety and alternatives fail. Data mixed: no clear major malformation signal; reports of prematurity/neonatal adaptation. ACOG allows cautious use.
Magnesium oxide (MgO) (± riboflavin)	Cofactor modulating NMDA/Ca2+; reduces cortical spreading depression	MgO 400–600 mg/day. Riboflavin 400 mg/day	Strength of recommendation: low. Evidence is limited, but a pregnancy cohort showed reductions in frequency/severity; generally considered low risk, though ACOG cautions about oral Mg in some contexts—use individualized risk–benefit. Riboflavin appears generally safe, but pregnancy data are limited.

Abbreviations: ACOG: American College of Obstetricians and Gynecologists, AHS: American Headache Society; qHS: Once a day at bedtime; SC: subcutaneous; BID: Two times a day, NMDA: N-Methyl-D-aspartate; GABA: Gamma-aminobutyric acid; AMPA: α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid.

Most available data on drug safety in pregnancy are derived from retrospective observational studies with potential biases and confounders. Randomized controlled trials (RCTs) in pregnant populations are rare due to ethical constraints. Consequently, many decisions rely on expert consensus and extrapolation from animal or general population data (26). Behavioral interventions remain the cornerstone of migraine prevention during pregnancy (24).

Maternal and fetal risks of migraine in pregnancy

MdP can significantly impair health-related quality of life, disrupting daily activities, self-care, and prenatal engagement. Symptoms such as severe headache, photophobia, nausea, and sleep disturbance are often compounded by psychiatric comorbidities. Pregnant migraineurs have higher rates of depression, anxiety, and mixed affective symptoms across all trimesters compared to non-migraineurs, and those experiencing MdP show an increased risk of postpartum depression (35–37). These comorbidities may hinder maternal-infant bonding and impact long-term child development.

Migraine, particularly with aura, is also linked to hypertensive disorders of pregnancy. Large cohort studies show increased odds of gestational hypertension (aOR 1.19), preeclampsia (aOR 1.36), and severe preeclampsia or eclampsia (aOR 1.51), independent of confounders (36). Risks are greatest in women with aura and in those with comorbidities such as chronic hypertension and diabetes.

Obstetric complications have also been reported. A prospective cohort of over 19,000 pregnancies found higher risks of preterm birth (aOR 1.21) and cesarean delivery (aOR 1.18) among women with untreated migraine (38), suggesting vascular dysregulation and impaired uteroplacental perfusion. Registry-based studies further associate maternal migraine—particularly MwA—with small-for-gestational-age infants and low birth weight (aHR 1.11), likely reflecting endothelial dysfunction and placental insufficiency (38).

MwA also increases maternal risk of ischemic stroke, sharing mechanisms with hypertensive disorders such as systemic inflammation and endothelial dysfunction (36). These overlapping pathways may amplify maternal and fetal risks, especially in women with cardiovascular or metabolic comorbidities. Additionally, certain acute migraine treatments (e.g., NSAIDs, triptans) may contribute to adverse outcomes depending on dose and timing, although evidence remains inconclusive (35).

Migraine in postpartum and breastfeeding women

Migraine postpartum relapse is common: over 50% recur in the first month and more than 75% by 3–6 months after delivery (38,39). Attacks in this period are typically more frequent and severe, with higher recurrence risk in women over 30. Breastfeeding has a protective effect, with a lower recurrence rate compared to bottle feeding, although the mechanisms remain unclear (38).

The abrupt drop in estrogen after childbirth is a major trigger, particularly in women with menstrual or hormone-sensitive migraine (40,41). Non-hormonal triggers such as sleep deprivation, irregular meals, stress, dehydration, and caffeine withdrawal are also common, while mood disorders—especially postpartum depression—may exacerbate vulnerability (42–44).

First-line management prioritizes non-pharmacological measures, including sleep hygiene, hydration, stress reduction, and trigger avoidance. When medication is required, safety during breastfeeding must be considered. Paracetamol and ibuprofen are compatible with breastfeeding, while sumatriptan—the best-studied triptan—appears safe with minimal infant exposure, especially if breastfeeding is delayed 8–12 hours after dosing. Naproxen and other NSAIDs may be used with caution, avoiding prolonged use, whereas ergot derivatives are contraindicated due to toxicity and suppression of lactation. Preventive therapy may be considered in severe, refractory cases, with beta-blockers (propranolol) and amitriptyline as options under close supervision (39,45).

Lifestyle interventions are essential. Key recommendations include consistent sleep patterns, regular meals, stress management, hydration, moderate exercise, and smoking cessation (46–52).

Dietary modifications for postpartum women with migraines should focus both on avoiding common food triggers and adopting evidence-based eating patterns that have been shown to reduce the frequency and severity of migraines. Strategies include:

- **Weight management:** For people who are overweight or obese, dietary strategies that promote gradual weight loss may reduce the burden of migraine, as obesity is associated with increased frequency and severity of attacks (53).
- **Omega-3 fatty acids and low intake of omega-6 linoleic acid**: the increase in dietary intake of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) to approximately 1.5 g/day, while reducing linoleic acid to ≤1.8% of energy, resulted in significant reductions in migraine days and decreased migraine intensity. The additional benefit of reducing linoleic acid suggests that postpartum women should emphasize omega-3-rich foods (such as fatty fish, flax seeds, and chia seeds) and limit sources of omega-6 (such as processed seed oils) (53,54).
- **Mediterranean-style or DASH (Dietary Approaches to Stop Hypertension) eating**

patterns: Systematic reviews and intervention studies support the role of the DASH diet and Mediterranean-style eating patterns in reducing migraine frequency and severity. These diets are characterized by high consumption of fruits, vegetables, whole grains, lean proteins, and healthy fats, with limited consumption of processed foods and red meat (55).

- **Maintain regular meal times and avoid fasting or skipping meals**: Irregular eating patterns and hypoglycemia are recognized triggers for migraines. It is recommended to maintain regular meal times and especially a balanced intake of macronutrients to stabilize blood glucose and reduce the risk of recurrence (56).
- **Reduce consumption of red and processed meats and refined carbohydrates**: Intervention data indicate that decreasing consumption of red and processed meats, as well as refined carbohydrates, is associated with lower migraine frequency and disability (57).
- **Identify and avoid individual food triggers**: Common triggers include alcohol, caffeine (both in excess and through withdrawal), chocolate, monosodium glutamate (MSG), such as ham, Parmesan cheese, and Roquefort cheese, mushrooms, and seaweed. Also nitrates (bacon, sausage) and foods containing tyramine (avocado, bananas, figs, raisins, cabbage). The use of a headache diary to identify personal triggers is supported by the literatura (58).
- **Ensure adequate hydration**: Increased water intake is associated with improved migraine outcomes, and dehydration is a well-established trigger (56).
- **Consider nutritional supplements**: There is evidence that nutrients such as magnesium and riboflavin may help relieve migraine symptoms, so ensuring adequate intake may be beneficial (58).

Gap evidence and future perspectives

MdP presents a complex clinical scenario shaped by hormonal, vascular, and metabolic changes. While many women experience remission in the second and third trimesters, those with MwA remain at increased risk for hypertensive disorders, preterm birth, and adverse fetal outcomes. The postpartum period represents a critical window, marked by high recurrence rates driven by abrupt hormonal withdrawal and lifestyle-related triggers, although breastfeeding appears protective. Management must therefore prioritize non-pharmacological strategies, safe acute options such as acetaminophen, ibuprofen, and sumatriptan, and carefully selected preventive agents like magnesium, propranolol, or amitriptyline in refractory cases.

Despite advances, important knowledge gaps persist. The precise mechanisms by which estrogens, progesterone, and their metabolites shape migraine pathophysiology remain unclear, as do the processes underlying symptom emergence or remission across reproductive milestones. The long-term vascular safety of CGRP antagonists in pregnancy is uncertain, and the paradoxical role of exercise highlights the complexity of non-hormonal modulators. Future research should focus on developing targeted hormonal therapies, sex-specific preclinical models, and biomarkers to guide personalized treatment. Exploration of novel pathways, including oxytocin, vasopressin, and prolactin, alongside longitudinal studies across the female reproductive lifespan, will be critical. Addressing these gaps through interdisciplinary collaboration will enable safer, more precise strategies to improve maternal and fetal outcomes while enhancing the quality of life for women affected by migraine.

In conclusion, migraine during a stage of a woman's life involving various physiological changes, such as pregnancy, requires further research to establish an adequate safety profile, biomarkers, and implement new groups of drugs, including CGRP monoclonal antibodies and neuromodulatory techniques, thereby obtaining a more effective treatment algorithm. A multidisciplinary approach is essential to balance efficacy and safety, particularly in regions where specific clinical guidelines are lacking.

References

1. Kim S, Park JW. Migraines in Women: A Focus on Reproductive Events and Hormonal Milestones. Headache and Pain Research 2024;25:3–15. Doi:10.62087/hpr.2024.0003.
2. Burch R. Epidemiology and Treatment of Menstrual Migraine and Migraine During Pregnancy and Lactation: A Narrative Review. Headache: The Journal of Head and Face Pain 2020;60:200–16. Doi:10.1111/head.13665.
3. Saldanha IJ, Cao W, Bhuma MR, Konnyu KJ, Adam GP, Mehta S, et al. Management of primary headaches during pregnancy, postpartum, and breastfeeding: A systematic review. Headache: The Journal of Head and Face Pain 2021;61:11–43. Doi:10.1111/head.14041.
4. Phillips K, Koonalintip P, Wakerley BR. Migraine and Pregnancy. Life 2024;14:1224. Doi:10.3390/life14101224.
5. Sances G, Granella F, Nappi R, Fignon A, Ghiotto N, Polatti F, et al. Course of Migraine During Pregnancy and Postpartum: A Prospective Study. Cephalalgia 2003;23:197–205. Doi:10.1046/j.1468-2982.2003.00480.x.
6. Kvisvik EV, Stovner LJ, Helde G, Bovim G, Linde M. Headache and migraine during pregnancy and puerperium: the MIGRA-study. J Headache Pain 2011;12:443–51. Doi:10.1007/s10194-011-0329-1.

7. Paškevičiūtė E, Bužinskienė D, Ryliškienė K. De novo migraine with aura in the third trimester of pregnancy: a case report and literature review. Acta Med Litu 2021;28:145–52. Doi:10.15388/Amed.2021.28.1.19.
8. Tiseo C, Vacca A, Felbush A, Filimonova T, Gai A, Glazyrina T, et al. Migraine and sleep disorders: a systematic review. J Headache Pain 2020;21:126. Doi:10.1186/s10194-020-01192-5.
9. Deneris A, Rosati Allen P, Hart Hayes E, Latendresse G. Migraines in Women: Current Evidence for Management of Episodic and Chronic Migraines. J Midwifery Womens Health 2017;62:270–85. Doi:10.1111/jmwh.12626.
10. Khan J, Asoom LI Al, Sunni A Al, Rafique N, Latif R, Saif S Al, et al. Genetics, pathophysiology, diagnosis, treatment, management, and prevention of migraine. Biomedicine & Pharmacotherapy 2021;139:111557. Doi:10.1016/j.biopha.2021.111557.
11. Barus J, Sudharta H, Adriani D. Study of the Mechanisms and Therapeutic Approaches of Migraine in Women and Pregnancy: A Literature Review. Cureus 2023. Doi:10.7759/cureus.35284.
12. Allais G, Chiarle G, Sinigaglia S, Airola G, Schiapparelli P, Benedetto C. Gender-related differences in migraine. Neurological Sciences 2020;41:429–36. Doi:10.1007/s10072-020-04643-8.
13. Godley F, Meitzen J, Nahman-Averbuch H, O'Neal MA, Yeomans D, Santoro N, et al. How Sex Hormones Affect Migraine: An Interdisciplinary Preclinical Research Panel Review. J Pers Med 2024;14:184. Doi:10.3390/jpm14020184.
14. Lackovic M, Jankovic M, Mihajlovic S, Milovanovic Z, Nikolic D. Exploring the Connection between Migraines and Pregnancy: The Impact of Physical Activity on Symptom Management. Medicina (B Aires) 2023;60:49. Doi:10.3390/medicina60010049.
15. Susanti R, Muhammad S. Migraine And Pregnancy: What Should We Know. Andalas Obstetrics And Gynecology Journal 2020;4:251–60. Doi:10.25077/aoj.4.2.251-260.2020.
16. Munoz-Ceron J, Marin-Careaga V, Peña L, Mutis J, Ortiz G. Headache at the emergency room: Etiologies, diagnostic usefulness of the ICHD 3 criteria, red and green flags. PLoS One 2019;14:e0208728. Doi:10.1371/journal.pone.0208728.
17. Cigarán-Mendez M, Pacho-Hernández JC, Fernández-Palacios FG, Tejera-Alonso Á, Valera-Calero JA, Gómez-Calero C, et al. Can Clinical, Psychophysical or Psychological Variables Help in Discriminating Women with Migraines from a Tertiary Center? A Diagnostic Accuracy Study. Diagnostics 2024;14:2805. Doi:10.3390/diagnostics14242805.
18. Magro I, Nurimba M, Doherty JK. Headache in Pregnancy. Otolaryngol Clin North Am 2022;55:681–96. Doi:10.1016/j.otc.2022.02.013.
19. Robbins MS, Farmakidis C, Dayal AK, Lipton RB. Acute headache diagnosis in pregnant women. Neurology 2015;85:1024–30. Doi:10.1212/WNL.0000000000001954.
20. Chansakul T, Young G. Neuroimaging in Pregnant Women. Semin Neurol 2017;37:712–23. Doi:10.1055/s-0037-1608939.
21. Haghdoost F, Togha M. Migraine management: Non-pharmacological points for patients and health care professionals. Open Medicine 2022;17:1869–82. Doi:10.1515/med-2022-0598.
22. Ghadiri-Sani M. Migraine in women: a review. Curr Opin Neurol 2025;38:271–6. Doi:10.1097/WCO.0000000000001372.
23. Smirnoff L, Bravo M, Hyppolite T. Neuromodulation for Headache Management in Pregnancy. Curr Pain Headache Rep 2025;29:14. Doi:10.1007/s11916-024-01344-1.
24. Parikh S, Delbono M, Silberstein S. Managing migraine in pregnancy and breastfeeding, 2020, p. 275–309. Doi:10.1016/bs.pbr.2020.07.011.
25. Puledda F, Sacco S, Diener H-C, Ashina M, Al-Khazali HM, Ashina S, et al. International Headache Society global practice recommendations for the acute pharmacological treatment of migraine. Cephalalgia 2024;44. Doi:10.1177/03331024241252666.
26. Nezvalová-Henriksen K, Spigset O, Nordeng H. Triptan Exposure During Pregnancy and the Risk of Major Congenital Malformations and Adverse Pregnancy Outcomes: Results From The Norwegian Mother and Child Cohort Study. Headache: The Journal of Head and Face Pain 2010;50:563–75. Doi:10.1111/j.1526-4610.2010.01619.x.
27. González-García N, Díaz de Terán J, López-Veloso AC, Mas-Sala N, Mínguez-Olaondo A, Ruiz-Piñero M, et al. Cefalea: embarazo y lactancia. Recomendaciones del Grupo de Estudio de Cefaleas de la Sociedad Española de Neurología (GECSEN). Neurología 2022;37:1–12. Doi:10.1016/j.nrl.2018.12.003.
28. Spiteri JA, Camilleri G, Piccinni C, Sultana J. Safety of drugs used for the treatment of migraine during pregnancy: a narrative review. Expert Rev Clin Pharmacol 2023;16:207–17. Doi:10.1080/17512433.2023.2181157.
29. Turankar T, Sorte A, Wanjari MB, Chakole S, Sawale S. Relation and Treatment Approach of Migraine in Pregnancy and Breastfeeding. Cureus 2023. Doi:10.7759/cureus.36828.
30. Stern JI, Chiang C, Kissoon NR, Robertson CE. Narrative review of peripheral nerve blocks for the management of headache. Headache: The Journal of Head and Face Pain 2022;62:1077–92. Doi:10.1111/head.14385.
31. Wahab S, Kataria S, Woolley P, O'Hene N, Odinkemere C, Kim R, et al. Literature Review: Pericranial Nerve Blocks for Chronic Migraines. Health Psychol Res 2023;11:1. Doi:10.52965/001c.74259.
32. Naghdi S, Underwood M, Madan J, Brown A, Duncan C, Matharu M, et al. Clinical effectiveness of

pharmacological interventions for managing chronic migraine in adults: a systematic review and network meta-analysis. J Headache Pain 2023;24:164. Doi:10.1186/s10194-023-01696-w.
33. Brin MF, Kirby RS, Slavotinek A, Adams AM, Parker L, Ukah A, et al. Pregnancy Outcomes in Patients Exposed to OnabotulinumtoxinA Treatment. Neurology 2023;101. Doi:10.1212/WNL.0000000000207375.
34. Russo A, Iannone LF, Orologio I, Rivi V, Boccalini A, Lo Castro F, et al. Safety of Onabotulinumtoxin-A for Chronic Migraine During Pregnancy and Breastfeeding: A Narrative Review. Toxins (Basel) 2025;17:192. Doi:10.3390/toxins17040192.
35. Buse DC, Reed ML, Fanning KM, Bostic R, Dodick DW, Schwedt TJ, et al. Comorbid and co-occurring conditions in migraine and associated risk of increasing headache pain intensity and headache frequency: results of the migraine in America symptoms and treatment (MAST) study. J Headache Pain 2020;21:23. Doi:10.1186/s10194-020-1084-y.
36. Welander NZ, Mwinyi J, Asif S, Schiöth HB, Skalkidou A, Fransson E. Migraine as a risk factor for mixed symptoms of peripartum depression and anxiety in late pregnancy: A prospective cohort study. J Affect Disord 2021;295:733–9. Doi:10.1016/j.jad.2021.08.119.
37. Reis ACS dos S e, Gonçalves ALR, Santos BOR dos, Silva JMP e, Câmara L de ÁGCD, Jurno ME. Prevalence of postpartum depression in patients with migraine. Headache Medicine 2022;13:222–5. Doi:10.48208/HeadacheMed.2022.24.
38. Hoshiyama E, Tatsumoto M, Iwanami H, Saisu A, Watanabe H, Inaba N, et al. Postpartum Migraines: A Long-term Prospective Study. Internal Medicine 2012;51:3119–23. Doi:10.2169/internalmedicine.51.8542.
39. Amundsen S, Nordeng H, Nezvalová-Henriksen K, Stovner LJ, Spigset O. Pharmacological treatment of migraine during pregnancy and breastfeeding. Nat Rev Neurol 2015;11:209–19. Doi:10.1038/nrneurol.2015.29.
40. Faubion SS, Batur P, Calhoun AH. Migraine Throughout the Female Reproductive Life Cycle. Mayo Clin Proc 2018;93:639–45. Doi:10.1016/j.mayocp.2017.11.027.
41. Chalmer MA, Kogelman LJA, Ullum H, Sørensen E, Didriksen M, Mikkelsen S, et al. Population-Based Characterization of Menstrual Migraine and Proposed Diagnostic Criteria. JAMA Netw Open 2023;6:e2313235. Doi:10.1001/jamanetworkopen.2023.13235.
42. Wells RE, Turner DP, Lee M, Bishop L, Strauss L. Managing Migraine During Pregnancy and Lactation. Curr Neurol Neurosci Rep 2016;16:40. Doi:10.1007/s11910-016-0634-9.
43. Gordon-Smith K, Ridley P, Perry A, Craddock N, Jones I, Jones L. Migraine associated with early onset postpartum depression in women with major depressive disorder. Arch Womens Ment Health 2021;24:949–55. Doi:10.1007/s00737-021-01131-6.
44. Marmura MJ. Triggers, Protectors, and Predictors in Episodic Migraine. Curr Pain Headache Rep 2018;22:81. Doi:10.1007/s11916-018-0734-0.
45. Parikh SK. Unique Populations with Episodic Migraine: Pregnant and Lactating Women. Curr Pain Headache Rep 2018;22:80. Doi:10.1007/s11916-018-0737-x.
46. Charles A. Migraine. New England Journal of Medicine 2017;377:553–61. Doi:10.1056/NEJMcp1605502.
47. Seng EK, Gosnell I, Sutton L, Grinberg AS. Behavioral Management of Episodic Migraine: Maintaining a Healthy Consistent Lifestyle. Curr Pain Headache Rep 2022;26:247–52. Doi:10.1007/s11916-022-01023-z.
48. Robblee J, Starling AJ. SEEDS for success: Lifestyle management in migraine. Cleve Clin J Med 2019;86:741–9. Doi:10.3949/ccjm.86a.19009.
49. Agbetou M, Adoukonou T. Lifestyle Modifications for Migraine Management. Front Neurol 2022;13. Doi:10.3389/fneur.2022.719467.
50. Airola G, Allais G, Castagnoli Gabellari I, Rolando S, Mana O, Benedetto C. Non-pharmacological management of migraine during pregnancy. Neurological Sciences 2010;31:63–5. Doi:10.1007/s10072-010-0276-7.
51. Qaseem A, Tice JA, Etxeandia-Ikobaltzeta I, Wilt TJ, Harrod CS, Cooney TG, et al. Pharmacologic Treatments of Acute Episodic Migraine Headache in Outpatient Settings: A Clinical Guideline From the American College of Physicians. Ann Intern Med 2025;178:571–8. Doi:10.7326/ANNALS-24-03095.
52. Bushnell C, McCullough LD, Awad IA, Chireau M V., Fedder WN, Furie KL, et al. Guidelines for the Prevention of Stroke in Women. Stroke 2014;45:1545–88. Doi:10.1161/01.str.0000442009.06663.48.
53. Gazerani P, Papetti L, Dalkara T, Cook CL, Webster C, Bai J. The Brain, the Eating Plate, and the Gut Microbiome: Partners in Migraine Pathogenesis. Nutrients 2024;16:2222. Doi:10.3390/nu16142222.
54. Razeghi Jahromi S, Ghorbani Z, Martelletti P, Lampl C, Togha M. Association of diet and headache. J Headache Pain 2019;20:106. Doi:10.1186/s10194-019-1057-1.
55. Roldán-Ruiz A, Bertotti G, López-Moreno M. Effects of Dietary Interventions in Patients With Migraine: A Systematic Review. Nutr Rev 2025;83:e1815–27. Doi:10.1093/nutrit/nuae188.
56. Moskatel LS, Zhang N. Migraine and Diet: Updates in Understanding. Curr Neurol Neurosci Rep 2022;22:327–34. Doi:10.1007/s11910-022-01195-6.
57. Tu Y-H, Chang C-M, Yang C-C, Tsai I-J, Chou Y-C, Yang C-P. Dietary Patterns and Migraine: Insights and Impact. Nutrients 2025;17:669. Doi:10.3390/nu17040669.
58. Altamura C, Cecchi G, Bravo M, Brunelli N, Laudisio A, Di Caprio P, et al. The Healthy Eating Plate Advice for Migraine Prevention: An Interventional Study. Nutrients 2020;12:1579. Doi:10.3390/nu12061579.

María Karina Vélez-Jiménez
https://orcid.org/0000-0002-8288-8765
Pablo Schubaroff
https://orcid.org/0009-0004-1382-5535
Maria Loreto Cid-Jeffs
https://orcid.org/0009-0004-1515-7235
Ana Mercedes-Robles
https://orcid.org/0009-0005-6840-5228
Adriana Patricia Martinez-Mayorga
https://orcid.org/0000-0001-9354-2295
Ildefonso Rodriguez-Leyva
https://orcid.org/0000-0002-3316-1471
Daniel San-Juan
https://orcid.org/0000-0001-6685-5851
Javier Andrés Galnares-Olalde
https://orcid.org/0000-0003-3004-6221
Miguel Angel Morales-Morales
https://orcid.org/0000-0002-0299-0525
Kevin Espejo-Campos
https://orcid.org/0009-0006-1534-1168

Author´s contributions: MKVJ: project administration, conceptualization, Methodology, supervision, writing – original draft; PS: writing – original draft, conceptualization, investigation, validation; MLCJ, AMR, APMM, JAGO: writing – original draft, investigation, validation; IRL: writing – original draft, supervision; DSJ: supervision, Writing – original draft, investigation, validation; MAMM: data curation, resources, formal analysis, writing – review & editing and visualization; KEC: writing – original draft, investigation, visualization.
Conflicts of interest: The authors declare no conflict of interest related to this work.
Funding: This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.